Int. J. Open Problems Compt. Math., Vol. 18, No. 4, December 2025
Print ISSN: 1998-6262, Online ISSN: 2079-0376

Comorbidity Analysis of Diabetes and COVID-19 Patients under Vaccination

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 10 August 2025; Accepted 3 October 2025

Abstract

There is always a high probability of finding individuals infected by COVID-19 to be having other underlying diseases. Those who have these two or more diseases died at higher rate, four times, compared to those who are suffering from one disease. For individuals under comorbidity, attrition rate is higher meaning the rate of recovery is low and more resources are used in cases of 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. In this note, we analyze comorbidity under vaccination.

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

1 Introduction

There is existence of comorbidities of diseases amongst individuals who have COVID-19 and people suffering from comorbidities tend to have a weaker immunity system, making their bodies vulnerable to any disease attacks thereby leading to low recovery rate and high death rate [8]. Considering COVID-19 and diabetes as a comorbidities, it takes a longer period for recovery and death

Table 1: Parameters and description of SIR population

Symbol	**Parameter**
S	Susceptible population
I	Infected population
R	Recovery population
β	Transmission probability
γ	death/recovery rate

may occur faster compared to those who have COVID-19 only.
Mathematical epidemiologists are recognized as pioneers of mathematical epidemiology. The work of [25] came up with susceptibility concept and Sir Ross and Kermack and Mckendrick were the first people to formulate mathematical models and develpoed a malaria infection model, whose result showed malaria can be reduced if the population of mosquitoes is decreased as malaria spreads through mosquito bites [9]. From his observation, it was believed that he came up with the important concept in epidemiology and later it was known as R_0. Certain studies used R_0 on compartmental model for diseases, and they discovered that there was recovery as result of permanent immunity and re-infection was also possible [10]. Later, an expansion was done on epidemiology by [1] where they introduced latent (exposed) period in his model as one of the compartmental (infected individual cannot infect others in the population).
Leading cause of deaths worldwide remain to be infectious diseases including Ebola, COVID-19 among others. The transmission of these infections is being explored and analyzed using mathematical models. The author in [13] formulated the first mathematical model which he used to analyze how vaccination of a healthy individual against smallpox can be effective during attack with the disease. The study by [14] developed a discrete time model of measles epidemic that reoccurs within a population. From a differential equation done by [15] for malaria as a host-vector disease, he recommended that the mosquitoes population should decrease in order to control malaria. The work was expanded by [17] where they formulated the first compartmental model having susceptible-infected-recovery (SIR) as parameters. Another susceptible-exposed-infected-recovered (SEIR) populations model was formulated and numerically analyzed by [39] which examined seasonality in recurrent epidemics. The model was formulated as shown below:

$$\begin{aligned} \frac{dW}{dt} &= -\beta SI \\ \frac{dM}{dt} &= \beta SI - \kappa E \end{aligned}$$

Table 2: Parameters and description of SEIR population

Symbol	**Parameter**
Q	Susceptible population
M	Exposed population
A	Infected population
N	Recovery population
β	Transmission probability
γ	death/recovery rate
κ	rate exposed people

$$\begin{aligned} \frac{dA}{dt} &= \kappa E - \gamma I \\ \frac{dN}{dt} &= \gamma I. \end{aligned}$$

The COVID-19 is an on-going disease and spread in most counties and containment measures like quarantine and isolation of infected individuals among others are being applied. In this model [3], the disease progress has been determined by basic reproduction number. Data from different countries were considered and analysis showed that the infection peak was reached 10 days after restriction measures were introduced. It was suggested from the model that the introduction of quarantine was not sufficient and stricter measures were further needed for the control of corona virus infection [16]. This model had several limitations such as the assumption of single incubation period, where other available data contradicting the 10 days according to this model and state the incubation could take up to four weeks. Therefore, distribution delay was suggested to be used [20]. They came up with the model of Susceptible-infected-recovery model(SIR). Other parameters and variables were not included like dr and latent class.

$$\begin{aligned} \frac{dS}{dt} &= -\kappa IS \\ \frac{dI}{dt} &= \kappa IS - \beta I - \sigma I \\ \frac{dR}{dt} &= \beta I \end{aligned}$$

Another quarantine model was introduced where a sub-population of asymptomatic individuals was considered. As the incubation period ended, the disease symptoms manifested and individuals were isolated in the quarantine to

Table 3: Parameters and description of symptomatic individuals

Symbol	**Parameter**
S	Susceptible population
I	Infected population
R	Recovery population
κ	Transmission rate
β	Recovery rate
β	Mortality rate due to infection

Table 4: Parameters and description of asymptomatic individuals

Symbol	**Parameter**
S	Susceptible population
I	Latent Infected individuals
τ	Incubation period
$t-\tau$	Individuals infected rate before incubation period

avoid infecting others [23]. As a result, they could not spread the infection any more. The model did not consider the movement of people and how this can infect others. From the model, no co-morbidity, vaccination, effect of infection during asymptomatic period was not considered [24] of which are the interest in this work. The model became as follows:

$$\begin{aligned} \frac{dS}{dt} &= -\kappa I(t)S(t) \\ \frac{dI}{dt} &= \kappa I(t)S(t) - \kappa I(t-\tau)S(t-\tau) \end{aligned}$$

The model above in [13] showed exposure time as a parameter was developed and analyzed. For a local setting, such as large social gatherings when R_0 of 2 and a 14 day infection period, it was possible for an infected person staying more than 9 hours in a social gathering to infect other people. Recommendations from the model show that those who are attending a social gathering should have protection [29]. Due to continuous progress, surveillance and update predictions which are necessary, this can cause a change in the prescription of the model hence more research should be carried out. An exposed individual in such a setting can be protected from being infected by staying protected

Table 5: Parameters and description of SIE model without recovery

Symbol	**Parameter**
S	Susceptible population
I	Infected population
E	Exposed population
β	Rate of infection
α	Level of protection of exposed person
c	The arrival-departure rate of attendees
τ	Time scale and turning factor to adjust parameters

(via washing of hands and/or use of face mask) [28]. The β was derived based on the known R_0, population size of the susceptible S_0 hence $\beta = \frac{R_0}{\tau} + \frac{R_0}{\tau S_0}$. The model did not include the Recovery class as more people are recovering from the disease, vaccination class, parameters like natural death rate and death induced by COVID-19 infection and comorbidity such as diabetes were not used [30]. Model description is as follows:

$$\begin{aligned} \frac{dS}{dt} &= -\beta S\frac{1}{N} + \alpha E + c \\ \frac{dE}{dt} &= \beta S\frac{1}{N} - (1-\alpha)\tau E \\ \frac{dI}{dt} &= (1-\alpha)\tau E \end{aligned}$$

A model containing isolation class [31], has been formulated and analyzed. From analytical results, close physical human interaction causes the spread of COVID-19. They recommended that infected individuals should be isolated to reduce further disease spread. As disease infection cases rise, they recommend realtime data to be used and more complicated models such as models for co-morbidity which had not been yet studied.
It has been observed in [32] and [33] that the disease was transmitted from infected people and infected surfaces. When infected population recover permanently or get permanent protection, then we have $R_0 < 1$. But, when there was no permanent recovery or protection, then re-infection occurs and R_0=1 hence undergo backward bifurcation. Due to re-appearance of the disease, vaccination, screening and isolation of infected individual were recommended. Further research and development of models should be carried out as they did not factor in vaccination as a mean of re-infection and future mass transmission. This study develops a mathematical model for co-morbidity under vaccination to fill in some of the knowledge gaps.

