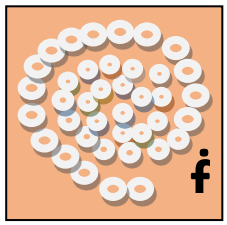

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Mathematical model for controllability of covid-19 investigating the impact of protective measures, for optimal solution to lower the disease burden in Nigeria

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Abstract

This work presents a non–linear deterministic mathematical model, analyzed to study the controllability of Covid-19 incorporating all-round protection, hospitalization, treatment and early burial of death due to covid-19 measures. We introduce a protection measure(A), that will cooperate with all government guidelines and observe personal hygiene, early burial of covid-19 death to extend B.I. Omede 2022 work. The existence of the disease-free equilibrium (DFE) state was established which shows the state in which there is no covid-19 infection for stability. The effective reproduction number and the local stability of the DFE were computed using the next generation and linearization approach respectively. The sensitivity analysis shows that the parameter with the highest positive sensitivity index is *a* proportion of susceptible ones that are not protecting themselves from covid-19 infection. The value, +1.014, of the sensitivity index of *a* shows that personal protection and other forms of protection, play important role in the control of spread of the disease. From numerical simulations, it was observed that the introduction of protective parameters leads to significant impact on the reduction of the spread of the disease. Moreso, it was discovered that effective implementation of protective measures without relaxing it will help in reducing the spread of the disease.

Keywords and phrases: early burial; Covid 19; reproduction number; model-fitting; protective measures

1 Introduction

The COVID-19 pandemic, first reported in Wuhan, China, in December 2019, rapidly escalated globally, with over 196 million confirmed cases and 4.2 million deaths by July 2021 [1]. Nigeria, among 210 affected countries, recorded its first case on February 27, 2020 a 44-year-old Italian citizen returning from Milan [16]. By May 2020, Nigeria reported 2,558 cases, 87 deaths (3.4% case fatality

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rate, CFR), and significant demographic trends: 69% male patients, 23% aged 21–30 years, and 15.2% regional CFR variation [3]. As of April 2024, Nigeria's cumulative totals reached 267,188 cases, 3,155 deaths, and 259,953 recoveries, though global tracking ceased due to inconsistent reporting [16]. Common symptoms included fever, dry cough, and breathing difficulties, with less frequent reports of muscle pain, diarrhea, and sore throat [1].

Transmission evolved from travel-related importation to community spread, including asymptomatic carriers [2], complicating containment efforts. Coronaviruses, known for causing illnesses from common colds to severe syndromes like SARS and MERS, were identified as the cause of COVID-19, declared a pandemic by the WHO in March 2020 [1]. Nigeria, flagged as high-risk by the WHO due to travel links with China [1], activated its highest public health emergency (Level 3) following the index case [3].

The Nigeria Centre for Disease Control (NCDC) spearheaded responses, deploying Rapid Response Teams, contact tracing, and designating COVID-19 treatment facilities [3]. Despite these measures, challenges emerged: public skepticism toward government motives, misinformation, and low adherence to protocols like masking and distancing [3]. Weak healthcare infrastructure, urban overcrowding, and poverty further strained responses, prompting calls for context-specific adaptations by global health experts like Dr. Wafaa El-Sadr [4].

Mathematical models played a critical role in predicting outbreak trajectories and evaluating intervention efficacy [4]. While unable to provide precise long-term forecasts, models helped estimate short-term healthcare demands (e.g., ICU beds, ventilators) and informed policy decisions on lockdowns and reopening timelines [4]. Early models in China suggested peak transmission by late January 2020 [5], but Nigeria's heterogeneous data highlighted regional disparities in CFR (0.2%–15%) and resource allocation [3]. Models emphasized prioritizing interventions (e.g., testing, isolation) over speculative death tolls [4], underscoring the importance of adaptive, evidence-based policies. Key challenges included reconciling public compliance with epidemiological guidelines [3], addressing healthcare inequities, and mitigating economic impacts. Nigeria's young population (median age 18) initially suggested lower vulnerability, yet comorbidities and limited healthcare access exacerbated risks [3]. The NCDC's daily updates aimed to enhance transparency, but persistent case growth reflected systemic gaps in enforcement and education [3].

Nigeria's COVID-19 experience underscores the interplay of global health governance [1], localized responses [16], and socioeconomic realities. While mathematical models provided strategic insights [5,4,2], their limitations highlighted the need for flexible, equity-focused policies. Strengthening healthcare systems, combating misinformation [16], and fostering community trust remain critical for future pandemic preparedness, particularly in low-resource settings. The pandemic's legacy emphasizes the value of integrating scientific rigor with contextual adaptability to mitigate health crises effectively [7].

2 Methodology/Model formulation

We present a deterministic compartmental model [7] which describes the transmission dynamics of covid-19 in the presence of some control measures. The model is a system of nine (9) nonlinear ordinary differential equations, which captures the dynamics of the disease in Nigeria. In this model, the human population is divided into nine (9) compartments namely:

Susceptible (S): This is a class of individuals who are prone to contract covid-19 disease. Since covid-19 is an epidemic that has affected every part of Nigeria, we assume that the entire population of Nigeria is susceptible to covid-19 infection.

Protection class (A): This is the class is a fraction of susceptible who employs several available protection measures against covid-19.
Exposed class (E): This is the class of people who have contracted covid-19 but have not shown symptoms of the disease.
Asymptomatically Infectious (I_{as}): This is the fraction of the exposed class who are not showing symptoms even after the incubation period has elapsed.
Symptomatically Infectious (I_s): This is the fraction of the exposed class who are showing symptoms at the expiration of the incubation period of the disease.
Hospitalized (H): The class contains a fraction of asymptomatically and symptomatically infectious classes who are isolated and are receiving treatments in isolation centers.
Quarantine class (Q): This is the class of symptomatically infectious humans who self-isolates themselves.
Dead (D): This comprises the fractions of the infectious classes who die due to the disease and the rate burial of the infected dead are done early to avoid re-infection
Recoveries(R): This contains fractions of the infected classes who have recovered from covid-19 infection naturally or after being treated.

