

MATHEMATICAL MODELLING OF EFFECTS OF NON-CLINICAL STRATEGIES IN COMBATING TRANSMISSION OF SARS-CoV-2 IN KENYA

Abstract

SARS-CoV-2 is a serious problem in Kenya today. It has put an unprecedented burden on worldwide economy and public health. The rapid spread of SARS-CoV-2 has been driven predominantly by aerosol transmissions. The objectives of this study were to formulate mathematical models on the spread of SARS –CoV-2 and incorporating the effects of nonclinical strategies like screening, facemask usage, hand washing and social distancing, determine well posedness of the model, validate the developed model and finally predict the effects of nonclinical strategies on the dynamics of the spread of SARS-CoV-2 in Kenya. The mathematical model was based on SIRS epidemiological classical model. In developing the model, the population was divided into six human compartments; susceptible, exposed, infected, isolated in hospital, at home and recovered. The basic reproduction number was determined using next generation method. The model was analyzed through the determination of the model steady states. The stabilities of steady states analyzed based on reproduction number (R_0) using: signs of Jacobi matrix evaluated at steady state, Lyapunov criteria, centre manifold theorem, Metzler matrix and Routh-Hurwitz. Numerical simulations were carried out using MATLAB inbuilt ode solver based on Runge Kutta method. Sensitivity analysis of the model parameters was carried out using partial differentiation of the reproduction number and also using normalized sensitivity analysis.

From this analysis, findings showed that adherence to the containment measures and contact tracing had the greatest negative impact on the reproduction number. It was found through simulation that adherence to the SARS-CoV-2 containment measures by the population would reduce the reproduction number to below 1 hence containing the pandemic. The findings of this study show the extent to which the nonclinical can be used to contain the spread of SARS-CoV-2 in Kenya. We recommended strict adherence to containment

KEYWORDS: COVID-19, MODELING, NON- CLINICAL, SIMULATION, MATLAB

Introduction

The coronavirus disease of 2019 (covid-19) pandemic reached Kenya in March 2020 with the initial cases reported in Nairobi and Mombasa cities according to the ministry of health, Kenya. It started at Wuhan in China in December 2019. The world health organization (WHO) declared

coronavirus disease a pandemic on March 11, 2020. It belongs to the family of viruses that cause viral pneumonia and the symptoms include fever, breathing difficulties, sore throat and lung infection etc. [49].

This trend of SARS-CoV-2 transmission had posed a big threat to global public health.

Understanding the transmission dynamics of this infection and evaluating the effectiveness of control measures like wearing facemasks, screening, social distancing in public places and regular hand washing was essential in containing the spread of the disease. [46]

The negative impact of coronavirus disease had trickled down to household welfare. Many Kenyans had lost jobs and businesses experience lower earnings. Government was strained due to the eventualities of the pandemic. The cash transfer program which the government had started to caution the vulnerable citizens was no longer tenable. The spread of the pandemic was occurring on daily basis. The interaction of people could not be contained since the government was unable to cater for their daily expenses due to limited government resources and a large population of approximately 55,126,719 people as of August 2021 according to Kenya Bureau of Statistics. Learning in primary, secondary and tertiary institutions was going on in schools and the number of classes in all schools were too little to allow social distancing of at least one and half meter recommended by W.H.O. For these reasons, use of facemasks, hand-washing and social distancing remained the viable and applicable ways of controlling the spread of COVID-19 in order to contain it. However, no single intervention had ability to control the transmission on its own. There was need to understand the extent to which the combined use of use of facemasks, social distancing and hand washing could limit the spread of this pandemic in Kenya. This is the motive for carrying out of this study.

Mathematical modeling is the process of describing a system using language and concepts of mathematics. Our deterministic model took the form of differential equations with six variables. Analysis of the model included finding the reproduction (R_0) which is the number of secondary infections caused by a single infected person. [49], [44]), [8] and [21], [30], [31], [39], [5], [16], [26], [18], and [40] and [6] among other modeling scholars developed model for a single intervention leaving a gap for combined intervention measures. There was scanty information about mathematical models, which have been developed to show the combined effects of non-clinical strategies like facemask, screening, hand washing and social distancing in controlling SARS-CoV-2 transmission in Kenya. This study intended to develop and analyze mathematical models to explain the extent to which the spread of SARS-CoV-2 is mitigated by the combined use of non-clinical strategies like mass usage of facemask, screening, hands washing and social distancing compared to effects of a single containment measure in Kenya. This is the knowledge gap we intended to fill.

Methods

Model Formulation

There whole population was divided into six human compartments. These six compartments are; S- Susceptible, E-Exposed, I-Infected, I_S –Isolated at home (asymptomatic), I_H – isolated on hospital (symptomatic), R- recovered. The rate of recruitment into the population (S) is given by Λ . People are exposed due to the contact rate of λ , and then those infected are traced at the rate of γ . The infected people die at the rate of μ_1 before testing or isolation is done.

Asymptomatic people who test positive are isolated in homecare at the rate of β_2 while symptomatic are isolated in hospital at the rate of β_1 . Some people isolated in hospital die at the rate of α_2 while others isolated in homecare die at the rate of α_3 . People who have recovered after treatment can become susceptible and fall sick again at the rate of Θ if they are exposed to the virus. The people isolated in the homecare are taken to the hospital at the rate of Λ incase their condition worsens. The rate of recovery for those isolated in hospital is τ_1 while the rate of recovery for those isolated at home care is τ_2 . A person who recovers from the virus becomes susceptible at the rate of θ and can get the disease again if exposed. The natural death rate is μ .

Assumptions of the model

In this research, it is assumed that all people in the country are susceptible since SARS-CoV-2 is spread to all parts of the country. After recovery, people can get the disease again if exposed and no herd immunity. There is a constant interaction of people in the community and people who are exposed to the virus cannot transmit it unless incubation period is over. It is also assumed that some exposed people do not show any symptom even after the incubation period is over and some can show later. The infected individuals after testing positive are put into isolation in the hospital or home care hence not interacting with the community. The other assumption is that not all individuals who are infected are identified during contact tracing; the untraced infected people are taken to the hospital when they develop symptoms. It is also assumed that α_1 , α_2 , α_3 and α_4 interact linearly and cannot reduce the transmission 100%.

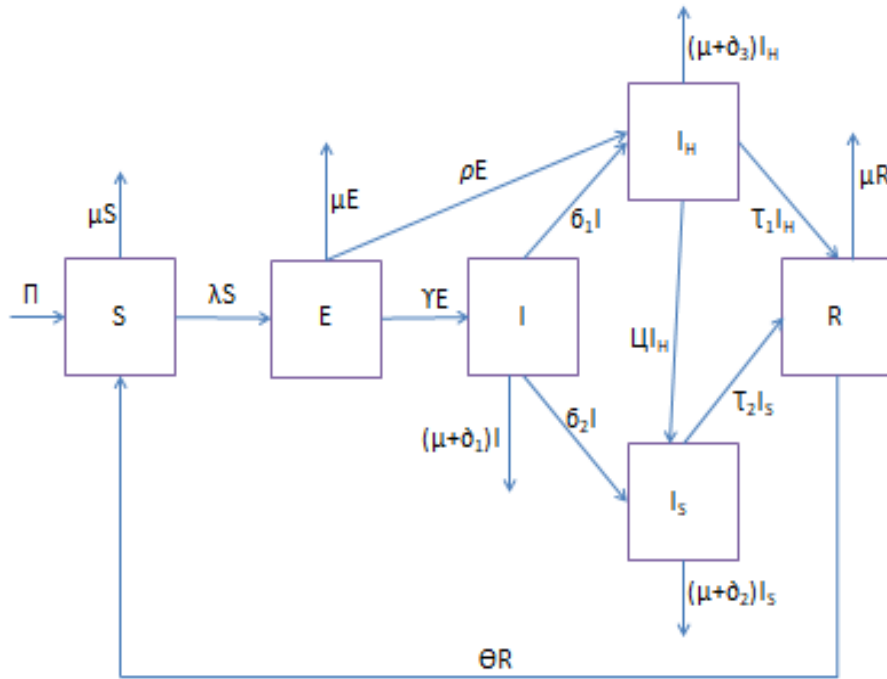


Figure 1: The flow chart of the model

Model equations

$$\frac{dS}{dt} = \Pi + \theta R - (\mu + (1-\lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S)) S \quad (1.1)$$

$$\frac{dE}{dt} = (1 - \lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S) S - (\mu + \gamma +) E \quad (1.2)$$

$$\frac{dI}{dt} = \gamma E - (\mu + \square_1 + \sigma_1 + \sigma_2) I \quad (1.3)$$

$$\frac{dI_H}{dt} = \sigma_1 I + \rho E - (\mu + \square_3 + \Upsilon + \tau_1) I_H \quad (1.4)$$

$$\frac{dI_S}{dt} = \sigma_2 I + \Upsilon I_H - (\tau_2 + \mu + \square_2) I_S \quad (1.5)$$

$$\frac{dR}{dt} = \tau_1 I_H + \tau_2 I_S - \theta E - \mu R \quad (1.6)$$

$$\text{Force of infection } \lambda = (1 - \Lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S) \quad (1.7)$$

$$\text{Where } \Lambda = (\alpha_1 + \alpha_2 + \alpha_3 + \alpha_4) \quad (1.8)$$

$$N(t) = S(t) + E(t) + I(t) + I_H(t) + I_S(t) + R(t)$$

Where N is the total population and 'β ' is the transmissibility parameter which is a function of α₁, α₂, α₃ and α₄ which are proportions of people screened, adhering to wearing facemasks, hand-washing and social distancing respectively.

