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Mathematical Model for Controlling the Spread of the Novel Covid-19 Virus: An Optimal Control Analysis. Case Study Ghana

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Abstract

Worldwide governments faced the challenge of developing well-planned tailored strategies for controlling the COVID-19 pandemic to provide effective and efficient health protection while allowing economic and social activities to go on. This study uses the Susceptible- Exposed- Infected- Super spreaders- Hospitalized and Recovered (SEIPHR) which assumed that persons can equally likely to be infected with the virus in case of contact with an infected person except they are immune. The study performed numerical and qualitative analysis and different state variables were determined. The local stability for the disease-free equilibrium point and the endemic equilibrium point of the infection were determined. The qualitative analysis results showed that the model has the disease-free equilibrium which was locally asymptotically stable for $R0 < 1$ and unstable for $R0 \geq 1$. which clearly shows that, in the long run, close to 11% of the population was expected to be susceptible to the disease. The study also conducted sensitivity analysis. This analysis revealed that, the most important parameter is the contact rate. The optimal control analysis was carried out using the Pontryagins's maximum principle to determine the optimal strategy to curtail the spread of the disease. It was observed that, time optimal control existed in the model. The overall effect of the activation of all the control strategies simultaneously reduced the spread of the disease.

Keywords: COVID-19, pandemic, Super spreaders- Hospitalized and Recovered (SEIPHR), population, parameter, disease-free equilibrium, control, contact, epidemiology, variables, infectious.

Introduction

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-Cov-2) is a novel coronavirus strain that is responsible for the coronavirus disease of 2019 (COVID-19). The World Health Organization (WHO) designated COVID-19 as a pandemic on March 11, 2020. It is a respiratory disease caused by a novel coronavirus (SARS-CoV-2) that was discovered in late December 2019 in Wuhan, Hubei province, China (Zhou et al., 2020). The virus, according to Surjawidjaja (2003), is a member of the Nidovirales order, a Coronaviridae family, and a subfamily of the Orthocoronavirine. Coronaviruses are enclosed viruses with a positive-sense, singlestranded

RNA and viral particles that resemble a crown, hence the name (Gumel et al., 2004). The COVID-19 pandemic virus is a highly infectious disease that can be transmitted directly or indirectly from an infectious person to a healthy person through the eyes, nose, mouth, and occasionally through the ears via droplets produced when coughing or sneezing, according to Srivastava and Gaur (2017). It's unclear what

caused the condition. Many biologists, however, believe rodents and bats are to blame. (Zou et al., 2008). SARS-Cov-2 can live for up to 8-10 hours on porous surfaces (paper, wood, sponge, and fabric, etc) and somewhat longer on nonpermeable surfaces (glass, plastics, metals, etc), according to Srivastava and Gaur (2017) and normally takes 2-14 days for it to incubate. It has symptoms that are comparable to a cold or u with fever, dry cough, loss of breath, and pneumonia among the other symptoms, according to Zou et al. (2008). Depending on the age and health situation of the infected, the severity of the illness can range from mild to severe symptoms, (Srivastava and Gaur, 2017). Almost 80% of COVID-19 patients are asymptomatic or have just minor symptoms, and they normally recover within two weeks. However, substantial fatality rates have been reported among the elderly and those with underlying chronic diseases. The Novel COVID 19 virus began to spread over the world on January 30, 2020, and the WHO designated the outbreak a Public Health Emergency of International Concern on January 30, 2020. (PHEIC). COVID 19 was subsequently declared a global pandemic by the WHO Director-General. Despite expectations that the virus did not thrive in hot climates, as of April 2020, practically every country on the planet has at least one positive case. Nigeria reported its first case on February 27, 2020, but by April 26, 2020, the number of illnesses had climbed to 1,314 with 45 deaths, barely 60 days later. COVID-19 had resulted in 4,271,689 confirmed cumulative cases as of May 11, 2020, with 287,613 recorded deaths. Europe (particularly Italy and Spain) became the hub of the outbreak, while the United States of America, Asia, and Australia saw hundreds of new infections per day and thousands of disease deaths, according to Gumel et al. (2004). As of May 2020, the new COVID-19 had grown into a global public health emergency, affecting 212 countries and territories worldwide (Worldometer, 2020). A differential equation is one with coefficients, (Bird, 2006). The maximum derivative