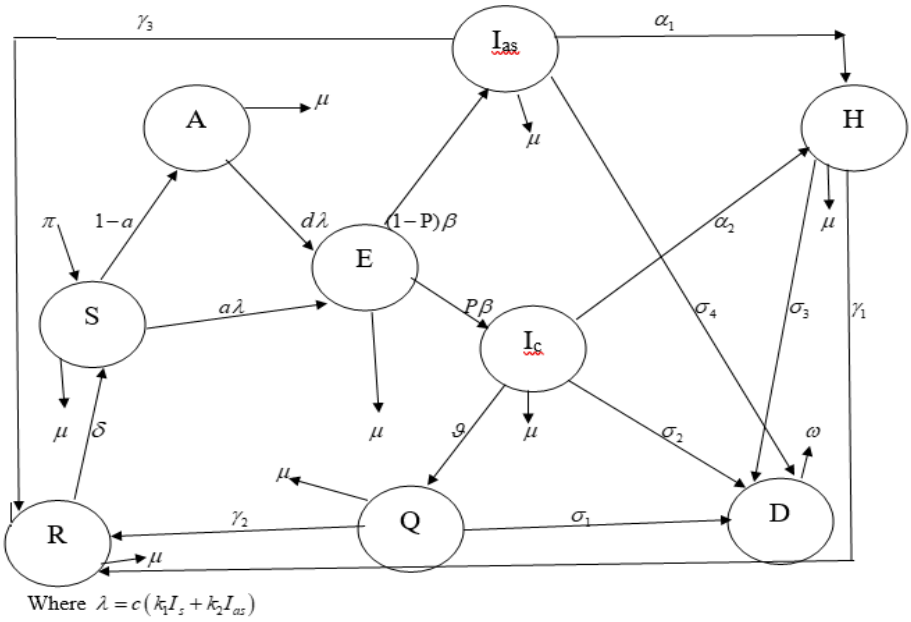
Hence, the total population N is given by $N = S + A + E + I_{as} + I_s + H + Q + D + R$. The recruitment of humans into the susceptible class is through migration into the country from foreign countries, and through birth of young ones. Here, the recruitment is put at the rate π. It is assumed that a fraction a of the susceptible contracts covid-19 through contact with asymptomatically and symptomatically infectious humans at the rate $ac(\varkappa_1 I_s + \varkappa_2 I_{as})$, where c is the rate of contact with the infectious classes, $\varkappa_1$ and $\varkappa_1$ are the probabilities a contact with I_s and I_{as}, respectively will result to infections. The remaining fraction (1-a) of the susceptible are assumed to protect themselves from being infected by the disease. However, the proportion d of this class is assumed to relax their protection and become infected with the disease at the rate $dc^0(\varkappa_1 I_s + \varkappa_2 I_{as})$, where c^0 is the rate at which the proportion d have contacts with the infectious classes. The exposed class becomes either symptomatically infectious or asymptomatically infectious in the proportions p and $(1-p)$, respectively, at the incubation rate β. The asymptomatically infectious and symptomatically classes are assumed to be hospitalized at the rates a_1 and a_2, respectively, whereas the proportion q of the symptomatically infectious class self-isolates themselves. The disease-induced death for the classes $I_{as}, I_s, H,$ and Q are put at σ_4, σ_2, and σ_3, and σ_1 respectively. The hospitalized class, the self-isolation class and the asymptomatically infectious class recover from the the disease at the rates γ_1, γ_2, and γ_3, respectively, while the natural death rate for all the classes is μ and ω is the rate infected dead burial are conducted early to avoid contacts and re-infection. However, since covid-19 is not known to confer life-long immunity after infection, we assume that the proportion δ, of recovery class becomes susceptible to the disease after recovery. Therefore, the rate of change of each class is given by the system of ordinary differential equations.

We present a deterministic compartmental model which describes the transmission dynamics of covid-19 in the presence of protective measures. The model is a system of nine (9) nonlinear ordinary differential equations, which captures the dynamics of the disease in Nigeria. In this model, the human population is divided into nine (9) compartments namely: Susceptible (S), Protection class (A), Exposed class (E), Symptomatically Infectious (I_s), Hospitalized (H), Quarantine class (Q), Dead (D), Recoveries(R) and Asymptomatically Infectious (I_{as})

Table 1: Description of the Parameter used in this model

Parameter	***Description***
π	rate of recruitment into the susceptible class
a	proportion of the susceptible class that are protected
c	rate of contact between infected class and those that are not protected
d	proportion of protected class that lose their protection
c_0	rate of contact between infected class and those that are protected
$\varkappa_1$	probability that a contact with symptomatomatically infectious persons will result to infection
$\varkappa_2$	probability that a contact with asymptomatomatically infectious persons will result to infection
a_1	the rate at which the asymptomatically infectious persons are hospitalized and treated
a_2	the rate at which the symptomatically infectious persons are hospitalized and treated
σ_1	death rate for self-isolated persons
σ_2	death rate for symptomatically infected persons
σ_3	death rate for hospitalized persons
σ_4	death rate for asymptomatically infected persons
ρ	proportion of exposed class that becomes symptomatically infectious
β	incubation rate of corona virus
γ_1	rate of recovery for hospitalized persons
γ_2	rate of recovery for self-isolated persons
γ_3	rate of recovery for asymptomatically infected persons
q	the proportion of symptomatically infectious humans that self-isolate
δ	proportion of recovered class that becomes susceptible
μ	human natural death rate
ω	The rate of early burials of death due to covid-19 to avoid re-infection

Figure 1: A compartmentalized diagram showing the covid-19 model with control measure



The System equation of the model

$$\frac{dS(t)}{dt} = \pi + \delta R - ac(k_1 I_s + k_2 I_{as})S - (1-a)S - \mu S$$

$$\frac{dA(t)}{dt} = (1-a)S(t) - cd(k_1 I_s + k_2 I_{as})A - \mu A$$

$$\frac{dE(t)}{dt} = ac(k_1 I_s + k_2 I_{as})S + cd(k_1 I_s + k_2 I_{as})\,\mathrm{A} - (\beta + \mu)\,\mathrm{E(t)}$$

$$\frac{dI_{as}(t)}{dt} = (1-p)\beta E - (\alpha_1 + \sigma_4 + \gamma_3 + \mu)I_{as}(t)$$

$$\frac{dI_s(t)}{dt} = p\beta E - (\alpha_2 + \sigma_2 + q + \mu)I_s(t) \qquad (1)$$

$$\frac{dH(t)}{dt} = \alpha_1 I_{as} + \alpha_2 I_s - (\sigma_3 + \gamma_1 + \mu)H(t)$$

$$\frac{dQ(t)}{dt} = qI_s - (\sigma_1 + \gamma_2 + \mu)Q(t)$$

$$\frac{dD(t)}{dt} = \sigma_1 Q + \sigma_2 I_s + \sigma_3 H + \sigma_4 I_{as} - \omega D(\mathrm{t})$$

$$\frac{dR_h(t)}{dt} = \gamma_1 H + \gamma_2 Q + \gamma I_{as} - (\delta + \mu)R_h(t)$$

2.1 Disease-free equilibrium

The equilibrium points of the model system are the steady state solutions obtained when the right hand of the system (1) is to zero. That is the solution to the equations
Disease-free equilibrium points of the model are the solutions to (1) when equated to (0) obtained when there is no disease in the population. That is the solution obtained when
$A = E = I_{as} = I_s = H = Q = D = 0$.