BOUNDEDNESS AND POSITIVITY OF THE MODEL

We state and prove the theorem below;

Theorem 1. The region Q given by

$$\frac{dI_S}{dt} = \sigma_2 I + \Upsilon I_H - (\tau_2 + \mu + \sigma_2) I_S \quad (1.9)$$

is positive and invariant with respect to equations 1-6

Proof: let S(t), E(t), I(t), I_H(t), I_S(t) and R(t) be any solution of the system with zero or positive initial conditions S(0) ≥ 0, E(0) ≥ 0, I(0) ≥ 0, I_H(0) ≥ 0, I_S(0) ≥ 0, R(0) ≥ 0

Using comparison theorem, we solve the equations (1) to (6) to get:

$$\frac{dS}{dt} = \Pi + \theta R - (\mu + (1 - \Lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S)) S \quad (1.10)$$

Since parameter Π and θR are positive,

$$\frac{dS}{dt} \geq -(\mu + (1 - \Lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S)) S \quad (1.11)$$

Dividing both sides by S and integrating with respect to t yields

$$\ln S \geq - \int_0^t (\mu + (1 - \Lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S)) dt$$

Substituting the initial condition S(0) yields:

$$S(t) \geq S(0) e^{-(\mu + (1 - \Lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S)) t} \geq 0. \quad (1.12)$$

The same process is done to the equations 2,3,4,5, and 6 respectively to get:

$$\frac{dE}{dt} \geq -(\mu + \gamma + \theta)E, E(t) \geq E(0) e^{-(\mu + \gamma + \theta)t} \geq 0 \quad (1.13)$$

$$\frac{dI}{dt} \geq -(\mu + \sigma_1 + \sigma_2) I, I(t) \geq I(0) e^{-(\mu + \sigma_1 + \sigma_2)t} \geq 0 \quad (1.14)$$

$$\frac{dI_H}{dt} \geq -(\mu + \tau_1 + \Upsilon) I_H, I_H(t) \geq I_H(0) e^{-(\mu + \tau_1 + \Upsilon)t} \geq 0 \quad (1.15)$$

$$\frac{dI_S}{dt} - (\tau_2 + \mu + \sigma_2) I_S, I_S(t) \geq I_S(0) e^{-(\tau_2 + \mu + \sigma_2)t} \geq 0 \quad (1.16)$$

$$\frac{dR}{dt} \geq -(\mu + \theta) R, R(t) \geq R(0) e^{-(\mu+\theta)t} \geq 0 \quad (1.17)$$

Taking the time derivative of N along its solution path yields;

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dI_H}{dt} + \frac{dI_S}{dt} + \frac{dR}{dt}$$

In absence of mortality rate due to SARS-COV-2infection,

$$\frac{dN}{dt} \leq \Pi - \mu N \quad (1.18)$$

Solving the differential equation yield,

$$\frac{\Pi}{\mu} - N \geq \frac{(\Pi - \mu N_0)}{\mu} e^{-\mu t} \quad (1.19)$$

As $t \rightarrow \infty$, the population size $N \rightarrow \frac{\Pi}{\mu}$

This implies that:

$$\lim_{t \rightarrow \infty} N(t) \leq \frac{\Pi}{\mu} \quad (1.20)$$

And since the population must be greater than zero,

$$0 < N(t) \quad (1.21)$$

The feasible set of solutions of the system equations enters and is confined into the region Ω for all the future time t. This proves the positivity and boundedness of the solution Therefore the model is well posed. The dynamics of the model can be studied in Ω

DESEASE FREE EQUILIBRIUM POINT

This is the point where there is no disease in the population. At this point, $E = I = I_H = I_S = R = 0$ and $S = \frac{\Pi}{\mu}$. From the differential equations 1 to 6, DFE is represented by $E^0 =$

$$\{S^0, E^0, I^0, I_H^0, I_S^0, R^0\} = \left\{ \frac{\Pi}{\mu}, 0, 0, 0, 0, 0 \right\} \quad (1.22)$$

BASIC REPRODUCTION NUMBER

We use next generation matrix method to compute the basic reproduction number denoted by R_0 as described by O. Diekmann, J. Heesterbeek, and J.A. Metz (1990). Matrix \mathbf{F} denoting the new infection while \mathbf{V} denoting the transfer of infection between compartments.

$$\mathbf{F} = \begin{pmatrix} \lambda S \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} = \begin{pmatrix} (1 - A) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S) S \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (1.23) \text{ And}$$

$$V = \begin{pmatrix} p_1 E \\ -\gamma E + p_2 I \\ -\sigma_1 I - \rho E + p_3 I_S \\ -\sigma_2 I + p_4 I_S - \zeta I_H \end{pmatrix} \quad (1.24)$$

Where $p_1 = (\mu + \gamma + \epsilon)$, $p_2 = (\mu + \delta_1 + \delta_2)$, $p_3 = (\mu + \delta_3 + \zeta + \tau_1)$ and
 $p_4 = (\tau_2 + \mu + \delta_2)$, $p_5 = (\theta + \mu)$

The Jacobian F and V are computed to yield

$$F = \begin{pmatrix} \beta(1-\Lambda) & \beta\eta_1(1-\Lambda) & \beta\eta_2(1-\Lambda) & \beta\eta_3(1-\Lambda) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad (1.25)$$

$$V = \begin{pmatrix} p_1 & 0 & 0 & 0 \\ -\gamma & p_2 & 0 & 0 \\ -\rho & -\sigma_1 & p_3 & 0 \\ 0 & 0 & -u & p_4 \end{pmatrix} \quad (1.26)$$

$$V^{-1} = \begin{pmatrix} \frac{1}{p_1} & 0 & 0 & 0 \\ \frac{1}{p_1 p_2} & \frac{1}{p_2} & 0 & 0 \\ \frac{\rho p_2 p_4 + p_4 \sigma_1}{p_1 p_2 p_3 p_4} & \frac{\sigma_1}{p_2 p_3} & \frac{1}{p_3} & 0 \\ \frac{u \rho p_2 + u \sigma_1}{p_1 p_2 p_3 p_4} & \frac{u \sigma_1}{p_2 p_3 p_4} & \frac{u}{p_3 p_4} & \frac{1}{p_4} \end{pmatrix} \quad (1.27)$$

The largest Eigenvalue of $F \cdot V^{-1}$ is the basic Reproduction number which is given by

$R_0 =$

$$\frac{\beta}{p_1} - \frac{\beta\Lambda}{p_1} + \frac{\beta\eta_1}{p_1 p_2} - \frac{\beta\eta_1 \Lambda}{p_1 p_2} + \frac{\beta\eta_2 \rho}{p_1 p_3} - \frac{\beta\eta_2 \Lambda \rho}{p_1 p_3} + \frac{u\beta\eta_3 \rho}{p_1 p_3 p_4} - \frac{u\beta\eta_3 \Lambda \rho}{p_1 p_3 p_4} + \frac{\beta\eta_2 \sigma_1}{p_1 p_2 p_3} - \frac{\beta\eta_2 \Lambda \sigma_1}{p_1 p_2 p_3} + \frac{u\beta\eta_3 \sigma_1}{p_1 p_2 p_3 p_4} - \frac{u\beta\eta_3 \Lambda \sigma_1}{p_1 p_2 p_3 p_4} \quad (1.28)$$

ENDERMIC EQUILIBRIUM POINT (EEP)

Endemic equilibrium exists whenever $R_0 > 1$ and it is asymptotically stable, Heesterbeek (1991) and Watmough (2002). To determine existence EEP, the model differential equations 1 to 6 are equated to zero and solved in terms of force of infection at steady state λ^* where $\lambda^* = (1-\Lambda)\beta(E + \eta_1 I + \eta_2 I_H + \eta_3 I_S)$ to yield:

Taking $p_1 = (\mu + \gamma + \rho)$, $p_2 = (\mu + \delta_1 + \delta_2)$, $p_3 = (\mu + \delta_3 + \zeta + \tau_1)$,
 $p_4 = (\tau_2 + \mu + \delta_2)$, $p_5 = (\theta + \mu)$

$$\text{and } \Lambda = (\alpha_1 + \alpha_1 + \alpha_1 + \alpha_1) \quad (1.29)$$

$$0 = (1 - \Lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S) S - p_1 E \quad (1.30)$$

$$0 = \gamma E - p_2 I \quad (1.31)$$

$$0 = \sigma_1 I + \rho E - p_3 I_H \quad (1.32)$$

$$0 = \sigma_2 I + \Upsilon I_H - p_4 I_S \quad (1.33)$$

$$0 = \Upsilon_1 I_H + \Upsilon_2 I_I - p_5 R \quad (1.34)$$

If we consider the infectious classes E, I, I_H and I_S to be positive,

From equations 30, 31, 32, 33, we have

$$E < \frac{(1 - \Lambda) \beta (E + \eta_1 I + \eta_2 I_H + \eta_3 I_S)}{p_1} \quad (1.35)$$

$$I < \frac{\gamma E}{p_2} \quad (1.36)$$

$$I_H < \frac{\sigma_1 I + \rho E}{p_3} \quad (1.37)$$

$$I_S < \frac{\sigma_2 I + \Upsilon I_H}{p_4} \quad (1.38)$$

Substituting equations 36 into 37 and 38 we get

$$I_H < \frac{\sigma_1 \frac{\gamma E}{p_2} + \rho E}{p_3} \quad (1.39)$$

$$I_S < \frac{\sigma_2 \frac{\gamma E}{p_2} + \Upsilon I_H}{p_4} \quad (1.40)$$

Substituting 39 into 40 yields

$$I_S < \left(\sigma_2 \frac{\gamma E}{p_2} + \Upsilon \left(\frac{\sigma_1 \gamma E}{p_3} + \rho E \right) \right) \div p_4 \quad (1.41)$$

Substituting equations 36, 39 and 41 into 35 yields

$$E < \left\{ (1 - \Lambda) \beta \left(E + \eta_1 \frac{\gamma E}{p_2} + \eta_2 \frac{\sigma_1 \gamma E}{p_3} + \rho E + \left(\eta_3 \sigma_2 \frac{\gamma E}{p_2} + \Upsilon \left(\frac{\sigma_1 \gamma E}{p_3} + \rho E \right) \right) \right) \right\} \div p_1 \quad (1.42)$$

On simplifying equation 42 and factoring out E, we get

$$E < \left\{ \frac{\beta}{p_1} - \frac{\beta \Lambda}{p_1} + \frac{\beta \eta_1}{p_1 p_2} - \frac{\beta \eta_1 \Lambda}{p_1 p_2} + \frac{\beta \eta_2 \rho}{p_1 p_3} - \frac{\beta \eta_2 \Lambda \rho}{p_1 p_3} + \frac{\eta_3 \beta \eta_3 \rho}{p_1 p_3 p_4} - \frac{\eta_3 \beta \eta_3 \Lambda \rho}{p_1 p_3 p_4} + \frac{\beta \eta_2 \sigma_1}{p_1 p_2 p_3} - \frac{\beta \eta_2 \Lambda \sigma_1}{p_1 p_2 p_3} + \frac{\eta_3 \beta \eta_3 \sigma_1}{p_1 p_2 p_3 p_4} - \frac{\eta_3 \beta \eta_3 \Lambda \sigma_1}{p_1 p_2 p_3 p_4} \right\} E \quad (1.43)$$

Dividing both sides by E yields

$$1 < \frac{\beta}{p_1} - \frac{\beta\Lambda}{p_1} + \frac{\beta\eta_1}{p_1 p_2} - \frac{\beta\eta_1\Lambda}{p_1 p_2} + \frac{\beta\eta_2 \rho}{p_1 p_3} - \frac{\beta\eta_2\Lambda\rho}{p_1 p_3} + \frac{u\beta\eta_3 \rho}{p_1 p_3 p_4} - \frac{u\beta\eta_3\Lambda\rho}{p_1 p_3 p_4} + \frac{\beta\eta_2 \sigma_1}{p_1 p_2 p_3} - \frac{\beta\eta_2\Lambda \sigma_1}{p_1 p_2 p_3} + \frac{u\beta\eta_3 \sigma_1}{p_1 p_2 p_3 p_4} - \frac{u\beta\eta_3\Lambda \sigma_1}{p_1 p_2 p_3 p_4} \quad (1.44)$$

Therefore from equation (44), $R_0 > 1$

This shows that the endemic exist and is positive.