that occurs in differential equations is known as order, and it is used to classify them. They're also categorised by degree, which is the maximum power of the highest derivative in the equation after it's been simplified. Dependent and independent variables are two types of variables that can be grouped together. Independent variables are usually measures of time and position that have a range of values. The domain of the issue is the collection of all feasible values for the independent variables. A dependent variable is a quantity that changes depending on the values of the independent variables during a task. (Tawhir, 2012). Over the years, mathematical models have proven to be trustworthy, successful, and efficient approaches for designing control strategies for infectious illnesses, epidemics, and pandemics such as Ebola, SARS, and MERS, among others. (Madubueze et al.,2018). Mathematical modelling continues to play an important role in deciphering complicated systems with observable behaviours or features. Recently, a number of mathematical models based on real-world COVID-19 transmission data have been constructed to detect disease-causing factors at various population levels. (Asamoah et al., 2020). For instance, in Asamoah et al. (2020), researchers looked at COVID -19 dissemination dynamics with environmental influences utilizing cases from Ghana. Using the existing cumulative COVID-19 mortality data, a mathematical model for estimating the consequences of non-pharmaceutical interventions (NPI) on the transmission dynamics of the emerging Coronavirus pandemic in Nigeria was published in Iboi et al. (2020). (Tilahun and Alemneh, 2021). The study's findings also suggested that if stakeholders are unable to implement all of the control techniques at the same time, the pandemic virus may resurface, resulting in an increase in transmission instances across the country. This study did not include mass screening as a control approach, and the model did not account for super spreaders. Shah et al. (2020) proposed a generalised SEIR model of COVID-19 that investigated the transmission dynamics' behaviour under various control interventions. Bermejo-Martin et al. (2020) developed a compartmental

mathematical model for the COVID 19 pandemic virus, with a focus on super spreaders' transmissibility. The study determined the basic reproduction number threshold, local stability, disease free equilibrium in terms of reproduction number and investigated the sensitivity of the model with respect to the variation of each its key parameters. The study demonstrated the suitability of the formulated model of pandemic COVID -19 infectious disease outbreak in Wuhan, China (Bermejo-Martin et al., 2020). This study incorporated super spreaders and hospitalization into the SEIR model, but the study did not consider control intervention on the model. The aim of this thesis seeks to add additional compartment to the SEIR model with applied optimal control intervention measures. This study would use the extended SEIR model of the form SEIPHR where susceptible(S), exposed(E), infectious(I), super-spreaders(P), hospitalisation(H), and Recovered(R). To reduce the spread of the COVID-19 pandemic virus, the two-time dependent control variables introduced to the mathematical model are the prevention strategy targeted at preventing the pandemic virus from symptomatic, asymptomatic and hospitalised humans. This is accomplished by long-term public health campaigning for social distancing, good personal hygiene, the use of face masks in public areas, and the provision of protective equipment for health-care professionals. The management approach is the best control variable strategy for managing hospitalised individuals with the goal of ensuring quick recovery and preventing deaths from COVID-19 pandemic virus sequelae. For hospitalised persons suffering with severe COVID-19, this can be performed by providing supplementary oxygen or a breathing equipment as soon as possible.

Methodology

Model Formulation

A new mathematical epidemiological model is constructed that takes into consideration the super-spreading phenomena of some individuals, based on the mathematical model of Kim (2016). The population of size N is divided into six classes: susceptible (S), exposed (E), symptomatic and infectious (I), super-spreaders (P), hospitalised (H), and recovered (R).

