

Modelling The Impact of Screening, Treatment and Underlying Health Conditions on Dynamics of Covid-19

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Abstract

This study formulated a SIRS classical mathematical model which is modified to incorporate the exposed and the treated individuals where COVID-19 is modeled. The model stratifies the population into two categories depending whether they have underlying health conditions or not, and describes disease transmission within or between the groups. Five compartments are considered in the model for each group that is; Susceptible individuals, exposed population, Infected individuals, treated population and the Recovered population. The objectives were to; Formulate a mathematical deterministic model based on classical SIRS model incorporating screening, treatment and underlying health conditions on covid-19 dynamics. Determine the Reproduction number and use it to analyze the model. Determining sensitivity analysis and Bifurcation. Simulating the model using data from the ministry of health. The Next generation matrix method was used to determine the basic reproduction number denoted R_0 of the proposed model. The results of the simulation indicated that the Disease Free Equilibrium is locally asymptotically stable whenever $R_0^* < 1$ and globally asymptotically stable if $R_0^* \leq 1$. On the other hand, Endemic Equilibrium was globally asymptotically stable if $R_0^* > 1$. The results obtained showed that increasing the rate of screening and treatment on the exposed population and weakening the disease transmission route between the susceptible, exposed and infected population are crucial to curb the spread of COVID-19 virus. The Government of Kenya should advocate treatment and screening of the exposed and infected individuals.

Keywords:

COVID-19 transmission, underlying health conditions, Screening, Treatment, Sustained Development Goals, Severe Acute Respiratory Syndrome Coronavirus 2

1. INTRODUCTION

COVID-19 is an infectious disease triggered by severe acute respiratory syndrome coronavirus 2 viruses that belongs to the family of viruses that cause viral pneumonia (WHO 2021). The WHO declared COVID-19 as a pandemic on 11th March 2020 and on 13th March 2020, Kenya reported the first confirmed COVID-19 case and thereafter the cases increased gradually. The virus can be transmitted from an infectious person's mouth or nose in small liquid particles when they speak, sing, breathe or cough to another person and is transmitted between peoples who are in close contact with one another. Also through touching surfaces or objects that has been contaminated by the virus and then touching their mouth, nose and their eyes. The common symptoms for COVID-19 are tiredness, fever, loss of smell or taste and dry cough. The less commonly symptoms are sore throat, headache, red or irritated eyes, aches and pains, diarrhea and rashes on skin. Serious symptoms for COVID-19 are chest pain, difficulty breathing or shortness of breath, loss of speech or mobility or confusion. (WHO, 2020).

Amongst many intervention strategies to prevent transmission of Coronavirus disease the government advocated the use of face masks, social distancing, hygiene, lockdown, quarantine, curfew, closure of schools and places of worship, ban of public meetings, and regulation of the number of people attending burials among others (Titus et al 2020). To curb the spread of the virus, the MOH in Kenya adopted treatment of confirmed cases and active screening which entailed a sequence of questions about recent travel, symptoms of any illness and contact with individuals who have tested positive for coronavirus disease (Diagne et al, 2021). It was noted that active screening, testing and treatment reduced the number of infectious individuals (Abioye et al, 2021).

22% of the global population is estimated to have relatively one underlying medical conditions that put them at higher threat for severe Coronavirus related infection (Clark et al. 2020). A research carried on by CDC and prevention addresses that amongst COVID-19 confirmed cases, the most familiar underlying medical conditions are chronic respiratory disease (18%), diabetes (30%) and cardiovascular infection (32%). In Kenya, a study done by (Ombajo et al 2022) on 787 COVID-19 patients admitted in six facilities (Kenyatta National Referral Hospital, Mbagathi Hospital, Coast General Teaching and Referral Hospital, Nairobi Hospital, Aga Khan University Hospital and Avenue Hospital), 43% had underlying conditions, with the most common being cardiovascular diseases (17%), diabetes (15%), HIV (7%), cancer (4%), chronic renal disease (3%) and chronic obstructive pulmonary disease (3%).

The virus has spread worldwide resulting to 233,136,147 confirmed cases, 4,771,408 deaths while approximately 211.3 million recoveries from the disease as of 30 September 2021 (WHO, 2021). In Africa the number of confirmed cases of COVID-19 as of 30th September 2021, amounted to 8,391,451 which represents around 3.58% of the infections around the world. (CDC, 2021). In an attempt to curb the spread of the disease, different governments took severe restrictive measures. Nevertheless, the virus still managed to have its impacts on several countries. In Kenya, according to the ministry of health as of 26 August 2021 the positivity rate was 12.9% with the number of confirmed cases being 232,869 and fatalities of 4,635 people, which poses a huge risk to economies and the global public health.

The virus has collapsed the economies of different countries and changed the people's way of life which immensely affected the social, economic and political pillar of Kenya Vision 2030 which reverberate very intimately to the sustainable Development Goals 2030. Kenya, like all other developing countries is challenged with the duty of strategically working towards the

attainment of the SDGs 2030 whose time limit of achievement corresponds with those of the state blueprint. Towards attainment of the SDG's and Kenya Vision 2030, the countries focus is on the "Big Four Agendas" that is food security, affordable health care, affordable housing and manufacturing as well as wealth and Job creation (Aminga, 2018).

Many mathematical models have already been formulated in various countries to analyze the complex transmission pattern of the COVID-19 pandemic, using ordinary differential equations, stochastic differential equations and fractional order Caputo derivative (Riyapan et al, 2021) to develop supportive strategies for efficient elimination of infection. Threshold theorems involving the basic reproduction number have been determined and applied to specific diseases. Similar results with new expressions for were obtained for various classical epidemiology models that are often based on the flow patterns between the compartments such as SEIR, SEIAR, SEIAHR, SSqEIR, SEEqIR, SEIHR, SEIFR, SEIPFR, SEIPAR, SEIRF, SEIHRF endemic models (Shanmugapriya et al, 2020).

Yang et al, 2021, modelled dynamics of COVID-19 and underlying health conditions but did not include effect of screening and treatment. Baek et al, 2020, studied COVID-19 transmission in a tertiary hospital and evaluation of the impacts of different intervention strategies using SEIR model but did not include underlying health conditions and effects of screening and treatment. Diagne et al 2021, studied dynamics of COVID-19 with vaccination and treatment but they did not include screening and underlying health conditions. Peter et al, 2021, developed a SEIQR mathematical model of COVID-19 with actual data from Pakistan. Titus et al 2020, developed an SEIR mathematical model of COVID-19 disease dynamics and analysis of intervention strategies but did not include effects of underlying health conditions. Clark et al, 2020, modelled a global, regional and national estimates of the population at increased risk of severe COVID-19 due to underlying health conditions but did not include effect of screening and treatment.

Masandawa et al, 2021 modelled COVID-19 transmission dynamics between healthcare workers and community but did not include effect of underlying health conditions. Bandekar et al, 2021 studied modelling and analysis of COVID-19 in India with treatment function through different phases of lockdown and unlock but did not include effect of underlying health conditions.

Nyamu et al, 2020 modelled COVID-19 in Kenya using SIR model but did not include effect of underlying health conditions. Ngari et al, 2020 studied parameters and state estimates of COVID-19 model using Lagrange polynomial, least square approximation and Kenya quarantine data but did not include effect of underlying health conditions. Ndairou et al, 2020 developed a compartmental mathematical modelling of COVID-19 transmission dynamics with case study of Wuhan but their study did not look into effect of underlying health conditions.

Although research has been done on dynamics of Covid-19 there is no research that has incorporated the impact of screening, treatment as a control strategy and underlying health conditions on control of covid-19 dynamics. The qualitative behavior, sensitivity analysis and numerical simulation are performed in this study. The paper is organized as follows; The proposed model is presented in Model Formulation. The numerical analysis of the model is presented in Model Analysis. The results obtained from numerical simulations of the model are provided in Numerical simulation. Finally, the conclusion drawn from this study is given in Conclusion.

2. MODEL FORMULATION

Our study is divided into two groups: group 1 those without underlying health conditions divided

into 5 compartments; S_n -Susceptible individuals, E_n - exposed individuals, I_n -infected individuals, T_n –treatment and R_n - the recovered individuals. Group 2 those with underlying health conditions divided into 5 compartments for the host population; S_u - Susceptible individuals, E_u - exposed individuals, I_u - infected persons, T_u -treatment and R_u -Recovered individuals. The progress from one compartment to another was formulated by 5 nonlinear ordinary differential equations for each group. The force of infection denoted by $\lambda(t) = \beta[E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u]$ where, $0 < \eta_5 < \eta_4 < \eta_3 < \eta_2 < \eta_1 < 1$ and β is the transmissibility parameter. The assumptions of the study are as follows, all individuals in the country are susceptible to COVID-19 for the disease has spread to all parts of the country. Screening of the exposed and infected individuals reduces the rate of transmission of COVID-19 since those identified are isolated. Individuals with underlying medical conditions are at high risk of being affected by COVID-19 disease than those without since they spend most times in hospitals. Individuals who have recovered from the disease can get re-infected by the virus if they get exposed to it.