With this definition, the components of the disease-free equilibrium point corresponding to the classes where there can be disease infections are zero. Let E_0 denote the disease-free equilibrium of the model, with this definition, the components of the disease-free equilibrium point corresponding to the classes where there can be disease infections are zero. Let E_0 denote the disease-free equilibrium of the model, then we have

$$E_0 = (S^*, \mathrm{A}^*, \mathrm{E}^*, \mathrm{I}^*_{\mathrm{as}}, \mathrm{I}^*_{\mathrm{s}}, H^*, \mathrm{Q}^*, \mathrm{D}^*, \mathrm{R}^*) = \left(\frac{\pi}{1-a+\mu}, \frac{(1-a)\pi}{\mu(1+\mu-a)}, 0,0,0,0,0,0,0 \right). \quad (2)$$

The disease-free equilibrium is necessary in the determination of the basic reproduction number of the disease.

Using the next generation matrix, we got the reproduction number as;

$$\frac{dE(t)}{dt} = ac(k_1 I_s + k_2 I_{as})S + cd(k_1 I_s + k_2 I_{as})A - (\beta+\mu)E(t),$$

$$\frac{dI_{as}(t)}{dt} = (1-p)\beta E - (\alpha_1 + \sigma_4 + \gamma_3 + \mu)I_{as}(t),$$

$$\frac{dI_s(t)}{dt} p\beta E - (\alpha_2 + \sigma_2 + q + \mu)I_s(t), \quad (3)$$

$$\frac{dH(t)}{dt} = \alpha_1 I_{as} + \alpha_2 I_s - (\sigma_3 + \gamma_1 + \mu)H(t),$$

$$\frac{dQ(t)}{dt} = qI_s - (\sigma_1 + \gamma_2 + \mu)Q(t),$$

From the subsystem, we have

$$F = \begin{pmatrix} ac(k_1 I_s + k_2 I_{as})S + cd(k_1 I_s + k_2 I_{as})A \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, V = \begin{pmatrix} (\beta+\mu)E \\ -(1-p)\beta E + (\alpha_1 + \sigma_4 + \gamma_3 + \mu)I_{as} \\ -p\beta + (\alpha_2 + \sigma_2 + q + \mu)I_s \\ -\alpha_1 I_{as} - \alpha_2 I_s + (\sigma_3 + \gamma_1 + \mu)H \\ -qI_s + (\sigma_1 + \gamma_2 + \mu)Q(t) \end{pmatrix}$$

Then, F is the Jacobian matrix of F evaluated at the disease-free equilibrium and V is the Jacobian matrix of V evaluated at the disease-free equilibrium. Hence, we have

$$F = \begin{pmatrix} 0 & \frac{ac\kappa_2\pi}{(1-a+\mu)} + \frac{dc\kappa_2\pi(1-a)}{\mu(1+\mu-a)} & \frac{ac\kappa_1\pi}{(1-a+\mu)} + \frac{dc\kappa_1\pi(1-a)}{\mu(1+\mu-a)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \beta+\mu & 0 & 0 & 0 & 0 \\ -(1-p)\beta & \alpha_1+\sigma_4+\gamma_3+\mu & 0 & 0 & 0 \\ -p\beta & 0 & \alpha_2+\sigma_2+q+\mu & 0 & 0 \\ 0 & -\alpha_1 & -\alpha_2 & \sigma_3+\gamma_1+\mu & 0 \\ 0 & 0 & -q & 0 & \sigma_1+\gamma_2+\mu \end{pmatrix}$$

The inverse of V is non-negative, and is given by

$$V^{-1} = \begin{pmatrix} \frac{1}{\beta+\mu} & 0 & 0 & 0 & 0 \\ \frac{(1-p)\beta}{(\beta+\mu)(\alpha_1+\sigma_4+\gamma_3+\mu)} & \frac{1}{(\alpha_1+\sigma_4+\gamma_3+\mu)} & 0 & 0 & 0 \\ \frac{(\alpha_1(1-p)\beta(\alpha_2+\sigma_2+q+\mu)+p\beta\alpha_2(\alpha_1+\sigma_4+\gamma_3+\mu)}{(\beta+\mu)(\alpha_1+\sigma_4+\gamma_3+\mu)(\alpha_2+\sigma_2+q+\mu)(\sigma_3+\gamma_1+\mu)} & \frac{\alpha_1}{(\alpha_1+\sigma_4+\gamma_3+\mu)(\sigma_3+\gamma_1+\mu)} & \frac{\alpha_2}{(\alpha_2+\sigma_2+q+\mu)(\sigma_3+\gamma_1+\mu)} & \frac{1}{(\sigma_3+\gamma_1+\mu)} & 0 \\ \frac{pq\beta}{(\beta+\mu)(\alpha_2+\sigma_2+q+\mu)(\sigma_1+\gamma_2+\mu)} & 0 & \frac{q}{(\alpha_2+\sigma_2+q+\mu)(\sigma_1+\gamma_2+\mu)} & 0 & \frac{1}{\sigma_1+\gamma_2+\mu} \end{pmatrix}$$

The next generation matrix is therefore given by

$$K = FV^{-1} = \begin{pmatrix} \frac{ack_2\pi(1-p)\beta}{(1-a+\mu)(\beta+\mu)(\alpha_1+\sigma_4+\gamma_3+\mu)} + \frac{ack_1\pi p\beta}{(1-a+\mu)(\beta+\mu)(\alpha_2+\sigma_2+q+\mu)} & \frac{ac\pi}{(1-a+\mu)(\alpha_1+\sigma_4+\gamma_3+\mu)} & \frac{ac\pi}{(1-a+\mu)(\alpha_2+\sigma_2+q+\mu)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

The egienvalues of K are 0,0,0,0 and

$\frac{(1-p)\beta acK_2\pi}{(1-a+\mu)(\beta+\mu)(\alpha_1+\sigma_4+\gamma_3+\mu)} + \frac{p\beta acK_1\pi}{(1-a+\mu)(\beta+\mu)(\alpha_2+\sigma_2+q+\mu)}$. Therefore, the basic reproduction number of the disease is

$R_0 = \frac{(1-p)\beta acK_2\pi}{(1-a+\mu)(\beta+d)(\alpha_1+\sigma_4+\gamma_3+\mu)} + \frac{p\beta acK_1\pi}{(1-a+\mu)(\beta+\mu)(\alpha_2+\sigma_2+q+\mu)}$. The basic reproduction number can be written in the form

$R_0 = R_{0a} + R_{0s}$

where $R_{0a} := \frac{(1-p)\beta acK_2\pi}{(1-a+\mu)(\beta+\mu)(\alpha_1+\sigma_4+\gamma_3+\mu)}$ is the average number of people that would be asymptomatically infected by a single infectious individual, where

$R_{0s} := \frac{p\beta acK_1\pi}{(1-a+\mu)(\beta+\mu)(\alpha_2+\sigma_2+q+\mu)}$ is the average number of people that would be asymptomatically infected by single infectious individuals.