2 Literature review

Diabetes is known as a globally silent sweeping epidemic mostly contributed by increasing number of people becoming sick of the disease [34]. A model wasndeveloped that had a resonance period of 2.9847134 hours which was far below that of existing model of 3.5232581 hours illustrating that the glucose concentration normalized quickly [35]. The model only focussed on internal rate of increase of the blood glucose concentration. Recommendation was made for a model that would factor the external rate of increase of the blood glucose concentration [36]. The model was based on detection of diabetes but a co-morbidity of COVID-19 and vaccination was not considered. Below is the model formulated:

$$\begin{aligned}\frac{dg}{dt} &= -ag - bh + fe \\ \frac{dh}{dt} &= cg - dh + ke \\ \frac{de}{dt} &= -lg - mh + ne.\end{aligned}$$

In [37] the author developed a malaria-Rotavirus co-infection model. The results showed that when there is treatment of co-infected individuals, there is reduction on the effect of the two diseases. Global stability can be achieved if maximum protection was given to co-infection. Co-infection analysis of the model showed that it underwent forward bifurcation. Numerical simulation done using reasonable parameter values indicated that co-morbidity sustained whenever R_{mr} was more than unit but failed when it was less than one [38]. They dwelt on the co-infection of malaria and rotavirus but we will be dealing with diabetes and COVID-19 under vaccination comparing the rate of recovery when you have co-morbidity. The model was developed as follows:

$$\begin{aligned}\frac{dS_H}{dt} &= \Lambda_H - \frac{\beta_m b_m I_v}{N_H} S_H - \beta_R \frac{L_R + L_{MR} + \phi(L_R + L_{MR})}{N_H} S_H \\ &- \mu_H S_H + \gamma_1 L_M + \gamma_2 L_R + \gamma_3 L_{MR} \\ \frac{dI_M}{dt} &= \frac{\beta_m b_m I_v}{N_H} S_H - \theta\beta_R \frac{L_R + L_{MR} + \phi(L_R + L_{MR})}{N_H} I_M - \gamma_1 I_M - \vartheta_M I_M \\ \frac{dL_R}{dt} &= \beta_R \frac{L_R + L_{MR} + \phi(I_R + I_{MR})}{N_H} S_H - \frac{\beta_m b_m I_v}{N_H} L_R - \mu_H L_R - \psi_H L_R\end{aligned}$$

which is equal to

$$\begin{aligned}\frac{dS_H}{dt} &= \Lambda_H - \frac{\beta_m b_m I_v}{N_H} S_H - \beta_R \frac{L_R + L_{MR} + \phi(L_R + L_{MR})}{N_H} S_H \\ &- \mu_H S_H + \gamma_1 L_M + \gamma_2 L_R + \gamma_3 L_{MR}\end{aligned}$$

$$\begin{aligned}\frac{dI_M}{dt} &= \frac{\beta_m b_m I_v}{N_H} S_H - \theta\beta_R \frac{L_R + L_{MR} + \phi(L_R + L_{MR})}{N_H} I_M \\ &- \gamma_1 I_M - \mu_H I_M - \vartheta_M I_M.\end{aligned}$$

In [40], the researchers developed and analyzed model of children with co-morbidity of malaria and pneumonia. The results showed that co-infection reduces due to low transmission rate. The rate of co-infection should be lowered by treating both diseases earlier before they become comorbidity. While the model focussed on malaria and pneumonia, we have developed a model of COVID19-Diabetes comorbidity considering vaccination. The model was developed as below;

$$\begin{aligned}\frac{dS_H}{dt} &= \Lambda_H - \lambda_M S_H - \lambda_P S_H - \mu_H)S_H + \pi I_M + \tau I_P + \phi I_{MP} \\ \frac{dI_M}{dt} &= \lambda_M S_H - \vartheta\lambda_P I_M - \mu_H I_M - \pi I_M - \sigma_M I_{MP} \\ \frac{dI_P}{dt} &= \lambda_P S_H - \varepsilon\lambda_M I_P - \mu_H)I_P - \tau I_P - \sigma_p I_p \\ \frac{dI_{MP}}{dt} &= \varepsilon\lambda_M I_P - -\vartheta\lambda_P I_M - (\mu_H - \sigma_{MP} + \sigma_p + \sigma_M + \phi)I_{MP} \\ \frac{dS_V}{dt} &= \Lambda_V N_V - \lambda_V S_V - \mu_V S_V \\ \frac{dS_V}{dt} &= \lambda_V S_V - \mu_V I_V\end{aligned}$$

The diabetes burden and its complications [6] was developed and analyzed. The findings were given in different scenarios to stress on its important features. Those with complications took time to recovery and the rate of death was high compared to those who did not have complications given COVID-19 was one of the complications. A lot of care should be taken when managing diabetic individuals with complications compared to those without, hence there is need to study the co-morbidity of CDUV.

The work of [41] developed and analysed a very instrumental model. As a result, the transmission and contacting disease again after recovery was higher. From the model, they did not consider the asymptomatic class for COVID-19 and COVID-19 with comorbidity. In our model, we consider diabetes as a comorbidity and include asymptomatic class for COVID-19 and latent class for CDUV. Individuals can infectious while having or without symptoms and so can infect people with diabetes hence it is necessary to study a model for COVID-19 and diabetes [42]. The model developed as shown below:

$$\frac{dS_H}{dt} = \Lambda_H - (\lambda_{CV} + \Theta_{CM} + \mu_H)S_H$$

$$\begin{aligned}
\frac{dS_{CM}}{dt} &= \Theta_{CM}S_H - \chi_{CM}\lambda_{CV}S_{CM} - \mu_H S_{CM} \\
\frac{dI_{CV}}{dt} &= \lambda_{CV}S_H - (\eta_1 + \varphi_{I1} + \mu_H)I_{CV} + \psi_1\lambda_{CV}R_{CV} \\
\frac{dQ_{CV}}{dt} &= \eta_1 I_{CV} + (\varphi_{Q1} + \delta_1 + \mu_H)Q_{CV} \\
\frac{dR_{CV}}{dt} &= \varphi_{I1}I_{CV} + \varphi_{Q1}Q_{CV} - \mu_H R_{CV} - \psi_1\lambda_{CV}R_{CV} \\
\frac{dI_{CVCM}}{dt} &= \chi_{CM}\lambda_{CV}S_{CM} - (\eta_2 + \varphi_{I2} + \mu_H)I_{CVCM} + \psi_2\lambda_{CV}R_{CV} \\
\frac{dQ_{CVCM}}{dt} &= \eta_2 I_{CVCM} + (\delta_1 + \delta_2 + \varphi_{Q2} + \mu_H)Q_{CVCM} \\
\frac{dR_{CVCM}}{dt} &= \varphi_{I2}I_{CVCM} + \varphi_{Q2}Q_{CVCM} - \mu_H R_{CM} - \psi_2\lambda_{CV}R_{CM},
\end{aligned}$$