Table 1: Parameters and Description

Parameters	Description
π	Recruitment rate into susceptible population
$\theta\pi$	Proportion of the recruitment rate to susceptible without underlying health conditions
$(1-\theta)\pi$	Proportion of the recruitment rate to susceptible with underlying health condition
λ_n	Rate at which susceptible without underlying conditions are exposed
λ_u	Rate at which susceptible with underlying conditions are exposed
μ	Natural death
ξ_n	The rate at which the treated without underlying conditions recover
ξ_u	The rate at which treated with underlying conditions recover
Ω_n	The rate at which the infected without underlying conditions get treated
Ω_u	The rate at which the infected with underlying health conditions get treated
δ_n	Rate at which the exposed without underlying conditions get infected
δ_u	Rate at which the exposed with underlying conditions get infected
α_n	Death caused by COVID-19 for persons without underlying conditions
α_u	Death caused by COVID-19 for persons with underlying health conditions
$\gamma_1, \gamma_2, \gamma_3, \gamma_4, \gamma_5$	People who develop underlying conditions in the course of Covid-19 to S_u, E_u, I_u, T_u and R_u
ε_n	Rate at which the recovered without underlying conditions are susceptible
ε_u	Rate at which recovered with underlying conditions are susceptible

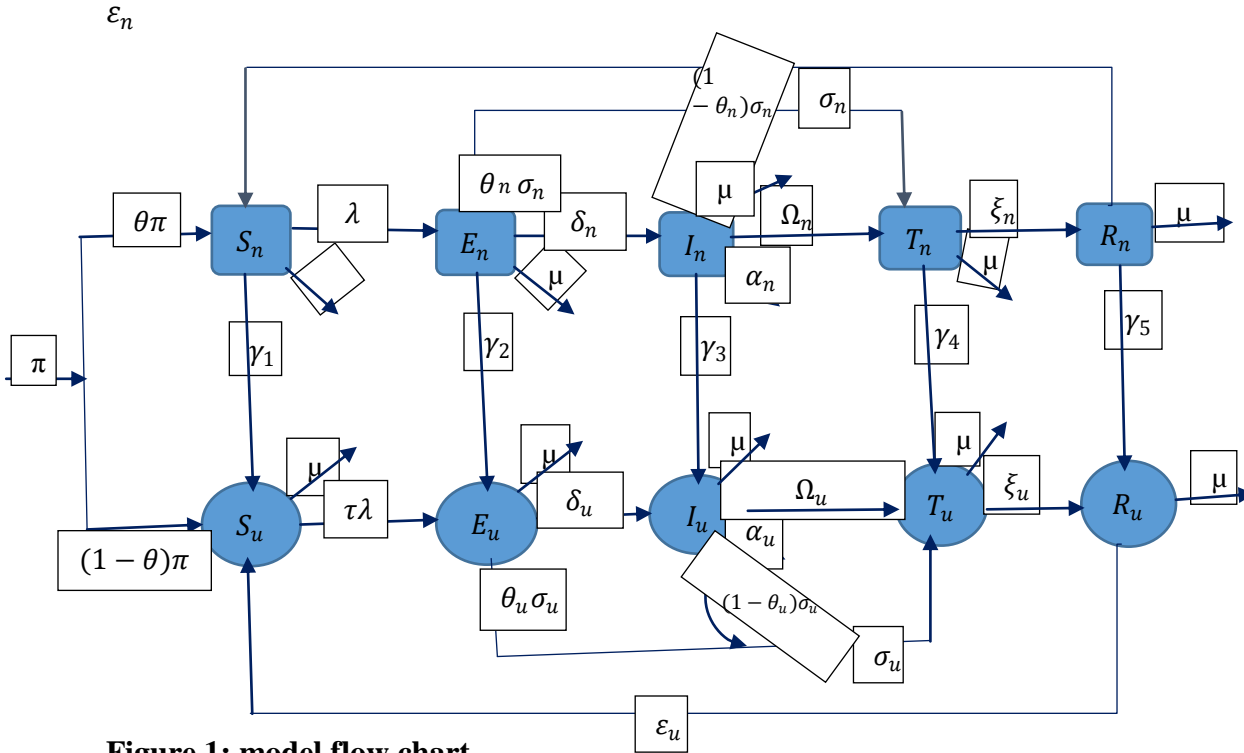


Figure 1: model flow chart
2.1 Model Equations

$$\frac{dS_n}{dt} = \theta\pi + \varepsilon_n R_n - (\beta[E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u] + \gamma_1 + \mu) S_n \quad (1)$$

$$\frac{dE_n}{dt} = (\beta[E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u]) S_n - (\delta_n + \mu + \gamma_2 + \theta_n \sigma_n) E_n \quad (2)$$

$$\frac{dI_n}{dt} = \delta_n E_n - (\Omega_n + \alpha_n + \gamma_3 + \mu + (1 - \theta_n) \sigma_n) I_n \quad (3),$$

$$\frac{dT_n}{dt} = \Omega_n I_n + \theta_n \sigma_n E_n + (1 - \theta_n) \sigma_n I_n - (\xi_n + \mu + \gamma_4) T_n \quad (4),$$

$$\frac{dR_n}{dt} = \xi_n T_n - (\varepsilon_n + \mu + \gamma_5) R_n \quad (5),$$

$$\frac{dS_u}{dt} = (1 - \theta)\pi + \varepsilon_u R_u + \gamma_1 S_n - (\beta[E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u] + \mu) S_u \quad (6)$$

$$\frac{dE_u}{dt} = (\beta[E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u]) S_u + \gamma_2 E_n - (\delta_u + \mu + \theta_u \sigma_u) E_u \quad (7)$$

$$\frac{dI_u}{dt} = \delta_u E_u + \gamma_3 I_n - (\Omega_u + \mu + \alpha_u + (1 - \theta_u) \sigma_u) I_u \quad (8),$$

$$\frac{dT_u}{dt} = \Omega_u I_u + \theta_u \sigma_u E_u + (1 - \theta_u) \sigma_u I_u + \gamma_4 T_n - (\xi_u + \mu) T_u \quad (9),$$

$$\frac{dR_u}{dt} = \xi_u T_u + \gamma_5 R_n - (\varepsilon_u + \mu) R_u \quad (10),$$

3. Model Analysis

3.1 Boundedness and Positivity

The dynamic system is uniformly bounded in the proper subset $Q \in R_+^{10}$

$$Q = \{S_n(t), E_n(t), I_n(t), T_n(t), R_n(t), S_u(t), E_u(t), I_u(t), T_u(t), R_u(t) \in R_+^{10} \}$$

In the absence of the disease, there are no exposure to disease, no infected, no treated and no recovery. Then initially $N(0) = S(0)$

$$\frac{dN}{dt} = \pi - \mu N - \alpha_n I_n - \alpha_u I_u \quad (11)$$

In absence of mortality rate caused by COVID-19, the equation (11) becomes;

$$\frac{dN}{dt} \leq \pi - \mu N \quad (12)$$

$$\frac{1}{\mu} \ln \left| \frac{\pi - \mu N}{\pi - \mu N_0} \right| \leq t + A$$

$$\pi - \mu N \geq C e^{-\mu t}$$

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

$$\lim_{t \rightarrow \infty} N(t) \leq \frac{\pi}{\mu} \quad (13)$$

And this implies that; $0 \leq N < \frac{\pi}{\mu}$ and $N \leq \frac{\pi}{\mu}$

Therefore, the model is bounded in the domain

$$Q_t = \left\{ (S_n(t), E_n(t), I_n(t), T_n(t), R_n(t), S_u(t), E_u(t), I_u(t), T_u(t), R_u(t)) \in R_+^{10} : N \leq \frac{\pi}{\mu} \right\}$$

Thus, the model makes biological sense and is well posed in the region.

Positivity

The positivity of the model is calculated by first letting:

$$S_n(0) \geq 0, E_n(0) \geq 0, I_n(0) \geq 0, T_n(0) \geq 0, R_n(0) \geq 0, S_u(0) \geq 0, E_u(0) \geq 0, I_u(0) \geq 0, T_u(0) \geq 0, R_u(0) \geq 0.$$

Proof. The prove of the theorem follows that, from the first differential equations

$$\begin{aligned} \frac{dS_n}{dt} &= \theta \pi + \varepsilon_n R_n - (\beta [E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u] + \gamma_1 + \mu) S_n \\ \frac{dS_n}{S_n} &\geq -(\beta [E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u] + \gamma_1 + \mu) dt \end{aligned} \quad (14)$$

On integration of (14) with respect to t and substituting t=0.

$$\frac{dS_n}{dt} \geq \frac{d}{dt} \left[S_n(0) e^{\int_0^t -(\beta [E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u] + \gamma_1 + \mu) dt} \right] \geq 0. \quad (15)$$

Clearly, $S_n(0) e^{\int_0^t -(\beta [E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u] + \gamma_1 + \mu) dt}$ is a non-negative function of t, therefore, $S_n(t)$ stays positive.