3 Analysis of the model

The stability or otherwise of the disease-free equilibrium point, E_0 is determined by the sign of the real part of all the eigenvalues of the Jacobian matrix of $f(X)$ evaluated at E_0. Let $J(E_0)$ be the Jacobian matrix of $f(X(t))$ evaluated at E_0. If all the eigenvalues of $J(E_0)$ have negative real parts, then E_0 is unstable. Therefore, we need to show that all the eigenvalues of $J(E_0)$ have negative real part. First we write $f(X(t))$ in the form

$$f(X(t)) = \begin{pmatrix} f_1 \\ f_2 \\ f_3 \\ f_4 \\ f_5 \\ f_6 \\ f_7 \\ f_8 \\ f_9 \end{pmatrix} = \begin{pmatrix} \pi + \delta R - ac(k_1 I_s + k_2 I_{as})S - (1-a)S - \mu S \\ (1-a)S - cd(k_1 I_s + k_2 I_{as})A - \mu A \\ ac(\mathrm{k}_1 I_s + k_2 I_{as})S + cd(k_1 I_s + k_2 I_{as})A - (\beta + \mu)E \\ (1-p)\beta E - (\alpha_1 + \sigma_4 + \gamma_3 + \mu)I_{as} \\ P\beta - (\alpha_2 + \sigma_2 + q + \mu)I_s \\ \alpha_1 I_{as} + \alpha_2 I_s - (\sigma_3 + \gamma_1 + \mu)H \\ qI_s - (\sigma_1 + \gamma_2 + \mu)Q \\ \sigma_1 Q + \sigma_2 I_s + \sigma_3 H + \sigma_4 I_{as} - \omega D(t) \\ \gamma_1 H + \gamma_2 Q + \gamma_3 I_{as} - (\delta + \mu)R \end{pmatrix}$$

The Jacobian matrix of $f(X)$ valuated at the disease-free equilibrium point E_0 is

$$\mathrm{J}(E_o) = \begin{pmatrix} (-d+1-a) & 0 & 0 & -\mathrm{ack}_2 S^* & -\mathrm{ack}_1 S^* & 0 & 0 & 0 & \delta \\ 1-a & -d & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\beta+d) & ack_2 S^* & ack_1 S^* & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-p)\beta & -G_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & p\beta & 0 & -G_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_1 & \alpha_2 & -G_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & q & 0 & -G_4 & 0 & 0 \\ 0 & 0 & 0 & \sigma_4 & \sigma_2 & \sigma_3 & \sigma_1 & 0 & 0 \\ 0 & 0 & 0 & \gamma_3 & 0 & \gamma_1 & \gamma_2 & 0 & -(\delta+\mu) \end{pmatrix}$$

where $G_1 = \alpha_1 + \sigma_4 + \gamma_3 + \mu$, $G_2 = \alpha_2 + \sigma_2 + q + \mu$, $\sigma_3 + \gamma_1 + \mu$, $G_4 = \sigma_1 + \gamma_2 + \mu$. The eigenvalues of $J(E_0)$ are $0, -\mu, -(\mu + 1 - a), -(\delta + \mu)$ and the eigenvalues of the sub-matrix

$$J_s(E_0)=\begin{pmatrix} -(\beta+d) & ack_2S^* & ack_1S^* & 0 & 0 \\ (1-p)\beta & -(\alpha_1+\sigma_4+\gamma_3+\mu) & 0 & 0 & 0 \\ p\beta & 0 & -(\alpha_2+\sigma_2+q+\mu) & 0 & 0 \\ 0 & \alpha_1 & \alpha_2 & -(\sigma_3+\gamma_1+\mu) & 0 \\ 0 & 0 & q & 0 & -(\sigma_1+\gamma_2+\mu) \end{pmatrix}$$

At this stage, notice that the matrices F,V and $J_s(E_0)$ are related by $F-V=J_s(E_0)$. In Driesseche and Watmough (2002). It is shown that all the eigenvalues of eigenvalues of the matrix F-V or equivalently, $J_s(\mathrm{E}_0)$ are all negative or have negative real parts if $p(FV^{-1})<1$. This shows that the disease -free equilibrium E_0 is locally asymptotically stable if $R_0<1,$ and unstable if $R_0>1$. Epidemiologically, this means that the disease can be eradicated in the population when, if the initial sizes of the compartments are in the basin of attraction of the disease –free equilibrium.

3.1 Global stability of the disease-free equilibrium

In the last subsection, we proved that the disease-free equilibrium E_0. Is locally asymptotically stable when the control reproduction number $\mathrm{R_e}<1$. The aim of this subsection is to determine the stability or otherwise of the disease-free equilibrium E_0 in the global sense. That is if the solution to the system of equations tends to the disease-free equilibrium

$$\frac{dX_1(t)}{dt}=F_1(X_1,X_2) \qquad 4$$

$$\frac{dX_2(t)}{dt}=F_2(X_1,X_2),F2(X_1,0)=0 \qquad 5$$

where the first equation represents the system for the uninfected compartments, while the second equation represents the system for the infected compartment in this case, $X_1(t)=(S(t),\mathrm{A(t)}),R(t)$ and $X_2(t)=(E(t),I_{as}(t),I_s(t),H(t),Q(t),D(t)$. Also, the right-hand sides are given by

$$F_1(X_1,X_2)=\begin{pmatrix} \pi+\delta R-ac(K_1I_s+K_2I_{as})\mathrm{S}-(1-\mathrm{a})\mathrm{S}-\mu\mathrm{S} \\ (1-\mathrm{a})\mathrm{S(t)}-\mathrm{cd}(\mathrm{K}_1I_s+K_2I_{as})A-\mu A \\ \gamma_1H+\gamma_2Q+\gamma_3I_{as}-(\delta+\mu)R_h(t) \end{pmatrix}$$

$$F_2(X_1, X_2) = \begin{pmatrix} ac(K_1 I_s + K_2 I_{as})S + cd(K_1 I_s + K_2 I_{as})A - (\beta + \mu)E(t) \\ (1-P)\beta E - (\alpha_1 + \sigma_4 + \gamma_3 + \mu)I_{as}(t) \\ \mathrm{p}\,\beta\,\mathrm{E} - (\alpha_2 + \sigma_2 + q + \mu)I_s(t) \\ \alpha_1 I_{as} + \alpha_2 I_s - (\sigma_3 + \gamma_1 + \mu)H(t) \\ \mathrm{qI_s} - (\sigma_1 + \gamma_2 + \mu)Q(t) \\ \sigma_1 Q + \sigma_2 I_s + \sigma_3 H + \sigma_4 I_{as} - \omega D(t) \end{pmatrix}$$