To characterize the optimal controls, Pontryagins maximum principle [43] was used and iterative method was used to solved optimality system. They performed numerical simulations and the effects of diabetics on recovery of these complication was not considered. The complications were general and they did not analyze COVID-19 as a comorbidity [18].
It was observed in [2] that those who were diabetic had higher chances of being infected with COVID-19 compared to non diabetic. From the study vaccination class, rate of recovery were not considered as well as the rate of death which will be done in this paper. The following sets of linear equations were applied in the study:

$$\begin{aligned}
\frac{dS_d}{dt} &= \Phi - \alpha_1 S_d - \alpha_2 S_d I_c - \mu S_d \\
\frac{dD}{dt} &= \alpha_1 S_d + \delta R_c - \rho D I_c - \mu D \\
\frac{dS_c}{dt} &= \Lambda + \varrho R_c - \beta S_c I_c - \mu S_c \\
\frac{dI_c}{dt} &= \beta S_c I_c + \rho D I_c + \alpha_2 S_d I_c - \gamma I_c - \mu I_c - \mu_1 I_c \\
\frac{dR_c}{dt} &= \gamma I_c - \varrho R_c - \delta R_c - \mu R_c,
\end{aligned}$$

It is worthnoting that mathematical models for COVID-19, Diabetes and comorbidity have been developed, analyzed and several parameters used. Most of mathematical models of underlying and comorbidity of COVID-19 and diabetes have been analysed under rate of transmission and the optimal controls suggested and tested. This study focuses on the low rate of recovery and how to lower the death rate caused by the underlying condition under vaccination. This includes classes such as vaccination as many people are undergoing vacci-

nation including diabetic people and the management of the comorbidity after infection of COVID-19 on diabetic population.

3 Basic concepts

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

Definition 3.1 *([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 3.2 *([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 3.3 *([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 3.4 *([19], Definition 5.3) Diabetes is a metabolism malfunction condition that make blood to have a lot of sugar in the human body.*

Definition 3.5 *([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 3.6 *([23], Definition 2.3) The parameter* R_0 *is the number of times infected individual infect other people in their entire infectious life.*

4 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.

4.1 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.

4.2 Gronwall's inequality

Gronwall s 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 [14]. The inequality yields an exponential-type upper bound that controls the function s growth in terms of the given coefficient and initial constant.

4.3 Routh-Hurwitz stability criterion

The Routh Hurwitz stability criterion is an algebraic method for determining whether all roots of a polynomial lie strictly within the complex $\mathcal{C}$. This criterion provides a systematic procedure to test stability without explicitly computing the roots of a polynomial.

4.4 Lyapunov technique (LT)

The LT is used to investigate stability properties of equilibrium points of dynamical systems without requiring explicit solutions of equations that govern the dynamical system. In LT we construct an auxiliary scalar function, called a Lyapunov function (LF), whose behavior along system trajectories provides information about stability.

4.5 The next generation matrix (G)

This is a matrix denoted as G and used in 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].

4.6 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

$$y = \begin{pmatrix} g_1(X) \\ g_2(X) \\ . \\ . \\ g_n(X) \end{pmatrix}. \tag{1}$$

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

4.7 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.

4.8 Numerical simulation technique

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 give graphs for 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]. The numerical simulations is left for the next paper after this current one.

5 Main results

5.1 Model formulation for COVID-19 Diabetes Comorbidity under vaccination

The total population size N_H has Infected individuals (symptomatic) but diabetic population ($I_{CD}(t)$), Vaccinated individuals ($V_D(t)$), Recovered individuals diabetic population ($R_D(t)$). Modification parameters $\chi 1$ and $\chi 2$ are accounting for the relative rate of recovery for those with the two diseases as compared to those who are diabetes free. Other parameters remain the same as in Diabetes free model. The following are some of the assumptions of the model:

(i). Vaccinated individuals are free from COVID-19 diabetic population and free diabetic population.

(ii). Individuals recovered from COVID-19 diabetic population.

(iii). Illness can cause death for diabetic population or free diabetic population

(iv). Re-infection occur after recovered for diabetic population for COVID-19

(v). Vaccination can be done after recovery for those who were infected before vaccination and susceptible for diabetic population or diabetic free.

We first consider compartment S_H for susceptible individuals diabetic population and free diabetic population. In this class, population is increased by those who are diabetic and those who are not diabetic at the assumed a constant recruitment ρ_H. The reduction is done by those who are infected by diabetes and vaccinated. These reduced Susceptible class while death μ due tnatural cause reduces susceptible individuals whether they are diabetic or not.

$$\frac{dS_H}{dt} = \rho_H - \lambda_D S_H - \mu S_H. \tag{2}$$

Considering compartment, D_H, of susceptible individuals of diabetic population. Individuals who are having diabetes are the one in this class. Recruitment is done from susceptible class at the rate of λ_D which increases the number of individuals. Some of individuals are infected by COVID-19 as asymptomatic or symptomatic cause movement into two classes asymptomatic L_{CD} and symptomatic I_{CD} class and rates of λ_2 and λ_2 respectively reducing the number of individuals. In this class vaccination is done at rate θ_D reducing number to vaccinated class V_D. Both natural death and diabetes induced death occur at this class reducing the number of individuals at the rate μ and δ_d respectively.

$$\frac{dD_H}{dt} = \lambda_D S_H - (\lambda_1 + \lambda_2 + \theta + \mu + \theta_D) S_H \tag{3}$$

Compartment V_D (vaccinated individuals).
In this class individuals are vaccinated against COVID-19 to reduce the rate of infection and re-infection, this can boost stability of the immunity system. From this study, we assumed those who are vaccinated are free from COVID-19 infection. Those who are vaccinated are increasing the number of individuals. From S_H class at the rate of θ, D_H class to V_D at the rate of θ_D and R_D class those who were infected before vaccination, recovered and now being vaccinated at the rate of κ. Individuals can be vaccinated but still die and when death occurs, the number of individuals decreases.

$$\frac{dV_D}{dt} = \kappa R_D + \theta S_H + \theta_D D_H - \mu V_D \tag{4}$$

During COVID-19 infection, individuals get infected with disease but have no signs and symptoms and they are infectious. Individuals get into this class from diabetic class D_H and recovery class if there is re-infection. and this increases the number of individuals. On the other hand, when there is recovery the number of asymptomatic individuals reduce. If asymptomatic persons developed signs and symptoms then they move to infected class I_{CD} at rate of τ. Both natural death, death due diseases or two diseases can occur at the rate of μ, δ or δ_{CD} respectively.Modifying factor for recovery is χ_1 help in comparing rate of recovery for asymptomatic and symptomatic.

$$\frac{dL_{CD}}{dt} = \lambda_1 S_H + \psi_1 R_D - (\mu + \delta + \tau + \chi_1\gamma_1)L_{CD} \tag{5}$$

Now L_{CD} consists of those who are infected and showing both sign and symptoms known are as symptomatic individuals. Individuals in this class have COVID-19 and Diabetes. The recruitment is done in this class from diabetic class D_D, asymptomatic class L_{CD} and recovery class R_D if there is re-infection. The rate at which movement from these classes are done: λ_2, ψ_2 and τ respectively and increase the number of individuals. When people recover from this class, they move to recovery class R_D at γ_2. Natural attrition, death caused by two diseases or COVID-19 alone can reduce the number of individuals significantly. The rate of recovery for asymptomatic is not the same as for symptomatic hence we have modifying factor χ_2. Therefore, we have

$$\frac{dI_{CD}}{dt} = \lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \delta_{CD} + \chi_2\gamma_2)I_{CD} \tag{6}$$

For R_D, people recover from COVID-19 infection but still remain diabetic, they come from asymptomatic class L_{CD} and symptomatic I_{CD}. The rr differ from one class to another hence modifying factors χ_1 and χ_2 respectively. For those who recovered need to be vaccinated, if they were not vaccinated before infection,this done at rate of κ to vaccination class V_D. Re-infection can occur