The positivity of $E_n(t), I_n(t), T_n(t), R_n(t), S_u(t), E_u(t), I_u(t), T_u(t)$ and $R_u(t)$ are proved the same way as follows:

$$E_n(t) \geq E_n(0) e^{-(\delta_n + \mu + \gamma_2 + \theta_n \sigma_n)t} \geq 0.$$

$$\begin{aligned}
 I_n(t) &\geq I_n(0)e^{-(\Omega_n + \alpha_n + \gamma_3 + \mu + (1-\theta)\sigma_n)t} \geq 0. \\
 T_n(t) &\geq T_n(0)e^{-(\xi_n + \mu + \gamma_4)t} \geq 0. \\
 R_n(t) &\geq R_n(0)e^{-(\varepsilon_n + \mu + \gamma_5)t} \geq 0. \\
 S_u(t) &\geq S_u(0)e^{-(\beta[E_n + \eta_1 I_n + \eta_2 T_n + \eta_3 E_u + \eta_4 I_u + \eta_5 T_u] + \mu)t} \geq 0. \quad (16) \\
 E_u(t) &\geq E_u(0)e^{-(\delta_u + \mu + \theta_u \sigma_u)t} \geq 0. \\
 I_u(t) &\geq I_u(0)e^{-(\Omega_u + \mu + \alpha_u + (1-\theta)\sigma_u)t} \geq 0. \\
 T_u(t) &\geq T_u(0)e^{-(\xi_u + \mu)t} \geq 0. \\
 R_u(t) &\geq r_u(0)e^{-(\varepsilon_u + \mu)t} \geq 0.
 \end{aligned}$$

3.2 Disease Free Equilibrium point (DFE)

The disease free equilibrium point of the system of equations [(1) -(10)] is obtained by setting all the exposed class, the treated, the infectious class and recovered classes to zero.

For those people with underlying health conditions

$$E_n^0 = (S_n^0, E_n^0, I_n^0, T_n^0, R_n^0) = \left(\frac{\theta\pi}{\mu}, 0, 0, 0, 0\right).$$

For those with underlying conditions

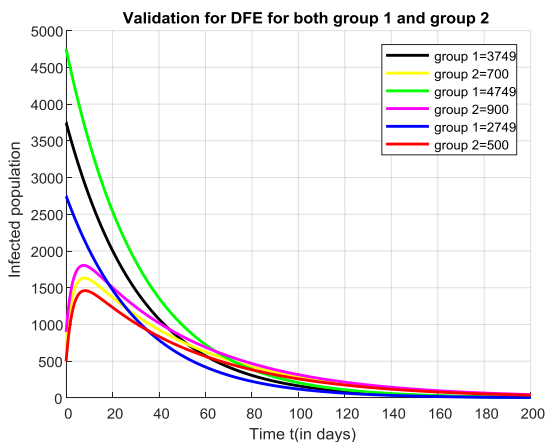
$$E_u^0 = (S_u^0, E_u^0, I_u^0, T_u^0, R_u^0) = \left(\frac{(1-\theta)\pi}{\mu}, 0, 0, 0, 0\right).$$

For the whole system

$$E^0 = (S_n^0, E_n^0, I_n^0, T_n^0, R_n^0, S_u^0, E_u^0, I_u^0, T_u^0, R_u^0) = \left(\frac{\theta\pi}{\mu}, 0, 0, 0, 0, \frac{(1-\theta)\pi}{\mu}, 0, 0, 0, 0\right). \quad (17)$$

This was validated by numerical method as shown below

Figure 2: Validation for the DFE for both group 1 and group 2



3.3 Computation of Basic Reproduction Number (R_o)

We use the Next Generation Matrix Method to determine Reproduction number R_o of the model (Chevez et al; 2002). Using the notation f for a matrix of new infection terms and v for matrix of the remaining transfer of infection terms in this system. The Reproduction number for the whole model is obtained as;

$$f = \begin{pmatrix} \lambda_n S_n \\ 0 \\ 0 \\ \kappa \lambda_u S_u \\ 0 \\ 0 \end{pmatrix}, v = \begin{pmatrix} (\delta_n + \mu + \gamma_n + \theta_n \sigma_n) E_n \\ -\delta_n E_n + (\Omega_n + \alpha_n + \gamma_3 + \mu + (1 - \theta_n) \sigma_n) I_n \\ -\Omega_n I_n - \theta_n \sigma_n E_n - (1 - \theta_n) \sigma_n I_n + (\xi_n + \mu + \gamma_4) T_n \\ -\gamma_2 E_n + (\delta_u + \mu + \theta_u \sigma_u) E_u \\ -\delta_u E_u - \gamma_3 T_n + (\Omega_u + \mu + \alpha_u + (1 - \theta_u) \sigma_u) I_u \\ -\Omega_u I_u - \theta_u \sigma_u E_u - (1 - \theta_u) \sigma_u I_u - \gamma_4 T_n + (\xi_u + \mu) T_u \end{pmatrix} \quad (18)$$

We obtain the matrices F and V by finding the Jacobian matrices of f and v evaluated at DFE respectively to get,

The Jacobian matrix f for the whole system

$$F = \begin{pmatrix} S\beta & S\beta\eta_1 & S\beta\eta_2 & S\beta\eta_3 & S\beta\eta_4 & S\beta\eta_5 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ S\beta\kappa & S\beta\kappa\eta_1 & S\beta\kappa\eta_2 & S\beta\kappa\eta_3 & S\beta\kappa\eta_4 & S\beta\kappa\eta_5 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (19)$$

To get the Jacobian of v for whole model we let:

$$\begin{aligned} \Delta_1 &= (\delta_n + \mu + \gamma_2 + \theta_n \sigma_n) \\ \Delta_2 &= (\Omega_n + \alpha_n + \gamma_3 + \mu + (1 - \theta_n) \sigma_n) \\ \Delta_3 &= (\xi_n + \mu + \gamma_4) \\ \Delta_4 &= (\delta_u + \mu + \theta_u \sigma_u) \\ \Delta_5 &= (\Omega_u + \mu + \alpha_u + (1 - \theta_u) \sigma_u) \\ \Delta_6 &= (\xi_u + \mu) \\ \Delta_7 &= (\varepsilon_n + \mu + \gamma_5) \end{aligned}$$

$$V = \begin{pmatrix} \Delta_1 & 0 & 0 & 0 & 0 & 0 \\ -\delta_n & \Delta_2 & 0 & 0 & 0 & 0 \\ -\theta_n \sigma_n & -(1 - \theta_n) \sigma_n - \Omega_n & \Delta_3 & 0 & 0 & 0 \\ -\gamma_2 & 0 & 0 & \Delta_4 & 0 & 0 \\ 0 & 0 & -\gamma_3 & -\delta_u & \Delta_5 & 0 \\ 0 & 0 & -\gamma_4 & -\theta_u \sigma_u & -(1 - \theta_u) \sigma_u - \Omega_u & \Delta_6 \end{pmatrix}$$

Let $k_1 = \theta_n \sigma_n$, $k_2 = -\Omega_n - (1 - \theta_n) \sigma_n$, $k_3 = \theta_u \sigma_u$ and $k_4 = -\Omega_u - (1 - \theta_u) \sigma_u$

We obtain V^{-1} of the whole model as

$$\begin{pmatrix} \frac{1}{\Delta_1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\delta_n}{\Delta_1 \Delta_2} & \frac{1}{\Delta_2} & 0 & 0 & 0 & 0 \\ \frac{-k_2 \delta_n \Delta_4 \Delta_5 \Delta_6 + k_1 \Delta_2 \Delta_4 \Delta_5 \Delta_6}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & -\frac{k_2}{\Delta_2 \Delta_3} & \frac{1}{\Delta_3} & 0 & 0 & 0 \\ \frac{\gamma_2}{\Delta_1 \Delta_4} & 0 & 0 & \frac{1}{\Delta_4} & 0 & 0 \\ \frac{\gamma_2 \delta_u \Delta_2 \Delta_3 \Delta_4 - k_2 \gamma_3 \delta_n \Delta_4 \Delta_6 + k_1 \gamma_3 \Delta_2 \Delta_4 \Delta_6}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & -\frac{k_2 \gamma_3}{\Delta_2 \Delta_3 \Delta_5} & \frac{\gamma_3}{\Delta_3 \Delta_5} & \frac{\delta_u}{\Delta_4 \Delta_5} & \frac{1}{\Delta_5} & 0 \\ \frac{-k_4 \gamma_2 \delta_u \Delta_2 \Delta_3 + k_2 k_4 \gamma_3 \delta_n \Delta_4 - k_1 k_4 \gamma_3 \Delta_2 \Delta_4 + k_3 \gamma_2 \Delta_2 \Delta_3 \Delta_5 - k_2 \gamma_4 \delta_n \Delta_4 \Delta_5 + k_1 \gamma_4 \Delta_2 \Delta_4 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & \frac{k_2 k_4 \gamma_3 \Delta_1 \Delta_4 - k_2 \gamma_4 \Delta_1 \Delta_4 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & \frac{-k_4 \gamma_3 \Delta_1 \Delta_2 \Delta_4 + \gamma_4 \Delta_1 \Delta_2 \Delta_4 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & \frac{-k_4 \delta_u \Delta_1 \Delta_2 \Delta_3 + k_3 \Delta_1 \Delta_2 \Delta_3 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & -\frac{k_4}{\Delta_5 \Delta_6} & \frac{1}{\Delta_6} \end{pmatrix}$$

Multiplying the matrices F and V^{-1} we obtain,

$$FV^{-1} = \begin{pmatrix} T_1 & T_2 & T_3 & T_4 & T_5 & T_6 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ T_7 & T_8 & T_9 & T_{10} & T_{11} & T_{12} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