Model Assumption

The mathematical model assumptions for the SEIPHR are as follows:

1. Individuals, with the exception of those who are immune, are equally likely to be infected by infectious individuals in the community when they come into contact.
2. There is no such thing as treatment failure; patients either recover or die.
3. Individuals who have recovered are immune for life.
4. Individuals who are infectious are identified early and isolated for treatment and public education.
5. All newborns are enrolled in the susceptible class.
6. Every individual in the population is susceptible to the novel coronavirus
7. The population is homogeneously mixed, which means that interactions are evenly random and independent between time intervals.
8. Interactions are done on proportions of the population.

The mathematical model flowchartt and the system of non-linear differential equations are as follows:

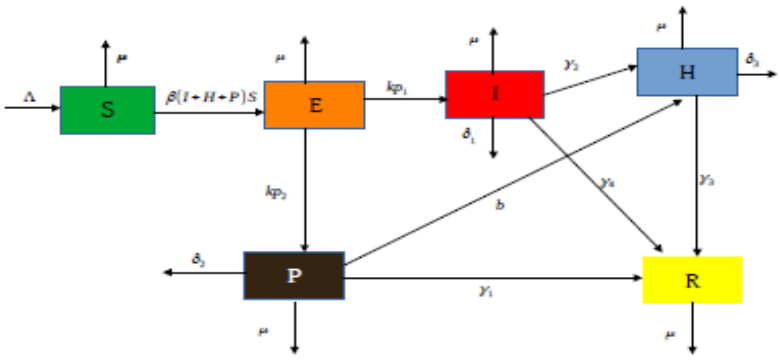

Figure 3.1: Model Flowchart

The parameters of the model are defined in the table below With regards to

Parameter	Parameter Definition
Λ	Recruitment rate(New borns and immigrants)
μ	Natural mortality rate
k	Rate at which exposed people become infected
δ_1	Disease induced death rate due to infected class
δ_2	Disease induced death rate due to super-spreaders class
δ_3	Disease induced death rate due to hospitalised class
β	Transmission coefficient from infected individuals
b	Average rate at which super-spreaders become hospitalised
γ_1	Recovery rate of super-spreaders without being hospitalised
γ_2	Average rate at which infectious individuals become hospitalised
γ_3	Recovery rate of hospitalised individuals
γ_4	Recovery rate without being hospitalised
p_1	Rate at which exposed individuals become infectious
p_2	Rate at which exposed individuals become superspreaders

Table 3.1: List of parameters for the model

the above model assumptions, the mathematical model is formulated by the law of balance and yields the following system of nonlinear differential equations.

$$\dot{S} = \frac{dS}{dt} = \Lambda - \beta(H + I + P)S - \mu S$$

$$\dot{E} = \frac{dE}{dt} = \beta(H + I + P)S - (\mu + kp_1 + kp_2)E$$

$$\dot{I} = \frac{dI}{dt} = kp_1E - (\mu + \delta_1 + \gamma_2 + \gamma_4)I$$

$$\dot{P} = \frac{dP}{dt} = kp_2E - (\delta_2 + \mu + b + \gamma_1)P$$

$$\dot{H} = \frac{dH}{dt} = \gamma_2I + bP - (\mu + \delta_3 + \gamma_3)H$$

$$\dot{R} = \frac{dR}{dt} = \gamma_1P + \gamma_4I + \gamma_3H - \mu R$$

Properties of the Model

A description of some basic aspects of the model represented in the system of equations in equation (1) is demonstrated in this part, such as feasible solution and positivity of solutions. The region in which the model equations' solutions are biologically meaningful is shown by the feasible solution, and the positivity of the solution describes the nonnegativity of the model equations' solutions in equation (1). The feasible solution set, which is a collection of positively invariant model solutions, is defined as follows:

$$\Omega = \left\{ (S, E, I, P, H, R) \in \mathbb{R}_+^6 : S + E + I + P + H + R = N \leqslant \frac{\Lambda}{\mu} \right\}$$