Table 6: Parameters and interpretations of model with diabetic population

Parameter	**Interpretations**
S_H	Susceptible population
D_H	Diabetic individuals
L_{CD}	Carriers individuals (infected and infectious but asymptomatic) with diabetes
I_{CD}	Infected individuals (symptomatic)
R_D	Recovered individuals
V_D	Vaccinated individuals
ρ_H	Rate of recruitment to the susceptible individuals
λ_1	Rate of recruitment to the carrier individuals
λ_2	Rate of recruitment to the infected individuals
λ_D	Rate of recruitment to the diabetic individuals
θ	Rate of recruitment from susceptible to the vaccinated individuals
θD	Rate of recruitment from diabetes to the vaccinated individuals
κ_D	Rate of recruitment from recovered to the vaccinated individuals
μ	Natural death rate
γ_{1CD}	Rate of recovery for carrier individuals
γ_{2CD}	Rate of recovery for infected individuals
τ	Rate of transfer of carrier individuals to the infected class
β	Effective contact rate for COVID-19 transmission
δ	Death rate due to corona virus
β_1	Rate of disease transmission directly from humans
χ_1	Modification parameter carrier to recovery with both diseases
χ_2	Modification parameter infected to recovery with both diseases
α	Modification parameter for infected on transmission

after recovery cause some individuals to be re-infected asymptomatically (L_{CD}) or symptomatically (I_{CD}) at the rate of ψ_1 or ψ_2 respectively.

$$\frac{dR_D}{dt} = \chi_1\gamma_2 I_{CD} + \chi_2\gamma_1 L_{CD} - (\mu + \psi_2 + \psi_1 + \kappa)R_D \tag{7}$$

The main model in this study is:

$$\frac{dS_H}{dt} = \rho_H - \lambda_D S_H - \mu S_H \tag{8}$$

$$\frac{dD_H}{dt} = \lambda_D S_H - (\lambda_1 + \lambda_2 + \theta + \mu + \theta_D)S_H \tag{9}$$

$$\frac{dL_{CD}}{dt} = \lambda_1 S_H + \psi_1 R_D - (\mu + \delta + \tau + \chi_1\gamma_1)L_{CD} \tag{10}$$

$$\frac{dV_D}{dt} = \kappa R_D + \theta S_H + \theta_D D_H - \mu V_D \tag{11}$$

$$\frac{dI_{CD}}{dt} = \lambda_2 S_H + \psi_2 R + \tau L - (\mu + \delta + \chi_2\gamma_2)I_{CD} \quad (12)$$

$$\frac{dR_D}{dt} = \chi_1\gamma_2 I_{CD} + \chi_2\gamma_1 L_{CD} - (\mu + \psi_2 + \psi_1 + \kappa)R_D. \quad (13)$$

At this juncture we do analysis of COVID-19 and Diabetes comorbidity.

5.2 Positivity of solution

Proposition 5.1 *From the Model 8 Let the initial conditions be be denoted as* (S_H,D_H,L_{CD},V_D,I_{CD} *and* R_D)(0) > 0. *There is complete positivity for the solution set* S_H,D_H,L_{CD},V_D,I_{CD} *and* $R_D(t)$.

Proof. Taking Model 8, we have

$$\frac{dS_H}{dt} = \rho_H - (\lambda_d + \mu)S_H \quad (14)$$

$$\frac{dD_H}{dt} = \lambda S_H - (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)D_H \quad (15)$$

$$\frac{dL_{cd}}{dt} = \lambda_1 D_H + \psi_1 R_d - (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})L_{cd} \quad (16)$$

$$\frac{dV_D}{dt} = \kappa R_d + \theta D_H - (\mu + \delta_d)V_d \quad (17)$$

$$\frac{dI_{cd}}{dt} = \lambda_2 D_H + \psi_2 R_d + \tau L_{cd} - (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})I_{cd} \quad (18)$$

$$\frac{dR_D}{dt} = \chi_2\gamma_2 I_{cd} + \chi_1\gamma_1 L_{cd} - (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)R_d \quad (19)$$

Now consider the first equation in the model above $\frac{S_H}{dt} = \rho_H - (\lambda_d + \mu)S_H$ then we have

$$\frac{dS_H}{dt} = \rho_H - (\lambda_d + \mu)S_H \quad (20)$$

$$\frac{dS_H}{dt} \geq (\lambda_d + \mu)S_H \quad (21)$$

Separating the variables yields

$$\frac{dS_H}{S_H} \geq (\lambda_d + \mu)dt \quad (22)$$

Integrating the differential inequality yields

$$\ln S_1(|\ \_s_0{}^s) \geq (\lambda_d + \mu)t_1(|\ \_t_0^t) \quad (23)$$

and applying the initial conditions $t = 0$,$S_H = S_0$ on

$$S_t \geq S_0 e^{(\lambda_d + \mu)} \geq 0 \quad (24)$$

since $\lambda_D + \mu \geq 0$. Applying same procedure to the remaining variables will indicate all are positive for all $t > 0$.

5.3 Boundedness of solution

We sum up the right hand side of four equations of the compartmentalized model 8 gives $N = S_H + D_H + L_{cd} + V_d + I_{cd} + R_d$, we have

$$\frac{dN}{dt} = \frac{dS_H}{dt} + \frac{dV_d}{dt} + \frac{dI_{cd}}{dt} + \frac{dL_{cd}}{dt} + \frac{dR_d}{dt}, \tag{25}$$

and substitution of corresponding value from model 8 and working out gives

$$\frac{dN}{dt} = \Lambda - \delta_d(D_H + V_d + R_d) - \delta_{dc}(L_{cd} + I_{cd}) - (S_H + D_H + L_{cd} + V_d + I_{cd} + R_d)\mu. \tag{26}$$

Thus,

$$\frac{dN}{dt} = \Lambda - \mu(S_H + D_H + L_{cd} + V_d + I_{cd} + R_d) \tag{27}$$

and so

$$\frac{dN}{dt} = \Lambda - \mu N. \tag{28}$$

Therefore we obtain

$$\frac{dN}{dt} \leq \Lambda - \mu N, \tag{29}$$

Hence, we have

$$\frac{dN}{dt} + \mu N \leq \Lambda. \tag{30}$$

Applying integrating factor and separation of variables and solving for 30 we get

$$N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t} \tag{31}$$

From Inequality 31, it can be clearly seen that

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t}, \tag{32}$$

where $N(0)$ is the initial population. Thus, as $t \to \infty$, we have

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} \tag{33}$$

This indicates that the model 8 is well-posed mathematically and epidemiologically and it is sufficient to consider its solution. It is uniformly bounded and represent human population which is non negative for all time $t \geq 0$.