Where;

$$\begin{aligned} T_1 &= \frac{S\beta}{\Delta_1} + \frac{S\beta\delta_n\eta_1}{\Delta_1\Delta_2} + \frac{S\beta(-k_2\delta_n\Delta_4\Delta_5\Delta_6 + k_1\Delta_2\Delta_4\Delta_5\Delta_6)\eta_2}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} + \frac{S\beta\gamma_2\eta_3}{\Delta_1\Delta_4} \\ &+ \frac{S\beta(\gamma_2\delta_u\Delta_2\Delta_3\Delta_6 - k_2\gamma_3\delta_n\Delta_4\Delta_6 + k_1\gamma_3\Delta_2\Delta_4\Delta_6)\eta_4}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ &+ \frac{S\beta(-k_4\gamma_2\delta_u\Delta_2\Delta_3 + k_2k_4\gamma_3\delta_n\Delta_4 - k_1k_4\gamma_3\Delta_2\Delta_4 + k_3\gamma_2\Delta_2\Delta_3\Delta_5 - k_2\gamma_4\delta_n\Delta_4\Delta_5 + k_1\gamma_4\Delta_2\Delta_4\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_2 &= \frac{S\beta\eta_1}{\Delta_2} - \frac{S\beta k_2\eta_2}{\Delta_2\Delta_3} - \frac{S\beta k_2\gamma_3\eta_4}{\Delta_2\Delta_3\Delta_5} + \frac{S\beta(k_2k_4\gamma_3\Delta_1\Delta_4 - k_2\gamma_4\Delta_1\Delta_4\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_3 &= \frac{S\beta\eta_2}{\Delta_3} + \frac{S\beta\gamma_3\eta_4}{\Delta_3\Delta_5} + \frac{S\beta(-k_4\gamma_3\Delta_1\Delta_2\Delta_4 + \gamma_4\Delta_1\Delta_2\Delta_4\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_4 &= \frac{S\beta\eta_3}{\Delta_4} + \frac{S\beta\delta_u\eta_4}{\Delta_4\Delta_5} + \frac{S\beta(-k_4\delta_u\Delta_1\Delta_2\Delta_3 + k_3\Delta_1\Delta_2\Delta_3\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_5 &= \frac{S\beta\eta_4}{\Delta_5} - \frac{S\beta k_4\eta_5}{\Delta_5\Delta_6} \\ T_6 &= \frac{S\beta\eta_5}{\Delta_6} \\ T_7 &= \frac{S\beta\kappa}{\Delta_1} + \frac{S\beta\kappa\delta_n\eta_1}{\Delta_1\Delta_2} + \frac{S\beta\kappa(-k_2\delta_n\Delta_4\Delta_5\Delta_6 + k_1\Delta_2\Delta_4\Delta_5\Delta_6)\eta_2}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} + \frac{S\beta\kappa\gamma_2\eta_3}{\Delta_1\Delta_4} \\ &+ \frac{S\beta\kappa(\gamma_2\delta_u\Delta_2\Delta_3\Delta_6 - k_2\gamma_3\delta_n\Delta_4\Delta_6 + k_1\gamma_3\Delta_2\Delta_4\Delta_6)\eta_4}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ &+ \frac{S\beta\kappa(-k_4\gamma_2\delta_u\Delta_2\Delta_3 + k_2k_4\gamma_3\delta_n\Delta_4 - k_1k_4\gamma_3\Delta_2\Delta_4 + k_3\gamma_2\Delta_2\Delta_3\Delta_5 - k_2\gamma_4\delta_n\Delta_4\Delta_5 + k_1\gamma_4\Delta_2\Delta_4\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_8 &= \frac{S\beta\kappa\eta_1}{\Delta_2} - \frac{S\beta\kappa k_2\eta_2}{\Delta_2\Delta_3} - \frac{S\beta\kappa k_2\gamma_3\eta_4}{\Delta_2\Delta_3\Delta_5} + \frac{S\beta\kappa(k_2k_4\gamma_3\Delta_1\Delta_4 - k_2\gamma_4\Delta_1\Delta_4\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_9 &= \frac{S\beta\kappa\eta_2}{\Delta_3} + \frac{S\beta\kappa\gamma_3\eta_4}{\Delta_3\Delta_5} + \frac{S\beta\kappa(-k_4\gamma_3\Delta_1\Delta_2\Delta_4 + \gamma_4\Delta_1\Delta_2\Delta_4\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_{10} &= \frac{S\beta\kappa\eta_3}{\Delta_4} + \frac{S\beta\kappa\delta_u\eta_4}{\Delta_4\Delta_5} + \frac{S\beta\kappa(-k_4\delta_u\Delta_1\Delta_2\Delta_3 + k_3\Delta_1\Delta_2\Delta_3\Delta_5)\eta_5}{\Delta_1\Delta_2\Delta_3\Delta_4\Delta_5\Delta_6} \\ T_{11} &= \frac{S\beta\kappa\eta_4}{\Delta_5} - \frac{S\beta\kappa k_4\eta_5}{\Delta_5\Delta_6} \\ T_{12} &= \frac{S\beta\kappa\eta_5}{\Delta_6} \end{aligned}$$

Using Mathematica to obtain the Eigenvalues $i=1,2,3,4,5,6$ of the matrix FV^{-1} we obtain;

$$X_1 = 0$$

$$\begin{aligned} X_2 &= 0 \\ X_3 &= 0 \\ X_4 &= 0 \\ X_5 &= 0 \end{aligned}$$

$$X_6 = \frac{1}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} S \beta (\delta_n \Delta_4 (\Delta_6 (\Delta_3 \Delta_5 \eta_1 - k_2 (\Delta_5 \eta_2 + \gamma_3 \eta_4)) + k_2 (k_4 \gamma_3 - \gamma_4 \Delta_5) \eta_5) + \Delta_2 (k_1 \Delta_4 (\gamma_3 (\Delta_6 \eta_4 - k_4 \eta_5) + \Delta_5 (\Delta_6 \eta_2 + \gamma_4 \eta_5)) + \Delta_3 (\Delta_4 \Delta_5 \Delta_6 + (\gamma_2 + \kappa \Delta_1) (\Delta_5 (\Delta_6 \eta_3 + k_3 \eta_5) + \delta_u (\Delta_6 \eta_4 - k_4 \eta_5)))))) \text{ which is the spectral radius or the dominant Eigenvalue } ,R_o. \quad (20)$$

For those with underlying health condition the reproduction number is given as;

$$R_n = \left\{ \frac{S_n^0 \beta_n (\mu + \gamma_4 + \delta_n \eta_2 + \xi_n + \eta_2 \theta_n \sigma_n + \frac{\delta_n (-(\mu + \alpha_n + \gamma_3) \eta_2 + \eta_1 (\mu + \gamma_4 + \xi_n))}{\mu + \alpha_n + \gamma_3 + \sigma_n - \theta_n \sigma_n + \Omega_n})}{(\mu + \gamma_4 + \xi_n) (\mu + \gamma_2 + \delta_n + \theta_n \sigma_n)} \right\} \quad (21)$$

And for those without underlying health conditions the reproduction number is

$$R_u = \left\{ \frac{S_u^0 \beta_u (\eta_3 + \frac{\eta_5 \theta_u \sigma_u + \frac{\delta_u (\eta_4 (\mu + \xi_u) + \eta_5 (\Delta_u - \Delta_u \theta_u + \Omega_u))}{\mu + \alpha_u + \sigma_u - \theta_u \sigma_u + \Omega_u}}{\mu + \xi_u})}{\mu + \delta_u + \theta_u \sigma_u} \right\} \quad (22)$$

3.4. Strength Number

Although reproduction number has been used in many epidemiological modelling with some success and great limitations, whereby it has been employed to determine either or not the spread will be severe, some great weaknesses has been pointed out. The number is not unique as it can be obtained by via different methods. Another issue was raised that; this number should have been a function of time not a constant value. Additionally, it was also noticed that a reproductive number cannot be used to indicate either a model will predict waves or not. Recently an alternative number was suggested and was called strength number, of course this number will be subjected to several test to see either it can be used to help to detect some complexities in the spread, at least it this number can help detect waves in a spread. Next generation matrix was used to derive the value, by taking the second derivative of infectious classes (Farman et al. 2023). In this section, we will present the strength number associate to SIRS model.

$$\begin{aligned} \frac{\partial}{\partial E_n} \left(\beta S \frac{E_n}{N} \right) &= \beta S \frac{\partial}{\partial E_n} \left(\frac{E_n}{N} \right) = \beta S \frac{\partial}{\partial E_n} \left(\frac{N - E_n(N^I)}{N^2} \right) = -\frac{\beta S}{N^2} \\ \frac{\partial}{\partial I_n} \left(\beta S \eta_1 \frac{I_n}{N} \right) &= \beta S \eta_1 \frac{\partial}{\partial I_n} \left(\frac{I_n}{N} \right) = \beta S \eta_1 \frac{\partial}{\partial I_n} \left(\frac{N - I_n(N^I)}{N^2} \right) = -\frac{\beta S \eta_1}{N^2} \\ \frac{\partial}{\partial T_n} \left(\beta S \eta_2 \frac{T_n}{N} \right) &= \beta S \eta_2 \frac{\partial}{\partial T_n} \left(\frac{T_n}{N} \right) = \beta S \eta_2 \frac{\partial}{\partial T_n} \left(\frac{N - T_n(N^I)}{N^2} \right) = -\frac{\beta S \eta_2}{N^2} \\ \frac{\partial}{\partial E_u} \left(\beta S \eta_3 \frac{E_u}{N} \right) &= \beta S \eta_3 \frac{\partial}{\partial E_u} \left(\frac{E_u}{N} \right) = \beta S \eta_3 \frac{\partial}{\partial E_u} \left(\frac{N - E_u(N^I)}{N^2} \right) = -\frac{\beta S \eta_3}{N^2} \\ (23) \frac{\partial}{\partial I_u} \left(\beta S \eta_4 \frac{I_u}{N} \right) &= \beta S \eta_4 \frac{\partial}{\partial I_u} \left(\frac{I_u}{N} \right) = \beta S \eta_4 \frac{\partial}{\partial I_u} \left(\frac{N - I_u(N^I)}{N^2} \right) = -\frac{\beta S \eta_4}{N^2} \\ \frac{\partial}{\partial T_u} \left(\beta S \eta_5 \frac{T_u}{N} \right) &= \beta S \eta_5 \frac{\partial}{\partial T_u} \left(\frac{T_u}{N} \right) = \beta S \eta_5 \frac{\partial}{\partial T_u} \left(\frac{N - T_u(N^I)}{N^2} \right) = -\frac{\beta S \eta_5}{N^2} \end{aligned}$$

For those with underlying condition were obtained in a similar way.