The two conditions that must be satisfied to guarantee global stability of the disease-free equilibrium are

i. In the system $\frac{dX_1(t)}{dt} = F_1(X_1, 0)$, the disease-free equilibrium $\left(\frac{\pi}{1+\mu-a}, \frac{\pi(1-a)}{\mu(1+\mu-a)}, 0\right)$ must be globally asymptotically stable. That is $\left(S(t), A(t), R(t)\right) \to \left(\frac{\pi}{1+\mu-a}, \frac{\pi(1-a)}{\mu(1+\mu-a)}, 0\right) as\, t \to \infty$.

ii. In the system $\frac{dX_2}{dt} = F_2(X_1, X_2)$, we must have $BX_2 - F_2\left(X_1, X_2\right) \geq 0$, where B is the Jacobian matrix of $F_2(X_1, X_2)$ evaluated at the disease-free equilibrium, E_0. The solution to (9) is

$$S(t) = \frac{\pi}{1+\mu-a} + \frac{\delta R(0)}{1+a-\delta} e^{-(\delta+\mu)t} + K_0 e^{-(1+\mu-a)t}$$

$$A(t) = A(0)e^{-\mu t} + \frac{\pi(1-a)}{\mu(1+\mu-a)}(1-e^{-\mu t}) + \frac{R(0)(1-a)}{1-a-\delta}\left(e^{-\mu t} - e^{-(\delta+\mu)t}\right) + K_0\left(e^{-\mu t} - e^{-(1+\mu-a)t}\right) \quad (6)$$

$$R(t) = R(0)e^{-(\delta+\mu)t},$$

where $K_0 = S(0) - \frac{\pi}{1+\mu-a} - \frac{\delta R(0)}{1-a-\delta}$.

We see that as $t \to \infty, \left(S(t), A(t), R(t)\right) \to \left(\frac{\pi}{1+\mu-a}, \frac{\pi(1-a)}{\mu(1+\mu-a)}, 0\right)$. That is $\left(S(t), A(t), R(t)\right)$ tends to the disease-free equilibrium. Therefore, the equilibrium point $\left(\frac{\pi}{1+\mu-a}, \frac{\pi(1-a)}{\mu(1+\mu-a)}, 0\right)$ is globally asymptotically stable. The matrix B is given by

$$B=\begin{pmatrix} -(\beta+\mu) & \frac{ack_2\pi}{1+\mu-a} & \frac{ack_1\pi}{1+\mu-a} & 0 & 0 & 0 \\ (1-p)\beta & -(\alpha_1+\sigma_4+\gamma_3+\mu) & 0 & 0 & 0 & 0 \\ p\beta & 0 & -(\alpha_2+\sigma_2+q+\mu) & 0 & 0 & 0 \\ 0 & \alpha_1 & \alpha_2 & -(\sigma_3+\gamma_1+\mu) & 0 & 0 \\ 0 & 0 & q & 0 & -(\sigma_1+\gamma_2+\mu) & 0 \\ 0 & \sigma_4 & \sigma_2 & \sigma_3 & \sigma_1 & 0 \end{pmatrix}$$

Therefore,

$$BX_2 - F_2(X_1, X_2) = \begin{pmatrix} ac(k_2I_s + k_1I_{as}) & \left(\frac{\pi}{1+\mu-a} - S(t)\right) - cd(k_2I_s + k_1I_{as}) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (7)$$

In (7), the quantity $ac(k_2I_s + K_1I_{as})\left(\frac{\pi}{1+\mu-a} - S(t)\right)$ is positive since from (11), we have that $S(t) \le \frac{\pi}{1+\mu-a} \forall t.$

Furthermore, one sees that if the parameter, d, which represents the fraction of individuals who are not consistent with their protection against Covid-19, is zero, the first element in the matrix becomes positive, which makes the matrix become nonnegative. In this case, the disease-free equilibrium becomes globally asymptotically stable. Let the contact rate, e be the bifurcation parameter such that $R_0 = 1$ implies that

$$\frac{ack_2\pi(1-p)\beta(\mu a + d(1-a))}{\mu(1-a+\mu)(\beta+\mu)(\alpha_1+\sigma_4+\gamma_3+\mu)} + \frac{ack_1\pi p\beta(\mu a + d(1-a))}{\mu(1-a+\mu)(\beta+\mu)(\alpha_2+\sigma_2+q+\mu)} = 1$$

$$\Rightarrow \frac{(1-p)\beta ack_2\pi(\mu a+d(1-a))(\alpha_2+\sigma_2+q+\mu)+p\beta ack_1\pi(\mu a+d(1-a))(\alpha_1+\sigma_4+\gamma_3+\mu)}{\mu(1-a+\mu)(\beta+d)(\alpha_1+\sigma_4+\gamma_3+\mu)(\alpha_2+\sigma_2+q+\mu)}=1$$

Therefore at $R_0=1$, the bifurcation parameter is

$$c^*=\frac{\mu(1-a+\mu)(\beta+d)(\alpha_1+\sigma_4+\gamma_3+\mu)(\alpha_2+\sigma_2+q+\mu)}{\beta a\pi\left[(1-p)k_2(\mu a+d(1-a))(\alpha_2+\sigma_2+q+\mu)\,pk_1(\mu a+d(1-a))(\alpha_1+\sigma_4+\gamma_3+\mu)\right]}$$

We now make change of variables in the model system as follows: $x_1=S(t), \mathrm{x}_2=A(t), \mathrm{x}_3=E(t), \mathrm{x}_4=I_{as}(t), X_5=I_s(t), x_6=H(t), x_7=Q(t), \mathrm{x}_8=D(t), x_9=R(t).$