LOCAL STABILITY OF DISEASE FREE EQILIBRIM (DFE)

Local stability of the model is investigated using the theorem by [10]

Theorem: The DFE (E^0) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$

Proof. To prove the theorem, we obtain the Jacobian matrix of the system of equations 1 to 6 as to get:

$$J_E = \begin{pmatrix} -\mu & (1-\Lambda)\beta & (1-\Lambda)\beta\eta_1 & (1-\Lambda)\beta\eta_2 & (1-\Lambda)\beta\eta_3 & \theta \\ 0 & (1-\Lambda)\beta - p_1 & (1-\Lambda)\beta\eta_1 & (1-\Lambda)\beta\eta_2 & (1-\Lambda)\beta\eta_3 & 0 \\ 0 & \gamma & -p_2 & 0 & 0 & 0 \\ 0 & \rho & \sigma_1 & -p_3 & 0 & 0 \\ 0 & 0 & \sigma_2 & \Upsilon & -p_4 & 0 \\ 0 & 0 & 0 & \Upsilon_1 & \Upsilon_2 & -p_5 \end{pmatrix} \quad (1.45)$$

One of the eigenvalues is $-\mu$.

The other matrix becomes:

$$\begin{pmatrix} (1-\Lambda)\beta - p_1 & (1-\Lambda)\beta\eta_1 & (1-\Lambda)\beta\eta_2 & (1-\Lambda)\beta\eta_3 & 0 \\ \gamma & -p_2 & 0 & 0 & 0 \\ \rho & \sigma_1 & -p_3 & 0 & 0 \\ 0 & \sigma_2 & \Upsilon & -p_4 & 0 \\ 0 & 0 & \tau_1 & \tau_2 & -p_5 \end{pmatrix} \quad (1.46)$$

The characteristic equation was obtained from (46), based on parameter κ and the resulting polynomial is;

$$(-\kappa - p_5) ((-\kappa - p_4) ((-\kappa - p_3)(-\beta\kappa + \kappa^2 + \beta\kappa\Lambda + \kappa p_1 - \beta p_2 + \kappa p_2 + \beta\Lambda p_2 + p_1 p_2 - \beta\gamma\eta_1 + \beta\gamma\Lambda\eta_1) + \beta(1-\Lambda)(\rho\kappa + \gamma\sigma_1 + \rho p_2)\eta_2) - \beta(1-\Lambda)(\Upsilon\rho\kappa + \Upsilon\gamma\sigma_1 + \gamma\kappa\sigma_2 + \Upsilon\rho p_2 + \gamma\sigma_2 p_3)\eta_3) = 0 \quad (1.47)$$

Equation 51 can be written as

$$(-\kappa - p_5) ((-\kappa - p_4) ((-\kappa - p_3) (K_1\kappa^2 + K_2\kappa + K_3) = 0 \quad (1.48)$$

Where $K_1 = 1$

$$K_2 = (-\beta + \beta\Lambda + p_1 + p_2 + \beta(1-\Lambda)\rho - \beta(1-\Lambda)(\Upsilon\rho + \gamma\sigma_2)\eta_3$$

$$K_3 = (-\beta p_2 + \beta\Lambda p_2 + p_1 p_2 - \beta\gamma\eta_1 + \beta\gamma\Lambda\eta_1) + \beta(1-\Lambda)(\gamma\sigma_1 + \rho p_2)\eta_2 - \beta(1-\Lambda)(\Upsilon\gamma\sigma_1 + \Upsilon\rho p_2 + \gamma\sigma_2 p_3)\eta_3$$

$$K_3 = \frac{p_1 p_2 p_3 p_4 (-p_2 + \lambda p_2 + p_1 p_2 - \gamma \eta_1 + \gamma \lambda \eta_1 + (1-\lambda) \eta_2 (\rho p_2 + \gamma \sigma_1) - (1-\lambda) \eta_3 (\Upsilon \rho p_2 + \Upsilon \gamma \sigma_1 + \gamma p_3 \sigma_2))}{(1-\lambda) (\gamma p_3 p_4 \eta_1 + p_2 (p_4 (p_3 + \rho \eta_2) + \rho v \eta_3) + \gamma (p_4 \eta_2 + v \eta_3) \sigma_1)} R_0 \quad (1.49)$$

From the expression (49), we apply Routh - Hurwitz Criteria where the solution of \square should have negative real root iff

$K_1 > 0$, $K_2 > 0$, $K_3 > 0$ and $K_1 K_2 > K_3$. In our case K_1 is positive because it is a positive constant. $K_2 > 0$ because only $-\beta$ is negative and $(\beta \lambda + p_1 + p_2 + \beta(1-\lambda)\rho) > \beta(1-\lambda)(\Upsilon \rho + \gamma \sigma_2)$. The product of $K_1 K_2 > 0$ since both are positive and if we choose $R_0 < 1$, then $K_1 K_2 > K_3$. For K_3 to be positive, $1-R_0$ must be positive. This means that $R_0 < 1$ hence DFE point E^0 is locally asymptotically stable.

GLOBAL STABILITY OF DFE

Global stability of the model is investigated using the theorem by [11]

Theorem: The fixed point $\tilde{U}_0 = (X^*, 0) = (\frac{\Pi}{\mu}, 0, 0, 0, 0, 0)$ is globally asymptotically stable, if $R_0 < 1$ is locally asymptotically stable and assumptions (H1) and (H2) are satisfied.

PROOF: The equations (1-6) are written in $\frac{dZ}{dt} = \hat{G}(X, Z)$, $\frac{dX}{dt} = F(X, Z)$, where X represent the disease free class R while $Z = (S, E, I, I_H, I_S)$ represents the disease infected classes. $G(X, 0) = 0$. $\tilde{U}_0 = (X^*, 0) = (\frac{\Pi}{\mu}, 0, 0, 0, 0, 0)$ denotes the disease free equilibrium (DFE) point of the model.

The conditions (H1) and (H2) are:

(H1) for $\frac{dX}{dt} = F(X, 0)$, X^0 is globally asymptotically stable. (H2), $\hat{G}(X, Z) = AZ - G(X, Z)$, $G(X, Z) \geq 0$ for $(X, Z) \in R_+^6$ where $A = D_Z G(X, 0)$ is an M-matrix (the off diagonal element of A are nonnegative) and R_+^6 is the region where the model makes biological sense.

In our case, $F(X, 0) = (\Pi - \mu R)$, $X = (R)$ and $Z = (S, E, I, I_H, I_S)$

$$A = \begin{pmatrix} -\mu & (1-\lambda)\beta & (1-\lambda)\beta\eta_1 & (1-\lambda)\beta\eta_2 & (1-\lambda)\beta\eta_3 \\ 0 & (1-\lambda)\beta - p_1 & (1-\lambda)\beta\eta_1 & (1-\lambda)\beta\eta_2 & (1-\lambda)\beta\eta_3 \\ 0 & \gamma & -p_2 & 0 & 0 \\ 0 & \rho & \sigma_1 & -p_3 & 0 \\ 0 & 0 & \sigma_2 & \Upsilon & -p_4 \end{pmatrix} \quad (1.50)$$

$$AZ = \begin{pmatrix} -\mu S + (1-\lambda)\beta E + (1-\lambda)\beta\eta_1 I + (1-\lambda)\beta\eta_2 I_H + (1-\lambda)\beta\eta_3 I_S \\ ((1-\lambda)\beta - p_1) E + (1-\lambda)\beta\eta_1 I + (1-\lambda)\beta\eta_2 I_H + (1-\lambda)\beta\eta_3 I_S \\ \gamma E - p_2 I \\ \rho E + \sigma_1 I - p_3 I_H \\ \sigma_2 I + \Upsilon I_H - p_4 I_S \end{pmatrix} \quad (1.51)$$

$$G(X,Z) = \begin{pmatrix} -\mu S + (1-\Lambda)\beta E + (1-\Lambda)\beta\eta_1 I + (1-\Lambda)\beta\eta_2 I_H + (1-\Lambda)\beta\eta_3 I_S \\ ((1-\Lambda)\beta - p_1)E + (1-\Lambda)\beta\eta_1 I + (1-\Lambda)\beta\eta_2 I_H + (1-\Lambda)\beta\eta_3 I_S \\ \gamma E - p_2 I \\ \rho E + \sigma_1 I - p_3 I_H \\ \sigma_2 I + \Upsilon I_H - p_4 I_S \end{pmatrix} \quad (1.52)$$

$$\hat{G}(X,Z) = AZ - G(X,Z) = 0 \quad (1.53)$$

Since $X \leq N$, $\frac{S}{N} \leq 1$, then $\hat{G}(X,Z) \geq 0$. This implies that the second condition of H2 is satisfied. Thus, $(X^* = (\frac{\Pi}{\mu}, 0, 0, 0, 0,))$ is globally asymptotically stable equilibrium of

$$\frac{dX}{dt} = F(X,0) \text{ when } R_0 < 1$$

GLOBAL STABILITY OF ENDEMIC EQUILIBRIUM

For the system of equations (1-6) we propose the following Lyapunov function $K(S, E, I, I_H, I_S, R)$

$$= S - S^* \ln \frac{S}{S^*} + y_1 (E - E^* - E^* \ln \frac{E}{E^*}) + y_2 (I - I^* - I^* \ln \frac{I}{I^*}) + y_3 (I_H - I_H^* - I_H^* \ln \frac{I_H}{I_H^*}) + y_4 (I_S - I_S^* - I_S^* \ln \frac{I_S}{I_S^*}) + y_5 (R - R^* - R^* \ln \frac{R}{R^*})$$

Where: y_1, y_2, y_3, y_4 and y_5 are constants to be determined.