…….. 2

From the model equations given in equation (1), it will be shown that the region is positively invariant. Let us consider the procedures below. From the mathematical model equations, the population of individuals is given by

$$S + E + I + P + H + R = N$$

…….3

To obtain the overall changes in the population, we sum the terms of the equation (1) which yields

$$\dot{S} + \dot{E} + \dot{I} + \dot{P} + \dot{H} + \dot{R} = \frac{dN}{dt} = \Lambda - \mu(S + E + I + P + H + R) - \delta_1 I - \delta_2 P - \delta_3 H$$

…….4

$$= \Lambda - \mu N - \delta_1 I - \delta_2 P - \delta_3 H$$

…….5

$$\Longrightarrow \quad \frac{dN}{dt} \leqslant \Lambda - \mu N$$

…….6

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

…….7

Which affirms that the population is not constant and also proves the equation (2). To demonstrate nonnegativity of the solutions, we start with the Susceptible compartment.

$$\dot{S} = \frac{dS}{dt} = \Lambda - \beta S(H + I + P) - \mu S$$

$$= \Lambda - (\beta(H + I + P) + \mu)S$$

$$\geqslant -(\beta(H + I + P) + \mu)S$$

$$\frac{dS}{S} \geqslant -(\beta(H + I + P) + \mu)dt$$

$$\ln(S(t)) \geqslant -(\beta(H + I + P) + \mu)t + c$$

$$S(t) \geqslant S(0)e^{-(\beta(H+I+P)+\mu)t}$$

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Due to the fact that all the rates are positive and that H; I; P will always be greater than zero which we will be proven later, it can be assumed that (β (H +I + P) +μ) > 0 such that S(t) ≥ 0 Similarly for the Exposed compartment we have,

$$\dot{E} = \frac{dE}{dt} = \beta(H + I + P)S - (\mu + kp_1 + kp_2)E$$

$$\geqslant -(\mu + kp_1 + kp_2)E$$

$$\frac{dE}{E} \geqslant -(\mu + kp_1 + kp_2)dt$$

$$\ln(E(t)) \geqslant -(\mu + kp_1 + kp_2)t + c$$

$$E(t) \geqslant E(0)e^{-(\mu+kp_1+kp_2)t}$$

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which also guarantees that the solution set of the exposed class is convergent and always greater than zero. Also, for the Infected class we have,

$$\dot{I} = \frac{dI}{dt} = kp_1E - (\mu + \delta_1 + \gamma_2 + \gamma_4)I$$

$$\geqslant -(\mu + \delta_1 + \gamma_2 + \gamma_4)I$$

$$\frac{dI}{I} \geqslant -(\mu + \delta_1 + \gamma_2 + \gamma_4)dt$$

$$\ln(I(t)) \geqslant -(\mu + \delta_1 + \gamma_2 + \gamma_4)t + c$$

$$I(t) \geqslant I(0)e^{-(\mu+\delta_1+\gamma_2+\gamma_4)t}$$

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This similarly guarantees the solution of Infected class to be convergent and always greater than zero. Analysing the Super-spreaders class also yields

$$\dot{P} = \frac{dP}{dt} = kp_2E - (\delta_2 + \mu + b + \gamma_1)P$$

$$\geqslant -(\delta_2 + \mu + b + \gamma_1)P$$

$$\frac{dP}{P} \geqslant -(\delta_2 + \mu + b + \gamma_1)dt$$

$$\ln(P(t)) \geqslant -(\delta_2 + \mu + b + \gamma_1)t + c$$

$$P(t) \geqslant P(0)e^{-(\delta_2+\mu+b+\gamma_1)t}$$

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Thus, the solution set is convergent and greater than zero. Also, for the Hospitalised class we have,