Then the right –hand side of the model system becomes

$$f\left(X(t),c^*\right)=\begin{pmatrix} f_1\\ f_2\\ f_3\\ f_4\\ f_5\\ f_6\\ f_7\\ f_8\\ f_9 \end{pmatrix}=\mu\begin{pmatrix} \pi+\delta x9-ac^*(k_1x_5+k_2x_4)x_1-(1-a)x_1-\mu x_1\\ (1-a)x_1-c^*d(k_1x_5+k_2x_4)x_2-\mu x_2\\ ac^*(k_1x_5+k_2x_4)x_1+c*d(k_1x_5+k_2x_4)x_2-(\beta+\mu)x_3\\ (1-p)\beta x_3-(\alpha_1+\sigma_4+\gamma_3+\mu)x_4\\ p\beta x_3-(\alpha_2+\sigma_2+q+\mu)x_5\\ \alpha_1x_4+\alpha_2x_5-(\sigma_3+\gamma_1+\mu)x_6\\ qx_5-(\sigma_1+\gamma_2+\mu)\,\mathrm{x}_7\\ \sigma_1x_7+\sigma_2x_5+\sigma_3x_6+\sigma_4x_4\\ \gamma_1x_6+\gamma_2x_7+\gamma_3x_4-(\delta+\mu)x_9 \end{pmatrix}$$

Then the Jacobian matrix of $f(X(t),c^*)$ at the disease-free equilibrium E_0 is given by

$$J(E_0,c^*)=\begin{pmatrix} -a_1 & 0 & 0 & -a_2 & -a_3 & 0 & 0 & 0 & \delta \\ a_4 & -\mu & 0 & -a_5 & -a_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & -a_7 & a_8 & a_9 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{10} & -a_{11} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{12} & 0 & -a_{13} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_1 & \alpha_2 & -a_{14} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & q & 0 & -a_{15} & 0 & 0 \\ 0 & 0 & 0 & \sigma_4 & \sigma_2 & \sigma_3 & \sigma_1 & 0 & 0 \\ 0 & 0 & 0 & \gamma_3 & 0 & \gamma_1 & \gamma_2 & 0 & -a_{16} \end{pmatrix} \quad (8)$$

where

$$a_1=(1+\mu-a),\ a_2=\frac{ac^*k_2\pi}{1+\mu-a},\ a_3=\frac{ac^*k_1\pi}{1+\mu-a},\ a_4=1-a,\ a_5=\frac{c^*d\pi k_2(1-a)}{\mu(1+\mu-a)},\ a_6=\frac{c^*d\pi k_1(1-a)}{\mu(1+\mu-a)},\ a_7=\beta+\mu$$

$$a_8=\frac{ac^*k_2\pi}{1+\mu-a}+\frac{c^*d\pi k_2(1-a)}{\mu(1+\mu-a)},\ a_9=\frac{ac^*k_1\pi}{1+\mu-a}+\frac{c^*d\pi k_1(1-a)}{\mu(1+\mu-a)},\ a_{10}=(1-P)\beta,\ a_{11}=\alpha_1+\sigma_4+\gamma_3+\mu,$$

$$a_{12}=\alpha_2+\sigma_2+q+\mu,\ a_{13}=\sigma_3+\gamma_1+\mu,\ a_{14}=\sigma_1+\gamma_2+\mu,\ a_{15}=\delta+\mu$$

The matrix $J(E_0;c^*)$ possesses one zero eigenvalue, while the remaining eigenvalues have negative real part. Therefore, the disease-free equilibrium E_0 is non-hyperbolic. Hence, we can apply the theorem above to analyze the dynamics of the model around the bifurcation parameter value $c=c^*$ .The right eigenvector $w(w_1\ w_2\ w_3 \ldots w_g)^T$ corresponding to the zero eigenvalue satisfies the following system of equations

$$-a_1w_1 - a_2w_4 - a_3w_6 + \delta w_9 = 0$$

$$a_4w_1 - \mu w_2 - a_5w_4 - a_cw_5 = 0$$

$$-a_7w_3 + a_8w_4 + a_9w_5 = 0$$

$$a_{10}w_3 - a_{11}w_4 = 0$$

$$a_{12}w_3 - a_{13}w_5 = 0$$

$$\alpha_1w_4 + \alpha_2w_5 - a_{14}w_6 = 0$$

$$qw_3 - a_{15}w_7 = 0$$

$$\sigma_4w_4 + \sigma_2w_5 + \sigma_3w_6 + \sigma_1w_7 = 0$$

$$\gamma_3w_4 + \gamma_1w_6 + \gamma_2w_7 - a_{16}w_9 = 0 \qquad (7)$$

On the other hand, the left eigenvector corresponding to the zero eigenvalue $w = (v_1\, v_2\, v_3 ... v_9)^T$ satisfies the system

$$-a_1v_1 + a_4v_2 = 0$$

$$-\mu v_2 = 0$$

$$-a_7v_3 + a_{10}v_4 + a_{12}v_5 = 0$$

$$-a_2v_1 - a_5v_2 + a_8v_3 - a_{11}v_4 + \alpha_1v_6 + \sigma_4v_8 + \gamma_3v_9 = 0$$

$$-a_3v_1 - a_6v_2 + a_9v_3 - a_{13}v_5 + \beta_2v_6 + qv_7 + \sigma_2v_8 = 0$$

$$-a_{14}v_6 + \sigma_3v_8 + \gamma_1v_9 = 0$$

$$-a_{15}v_7 + \sigma_1v_8 + \gamma_2v_9 = 0$$

$$\delta v_1 - a_{16}v_9 = 0 \qquad (9)$$

The solution to (14) is obtained in terms of w_3 as

$$w_1 = \left(-\frac{a_2a_{10}}{a_1a_{11}} - \frac{a_3}{a_1}\left(\frac{\alpha_1a_{10}}{a_{11}a_{14}} + \frac{\alpha_2a_{12}}{a_{13}a_{14}}\right) + \frac{\delta}{a_1}\left(\frac{\gamma_3a_{10}}{a_{16}a_{11}} + \frac{\alpha_1\gamma_1a_{10}}{a_{11}a_{14}a_{16}} + \frac{\alpha_2\gamma_1a_{12}}{a_{13}a_{14}a_{16}} + \frac{q\gamma_2a_{12}}{a_{13}a_{15}a_{16}} \right) w3 \right.$$

$$w_2 = \left(\frac{a_4}{\mu} \left(-\frac{a_2 a_{10}}{a_1 a_{11}} - \frac{a_3}{a_1} \right) \frac{\alpha_1 a_{10}}{a_{11} a_{14}} + \frac{\alpha_2 a_{12}}{a_{13} a_{14}} \right) + \frac{\delta}{a_1} \left(\frac{\gamma_3 a_{10}}{a_{16} a_{11}} + \frac{\alpha_1 \gamma_1 a_{10}}{a_{11} a_{14} a_{16}} + \frac{\alpha_2 \gamma_1 a_{12}}{a_{13} a_{14} a_{16}} + \frac{q \gamma_2 a_{12}}{a_{13} a_{15} a_{16}} \left(-\frac{a_4 a_{10}}{\mu a_{11}} - \frac{a_6 a_{12}}{\mu a_{13}} \right) \right) w3$$