The Lyapunov function $K(S, E, I, I_H, I_S, R)$ satisfy the conditions

$$K(S^*, E^*, I^*, I_H^*, I_S^*, R^*) = 0 \quad \text{and}$$

$$K(S, E, I, I_H, I_S, R) > 0 \text{ Hence its positive definite for}$$

$$\frac{dK(S, E, I, I_H, I_S, R)}{dt} \quad (1.54)$$

$$\text{To be negative definite, it must satisfy } \frac{dK(S^*, E^*, I^*, I_H^*, I_S^*, R^*)}{dt} = 0 \quad (1.55)$$

$$\text{And } \frac{dK(S^*, E^*, I^*, I_H^*, I_S^*, R^*)}{dt} < 0 \quad (1.56)$$

The EEP $(E^*) = (S^*, E^*, I^*, I_H^*, I_S^*, R^*)$ for the system satisfy

$$\Pi = (\mu + \lambda)S^{**} - \theta R^{**}$$

$$\lambda S^{**} = p_1 E^{**}$$

$$\gamma E^{**} = p_2 I^{**}$$

$$p_3 I_H^{**} = \sigma_1 I^{**} + \rho E^{**}$$

$$p_4 I_S^{**} = \sigma_2 I^{**} + \Upsilon I_H^{**}$$

$$p_5 R^{**} = \tau_1 I_H^{**} + \tau_2 I_S^{**}$$

$$dk(S, E, I, I_H, I_S, R) = (1 - \frac{S^{**}}{S}) \frac{dS}{dt} + y_1 (1 - \frac{E^{**}}{E}) \frac{dE}{dt} + y_2 (1 - \frac{I^{**}}{I}) \frac{dI}{dt} + y_3 (1 - \frac{I_H^{**}}{I_H}) \frac{dI_H}{dt} + y_4 (1 - \frac{I_S^{**}}{I_S}) \frac{dI_S}{dt} + y_5 \frac{dR}{dt} \quad (1.57)$$

Substituting for $\frac{dS}{dt}, \frac{dE}{dt}, \frac{dI}{dt}, \frac{dI_H}{dt}, \frac{dI_S}{dt}$ and $\frac{dR}{dt}$ into δ_2 to get

$$(1 - \frac{S^{**}}{S}) \Pi + \theta R - (\mu + \lambda) S + y_1 (1 - \frac{E^{**}}{E}) (\lambda S - p_1 E) + y_2 (1 - \frac{I^{**}}{I}) (\gamma E - p_2 I) + y_3 (1 - \frac{I_H^{**}}{I_H}) (\delta_1 I + \rho E - p_3 I_H) + y_4 (1 - \frac{I_S^{**}}{I_S}) (\delta_2 I - p_4 I_S) + y_5 (1 - \frac{R^{**}}{R}) (\tau_1 I_H + \tau_2 I_S - p_5 R) \quad (58)$$

Let $(1 - \Lambda) = M$, The force of infection $\lambda = M\beta(E + \eta_1 I + \eta_2 I_H + \eta_3 I_S) = M\beta E + M\beta\eta_1 I + M\beta\eta_2 I_H + M\beta\eta_3 I_S$

And when we let

$$E = -M\beta E + y_1 M\beta E$$

$$I = -M\beta\eta_1 I + y_2 M\beta\eta_1 I$$

$$I_H = -M\beta\eta_2 I_H + y_3 M\beta\eta_2 I_H \quad (1.59)$$

$$I_S = -M\beta\eta_3 I_S + y_4 M\beta\eta_3 I_S$$

$$dK(S, E, I, I_H, I_S, R) = \mu S^{**} + \lambda S^{**} + \theta R^{**} - (-M\beta E + y_1 M\beta E) - (-M\beta\eta_1 I + y_2 M\beta\eta_1 I) - (M\beta\eta_2 I_H + y_3 M\beta\eta_2 I_H) - (-M\beta\eta_3 I_S + y_4 M\beta\eta_3 I_S) + \theta R - \mu S + \frac{S^{**}}{S} \mu S^{**} - \frac{S^{**}}{S} \lambda S^{**} - \frac{S^{**}}{S} \theta R^{**} + \frac{S^{**}}{S} \theta R + \frac{S^{**}}{S} \mu + \frac{S^{**}}{S} \lambda S + y_1 \lambda S - y_1 p_1 E - y_1 \frac{E^{**}}{E} \lambda S + y_1 \frac{E^{**}}{E} p_1 E + y_2 \gamma E - y_2 p_2 I - y_2 \frac{I^{**}}{I} \gamma E + y_2 \frac{I^{**}}{I} p_2 I + y_3 \sigma_2 I + y_3 \rho E - y_3 p_3 I_H - y_3 \frac{I_H^{**}}{I_H} \sigma_1 I - y_3 \frac{I_H^{**}}{I_H} \rho E + y_3 \frac{I_H^{**}}{I_H} p_3 I_H + y_4 \sigma_2 I - y_4 p_4 I_S - y_4 \frac{I_S^{**}}{I_S} \sigma_2 I + y_4 \frac{I_S^{**}}{I_S} p_4 I_S + y_5 \tau_1 I_H + y_5 \tau_2 I_S - y_5 p_5 R - y_5 \frac{R^{**}}{R} \tau_1 I_H + y_5 \frac{R^{**}}{R} \tau_2 I_S - y_5 \frac{R^{**}}{R} p_5 R \quad (1.60)$$

From (60) we determine the values of y_1, y_2, y_3, y_4 and y_5 and separate positive from negative parts to yield;

$$P = \mu S^{**} + \lambda S^{**} + \theta R^{**} + \frac{S^{**}}{S} \theta R + \frac{S^{**}}{S} \mu + \frac{S^{**}}{S} \lambda S + y_1 \lambda S + y_1 \frac{E^{**}}{E} p_1 E + y_2 \gamma E + y_2 \frac{I^{**}}{I} p_2 I + y_3 \sigma_1 I + y_3 \rho E + y_3 \frac{I_H^{**}}{I_H} p_3 I_H + y_4 \sigma_2 I + y_4 \frac{I_S^{**}}{I_S} p_4 I_S + y_5 \tau_1 I_H + y_5 \tau_2 I_S + y_5 \frac{R^{**}}{R} \tau_2 I_S \quad (1.61)$$

$$\begin{aligned}
Q = & -\theta R - \mu S - \lambda S - \frac{S^{**}}{S} \mu S^{**} - \frac{S^{**}}{S} \lambda S^{**} - \frac{S^{**}}{S} \theta R^{**} - y_1 p_1 E - y_1 \frac{E^{**}}{E} \lambda S - y_2 p_2 I - \\
& y_2 \frac{I^{**}}{I} \gamma E - y_3 p_3 I_H - y_3 \frac{I_H^{**}}{I_H} \sigma_1 I - y_3 \frac{I_H^{**}}{I_H} \rho E - y_4 p_4 I_S - y_4 \frac{I_S^{**}}{I_S} \sigma_2 I - y_5 p_5 R - \\
& y_5 \frac{R^{**}}{R} \tau_1 I_H - y_5 \frac{R^{**}}{R} p_5 R
\end{aligned}
\tag{1.62}$$

The $\frac{dK}{dt} = 0$ holds only when $(S = S^*, E = E^*, I = I^*, I_H = I_H^*, I_S = I_S^*, R = R^*)$ so that maximum compact invariant set in $(S; E; I) \in \mathbb{J} : \frac{dV}{dt} = 0$ is a singleton E^{**} using Lasalle's invariant principle $\frac{dL(SIAR)}{dt} < 0$ if and only if $P > Q$ (Mukandavire et al 2010). This results shows that SARS-COV-2 would persist in the population whenever $P > 0$ irrespective of the initial conditions. And if $Q > P$, the disease will die out irrespective of initial conditions.

BIFURCATION ANALYSIS OF THE MODEL

The Centre Manifold Theorem by [10] is used to investigate the nature of the bifurcation of the model. Let $S = x_1, E = x_2, I = x_3, I_H = x_4, I_S = x_5, R = x_6$.

$$\text{Then } N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6. \tag{1.63}$$

The model can be written as

$$\frac{dN}{dt} = F(x) \text{ with } F = (f_1, f_2, f_3, f_4, f_5, f_6) \text{ and}$$

$$\frac{dx_1}{dt} = f_1 = \Pi + \theta x_6 - (\mu + (1 - \Lambda) \beta (x_2 + \eta_1 x_3 + \eta_2 x_4 + \eta_3 x_5)) x_1$$

$$\frac{dx_2}{dt} = f_2 = (1 - \Lambda) \beta (x_2 + \eta_1 x_3 + \eta_2 x_4 + \eta_3 x_5) x_1 - p_1 x_2$$

$$\frac{dx_3}{dt} = f_3 = \gamma x_2 - p_2 x_3 \tag{1.64}$$

$$\frac{dx_4}{dt} = f_4 = \sigma_1 x_3 + \rho x_2 - p_3 x_4$$

$$\frac{dx_5}{dt} = f_5 = \sigma_2 x_3 - p_4 x_5$$

$$\frac{dx_6}{dt} = f_6 = \tau_1 x_4 + \tau_2 x_5 - p_5 x_6$$

By choosing $\beta = \beta^*$ as the bifurcation parameter and investigating the case when the basic reproduction number $R_0 = 1$, it yields

$$R_0 = \frac{(1-\Lambda)(\mu\beta\eta_3\rho p_2 + \beta\eta_2\rho p_2 p_4 + \beta\eta_1 p_3 p_4 + \beta p_2 p_3 p_4 + \mu\beta\eta_3 \sigma_1 + \beta\eta_2 p_4 \sigma_1)}{p_1 p_2 p_3 p_4} = 1$$

$$\beta = \frac{p_1 p_2 p_3 p_4}{(1-\Lambda)(\mu\eta_3\rho p_2 + \eta_2\rho p_2 p_4 + \eta_1 p_3 p_4 + p_2 p_3 p_4 + \mu\eta_3 \sigma_1 + \eta_2 p_4 \sigma_1)} \tag{1.65}$$

To investigate the stability of the model we use the following model of [11].