5.4 Disease Free Equilibrium (DEF)

We consider the DEF denoted by $E^0(S_H^0, D_H^0, L_{cd}^0, V_d^0, I_{cd}^0)$. Using model 8 reduced as without R_d, we have

$$\begin{aligned}
\frac{dS_H}{dt} &= \rho_H - (\lambda_d + \mu)S_H \\
\frac{dD_H}{dt} &= \lambda S_H - (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)D_H \\
\frac{dL_{cd}}{dt} &= \lambda_1 D_H + \psi_1 R_d - (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})L_{cd} \\
\frac{dV_d}{dt} &= \kappa R_d + \theta D_H - (\mu + \delta_d)V_d \\
\frac{dI_{cd}}{dt} &= \lambda_2 D_H + \psi_2 R_d + \tau L_{cd} - (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})I_{cd}.
\end{aligned}$$

Consider

$$S_H^0 = \frac{\rho_H}{(\lambda_d + \mu)}. \tag{34}$$

Considering the compartmentalized System 8, we obtain from the second equation

$$D_H^0 = \frac{\lambda S_H}{(\theta_d + \mu + \delta_d)} \tag{35}$$

and substituting Equation 34 into Equation 35 we obtain

$$D_H^0 = \frac{\lambda \rho_H}{(\lambda_d + \mu)(\theta_d + \mu + \delta_d)}, \tag{36}$$

Using the forth equation of the compartmentalized system 8, we get

$$V_d^0 = \frac{\theta D_H}{(\mu + \delta_d)}. \tag{37}$$

Substituting Equation 36 and Equation 37 and solving for V_d^0, we get

$$V_d^0 = \frac{\theta\lambda\rho_H}{(\lambda_d + \mu)(\theta_d + \mu + \delta_d)(\mu + \delta_d)}. \tag{38}$$

Using Equation 34, Equation 36 and Equation 38, the DFE E^0, of model 8 is

$$E^0(S_H^0, D_H^0, L_{cd}^0, V_d^0, I_{cd}^0) = \frac{\rho_H}{(\lambda_d + \mu)}, \frac{\lambda\rho_H}{A}, \frac{\theta\lambda\rho_H}{B}, 0, 0, 0, \tag{39}$$

where $A = (\lambda_d + \mu)(\theta_d + \mu + \delta_d)$ and $B = (\lambda_d + \mu)(\theta_d + \mu + \delta_d)(\mu + \delta_d)$.

5.4.1 The Basic Reproduction Number

We consider a population with those having diabetes but are being affected with COVID-19. From the model infection classes are I_{cd} and L_{cd}. The infectious subsystem is reduced to:

$$\begin{aligned}\frac{dL_{cd}}{dt} &= \lambda_1 D_H + \psi_1 R_d - (\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})L_{cd};\\ \frac{dI_{cd}}{dt} &= \lambda_2 D_H + \psi_2 R_d + \tau L_{cd} - (\mu+\delta+\chi_2\gamma_2+\delta_{dc})I_{cd}.\end{aligned}$$

The infection rate matrix F can be constructed as; new infection is divided into two where a fraction of those who are causing new infection at L class diabetic population and those who are causing new infection diabetic population 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; $(1-n)\lambda D_H$ into L class and $(n)\lambda D_H$ into I so we have

$$F = \begin{pmatrix} (1-n)\lambda D_H \\ n\lambda D_H \end{pmatrix} \tag{40}$$

Substituting λ into Equation 40 we have

$$F = \begin{pmatrix} (1-n)(\beta_L \frac{L}{N} + \beta_I \frac{I}{N})D_H \\ n(\beta_L \frac{L}{N} + \beta_I \frac{I}{N})D_H \end{pmatrix}. \tag{41}$$

The Jacobian of F becomes

$$F = \frac{D_H}{N}\begin{pmatrix} (1-n)(\beta_L & (1-n)(\beta_I \\ n(\beta_L & n(\beta_I \end{pmatrix} \tag{42}$$

and the Jacobian matrix is deduced as

$$J_{L,I} = \begin{pmatrix} (\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc}) & 0 \\ -\tau & (\mu+\delta+\chi_2\gamma_2+\delta_{dc}) \end{pmatrix}. \tag{43}$$

Hence,

$$V = \begin{pmatrix} (\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc}) & 0 \\ -\tau & (\mu+\delta+\chi_2\gamma_2+\delta_{dc}) \end{pmatrix} \tag{44}$$

and to calculate the inverse of V, we get the determinant of V as

$$\begin{aligned} detV &= [-(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})][-(\mu+\delta+\chi_2\gamma_2+\delta_{dc})]\\ &= (\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})(\mu+\delta+\chi_2\gamma_2+\delta_{dc})\end{aligned}$$

So,

$$V^{-1} = \begin{pmatrix} \frac{1}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} & 0 \\ \frac{\tau}{((\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})(\mu+\delta+\chi_2\gamma_2+\delta_{dc}))} & \frac{1}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})} \end{pmatrix}. \tag{45}$$

Therefore,

$$FV^{-1} = \frac{D_H}{N}\begin{pmatrix} A & \frac{(1-n)\beta_I}{(\mu+\delta+\gamma_2)} \\ \frac{n\beta_L}{(\mu+\delta+\tau+\gamma_1)} + n\beta_I\frac{\tau}{(\mu+\delta+\tau+\gamma_1)(\mu+\delta+\gamma_2)} & \frac{n\beta_I}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})} \end{pmatrix}, \quad (46)$$

where $A = \frac{(1-n)\beta_L}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} + (1-n)(\beta_I\frac{\tau}{((\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})(\mu+\delta+\chi_2\gamma_2+\delta_{dc}))})$.

The two eigenvalues represent asymptomatic class population L_{cd} and symptomatic infected class I_{cd} diabetic population hence we have two R_0, L_{cd} and R_0, I_{cd}. We add the two and get the total basic reproduction number $R_0 = R_0, L + R_0, I$.
So, $\frac{D_H}{N}[\frac{(1-n)\beta_{L_{cd}}}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} + (1-n)\beta_{I_{cd}}\frac{\tau}{((\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})(\mu+\delta+\chi_2\gamma_2+\delta_{dc}))}] - \eta$,
and

$$\frac{D_H}{N}[\frac{n\beta_{I_{cd}}}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}] - \eta.$$

Hence,

$$\begin{aligned} R_0, L_{cd} &= \frac{D_H}{N}[\frac{(1-n)\beta_{L_{cd}}}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} \\ &+ (1-n)(\beta_{I_{cd}}\frac{\tau}{[(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})(\mu+\delta+\chi_2\gamma_2+\delta_{dc})]}, \end{aligned}$$

$R_0, I_{cd} = \frac{D_H}{N}\frac{n\beta_{I_{cd}}}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}$ and adding the two gives,
$R_0 = \frac{D_H}{N}[\frac{(1-n)\beta_{L_{cd}}}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} + (1-n)(\beta_{I_{cd}}\frac{\tau}{[(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})(\mu+\delta+\chi_2\gamma_2+\delta_{dc})} + \frac{n\beta_{I_{cd}}}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}]$
Rearranging R_0 we obtain
$R_0 = \frac{D_H}{N}[\frac{(1-n)\beta_{L_{cd}}}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} + \frac{\beta_{I_{cd}}}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}(\frac{(1-n)\tau}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} + n)]$
Now, at DFE where $D_H^0 = \frac{\lambda\rho_H}{(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$ and $N = \frac{\Lambda}{\mu}$ we substitute D_H^0 and N into R_0 to get $R_0 = \frac{\mu}{(\theta+\mu)}[\frac{(1-n)\beta_L}{(\mu+\delta+\tau+\gamma_1)} + \frac{\beta_I}{(\mu+\delta+\gamma_2)}(n + \frac{(1-n)\tau}{(\mu+\delta+\tau+\gamma_1)})]$.
From R_0 we can conclude that:

(i). $\frac{(1-n)\beta_{L_{cd}}}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})}$ infections caused by only those who are in L_{cd} class.

(ii). $\frac{\beta_{I_{cd}}}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}(\frac{(1-n)\tau}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})} + n)$ infections caused by those who went straight to I_{cd} class $n\frac{\beta_{I_{cd}}}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}$ and those who enter through L_{cd} showed signs and symptoms then moved to I $\frac{\beta_I}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}(\frac{(1-n)\tau}{(\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})})$.