$$w_3 > 0, w_4 = \frac{a_{10}}{a_{11}} w_3, w_5 = \frac{a_{12}}{a_{13}} w_3, w_6 = \left(\frac{\alpha_1 a_{10}}{a_{11} a_{14}} + \frac{\alpha_2 a_{12}}{a_{13} a_{14}} \right) w_3, \quad w_7 = \frac{q a_{12}}{a_{13} a_{15}} w_3$$

$$wg = \left(\frac{\gamma_3 a_{10}}{a_{16} a_{11}} + \frac{\alpha_1 \gamma_1 a_{10}}{a_{11} a_{14} a_{16}} + \frac{\alpha_2 \gamma_1 a_{12}}{a_{13} a_{14} a_{16}} + \frac{q \gamma_2 a_{12}}{a_{13} a_{15} a_{16}} \right) w_3$$

From (9), the compartments v_1, v_2, v_8 and v_9 are all zeros since they correspond to the states that are not infected by the virus disease [8]. Solving for other components in terms of v_3, we obtain $v_3 > 0, v_4 = \frac{a_8}{a_{11}} v_3, \; v_5 = \frac{a_9}{a_{13}} v_3, \; v_6 = 0, v_7 = 0.$ Since v_3, v_4 and v_5 are the only non-zero left eigenvectors, the relevant components of $f\left(X(t), c^*\right)$ are f_3, f_4 and f_5. Futhermore, the second order partial derivatives of f_4 and f_5 are all zeros since these components are linear in each variable, and do not contain the bifurcation parameter, c^*. Therefore, the parameters a and b in the bifurcation theorem become

$$\phi_1 = v_3 \sum_{i,j}^{9} w_i w_j \frac{\partial^2 f_3(0,0)}{\partial x, \partial x_j}, \qquad 9$$

$$\phi_2 = v_3 \sum_{i}^{9} w_i \frac{\partial^2 f_3(0,0)}{\partial x, \partial c^*} \qquad 10$$

The second orders partial derivatives with respect to each relevant variable evaluate at disease-free equilibrium are

$$\frac{\partial^2 f_3(0,0)}{\partial x_1^{\,2}} = 0, \; \frac{\partial^2 f_3(0,0)}{\partial x_1 \partial x_2} = 0, \frac{\partial^2 f_3(0,0)}{\partial x_1 \partial x_3} = 0, \frac{\partial^2 f_3(0,0)}{\partial x_1 \partial x_4} = a c^* k_2, \frac{\partial^2 f_3(0,0)}{\partial x_1 \partial x_5} = a c^* k_1, \frac{\partial^2 f_3(0,0)}{\partial x^2_{\,2}} = 0$$

$$, \frac{\partial^2 f_3(0,0)}{\partial x_2 \partial x_3} = 0, \frac{\partial^2 f_3(0,0)}{\partial x_2 \partial x_4} = d c^* K_2, \frac{\partial^2 f_3(0,0)}{\partial x_2 \partial x_5} = d c^* K_1, \; \frac{\partial^2 f_3(0,0)}{\partial x_1 \partial c^*} = 0, \frac{\partial^2 f_3(0,0)}{\partial x_4 \partial c^*} = \frac{a K_2 \pi}{1 + \mu - a} + \frac{d\pi(1-a) K_2}{\mu(1 + \mu - a)},$$

$\frac{\partial^2 f_3(0,0)}{\partial x_5 \partial c^*} = \frac{a K_1 \pi}{1 + \mu - a} + \frac{d\pi(1-a) K_1}{\mu(1 + \mu - a)}$. Using these partial derivatives, we have

$$\phi_1 = v_3 c^* (w_1 a + w_2 d)(w_4 K_2 + w_5 K_1), \qquad 12$$

$$\phi_2 = v_3 \left[K_2 w_4 (a x_1^* + d x^*_{\,2}) + K_1 w_5 (a x_1^* + d x^*_{\,2}) \right] \qquad 13$$

where $x_1^* = \frac{\pi}{1+\mu-a}$, $x_2^* = \frac{\pi(1-a)}{\mu(1+\mu-a)}$. We see that $\phi_2 > 0$. Therefore, forward bifurcation occurs in the model if $\phi_1 < 0$, otherwise, backward bifurcation occurs in the model.

The control reproduction number R_0, of the model is a function of parameters. In this section, we investigate how much and how R_0 depends on each of these parameters. That is, we conduct sensitivity analysis on the control.

4 Numerical simulations/sensitivity analysis

Parameter Values used in this model reproduction number. Sensitivity analysis helps to determine how sensitive R_0 is to various key parameters of the model. It also helps to discover parameters of the model that increase or decrease R_0.

Sensitivity analysis identifies parameters influencing COVID-19 spread. Parameters with positive indices (a, c, $\varkappa_1$, $\varkappa_2$, π, β, d) must be reduced; those with negative indices (α_1, α_2, σ_2, σ_4, q, p, γ_3) require increase. The highest positive index is a (+1.014), representing non-protected susceptibles, underscoring personal protection importance. Reducing a lowers exposure, decreasing transmission and the basic reproduction number. This highlights targeted interventions to curb spread by minimizing unprotected populations and enhancing protective measures.

Table 3:

PARAMETER	SENSITIVITY INDEX
c	+0.9992
c_0	+0.0007646
$\varkappa_2$	+0.2082
$\varkappa_1$	+0.0002669
π	+1
β	+0.0272
d	+0.00076469
a	+1.014
α_1	-0.1237
α_2	-0.2224
σ_2	-0.07113
σ_4	-0.0552
q	-0.4719
ρ	-0.447
γ_3	-0.4719