We obtain matrix F as;

$$F_A = \begin{pmatrix} -\frac{\beta S}{N^2} & -\frac{\beta S \eta_1}{N^2} & -\frac{\beta S \eta_2}{N^2} & -\frac{\beta S \eta_3}{N^2} & -\frac{\beta S \eta_4}{N^2} & -\frac{\beta S \eta_5}{N^2} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ -\frac{\beta S \kappa}{N^2} & -\frac{\beta S \kappa \eta_1}{N^2} & -\frac{\beta S \kappa \eta_2}{N^2} & -\frac{\beta S \kappa \eta_3}{N^2} & -\frac{\beta S \kappa \eta_4}{N^2} & -\frac{\beta S \kappa \eta_5}{N^2} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (24)$$

We obtain V^{-1} of the whole model as

$$\begin{pmatrix} \frac{1}{\Delta_1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\delta_n}{\Delta_1 \Delta_2} & \frac{1}{\Delta_2} & 0 & 0 & 0 & 0 \\ \frac{-k_2 \delta_n \Delta_4 \Delta_5 \Delta_6 + k_1 \Delta_2 \Delta_4 \Delta_5 \Delta_6}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & -\frac{k_2}{\Delta_2 \Delta_3} & \frac{1}{\Delta_3} & 0 & 0 & 0 \\ \frac{\gamma_2}{\Delta_1 \Delta_4} & 0 & 0 & \frac{1}{\Delta_4} & 0 & 0 \\ \frac{\gamma_2 \delta_u \Delta_2 \Delta_3 \Delta_6 - k_2 \gamma_3 \delta_n \Delta_4 \Delta_6 + k_1 \gamma_3 \Delta_2 \Delta_4 \Delta_6}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & -\frac{k_2 \gamma_3}{\Delta_2 \Delta_3 \Delta_5} & \frac{\gamma_3}{\Delta_3 \Delta_5} & \frac{\delta_u}{\Delta_4 \Delta_5} & \frac{1}{\Delta_5} & 0 \\ \frac{-k_4 \gamma_2 \delta_u \Delta_2 \Delta_3 + k_2 k_4 \gamma_3 \delta_n \Delta_4 - k_1 k_4 \gamma_3 \Delta_2 \Delta_4 + k_3 \gamma_2 \Delta_2 \Delta_3 \Delta_5 - k_2 \gamma_4 \delta_n \Delta_4 \Delta_5 + k_1 \gamma_4 \Delta_2 \Delta_4 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & \frac{k_2 k_4 \gamma_3 \Delta_1 \Delta_4 - k_2 \gamma_4 \Delta_1 \Delta_4 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & \frac{-k_4 \gamma_3 \Delta_1 \Delta_2 \Delta_4 + \gamma_4 \Delta_1 \Delta_2 \Delta_4 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & \frac{-k_4 \delta_u \Delta_1 \Delta_2 \Delta_3 + k_3 \Delta_1 \Delta_2 \Delta_3 \Delta_5}{\Delta_1 \Delta_2 \Delta_3 \Delta_4 \Delta_5 \Delta_6} & -\frac{k_4}{\Delta_5 \Delta_6} & \frac{1}{\Delta_6} \end{pmatrix}$$

$$A_0 = \frac{1}{2N^2 \Delta_1^2 \Delta_3^2 \Delta_5^2 \Delta_4^2 \Delta_5^2 \Delta_6^2} (k_4 \beta S \eta_5 \gamma_2 \delta_u \Delta_1 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^8 + k_4 \beta S \kappa \eta_5 \delta_u \Delta_1^2 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^8 - k_2 k_4 \beta S \eta_5 \gamma_3 \delta_n \Delta_1 \Delta_2^2 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^8 + k_1 k_4 \beta S \eta_5 \gamma_3 \Delta_1 \Delta_2^3 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^8 - k_3 \beta S \eta_5 \gamma_2 \Delta_1 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^8 - k_3 \beta S \kappa \eta_5 \Delta_1^2 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^8 + k_2 \beta S \eta_5 \gamma_4 \delta_n \Delta_1 \Delta_2^2 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^8 - k_1 \beta S \eta_5 \gamma_4 \Delta_1 \Delta_2^3 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^8 - \beta S \eta_4 \gamma_2 \delta_u \Delta_1 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^9 - \beta S \kappa \eta_4 \delta_u \Delta_1^2 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^9 + k_2 \beta S \eta_4 \gamma_3 \delta_n \Delta_1 \Delta_2^2 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^9 - k_1 \beta S \eta_4 \gamma_3 \Delta_1 \Delta_2^3 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^9 - \beta S \eta_3 \gamma_2 \Delta_1 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^9 - \beta S \kappa \eta_3 \Delta_1^2 \Delta_2^3 \Delta_3^5 \Delta_4^3 \Delta_5^8 \Delta_6^9 + k_2 \beta S \eta_2 \delta_n \Delta_1 \Delta_2^2 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^9 - k_1 \beta S \eta_2 \Delta_1 \Delta_2^3 \Delta_3^4 \Delta_4^4 \Delta_5^8 \Delta_6^9 - \beta S \eta_1 \delta_n \Delta_1 \Delta_2^2 \Delta_3^5 \Delta_4^4 \Delta_5^8 \Delta_6^9 - \beta S \Delta_1 \Delta_2^3 \Delta_3^5 \Delta_4^4 \Delta_5^8 \Delta_6^9 - \sqrt{(\alpha)})$$

Having obtained the strength number to be greater than zero will lead us to great conclusion that there will be another wave of COID-19 that will occur.

3.4 Existence of Endemic Equilibrium Point for the model (EEP).

For the sake of the analysis we consider a special case of the model for population without underlying health conditions, whose Reproduction number;

$$R_n = \left\{ \frac{S \beta_n (\mu + \gamma_4 + \delta_n \eta_2 + \xi_n + \eta_2 \theta_n \sigma_n + \frac{\delta_n (-(\mu + \alpha_n + \gamma_3) \eta_2 + \eta_1 (\mu + \gamma_4 + \xi_n))}{\mu + \alpha_n + \gamma_3 + \sigma_n - \theta_n \sigma_n + \Omega_n})}{(\mu + \gamma_4 + \xi_n)(\mu + \gamma_2 + \delta_n + \theta_n \sigma_n)} \right\}$$

A positive endemic equilibrium exists and is locally asymptotically stable whenever $R_o > 1$ and the infectious classes must be greater than zero. Setting the right hand of the model equations (4,3,2) for infectious classes to zero and solving for T_n then substitute it to I_n . Then solving for I_n and substituting it to E_n and finally solve for E_n .

$$\Omega_n I_n + \theta_n \sigma_n E_n + (1 - \theta_n) \sigma_n I_n - (\xi_n + \mu + \gamma_4) T_n = 0$$

$$T_n < \frac{\Omega_n I_n + \theta_n \sigma_n E_n + (1 - \theta_n) \sigma_n I_n}{(\xi_n + \mu + \gamma_4)}$$

$$T_n = \frac{\Omega_n I_n + \theta_n \sigma_n E_n + (1 - \theta_n) \sigma_n I_n}{\Delta_3} \quad (25)$$

$$I_n < \frac{\delta_n E_n}{\Omega_n + \alpha_n + \gamma_3 + \mu + (1 - \theta_n) \sigma_n}$$

Substituting I_n into T_n we get;

$$T_n = \frac{\Omega_n \delta_n E_n + \Delta_2 \theta_n \sigma_n E_n + (1 - \theta_n) \sigma_n \delta_n E_n}{\Delta_2 \Delta_3} \quad (26)$$

$$\lambda_n S_n = (\delta_n + \mu + \gamma_2 + \theta_n \sigma_n) E_n$$

$$E_n < \frac{\lambda_n S_n}{\delta_n + \mu + \gamma_2 + \theta_n \sigma_n} \text{ the reduced equation for } E_n \text{ is ; } E_n < \frac{\lambda_n S_n}{\Delta_1}$$

Substituting λ_n in E_n with force of infection it becomes;

$$E_n < \frac{\beta [E_n + \eta_1 I_n + \eta_2 T_n] S^0}{\Delta_1} \text{ substituting } T_n \text{ and } I_n \text{ into } E_n \text{ we obtain;}$$

$$1 < \frac{\beta S^0}{\Delta_1} + \frac{\beta \eta_1 \delta_n S^0}{\Delta_1 \Delta_2} + \frac{\beta \eta_2 \Omega_n \delta_n S^0}{\Delta_1 \Delta_2 \Delta_3} + \frac{\beta \eta_2 \Delta_2 \theta_n \sigma_n S^0}{\Delta_1 \Delta_3} + \frac{\beta \eta_2 (1 - \theta_n) \sigma_n \delta_n S^0}{\Delta_1 \Delta_2 \Delta_3} \quad (27)$$

which corresponds to endemic equilibrium point implying that the EEP for the system (6-10) which are the individuals with underlying conditions and (1-10) for the whole system exists.