We consider the general systems of ordinary differential equation according to [10] with parameter β

$$\frac{dx}{dt} = f(x, \beta), \mathbb{R}^n \times \mathbb{R} \rightarrow \mathbb{R} \text{ and } f \in C^2(\mathbb{R}^n \times \mathbb{R}),$$

Where 0 is the equilibrium point of the system (that is, $f(x, \beta) \equiv 0$ for all β) and assume that;

A1: $A = D_x f(0,0,0,0) = \left(\frac{df^i}{dx^j}\right)(0,0,0,0)$ is the linearization matrix of the system of equations (1)to (6) around the equilibrium 0 with β evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A contain negative real parts:

A2: Matrix a has a nonnegative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue. Let f_k be the k^{th} component of f and

$$a = \sum_{k,i,j=1}^n v_k w_i w_j \frac{d^2 f_k}{dx_i dx_j}(0,0,0,0) \quad (1.66)$$

$$b = \sum_{k,i=1}^n (0,0,0,0) v_k w_i \frac{d^2 f_k}{dx_i d\beta}(0,0,0,0) \quad (1.67)$$

The local dynamics of the system around 0 is totally determined by the signs of a and b obtained above.

Eigenvectors of $J_E = J_\beta$. The Jacobian of the model at β denoted by J_β has a right eigenvector denoted by $w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ given by:

$$J_\beta = \begin{pmatrix} -\mu & (1-\Lambda)\beta & (1-\Lambda)\beta\eta_1 & (1-\Lambda)\beta\eta_2 & (1-\Lambda)\beta\eta_3 & \theta \\ 0 & (1-\Lambda)\beta - p_1 & (1-\Lambda)\beta\eta_1 & (1-\Lambda)\beta\eta_2 & (1-\Lambda)\beta\eta_3 & 0 \\ 0 & \gamma & -p_2 & 0 & 0 & 0 \\ 0 & \rho & \sigma_1 & -p_3 & 0 & 0 \\ 0 & 0 & \sigma_2 & \Upsilon & -p_4 & 0 \\ 0 & 0 & 0 & \Upsilon_1 & \Upsilon_2 & -p_5 \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (1.68)$$

From the equations of 68, we get the nonnegative eigenvector w as shown below;

$$w_1 = \frac{(1-\Lambda)\beta w_2 + (1-\Lambda)\beta\eta_1 w_3 + (1-\Lambda)\beta\eta_2 w_4 + (1-\Lambda)\beta\eta_3 w_5 + \theta w_6}{\mu} > 0$$

$$w_2 = \frac{(1-\Lambda)\beta\eta_1 w_3 + (1-\Lambda)\beta\eta_2 w_4 + (1-\Lambda)\beta\eta_3 w_5}{(p_1 - (1-\Lambda)\beta)} > 0$$

$$w_3 = \frac{\gamma w_2}{p_2} > 0$$

$$w_4 = \frac{\rho w_2 + \sigma_2 w_3}{p_3} > 0$$

$$w_5 = \frac{\sigma_2 w_3 + \Upsilon w_4}{p_4} > 0$$

$$w_6 = \frac{\Upsilon_1 w_4 + \Upsilon_2 w_5}{p_5} > 0$$

The Jacobian matrix also has a left eigenvector denoted by v and is given by $v = (v_1, v_2, v_3, v_4, v_5, v_6)^T$

$$\begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 \\ (1-\Lambda)\beta & (1-\Lambda)\beta - p_1 & \gamma & \rho & 0 & 0 \\ (1-\Lambda)\beta\eta_1 & (1-\Lambda)\beta\eta_1 & -p_2 & \bar{\sigma}_1 & \bar{\sigma}_2 & 0 \\ (1-\Lambda)\beta\eta_2 & (1-\Lambda)\beta\eta_2 & 0 & -p_3 & \Upsilon & \Upsilon_1 \\ (1-\Lambda)\beta\eta_3 & (1-\Lambda)\beta\eta_3 & 0 & 0 & -p_4 & \Upsilon_2 \\ \theta & 0 & 0 & 0 & 0 & -p_5 \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \\ v_6 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (1.69)$$

Solution of equation (69) yields;

$$v_1 = 0$$

$$v_2 = -\frac{\gamma v_3 + \rho v_4}{(1-\Lambda)\beta - p_1} < 0$$

$$v_3 = \frac{(1-\Lambda)\beta\eta_1 v_2 + \bar{\sigma}_1 v_4 + \bar{\sigma}_2 v_5}{p_2} > 0$$

$$v_4 = \frac{(1-\Lambda)\beta\eta_2 v_2 + \Upsilon v_5}{p_3} > 0$$

$$v_5 = \frac{(1-\Lambda)\beta\eta_3 v_2}{p_4} > 0$$

$$v_6 = 0$$

The signs of \mathbf{a} and \mathbf{b} is found as follows:

$$v_k w_i w_j \frac{d^2 f_k}{dx_i dx_j}$$

for $k=2$; $i, j=1,2,4$,

$$(1.70)$$

The summation of the set of equations (70) gives the value of \mathbf{a} , which is given by;

$$\begin{aligned} \mathbf{a} = & -(1-\Lambda)\beta \frac{p_4 v_5}{(1-\Lambda)\beta\eta_3} \left(\frac{(1-\Lambda)\beta\eta_1 w_3 + (1-\Lambda)\beta\eta_2 w_4 + (1-\Lambda)\beta\eta_3 w_5}{(p_1 - (1-\Lambda)\beta)} \right) \\ & \left\{ \left(\frac{(1-\Lambda)\beta w_2 + (1-\Lambda)\beta\eta_1 w_3 + (1-\Lambda)\beta\eta_2 w_4 + (1-\Lambda)\beta\eta_3 w_5 + \theta w_6}{\mu} \right) + \right. \\ & \left. \left(\frac{(1-\Lambda)\beta w_2 + (1-\Lambda)\beta\eta_1 w_3 + (1-\Lambda)\beta\eta_2 w_4 + (1-\Lambda)\beta\eta_3 w_5 + \theta w_6}{\mu} \right) + \left(\frac{\rho w_2 + \bar{\sigma}_2 w_3}{p_3} \right) + \left(\frac{\rho w_2 + \bar{\sigma}_2 w_3}{p_3} \right) \right\} < 0 \end{aligned} \quad (1.71)$$

Finding the value of **b** according to **Theorem 3 of [10]** we let $k = 2, i = 2, 3, 4,$

For $V_k w_i w_j \frac{d^2 f_k}{dx_i dx_j}$ to generate the set of equations which are added to get the expression of **b** which is:

b =

$$\begin{aligned} & \frac{(1-\Lambda)\beta\eta_1 v_2 + \sigma_1 v_4 + \sigma_2 v_5}{p_2} \left(\frac{\gamma w_2}{p_2} \right) (1-\Lambda)\eta_1 x_1 + \frac{(1-\Lambda)\beta\eta_1 v_2 + \sigma_1 v_4 + \sigma_2 v_5}{p_2} \left(\frac{\rho w_2 + \sigma_2 w_3}{p_3} \right) (1-\Lambda)\eta_2 x_1 + \\ & \frac{(1-\Lambda)\beta\eta_1 v_2 + \sigma_1 v_4 + \sigma_2 v_5}{p_2} \left(\frac{(1-\Lambda)\beta w_2 + (1-\Lambda)\beta\eta_1 w_3 + (1-\Lambda)\beta\eta_2 w_4 + (1-\Lambda)\beta\eta_3 w_5 + \theta w_6}{\mu} \right) (1-\Lambda) (x_2 + \\ & \eta_1 x_3 + \eta_2 x_4 + \eta_3 x_5) + \frac{(1-\Lambda)\beta\eta_1 v_2 + \sigma_1 v_4 + \sigma_2 v_5}{p_2} \left(\frac{\sigma_2 w_3 + \Omega w_4}{p_4} \right) (1-\Lambda)\eta_3 x_1 > 0. \end{aligned}$$

(1.72)

Therefore, from theorem 3 item *iv*, β changes from negative to positive. 0 changes its stability from stable to unstable. There is a corresponding change of stability from a negative unstable equilibrium to a positive and locally asymptotically stable equilibrium.

SENSITIVITY ANALYSIS OF THE MODEL

Normalized Sensitivity analysis of some parameter was done using normalized sensitivity by [13] to determine the one with greatest impact on the reproduction number. The values in table 2 are used to generate sensitivity indices by substituting them in the partial derivatives obtained from Normalized sensitivity analysis.

Table 1: SENSITIVITY ANALYSIS

parameter	description	sensitivity index
σ_1	Rate of isolation of infected people (symptomatic) into hospital	0.00429
σ_2	Rate of isolation of infected people (Asymptomatic) into the homecare	0.02431
τ_1	Rate of recovery of infected people isolated in hospital	-0.0115868
τ_2	Rate of recovery of infected people isolated in homecare	-0.0081

Θ	Rate of reinfection after recovery	0
Λ .	Percentage of people adhering to SARS-COV-2 containment measures	-2.4519042
Υ	Rate at which exposed individuals who get infected are traced.	-0.6636.

Numerical Simulations

Table 2: value of parameters and their source references

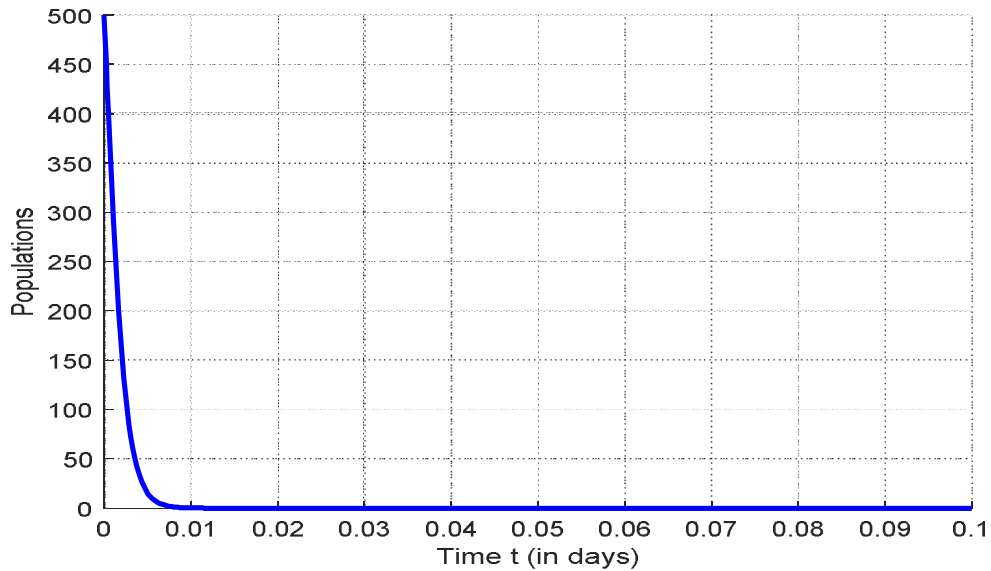
parameter	value	Reference
Π	0.026	[25]
μ	0.005	[25]
γ	0.75	[33]
Θ	0.067	[29]
τ_1	0.001	[31]
τ_2	0	[26]
τ_3	0.0328	[31]
σ_1	0.15	[42],
σ_2	0.85	[28]
ρ	0.2	Assumed
ζ	0.5	Assumed
τ_1	0.698	[26]