Numerical simulation of the results

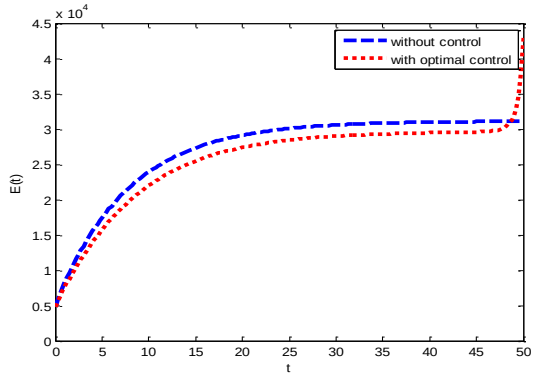


Figure 2: Exposed Humans

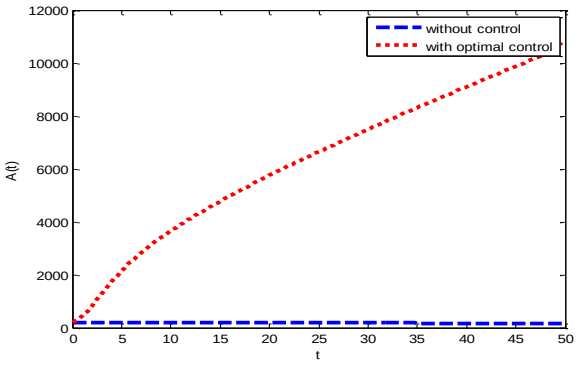


Figure 3: Graph of the protected class

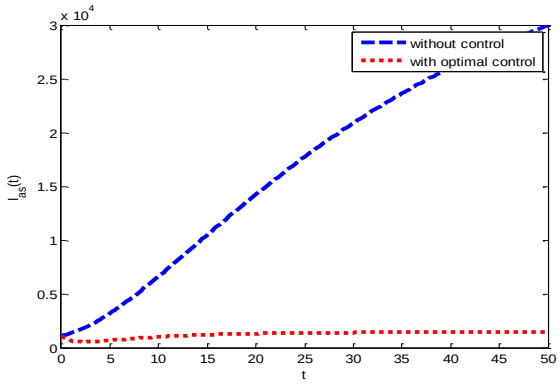


Figure 4: Graph of Asymptomatically infectious Humans

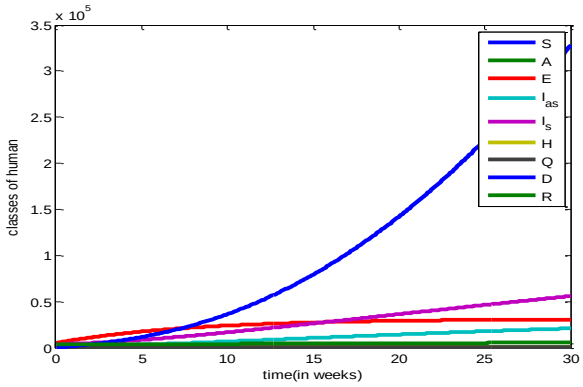


Figure 5: Graph for all the classes of Humans

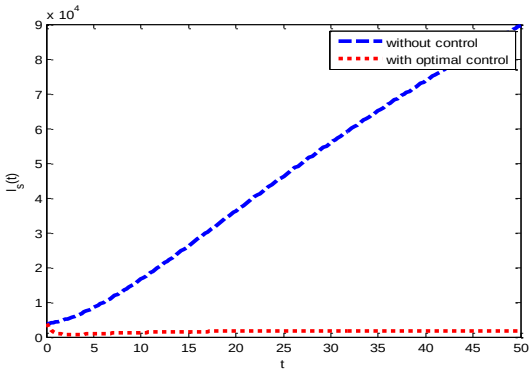


Figure 6: Symptomatically infectious Human

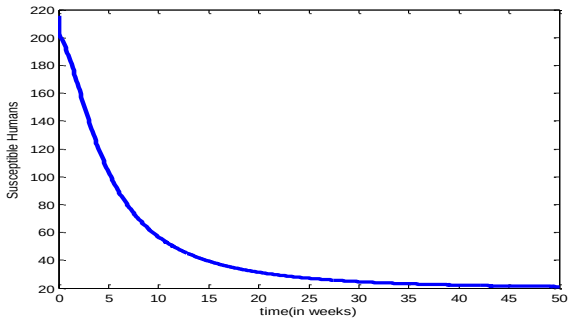


Figure 7: Susceptible humans

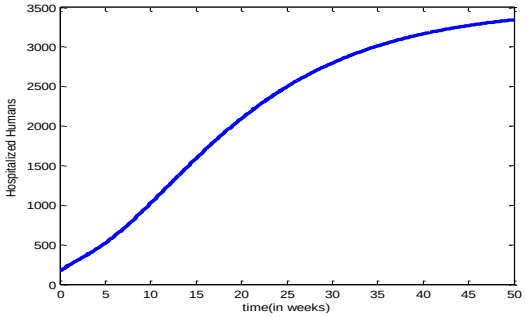


Figure 8: Hospitalized Humans

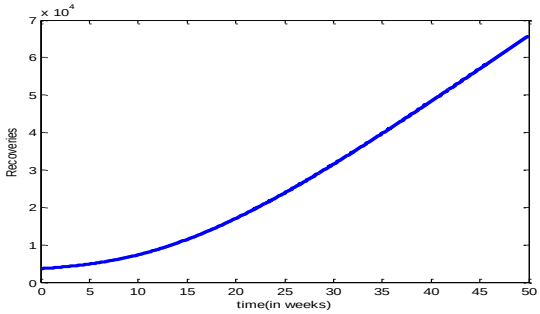


Figure 9: Recovered Humans

5 Discussion and Conclusion

The result of the implementation is presented graphically in figures 2,3,4 and 6. The figures show the effect of optimally applying the stated control measures in controlling the spread of covid-19. Specifically, we show the effect of optimal control on the number of persons that are exposed to covid-19, the number of persons that are symptomatically and asymptomatically infectious, and the number of covid-19 casualties. Each figure shows the number of people in these classes when there is no control and when optimal control is applied. The effect of applying optimal protection measures is shown in figure 2. We observe that when optimal control is applied, more people tend to control themselves as compared to when control is not applied. In figure 1, the effect of optimal control on the number of people exposed to covid-19 is presented. The graph shows that the number of people exposed to covid-19 is reduced when optimal control is applied. However, there is a sharp increase in the number of exposed persons when optimal control is applied. This sharp and sudden increase could be attributed to the inability of some people in the protection class to continue to observe adequate protection measures. In figure 4 and figure 6, we see that the number of people that are symptomatically and asymptomatically infectious are reduced when optimal control is applied. This is

the number of people that are exposed to covid-19 has been reduced. Lastly, the number of casualties can be seen to decrease when optimal control.

It can be concluded that if there are no interventions to control the spread of the disease, the disease will not be wiped out from the population. It was also observed that the best combination is protection, early burial of death due to covid-19, hospitalized and quarantine. We further noted that there is a direct correlation between asymptomatically class and the reproduction numbers, that is, as the rate of secondary infective increase also the rate of symptomatic increases. We finally observed that when protective measures are observed, quarantine, hospitalized and treatment policy is strictly adhered to and no delay in the burial of death due to covid-19, the disease will be completely wiped out of the population and thus reducing the rate of infection which increases the recovery rate in all classes.

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