(iii). When $\tau = 0$, we have $R_0 = \frac{\mu}{(\theta+\mu)}[\frac{(1-n)\beta_{L_{cd}}}{(\mu+\delta+\chi_1\gamma_1+\delta_{dc}))} + \frac{\beta_{I_{cd}}}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})}n]$.

5.5 The Local Stability of DFE for Diabetic Population

We now analyze the local stability of DFE for diabetic population. We state the result below.

Theorem 5.2 *The DFE for diabetic population of Model 8 is locally asymptotically stable.*

Proof. The DFE for diabetic population of Model 8 can be studied by evaluating its Jacobian matrix given as:

$$J_E^0 = \begin{pmatrix} X & 0 & 0 & 0 & 0 & 0 \\ \lambda & -Y & 0 & 0 & 0 & 0 \\ 0 & \lambda_1 & -Z & 0 & 0 & \psi_1 \\ 0 & \theta & 0 & -P & 0 & \kappa \\ 0 & \lambda_2 & \tau & 0 & -Q & \psi_2 \\ 0 & 0 & \chi_1\gamma_1 & 0 & \chi_2\gamma_2 & -R \end{pmatrix}, \tag{47}$$

where $X = -(\lambda_d + \mu)$, $Y = (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)$, $Z = (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})$, $P = (\mu + \delta_d)$, $Q = (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})$ and $R = (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)$. The characteristic equation is $[-((\lambda_d + \mu)][(\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)][-(\mu + \delta_d)][-(\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})][-(\mu + \delta + \chi_2\gamma_2 + \delta_{dc})][-(\mu + \psi_2 + \psi_1 + \kappa + \delta_d)] = 0$. Therefore, $\lambda = -(\mu + \delta + \chi_2\gamma_2 + \delta_{dc}), -(\lambda_d + \mu) and - (\mu + \delta_d)$ making R_0 negative. This will reduce matrix J_E^0 to

$$J_E^0 = \begin{pmatrix} -G & 0 & 0 \\ \lambda_1 & -H & \psi_1 \\ 0 & \chi_1\gamma_1 & -L \end{pmatrix}, \tag{48}$$

where $G = (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)$, $H = (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})$ and $L = (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)$. The negative trace of the Matrix 47 is $-[(\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d) + (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc}) + (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)]$, and $\det(J_E^0)$ is $(\lambda_1+\lambda_2+\theta_d+\mu+\delta_d)[(\psi_1\chi_1\gamma_1)-((\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})(\mu+\psi_2+\psi_1+\kappa+\delta_d))]$ which is positive provided $(\psi_1\chi_1\gamma_1) > ((\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})(\mu + \psi_2 + \psi_1 + \kappa + \delta_d))$. Routh-Hurwitz condition holds hence DFE for diabetic population is LAS.

5.6 The Global Stability of DFE for Diabetic population

We do the analysis of DFE for diabetic population of Model 8 to establish if it is GAS. From model 8, w have X=(S_H, D_H, V_d) and Z=(L_{cd},I_{cd}, R_d), therefore we obtain

$$H(X, 0) = \begin{pmatrix} \rho_H - (\theta + \mu)S_H \\ \theta S_H - \mu V \end{pmatrix} \tag{49}$$

which is equivalent to

$$H(X,0) = \begin{pmatrix} -C & 0 & 0 \\ \lambda_1 & -D & \psi_1 \\ 0 & \chi_1\gamma_1 & -E \end{pmatrix}, \tag{50}$$

where $C = (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)$, $D = (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})$ and $E = (\mu+\psi_2+\psi_1+\kappa+\delta_d)$. Equation 71 has a unique equilibrium point at $X = (\frac{\rho_H}{(\lambda_d+\mu)}, \frac{\lambda\rho_H}{(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}, \frac{\theta\lambda\rho_H}{(\lambda_d+\mu)(\theta_d+\mu+\delta_d)(\mu+\delta_d)}$ which is GAS. Now, we get

$$P = \begin{pmatrix} K & 0 & \psi_1 \\ \tau & J & \psi_2 \\ \chi_1\gamma_1 & \chi_2\gamma_2 & -O \end{pmatrix}, \tag{51}$$

where $K = \frac{\beta}{N}D_H - (\mu+\delta+\tau+\chi_1\gamma_1+\delta_{dc})$, $J = \frac{\beta}{N}D_H - (\mu+\delta+\chi_2\gamma_2+\delta_{dc})$ and $O = (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)$. From matrix P, we do an evaluation and obtain

$$\begin{aligned} PZ &= \frac{\beta}{N}D_H L - (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})L \\ &+ \psi_1 R\tau L + \frac{\beta}{N}D_H I - (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})I \\ &+ \psi_2 R\chi_1\gamma_1 L + \chi_2\gamma_2 I - (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)R. \end{aligned}$$

and

$$\begin{aligned} GZ &= \frac{\beta}{N}D_H L - (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})L \\ &+ \psi_1 R\tau L + \frac{\beta}{N}D_H I - (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})I \\ &+ \psi_2 R\chi_1\gamma_1 L + \chi_2\gamma_2 I - (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)R. \end{aligned}$$

So,

$$\widehat{G}Z = PZ - GZ = 0. \tag{52}$$

$\widehat{G}Z = [0]^T$. Therefore, $\widehat{G}Z = 0$ and hence E^0 is GAS.

5.7 The Endemic Equilibrium (EE)

We denote EE by $E^*(S_H^*, D_H^*, V_d^*, L_{cd}^*, I_{cd}^*, R_d^*)$ and we carry out the analysis as given by the next result.

Theorem 5.3 *The Endemic equilibrium $E^*(S_H^*, D_H^*, V_d^*, L_{cd}^*, I_{cd}^*, R_d^*)$ exists provided that $R_0 > 1$.*

Proof. Using model 8 and equating each equation to zero we have

$$\begin{aligned}
\frac{dS_H^*}{dt} &= \rho_H - (\lambda_d + \mu)S_H^* \\
\frac{dD_H^*}{dt} &= \lambda S_H^* - (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)D_H^* \\
\frac{dL_{cd}^*}{dt} &= \lambda_1 D_H^* + \psi_1 R_d^* - (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})L_{cd}^* \\
\frac{dV_D^*}{dt} &= \kappa R_d^* + \theta D_H^* - (\mu + \delta_d)V_d^* \\
\frac{dI_{cd}^*}{dt} &= \lambda_2 D_H^* + \psi_2 R_d^* + \tau L_{cd}^* - (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})I_{cd}^* \\
\frac{dR_D^*}{dt} &= \chi_2\gamma_2 I_{cd}^* + \chi_1\gamma_1 L_{cd}^* - (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)R_d^*
\end{aligned}$$

So,

$$S_H^* = \frac{\rho_H}{(\lambda_d + \mu)}. \tag{53}$$

From the the second equation we have

$$D_H^* = \frac{\lambda S_H^*}{(\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)}. \tag{54}$$

Substituting Equation 53 into equation 54, we obtain

$$D_H^* = \frac{\lambda\rho_H}{(\lambda_d + \mu)(\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)}. \tag{55}$$