τ_2	1.0	[26]
β	0.00005	Estimated
η_1	0.019	[36]
η_2	0.056	[41]
η_3	0.075	Assumed
α_1	0.25	Assumed
α_2	0.25	Assumed
α_3	0.25	Assumed
α_4	0.25	Assumed

TABLE 3: INITIAL CONDITIONS IN EACH COMPARTMENT

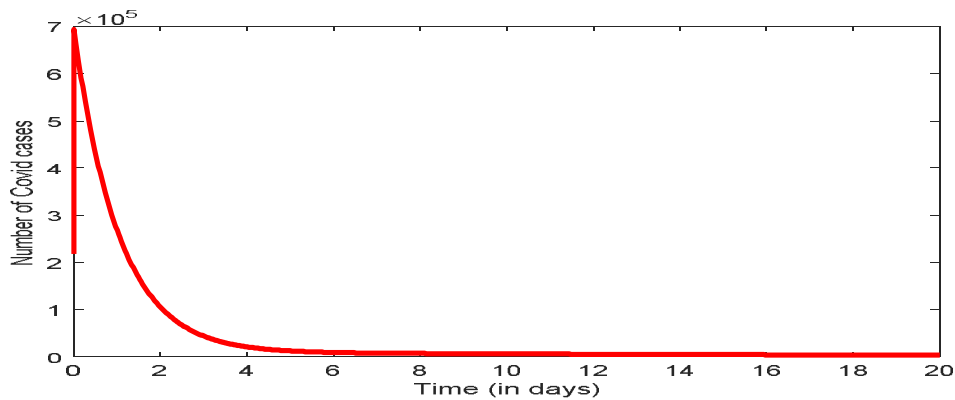
S	47,564,296	[25]
E	21,879,576	[23]
I	2,844	[24]
I_H	426	[44]
I_S	2,844	[44]
R	159	[24]

FIGURE 2: SUSCEPTIBLE POPULATION AGAINST TIME (IN DAYS)



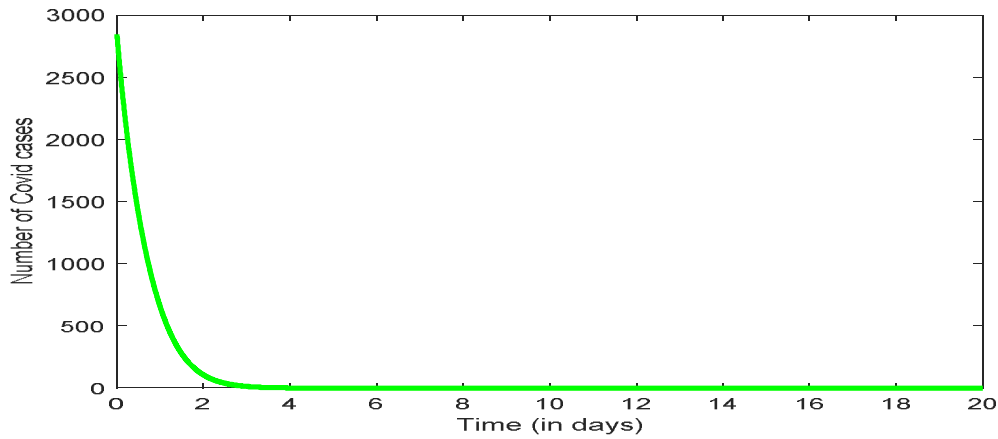
The susceptible population will decrease sharply and within a very short moment due to the effects of the containment measures. This is exhibited by the curve which has a negative gradient.

FIGURE 3: THE EXPOSED POPULATION AGAINST TIME (IN DAYS)



The rate of exposure will gradually decrease as shown by the negative curvature of the graph. As more people continue to adhere to these SARS-COV-2 containment measures, the disease will be suppressed and it will die off.

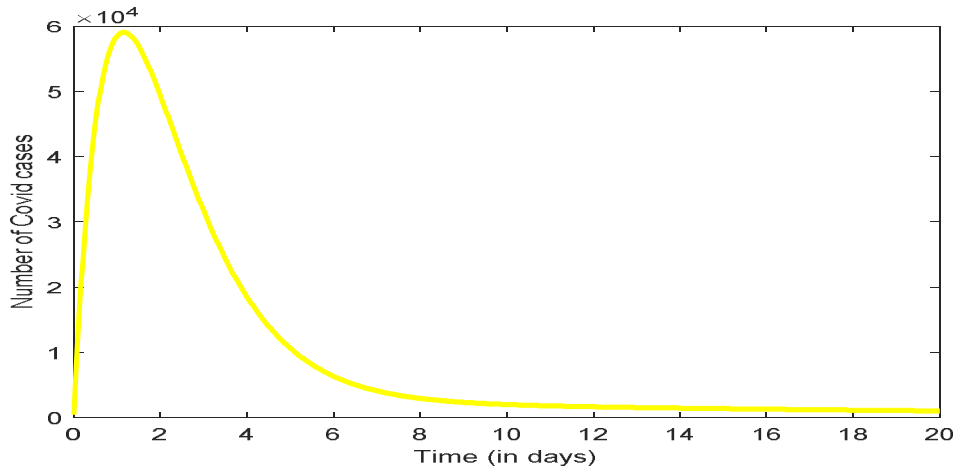
FIGURE 4: DAILY SARS-COV-2 INFECTIONS AGAINST TIME (IN DAYS)



The number of infected population decrease sharply due to adherence of the SARS-COV-2 containment measures among the population. This is exhibited by the negative curvature of the graph.

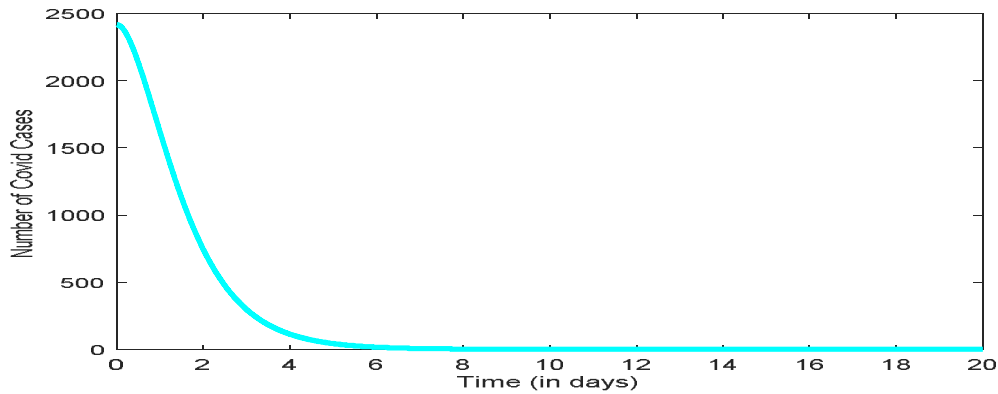
FIGURE 5: DAILY HOSPITALISED COVID 19 CASES AGAINST TIME (IN DAYS)

HOSPITAL TREATMENT



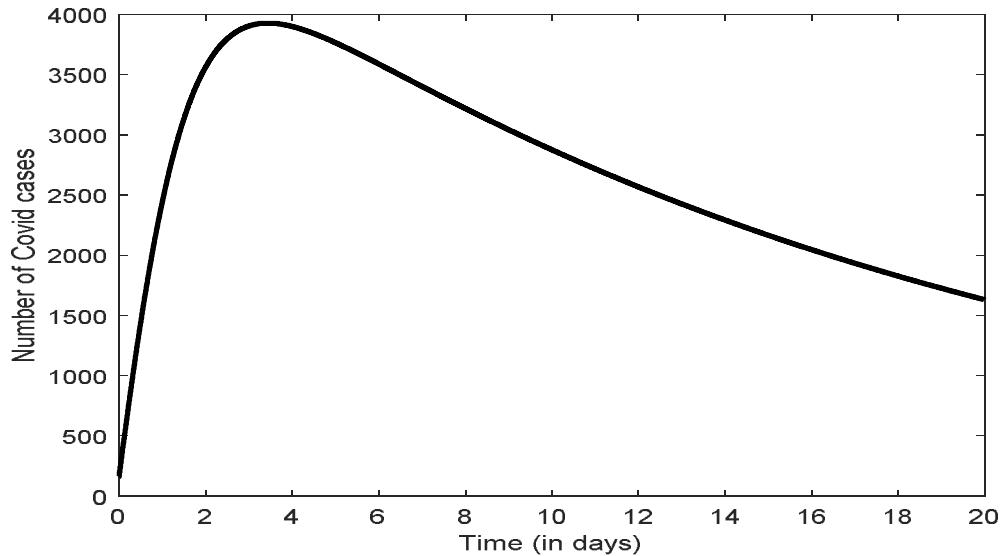
The change of the gradient of the curvature of the graph from positive to negative after the maxima shows that there would be increase in the number of people who will be taken to hospital for treatment since the disease is still persistence in the population but once the containment measures are adhered to by the people, the number of people taken to hospital will start decreasing since the transmission will be contained.

FIGURE 6: DAILY HOMECARE ISOLATION CASES AGAINST TIME (IN DAYS)



There is decrease in the number of homecare isolation with time. People without symptoms will not need to go for home isolation since they cannot transmit the disease if they are strictly following these measures meant to contain the pandemic.

FIGURE 7: DAILY RECOVERED SARS-COV-2 CASES AGAINST TIME (IN DAYS)



The graph shows that there is increase in the number of recovered patients since those who are in hospital treatment or home treatment will continue to recover and once the infection is suppressed, the number of the daily recoveries will also reduce consequently.