3.5 Local Stability of the disease free equilibrium point (D.F.E)

For the sake of analysis, we are going to consider a special case of the model for the population without underlying health conditions.

Theorem, The Disease Free Equilibrium of the system [1-10] is locally asymptotically stable whenever $R_o < 1$ and unstable if $R_o > 1$. Determining Jacobian Matrix of the system (1-5) at Disease Free Equilibrium is obtained

$$\begin{pmatrix} -(\gamma_1 + \mu) & \beta & \beta \eta_1 & \beta \eta_2 & \varepsilon_n \\ 0 & \beta - (\delta_n + \mu + \gamma_2 + \theta_n \sigma_n) & \beta \eta_1 & \beta \eta_2 & 0 \\ 0 & \delta_n & -(\Omega_n + \mu + \alpha_n + \gamma_3 + (1 - \theta_n) \sigma_n) & 0 & 0 \\ 0 & \theta_n \sigma_n & \Omega_n + (1 - \theta_n) \sigma_n & -(\varepsilon_n + \mu + \gamma_4) & 0 \\ 0 & 0 & 0 & \xi_n & -(\mu + \gamma_5 + \varepsilon_n) \end{pmatrix} \quad (28)$$

Through inspection method we obtained following Eigenvalues which are negative.

$$X_1 = -(\gamma_1 + \mu), \quad X_2 = -(\varepsilon_n + \mu + \gamma_5)$$

For the reduced matrix the characteristic polynomial is obtained as;

$$\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0 \quad (29)$$

From the characteristic polynomial the values $a_1, a_2,$ and a_3 are determined using Mathematica Software and expressed in terms of R_o . By Routh-Hurwitz Criteria for stability, the system (1)-(10) is locally asymptotically stable at DFE whenever $R_o < 1$ if and only if $a_1 > 0; a_2 > 0; a_3 > 0$ and $a_1 a_2 > a_3$.

3.6 Global Stability of the disease free equilibrium point (D.F.E)

For the sake of analysis, we are going to consider a special case of the model for the population without underlying health conditions. The global stability of disease free equilibrium is investigated using Metzler Matrix method proposed by (Catillo-Chevez et al, 2002).

$$\frac{dX}{dt} = F(X, Z); \frac{dZ}{dt} = G(X, Z), G(X, 0) = 0$$

Where ; $X = (S_n, R_n) \in R_+^2$ denote non-infectious COVID-19 compartments and $Z = (E_n, I_n, T_n) \in R_+^3$ denote the infectious COVID-19 compartments $E_o = (X^*, 0)$ represents the disease free equilibrium of the system if this point satisfies following conditions.

- i. $\frac{dX}{dt} = F(X, 0)$, where X^* is globally asymptotically stable.
- ii. $\frac{dZ}{dt} = D_z G(X, 0) Z - G(X, Z) \geq 0$ for all $X, Z \in \Omega$, then we can conclude that E_o is locally asymptotically stable if the following theorems holds.

Theorem; The equilibrium point $E_o = (X^*, 0)$ of the system [1-5] is globally asymptotically stable if $R_o^* \leq 1$ and the conditions (i) and (ii) are satisfied, otherwise unstable. From equation (1) two vectors functions $G(X, Z)$ and $F(X, Z)$, we consider systems

$$F(X, 0) = \begin{pmatrix} \theta\pi - \mu S_n \\ 0 \end{pmatrix}$$

Letting $A = D_z G(X^*, 0)$, which is the Jacobian of $\hat{G}(X, Z)$ taken in (E_n, I_n, T_n) and evaluated at $(X^*, 0)$ such that the matrix A is given by;

A=

$$\begin{pmatrix} \beta - (\delta_n + \mu + \gamma_2 + \theta_n \sigma_n) & \beta \eta_1 & \beta \eta_2 \\ \delta_n & -(\Omega_n + \mu + \alpha_n + \gamma_3 + (1 - \theta_n) \sigma_n) & 0 \\ \theta_n \sigma_n & \Omega_n + (1 - \theta_n) \sigma_n & -(\varepsilon_n + \mu + \gamma_4) \end{pmatrix}$$

(27)

$$AZ = \begin{pmatrix} T_n \beta \eta_2 + I_n \beta \eta_1 + E_n (\beta - (\delta_n + \mu + \gamma_2 + \theta_n \sigma_n)) \\ E_n \delta_n - I_n (\Omega_n + \mu + \alpha_n + \gamma_3 + (1 - \theta_n) \sigma_n) \\ E_n \theta_n \sigma_n + I_n (\Omega_n + (1 - \theta_n) \sigma_n) - T_n (\varepsilon_n + \mu + \gamma_4) \end{pmatrix} \quad (30)$$

$$\hat{G}(X, Z) = \begin{pmatrix} (1 - \frac{S_n}{N}) \beta (E_n + \eta_1 I_n + \eta_2 T_n) \\ 0 \\ 0 \end{pmatrix} \quad (31)$$

Therefore, if $\hat{G}(X, Z) \geq 0$, then the Disease Free Equilibrium, E_o is globally asymptotically stable otherwise it is unstable. Thus $G(X, Z) \geq 0$ for all $X, Z \in R_+^3$. It's clear that matrix A is a M-matrix since the off-diagonal elements of A are non-negative. It also implies that the local stability for individuals with underlying health conditions and for the whole system exists.

3.8 Global stability of the Endemic Equilibrium point (EEP)

The system (1)-(10) is globally asymptotically stable if the $R_o \geq 1$. We propose following Lyapunov Functions for system (1-5) for individuals without underlying health conditions.

$$k(S_n, E_n, I_n, T_n, R_n) = S_n - S_n^* - S_n^* \ln \frac{S_n}{S_n^*} + y_1 \left(E_n - E_n^* - E_n^* \ln \frac{E_n}{E_n^*} \right) + y_2 \left(I_n - I_n^* - I_n^* \ln \frac{I_n}{I_n^*} \right)$$

+ $y_3 \left(T_n - T_n^* - T_n^* \ln \frac{T_n}{T_n^*} \right) + y_4 \left(R_n - R_n^* - R_n^* \ln \frac{R_n}{R_n^*} \right)$ where y_1, y_2, y_3 and y_4 were positive constants to be determined. After solving we obtain the value of P and Q as follows;

$$P = \lambda_n^{**} S_n^{**} + \gamma_1 S_n^{**} + \mu S_n^{**} + \varepsilon_n R_n + \frac{S_n^{**}}{S_n} \varepsilon_n R_n^{**} + \frac{S_n^{**}}{S_n} \lambda_n S_n + \frac{S_n^{**}}{S_n} S_n + \frac{S_n^{**}}{S_n} \mu S_n + y_1 \lambda_n S_n + \frac{E_n^{**}}{E_n} y_1 \Delta_1 E_n + y_2 \delta_n E_n + \frac{I_n^{**}}{I_n} y_2 \Delta_2 I_n + y_3 \Omega_n I_n + y_3 \theta_n \sigma_n E_n + y_3 \sigma_n I_n + \frac{T_n^{**}}{T_n} y_3 \theta_n \sigma_n I_n + \frac{T_n^{**}}{T_n} y_3 \Delta_3 T_n + y_4 \xi_n T_n + \frac{R_n^{**}}{R_n} y_4 \Delta_7 R_n$$

$$Q = -\varepsilon_n R_n^{**} - \left(-\frac{\beta E_n(s)}{N} + y_1 \frac{\beta E_n(s)}{N} \right) - \left(-\frac{\beta \eta_1 I_n(s)}{N} + y_1 \frac{\beta \eta_1 I_n(s)}{N} \right) - \left(-\frac{\beta \eta_2 T_n(s)}{N} + y_1 \frac{\beta \eta_2 T_n(s)}{N} \right) - \gamma_1 S_n - \mu S_n - \frac{S_n^{**}}{S_n} \lambda_n^{**} S_n^{**} - \frac{S_n^{**}}{S_n} \gamma_1 S_n^{**} - \frac{S_n^{**}}{S_n} \mu S_n^{**} - \frac{S_n^{**}}{S_n} \varepsilon_n R_n - y_1 \Delta_1 E_n -$$

$$\begin{aligned} & \frac{E_n^{**}}{E_n} y_1 \lambda_n S_n - y_2 \Delta_2 I_n - \frac{I_n^{**}}{I_n} y_2 \delta_n E_n - y_3 \theta_n \sigma_n I_n - y_3 \Delta_3 T_n - \frac{T_n^{**}}{T_n} y_3 \Omega_n I_n - \frac{T_n^{**}}{T_n} y_3 \theta_n \sigma_n E_n - \\ & \frac{T_n^{**}}{T_n} y_3 \sigma_n I_n - y_4 \Delta_7 R_n - \frac{R_n^{**}}{R_n} y_4 \xi_n T_n \end{aligned} \quad (32)$$

Then $\frac{dk}{dt} = 0$ holds only when $S_n = S_n^{**}, E_n = E_n^{**}, I_n = I_n^{**}, T_n = T_n^{**}$ and $R_n = R_n^{**}$ so the maximal compact invariant set in $(S;E;T) \in \Pi: \frac{dV}{dt} = 0$ is the singleton E_* using Lassalet's invariance principle $\frac{dK(S,I,A,R)}{dt} < 0$ if and only if $P > Q$ (Mukandavire et al,2010). This result shows that COVID-19 would persist whenever $P > Q$ irrespective of the initial conditions and if $Q > P$ the disease will die out irrespective of initial conditions. The global stability for EEP exists for people without underlying health conditions, hence implying that the global stability for EEP for system (1-5) and (1-10) for the whole system exists.