Considering the third equation

$$L_{cd}^* = \frac{\lambda_2 D_H^* + \psi_2 R_d^*}{(\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})}, \tag{56}$$

and substituting Equation 55 into Equation 56 we get

$$L_{cd}^* = \frac{\lambda\rho_H\lambda_1}{(\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})(\lambda_d + \mu)(\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)} + \frac{\psi_1 R_d^*}{(\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})}, \tag{57}$$

and from the fifth equation we get

$$I_{cd}^* = \frac{\lambda_2 D_H^* + \tau L_{cd}^* + \psi_2 R_d^*}{(\mu + \delta + \chi_2\gamma_2 + \delta_{dc})}. \tag{58}$$

Now, substituting equation 55 into equation 58, we get

$$I_{cd}^* = \frac{\lambda_2\lambda\rho_H}{(\lambda_d + \mu)(\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)(\mu + \delta + \chi_2\gamma_2 + \delta_{dc})} + \frac{\tau L_{cd}^* + \psi_2 R_d^*}{(\mu + \delta + \chi_2\gamma_2 + \delta_{dc})}. \tag{59}$$

From the last equation we have

$$R_d^* = \frac{\gamma_2 I_{cd}^* + \chi_1\gamma_1 L_{cd}^*}{(\mu + \psi_2 + \psi_1 + \kappa + \delta_d)}, \tag{60}$$

Now, substituting R_0 into equation 58, we have

$$I_{cd}^* = \frac{R_0\rho_H}{(\lambda_d + \mu)(\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)(\mu + \delta + \chi_2)} + \frac{(\tau L_{cd}^* + \psi_2 R_d^*)\lambda_2}{R_0}. \tag{61}$$

Let $x = \mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc}$,
$x_1 = \lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d$
$x_2 = \lambda_d + \mu$
$x_3 = \mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc}$
$x_4 = \mu + \psi_2 + \psi_1 + \kappa + \delta_d$
$x_5 = \mu + \delta + \chi_2$.
Then we have

$$L_{cd}^* = \frac{\lambda\rho_H\lambda_1}{xx_2x_1} + \frac{\psi_1 R_d^*}{x_3}, \tag{62}$$

$$R_d^* = \frac{\gamma_2 I_{cd} + \chi_1\gamma_1 L_{cd}}{x_4}, \tag{63}$$

$$I_{cd}^* = \frac{R_0\rho_H}{x_2x_1x_5} + \frac{(\tau L_{cd} + \psi_2 R_d^*)\lambda_2}{R_0}, \tag{64}$$

Substituting Equation 63 into equation 62, we obtain

$$L_{cd}^* = \frac{\lambda\rho_H\lambda_1 x_3 x_4}{(xx_1x_2)(x_3x_4 - \psi_1\chi_1\gamma_1)} + \frac{\psi_1\gamma_2 I_{cd}}{(x_3x_4 - \psi_1\chi_1\gamma_1)}. \tag{65}$$

Again substituting Equation 63 into Equation 64

$$I_{cd}^* = \frac{R_0\rho_H}{x_2x_1x_5} + \frac{\psi_2\lambda_2 I_{cd}}{R_0x_4} + \frac{\tau x_4\lambda_2 + R_0\psi_2\lambda_2 L_{cd}}{R_0x_4}. \tag{66}$$

Let $N = x_3x_4 - \psi_1\chi_1\gamma_1$
$N_1 = xx_1x_2$
$N_2 = x_2x_1x_5$
$N_3 = x_3x_4$.
Substituting Equation 65 into Equation 66 and working out gives

$$I_{cd}^* = R_0^2\rho_H x_4 N - (\frac{N_1 N x_4 - \lambda\rho_H\lambda_1\psi_2\lambda_2 N_3}{N_1})R_0 + (\lambda_2 N x_4 + \psi_2\lambda_2\psi_1\gamma_2 + N\psi_2\lambda_2). \tag{67}$$

Equating Equation 67 to zero gives,

$$R_0^2\rho_H x_4 N - (\frac{N_1 N x_4 - \lambda\rho_H\lambda_1\psi_2\lambda_2 N_3}{N_1})R_0 + (\lambda_2 N x_4 + \psi_2\lambda_2\psi_1\gamma_2 + N\psi_2\lambda_2 - I_{cd}^*) = 0. \tag{68}$$

We conclude by saying there is only one possible sign change at (+,-,+). When R_0 is positive then $S_H^*, D_H^*, V_d^*, L_{cd}^*, I_{cd}^*, R_d^*$ are also positive.

5.8 The Local Stability of EE for Diabetic population

The Jacobian at the endemic equilibrium of system 8 below

$$\begin{aligned}
\frac{dS_H^*}{dt} &= \rho_H - (\lambda_d + \mu)S_H^* \\
\frac{dD_H^*}{dt} &= \lambda S_H^* - (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)D_H^* \\
\frac{dL_{cd}^*}{dt} &= \lambda_1 D_H^* + \psi_1 R_d^* - (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})L_{cd}^* \\
\frac{dV_D^*}{dt} &= \kappa R_d^* + \theta D_H^* - (\mu + \delta_d)V_d^* \\
\frac{dI_{cd}^*}{dt} &= \lambda_2 D_H^* + \psi_2 R_d^* + \tau L_{cd}^* - (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})I_{cd}^* \\
\frac{dR_D^*}{dt} &= \chi_2\gamma_2 I_{cd}^* + \chi_1\gamma_1 L_{cd}^* - (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)R_d^*,
\end{aligned}$$

is given by

$$J(E^*) = \begin{pmatrix}
-\Gamma & 0 & 0 & 0 & 0 & 0 \\
\lambda & -\Delta & 0 & 0 & 0 & 0 \\
0 & \lambda_1 & -\Theta & 0 & 0 & \psi_1 \\
0 & \theta & 0 & -\Xi & 0 & \kappa \\
0 & \lambda_2 & \tau & \gamma_2 & -\Pi & \psi_2 \\
0 & 0 & \chi_1\gamma_1 & 0 & \chi_2\gamma_2 & -\Sigma
\end{pmatrix}, \tag{69}$$

where $\Gamma = (\lambda_d + \mu)$, $\Delta = (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)$, $\Theta = (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})$, $\Xi = (\mu + \delta_d)$, $\Pi = (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})$ and $\Sigma = (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)$.
From matrix 69, there is one eigenvalue $-(\mu + \delta_d)$. The other eigenvalues can be deduced from the reduced matrix define as

$$J(E^*) = \begin{pmatrix}
-\Upsilon & 0 & 0 & 0 & 0 \\
\lambda & -\Phi & 0 & 0 & 0 \\
0 & \lambda_1 & -\Psi & 0 & \psi_1 \\
0 & \lambda_2 & \tau & -\Omega & \psi_2 \\
0 & 0 & \chi_1\gamma_1 & \chi_2\gamma_2 & -\varpi
\end{pmatrix}, \tag{70}$$

where $\Upsilon = (\lambda_d + \mu)$, $\Phi = (\lambda_1 + \lambda_2 + \theta_d + \mu + \delta_d)$, $\Psi = (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})$, $\Omega = (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})$ and $\varpi = (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)$.
Let, $M = (\lambda_d + \mu)$

$M_1 = (\lambda_d + \mu)$

$M_2 = (\mu + \delta + \tau + \chi_1\gamma_1 + \delta_{dc})$

$M_3 = (\mu + \delta + \chi_2\gamma_2 + \delta_{dc})$

$M_4 = (\mu + \psi_2 + \psi_1 + \kappa + \delta_d)$

Then,

$$J(E^*) = \begin{pmatrix} -M & 0 & 0 & 0 & 0 \\ \lambda & -M_1 & 0 & 0 & 0 \\ 0 & \lambda_1 & -M_2 & 0 & \psi_1 \\ 0 & \lambda_2 & \tau & -M_3 & \psi_2 \\ 0 & 0 & \chi_1\gamma_1 & \chi_2\gamma_2 & -M_4 \end{pmatrix} \tag{71}$$

5.9 The Global Stability of EE for Diabetic population

We use Lyapunov's direct method and LaSalle's principle to establish the global stability of equilibria.