SIMULATION RESULTS SHOWING EFFECTS OF NON-CLINICAL STRATEGIES

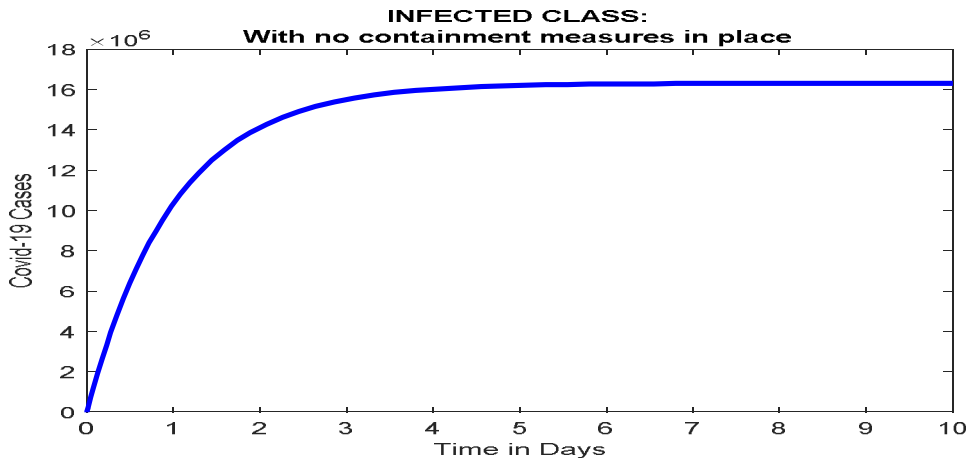


Figure 8: infected class with no containment measures

There is increase in reported infected individuals with time due to lack of containment measures.

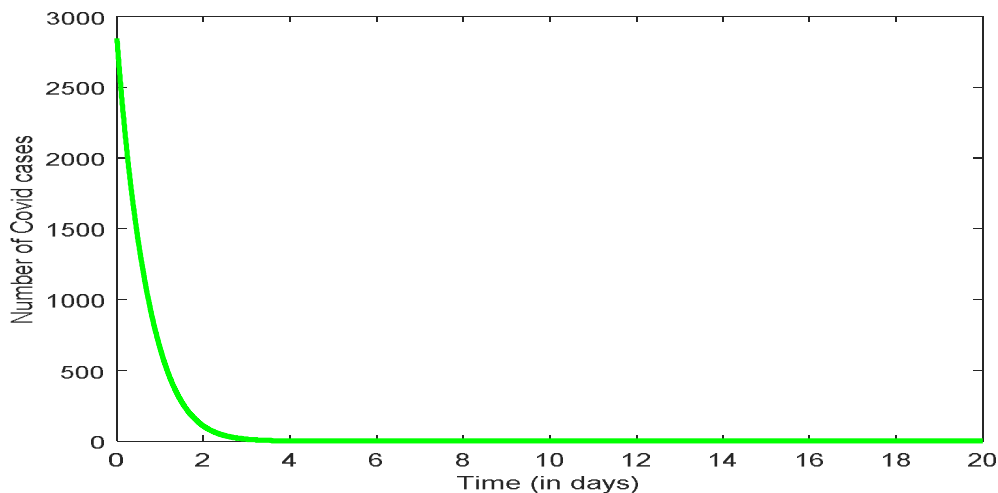


Figure 9: daily SARS-CoV-2 infections with containment measures in place

The number of infected population decrease sharply due to adherence of the SARS-COV-2 containment measures among the population. This is exhibited by the negative curvature of the graph. Exposure is due to the contact between people who are infected and those who are not. This contact is reduced when people start adhering to social distancing, facemask wearing, screening and hand washing.

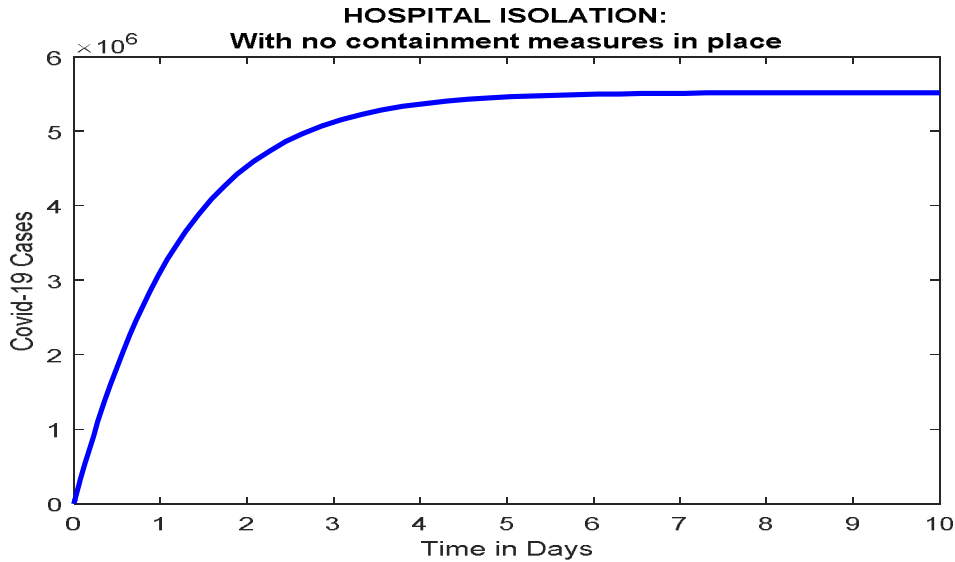


Figure 10: hospital isolation with no containment measures in place

Symptomatic SARS-COV-2 cases will continue piling up with time in the hospital. This is due to continuous increase in infection in the population.

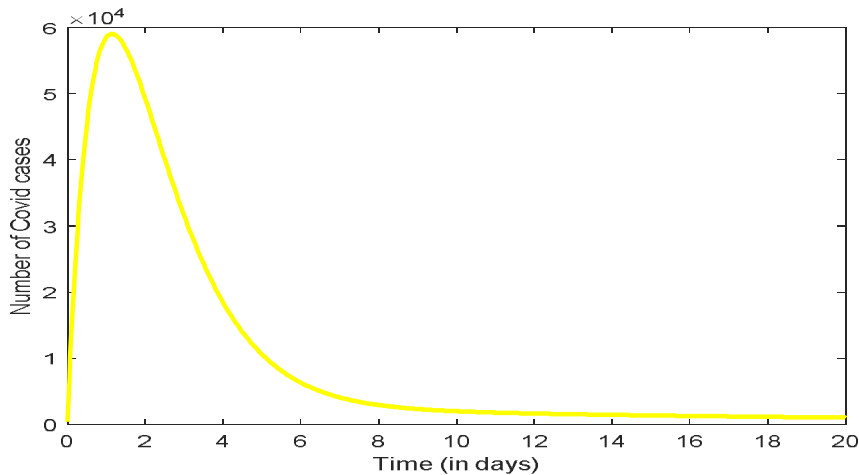


Figure 11: daily SARS-CoV-2 hospital isolation with containment measures in place

The change of the gradient of the curvature of the graph from positive to negative after the maxima shows that there would be increase in the number of people who will be taken to hospital for treatment since the disease is still persistence in the population but once the containment measures are adhered to by the people, the number of people taken to hospital will start decreasing since the transmission will be contained.

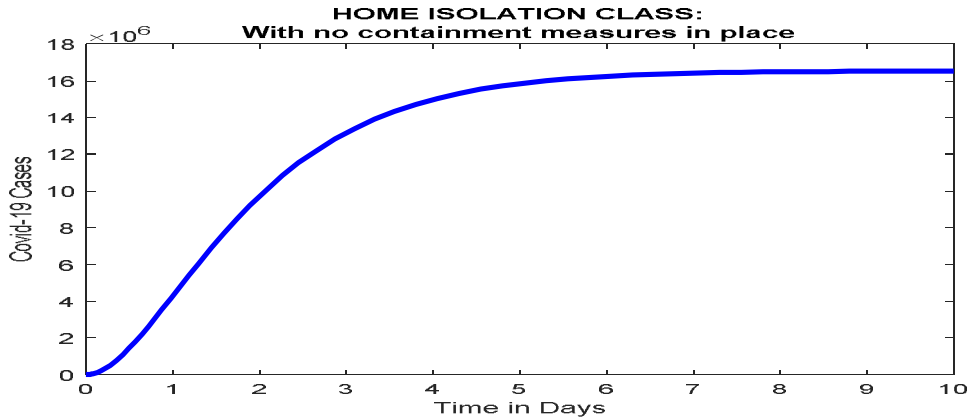


Figure 12: home isolation with no containment measures

There is increase in the number of SARS-COV-2 cases with time among the asymptomatic individuals. This is due to increasing number of infected people in the population.

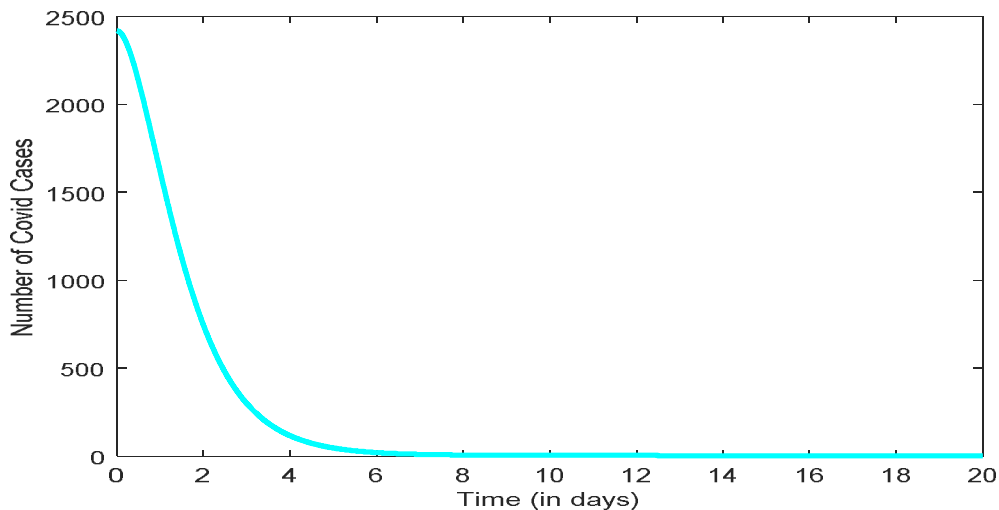


Figure 13: daily homecare isolation with containment measures in place

Home treatment was for people with mild symptoms. The graph has a curve with continuous negative gradient showing that the people isolated at home decreases since those were there have recovered. There is no increase in the number of people in the compartment since those who are recovering are more than those who are getting the infection. People without symptoms will not need to go for home isolation since they cannot transmit the disease if they are strictly following these measures meant to contain the pandemic. Continuous adherent to these SARS-COV-2 containment measures suppresses the disease completely.