3.9 Bifurcation analysis

For the sake of analysis, we are going to consider a special case of the model for the population without underlying health conditions. Centre Manifold Theorem was used to investigate the nature of the bifurcation of the model (Liu and Zhang, 2011). For simplicity of the remaining variables is made by letting; $S_n = y_1, E_n = y_2, I_n = y_3, T_n = y_4$ and $R_n = y_5$

$$N = y_1 + y_2 + y_3 + y_4 + y_5 \quad (33)$$

Further, by using vector notation, $y = (y_1 + y_2 + y_3 + y_4 + y_5)^T$ the COVID-19 model [1-5] was written in the form $\frac{dy}{dt} = F(y)$ with $F = (p_1, p_2, p_3, p_4, p_5)^T$ where the Jacobian of the system was evaluated at DFE point $E_*^0 = (S_{n*}^0, E_{n*}^0, I_{n*}^0, T_{n*}^0, R_{n*}^0) = (\frac{\theta\pi}{\mu}, 0, 0, 0, 0)$, denoted by $J(E_*^0)$.

We obtained

$$J(E_*^0) = \begin{pmatrix} -(\gamma_1 + \mu) & \beta^* & \beta^* \eta_1 & \beta^* \eta_2 & \varepsilon_n & 0 \\ 0 & \beta^* - \Delta_1 & \beta^* \eta_1 & \beta^* \eta_2 & 0 & 0 \\ 0 & \delta_n & -\Delta_2 & 0 & 0 & 0 \\ 0 & \theta_n \sigma_n & \Omega_n + (1 - \theta_n) \sigma_n & -\Delta_3 & 0 & 0 \\ 0 & 0 & 0 & \xi_n & -\Delta_7 & 0 \end{pmatrix} \quad (34)$$

The Jacobian of $\frac{dy}{dt} = F(y)$ at Disease Free Equilibrium point, with $\beta = \beta^*$, denoted by $J(E_*^0)$, has eigenvalues $(-(\gamma_1 + \mu), -\Delta_7)$ which are negative then the matrix reduces to;

$$\begin{bmatrix} \beta^* - \Delta_1 & \beta^* \eta_1 & \beta^* \eta_2 \\ \delta_n & -\Delta_2 & 0 \\ \theta_n \sigma_n & (1 - \theta_n) \sigma_n + \Omega_n & -\Delta_3 \end{bmatrix} \quad (35)$$

Eigenvectors of J_{β^*} for a case where $R_o^* = 1$, it can be shown that the Jacobian $[J E_{n*}^0]$ at $\beta = \beta^*$ denoted by J_{β^*} has a right eigenvector given by $u = [u_1, u_2, u_3, u_4, u_5]^T$ given by:

After solving the equations above, we obtained;

$$\begin{aligned}
 u_1 &= \frac{\beta^* u_2 + \beta^* \eta_1 u_3 + u_4 \beta^* \eta_2 + \varepsilon_n u_5}{k_1} > 0, & u_2 &= -\frac{(\beta^* \eta_1 u_3 + u_4 \beta^* \eta_2)}{(\beta^* - \Delta_1)} < 0, \\
 u_3 &= \frac{\delta_n u_2}{\Delta_2} > 0, & u_4 &= \frac{\theta_n \sigma_n u_2 + k_2 u_3}{\Delta_3} > 0, \\
 & & u_5 &= \frac{\xi_n u_4}{\Delta_7} > 0
 \end{aligned}$$

The Jacobian matrix has a left eigenvector denoted by $v = (v_1, v_2, v_3, v_4, v_5)^T$
After solving the equations above we obtained;

$$\begin{aligned}
 v_1 = v_5 &= 0, & v_2 &= -\frac{(\delta_n v_3 + \theta_n \sigma_n v_4)}{(\beta^* - \Delta_1)} < 0 \\
 v_3 &= \frac{\beta^* \eta_1 v_2 + k_2 v_4}{\Delta_2} > 0, & v_4 &= \frac{\beta^* \eta_2 v_2}{\Delta_3} > 0
 \end{aligned}$$

The value of a is obtained as;

$$a = v_2 \beta (2u_1 u_2 + 2\eta_1 u_1 u_3 + 2\eta_2 u_1 u_4) < 0.$$

To find the value of b as per the above theorem we have, Since v_1, u_2, u_3 and u_4 are greater than zero, it follows that;

$$b = v_2 S^0 (u_2 + \eta_1 u_3 + \eta_2 u_4) > 0$$

Therefore, $a < 0$ and $b > 0$, when $\beta^* < 0$ with $|\beta^*| \ll 1$, $(0,0)$ is unstable and there exists a negative and locally asymptotically stable equilibrium; when $0 < \beta^* \ll 1$, $(0,0)$ is stable and there exists a positive unstable equilibrium. Absence of backward bifurcation show that it is possible to eradicate COVID-19 disease whenever $R_0^* < 1$.

4.0 NUMERICAL SIMULATION

MATLABR2017b was used in numerical simulation do demonstrate the dynamical behavior of the non-linear ordinary differential equations in the system (1)- (10). Simulations of the system were carried out using initial conditions and parameter values in the table below which shows a summary of data, variables and parameter available in Literature and graphically presented.

Table 2: Parameter values for numerical simulation

Parameters	Value	Citation
π	9.8	Kimathi et al(2022)
B	3.11×10^{-8}	Assumed
μ	5.09×10^{-3}	Kenya demographic profile
ξ_n	0.12 per day	WHO, Wang & Yang(2021)
ξ_u	0.08 per day	WHO, Wang & Yang(2021)
Ω_n	0.03	WHO, Wang & Yang(2021)
Ω_u	0.18	Wang & Yang(2021)
δ_n	0.0033	Titus et.al 2020
δ_u	1/7	Wang & Yang(2021)
α_n	0.0012 per day	Wang & Yang(2021)
α_u	0.0144 per day	Wang & Yang(2021)
σ_n	0.5	Rachel et al (2021)
σ_u	0.52	Assumed
ε_n	0.0167	Dwomoh et al 2021
ε_u	0.59	Assumed
θ	0.49	Assumed
θ_n	0.002	Assumed

θ_u	0.012	Assumed
γ_1	0.2	Okyere et al (2022)
γ_2	0.80	Wangari et al (2021)
γ_3	0.3	Kouidere et al (2021)
γ_4	0.20	Wangari et al (2021)
γ_5	0.069	Assumed
η_1	0.49	Rachel et al (2021)
η_2	0.4605	Rachel et al (2021)
η_3	0.009	Rachel et al (2021)

4.1 Simulation graphs for both individuals with and without underlying health conditions

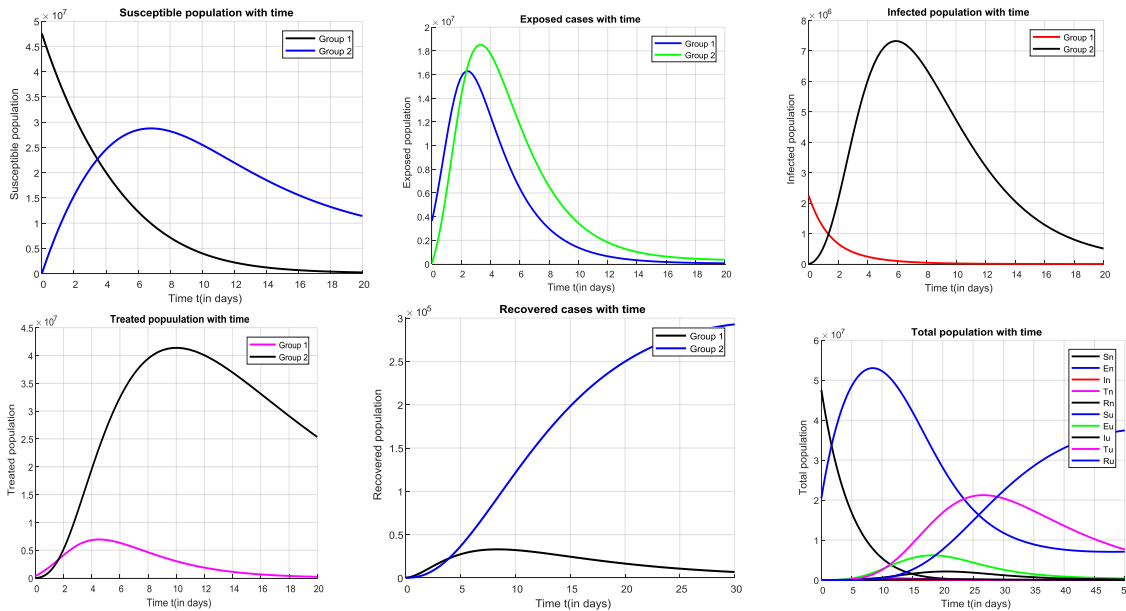


Figure 3: Simulation graphs for individuals with and without underlying health condition