Theorem 5.4 *If $R_0 > 1$ then the EE for diabetic population given and denoted by $E^*(S_H^*, D_H^*, V_d^*, L_{cd}^*, I_{cd}^*, R_d^*))$ is GAS strictly inside Ω.*

Proof. Consider a non-linear LF V_e on (S_H,D_H, V_d, L_{cd}, I_{cd},R_d):$\in \Omega \subset \Re_+^6$: whereby ($S_H$,$D_H$,$V_d$, L_{cd}, I_{cd},R_d) > 0. Then we have that

$$\begin{aligned} P_e : (S_H, D_H, V_d, L_{cd}, I_{cd}, R_d) &= \frac{1}{2}(\frac{S_H}{S_H^*} - 1)^2 + \frac{1}{2}(\frac{D_H}{D_H^*} - 1)^2 + \frac{1}{2}(\frac{V_D}{V_D^*} - 1)^2 \\ &+ \frac{1}{2}(\frac{L_{cd}}{L_{cd}^*} - 1)^2 + \frac{1}{2}(\frac{I_{cd}}{I_{cd}^*} - 1)^2 + \frac{1}{2}(\frac{R_d}{R_d^*} - 1)^2. \end{aligned}$$

So, P_e is strictly inside Ω. Therefore, P_e has attained its minimum at E^* globally on Ω and P_e:(S_H,D_H,V_d, L_{cd}, I_{cd},R_d) $= 0$. Moreover,

$$\begin{aligned} \frac{dP_e}{dt} &= \frac{1}{S_H^*}(\frac{S_H}{S_H^*} - 1)\frac{dS_H}{dt} + \frac{1}{D_H^*}(\frac{D_H}{D_H^*} - 1)\frac{dD_H}{dt} + \frac{1}{L_{cd}^*}(\frac{L_{cd}}{L_{cd}^*} - 1)\frac{dL_{cd}}{dt} \\ &+ \frac{1}{V_d^*}(\frac{V_d}{V_d^*} - 1)\frac{dV_d}{dt} + \frac{1}{I_{cd}^*}(\frac{I_{cd}}{I_{cd}^*} - 1)\frac{dI_{cd}}{dt} + \frac{1}{R_d^*}(\frac{R_d}{R_d^*} - 1)\frac{dR_d}{dt}. \end{aligned}$$

The derivatives of (S_H,D_H, V_d, L_{cd}, I_{cd},R_d) from 8 are converted and after some manipulation shows stability. Hence, $V_e' < 0$. $V_e' = 0$ if and only if $S_H = S_H^*$, $L_{cd} = L_{cd}^*$, $I_{cd} = I_{cd}^*$, $V_d = V_d^*$, $D_H = D_H^*$ and $R_d = R_d^*$. Thus, E^* is GAS in the interior of the region Ω.

5.10 Sensitivity Analysis

Parameter sensitivity shows how some parameters within the system, have higher or lower degree of influencing model's stability We consider the variables below as given in the model.

$R_0 = \frac{\lambda\mu\beta\alpha\rho_H}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$
$\frac{dR_0}{d\alpha} = \frac{\lambda\mu\beta\rho_H}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$
$\frac{dR_0}{d\lambda} = \frac{\mu\beta\alpha\rho_H}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$

Table 7: Parameters values, source and description for stability

Parameter	**Interpretations**
ρ_H	RRTS individuals
θ	RRFS to the vaccinated individuals DP
θD	RRFD to the vaccinated individuals
κ_D	RRFR to the vaccinated individuals DP
γ_{1CD}	Rate of recovery for carrier individuals DP
γ_{2CD}	Rate of recovery for infected individuals but DP
τ_{CD}	Rate of transfer of carrier individuals to the infected class DP
β	Effective contact rate for COVID-19 transmission DP
μ	Natural death rate diabetic population
δ	Death rate due to corona virus but with diabetes
α	Modification parameter for infected on transmission
χ_1	Modification parameter carrier to recovery with both diseases
χ_2	Modification parameter infected to recovery with both diseases
ψ_{1CD}	Rate of reinfections for carrier individuals but with diabetes
ψ_{2CD}	Rate of reinfections for infected individuals but with diabetes

$$\frac{dR_0}{d\rho_H} = \frac{\mu\beta\alpha\lambda}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$$
$$\frac{dR_0}{d\beta} = \frac{\rho_H\mu\alpha\lambda}{(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$$
$$\frac{dR_0}{d\theta_d} = -\frac{\beta\rho_H\mu\alpha\lambda}{\theta_d(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$$
$$\frac{dR_0}{d\gamma_2} = -\frac{\beta\rho_H\mu\alpha\lambda}{\gamma_2(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$$
$$\frac{dR_0}{d\chi_2} = -\frac{\beta\rho_H\mu\alpha\lambda}{\chi_2(\mu+\delta+\chi_2\gamma_2+\delta_{dc})(\lambda_d+\mu)(\theta_d+\mu+\delta_d)}$$

therefore $S_P^{R_0=\frac{dR_0}{dP}} \times \frac{P}{R_0}$

$S_\alpha^{R_0} = 1$, $S_\lambda^{R_0} = 1$, $S_{\rho_H}^{R_0} = 1$, $S_\beta^{R_0} = 1$, $S_{\theta_d}^{R_0} = -\frac{1}{\theta_d}$, $S_{\chi_2}^{R_0} = -\frac{1}{\chi_2}$, $S_{\chi_1}^{R_0} = -\frac{1}{\chi_1}$, $S_{\gamma_2}^{R_0} = -\frac{1}{\gamma_2}$, $S_{\gamma_1}^{R_0} = -\frac{1}{\gamma_1}$.

Parameters such as β, α, θ_d and ρ_H 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, χ_2, andχ_1 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 on diabetic population and less people will have severe illness, less deaths and less admission in 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. The analytical results in this work can be done using Python under jupyter notebook to conduct numerical simulations for model 8 using parameter given in the table below. The higher the rate of recovery, the more individuals get recovered. As rate of recovery reduces,

less people get recovered. As more people get recovered and vaccinated, less individuals get reinfected as diabetic population take control measurers and administer their medicine proper. As more people get vaccinated, few become asymptomatic and symptomatic and the attack is not persisting. This make more people to recover hence less asymptomatic and symptomatic population. The effect of varying gamma is low generally compare to other populations. As gamma increases, more individuals become vaccinated and if gamma is low then few will be vaccinated. In a lower or higher gamma, the vaccinated population continues to increase as more people get vaccinated and few are infected. General observation is that the rate of recovery is slow when there is co-morbidity. We conclude that recovery rate is low on individuals with COVID-19 disease under diabetic population but high on individuals with only COVID-19 under free diabetic population. This make the death rate, number of individuals admitted in the hospital to be higher on those with COVID-19 and Diabetes compared to those with COVID-19 only.

6 Open Problems

Certain natural problems emanate from this research. The following problems and questions can be considered for future research and further analysis. **Problem 1:** Can one carry out a numerical analysis of the comorbidity? **Problem 2:** There is need for bifurcation analysis of the model for robustness.

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.