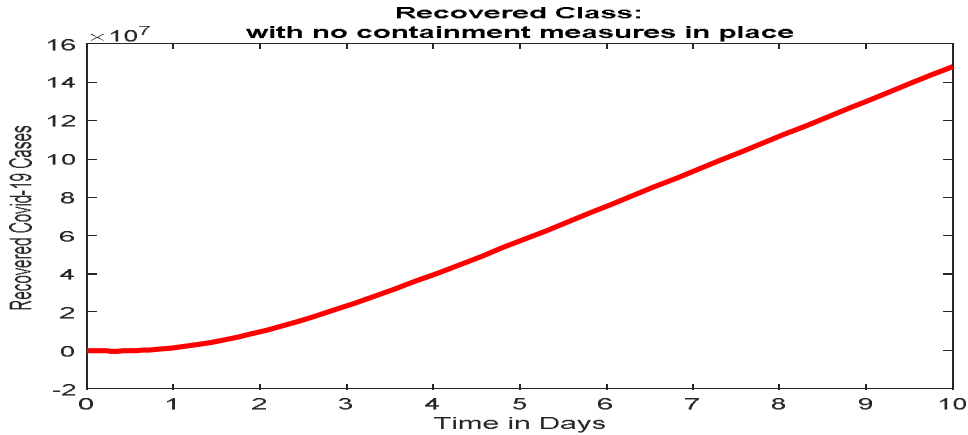


Figure 14: recovered compartment with no containment measures in place

There is continuous increase in SARS-COV-2 recovery cases with time. This is as a result of more infected people recovering from the disease in the hospital and home isolation on daily basis as a result of treatment.

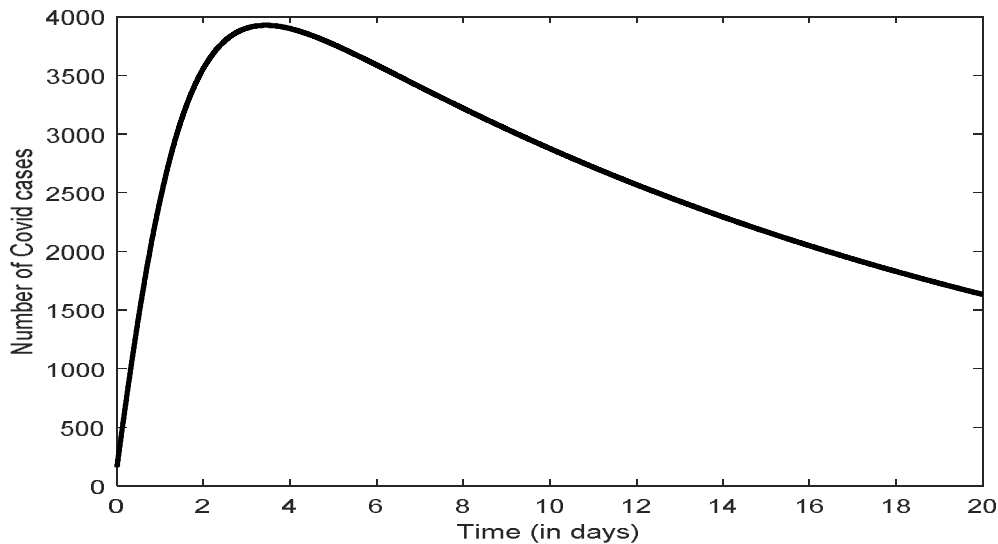


Figure 15: daily recovered SARS-CoV-2 cases with containment measures in place

The graph shows a curve that is having a positive gradient, proceeds to maxima and later the gradients is negative all through. This shows that there is increase in the number of recovered patients since those who are in hospital treatment or home treatment will continue to recover and once the infection is suppressed, the number of the daily recoveries will also reduce consequently.

EFFECTS OF EACH CONTAINEMENT MEASURES ON EXPOSED CLASS

The four SARS-COV-2 containment measures (screening, social distancing, hand-washing and facemask wearing) were meant to reduce the contact rate between the infectious people and those who are not affected. This effectively reduces the rate of exposure. The four SARS-COV-2 containment measures were simulated to investigate their individual effects on the exposed class compared to their total contribution when they are combined together (Λ). It was assumed that they had equal contribution in reducing exposure to the disease hence each was given a reduction rate of $1 \div 4 = 0.25$. The combined contribution for the four is 1.

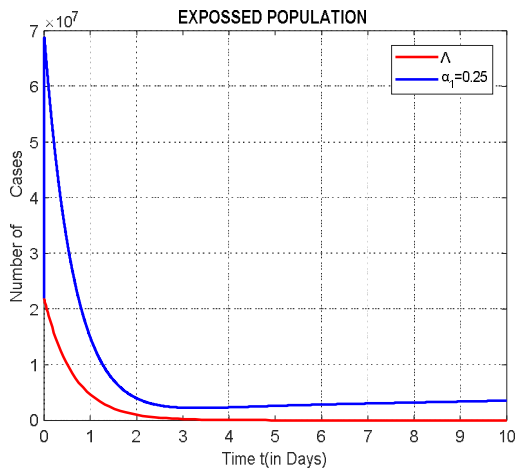


Figure 16a: Effects of facemask vs effects of Λ on Exposed class

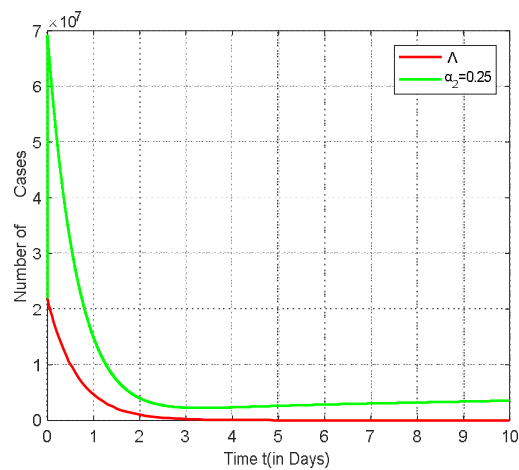


Figure 16b: Effects of hand washing vs effects of Λ on Exposed class

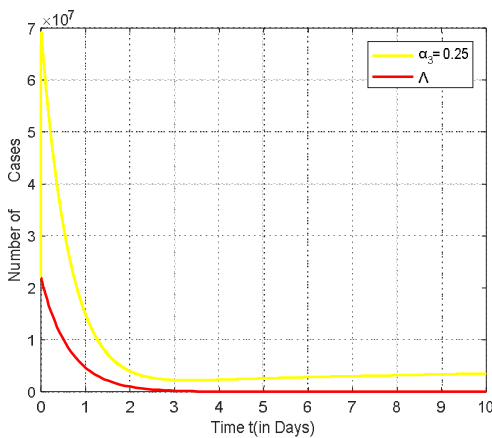


Figure 16c: Effects of social distancing vs Effects of Λ on Exposed class

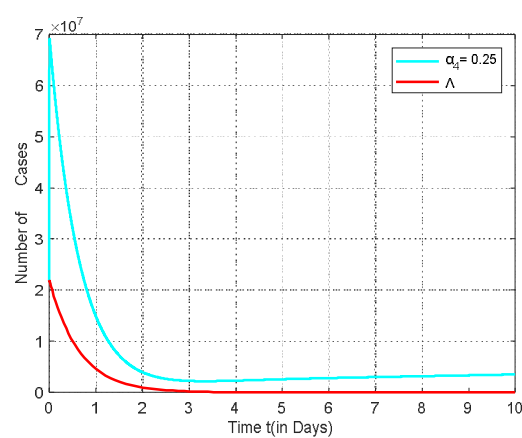


Figure 16d: Effects of screening vs Effects of Λ on Exposed class

The simulation result shows that each containment measure causes reduction in the exposure to the disease but none can contain the pandemic alone. The graph shows that no individual measure is reducing the exposure to zero.

The combination of all four measures has an effect of reducing the exposure to zero. This is exhibited by the graph in red color on all the four Cartesian planes.

Results and discussion

The model was found to be well posed and stabilities existed. In absence of the use of these preventive measures, the basic reproduction number was found to be 4.0682. This means that SARS-COV-2 will persist in the community. Isolation is not enough to stop the spread. In order to decrease the basic reproduction number to below 1 hence suppressing the disease, it can be calculated from the Reproduction number equation that at least 75.42% of the population must adhere to these measures which are meant to curtail transmission. From table 1, it was found that Λ has the greatest negative impact to the reproduction number (-3.251) followed by γ (-0.8848). This shows the need to emphasize on adherence to the containment measures in addition to contact tracing.

On simulation of the variables, it was found that the effectiveness of each individual containment measure was unable to lonely reduce the exposure to zero as shown in figures 16a, 16b, 16c and 16d. The combined effects of all the four containment measures collectively were shown to prevent the exposure completely.

Conclusion

Our task was to develop a mathematical deterministic model of the effects of screening and non-clinical strategies like social distancing, hand washing and use of facemask on the transmission of SARS-COV-2 in Kenya. Figure one shows the flow chart of the model detailing the six human compartments that makes up the whole population. The progression from one compartment to another is shown. Boundedness and positivity of the model was done successfully. We determined the two equilibrium points; Disease free Equilibrium and Endemic Equilibrium Points of the system. The basic reproduction number was determined using Next Generation Method. The model was analyzed through the determination of the model steady states. The stabilities of steady states analyzed based on reproduction number using: signs of Jacobi Matrix evaluated at steady state, Lyapnov Criteria, Centre Manifold theorem, Metzler matrix and Routh-Hurwitz. The stabilities analyzed were; local stability of the DFE, Global stability of DFE and Global stability of Endemic Equilibrium. Bifurcation analysis was carried out using Centre Manifold Theorem by Carlos Castillo-Chavez Z. F. (2001). Numerical simulations were carried out using MATLAB inbuilt ODE solver based on Runge Kutta Method. Sensitivity analysis of the model parameters was carried out using partial differentiation of the reproduction number and also using Normalized sensitivity analysis. From this analysis, findings showed that adherence to the containment measures and contact tracing had the greatest negative impact on the reproduction number. From simulations, it was found that the rate of infection is highly reduced as a result of adherence to the combined containment measures (Λ). No single intervention was found through simulation that adherence to the combined SARS-CoV-2

containment measures by the population would reduce the reproduction number hence containing the pandemic.

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