4.2 Effect of Screening the Exposed population for those with and without underlying health conditions.

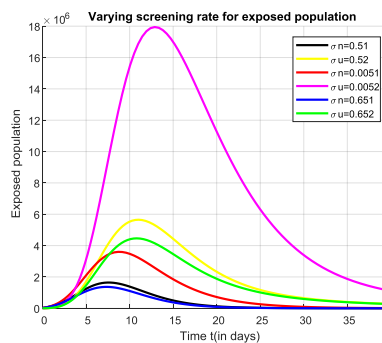


Fig. 4 Exposed population Screening rate was varied from $\sigma_n = 0.0051$ to $\sigma_n = 0.651$ group 1 while for group 2 it was varied from $\sigma_u = 0.0052$ to $\sigma_u = 0.652$ and the other parameters were held constant. From fig. 4 reducing the rate of screening it increases the number of the exposed people hence increasing the number treated individuals for both group 1 and group 2 hence reducing the number of the susceptible population being exposed and infected. Also when the rate of screening is increased the number of the exposed people reduces hence reducing the number of the infected treated individuals. From fig. 2 the rate of exposure is higher for population with underlying health conditions as compared to those without.

4.3 Effects of Screening for Infected population with or without underlying health conditions.

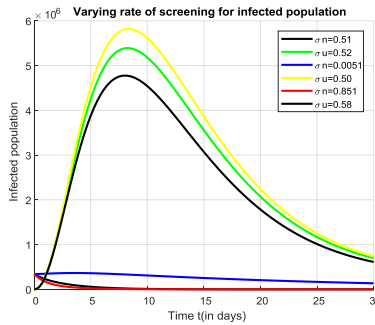


Fig. 5 Infected population Screening rate for the infected population was varied from $\sigma_n = 0.0051$ to $\sigma_n = 0.851$ for group 1 and $\sigma_u = 0.50$ to $\sigma_u = 0.58$ for group 2 while the other parameters were held constant. The result shows that if the rate of screening is reduced on the infected population it increases the number of the infected persons hence increasing the treated population. While if the screening rate of the infected individual is increased it reduces the number of the infected individuals hence reducing the number of the treated population. The rate infectious was higher for group 1 as compared to group 2.

4.4 Effects of treatment rate for exposed and infected populations screened for both group 1 and group 2.

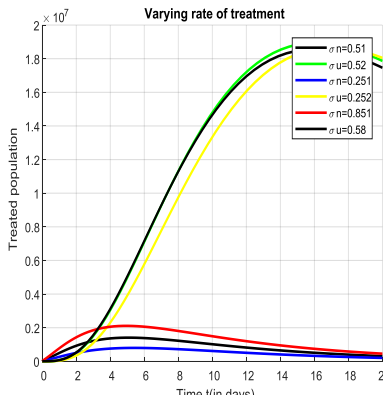


Fig. 6 Treated population The treatment rate was varied from $\sigma_n = 0.0051$ to $\sigma_n = 0.851$ and $\sigma_u = 0.52$ to $\sigma_u = 0.58$ while all the other parameters were held constant. The results in fig. 6 shows that if the treatment rate is increased it increases the number of the treated population for the exposed and infected population screened. Also we can conclude that if treatment rate is reduced the number of the treated population for the exposed and infected classes screened reduces.

4.5 Sensitivity Analysis of the model

Sensitivity analysis was done and the technique employed is based on the reproduction number. Sensitivity index of the model parameter is given by the relation;

$$S_X^{R_n} = \frac{\partial R_n}{\partial X} * \frac{X}{R_n}$$

Sensitivity analysis for the whole model

Table 3: Sensitivity analysis for the whole model

Symbols	Sensitivity index for both group 1 and group 2
B	1
R_0	2.063014395488956
γ_2	-0.2598140616507
θ_n	-0.00143981600292
σ_n	-0.00216800555539
Ω_n	-0.00004369137314
α_n	-0.000005706747435
γ_3	0.0000210433217498
μ	-0.05525356159285
ξ_n	-0.00181788839435
γ_4	-0.00161146558593
δ_n	-0.000020760629324
δ_u	0.02028562046267790
θ_u	-0.0167187823640163
σ_u	-0.8062435596375214
Ω_u	-0.0794581175610974
α_u	-0.0137696019461304
ξ_u	-0.3600793474296689
η_1	0.000804944575963319
η_2	0.00347353036743719
η_3	0.02445896527941908
η_4	0.3230496508014381
η_5	0.3829893959098814
κ	0.3695858996240922

The parameters with negative partial derivative of the reproduction number $\mu, \theta_u, \sigma_u, \Omega_u, \alpha_u, \xi_u, \gamma_4, \xi_n, \alpha_n, \Omega_n, \delta_n, \theta_n, \sigma_n$ and γ_2 means that if the values are increased, may greatly reduce COVID-19 in the community, while the other parameters are not changed. The parameters reduce the reproduction number which indicates that COVID-19 is reduced in the community. The positive partial derivatives of the reproduction number (with respect to $\beta, \eta_2, \eta_1, \gamma_3, \eta_3, \eta_4, \eta_5, \delta_u$ and κ) shows that an increase of these parameters increases the reproduction number hence increasing the risk of COVID-19 in the community. The sensitivity analysis of the model for individuals without underlying conditions exist indicating that the sensitivity analysis for the whole model and for the individuals with underlying health conditions also exist.

5.0 CONCLUSION.

This study sought to formulate a deterministic model on the impact of screening and treatment on the control of COVID-19 dynamics incorporating effects of underlying health conditions. The study considered the general SIS model which was modified to incorporate the exposed population, treated population and the recovered population. Our study incorporated the findings of study by

(Yang et al,2021) which had divided the population in two categories. This objective was achieved in the section 2.1 whereby the system of the first order nonlinear differential equation describing the dynamics of COVID-19 were deduced from the flow chart.

In this study, the behavior of the deterministic model was analyzed by obtaining the positivity and boundedness of the solution, equilibrium points, basic reproduction number was studied by next generation matrix method, local stability of the DFE was obtained by Routh-Hurwitz criteria for stability. Global stability was also obtained by Castillo-Chavez method, global stability of the EEP was carried out by a Lyapunov function and Bifurcation analyses was also obtained. From the analysis we found that, the model was bounded and lies in the positive region, DFE existed in absence of the disease, the endemic Equilibrium point exist when the $R_0^n > 1$, local stability of the DFE is locally asymptotically stable whenever $R_0^* < 1$ and unstable if $R_0^* > 1$. Global stability of EEP is asymptotically stable when $R_0^n > 1$.

Further analysis was carried out using the reduced system of the model (1) -(5) whereby global stability of the Disease Free Equilibrium is asymptotically stable whenever $R_0^* < 1$ and absence of backward bifurcation show that it is possible to eradicate COVID-19 disease. To simulate the model the numerical value of the reproduction number was obtained to be 3.1273739895. Every effort should be made to lower this value of the reproduction number to less than one. Also sensitivity was for the system of the model was performed based on the reproduction number. It was observed that increased these parameters $\mu, \theta_u, \sigma_u, \Omega_u, \alpha_u, \xi_u, \gamma_4, \xi_n, \alpha_n, \Omega_n, \delta_n, \theta_n, \sigma_n$ and γ_2 will reduce the burden of the disease in the community for they greatly reduce the reproduction number to less than one. Also increasing these parameters $\beta, \eta_2, \eta_1\gamma_3, \eta_3, \eta_4, \eta_5, \delta_u$ and κ increases the reproduction number hence increasing the risk of COVID-19 in the community. Our simulations confirm the high risk of individuals with underlying health conditions. The sensitivity analysis for those with underlying health conditions have more impact on COVID-19 transmission. Finally, we have performed numerical simulation outlining the population of the treated and the screened individuals. The figures on treated population show that if we increase the rate of treatment the treated population widens. Also if we increase the rate of screening on the exposed and infected persons it reduces the number of the infected and exposed population hence decreasing the number of the treated population. Therefore, screening and treatment have a greater impact in reducing the transmission of COVID-19. This study shows that the best alternative ways to curb the spread of COVID-19 is by reducing the contact rate between the susceptible and the exposed individuals and improving the treatment rate of COVID-19 virus. Particularly, we find that reducing the between-group contact is effective in protecting the vulnerable group against the COVID-19 infection, and this control strategy seems to be productive in bringing down the numbers of infections and hospitalizations for the group with chronic conditions.

AVAILABILITY OF DATA

The secondary data used in the study is obtained from the literature reviewed and the daily updates of COVID-19 from the Ministry of Health in Kenya.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS CONTRIBUTIONS

The correspondence author wrote the manuscript and the supervisors assisted in developing the matlab codes, proof reading and correcting the work, offering guidance and guidelines.

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