$$\dot{H} = \frac{dH}{dt} = \gamma_2 I + \gamma_4 P - (\mu + \delta_3 + \gamma_3)H$$

$$\geqslant -(\mu + \delta_3 + \gamma_3)H$$

$$\frac{dH}{H} \geqslant -(\delta_2 + \mu + b + \gamma_1)dt$$

$$\ln(H(t)) \geqslant -(\mu + \delta_3 + \gamma_3)t + c$$

$$H(t) \geqslant H(0)e^{-(\mu+\delta_3+\gamma_3)t}$$

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which also guarantees that the solution set of the hospitalised class is convergent and always greater than 0. Lastly for the Recovered class we have,

$$\dot{R} = \frac{dR}{dt} = \gamma_1 P + \gamma_4 I + \gamma_3 H - \mu R$$

$$\geqslant -\mu R$$

$$\frac{dR}{R} \geqslant -\mu R dt$$

$$\ln(R(t)) \geqslant -\mu t + c$$

$$R(t) \geqslant R(0)e^{-\mu t}$$

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This also shows that the solution set of the Recovered class is convergent and guaranteed to be greater than 0.

Equilibrium Points

Given the set of equations in (1), the left-hand side of the equations which corresponds to the rates of change are set to zero which yields the equilibrium points. To aid simplification, the following substitutions are made into the system of equations in (1). We let

$$m_1 = \mu + kp_1 + kp_2$$

$$m_2 = \mu + \delta_1 + \gamma_2 + \gamma_4$$

$$m_3 = \mu + \delta_2 + b + \gamma_1$$

$$m_4 = \mu + \delta_3 + \gamma_3$$

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The simplification of equation (1) gives,

$$\dot{S} = \frac{dS}{dt} = \Lambda - \beta(H + I + P)S - \mu S$$

$$\dot{E} = \frac{dE}{dt} = \beta(H + I + P)S - m_1 E$$

$$\dot{I} = \frac{dI}{dt} = kp_1 E - m_2 I$$

$$\dot{P} = \frac{dP}{dt} = kp_2 E - m_3 P$$

$$\dot{H} = \frac{dH}{dt} = \gamma_2 I + bP - m_4 H$$

$$\dot{R} = \frac{dR}{dt} = \gamma_1 P + \gamma_4 I + \gamma_3 H - \mu R$$

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Hence substituting the left-hand side of equation (16) to zero also yields,

$$\Lambda - \beta S(H + I + P) - \mu S = 0$$

$$\beta(H + I + P)S - m_1 E = 0$$

$$kp_1 E - m_2 I = 0$$

$$kp_2 E - m_3 P = 0$$

$$\gamma_2 I + pP - m_4 H = 0$$

$$\gamma_1 P + \gamma_4 I + \gamma_3 H - \mu R = 0$$

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We firstly find the Disease-Free Equilibrium point by setting all other compartments except the Susceptible compartment to zero i.e. (S >0, E = I = P = H = R = 0). However, following the steps, we arrived at the final equation of the endemic equilibrium points.

$$y_2^* = \{s_2^*, e_2^*, i_2^*, p_2^*, h_2^*, r_2^*\} = \left\{\frac{n_1}{kn_2}, \frac{n_5}{m_1 kn_2}, \frac{p_1 n_5}{m_1 m_2 n_2}, \frac{p_2 n_5}{m_1 m_3 n_2}, \frac{n_3 n_5}{\mu n_1 n_2}, \frac{n_4 n_5}{\mu n_1 n_2}\right\}$$

……18

$$n_1 = m_1 m_2 m_3 m_4$$

$$n_2 = \beta(m_2 p_2(b + m_4) + m_3 p_1(\gamma_2 + m_4))$$

$$n_3 = (bm_2 p_2 + \gamma_2 m_3 p_1)$$

$$n_4 = (m_2 p_2 (b\gamma_3 + \gamma_1 m_4) + m_3 p_1 (\gamma_2 \gamma_3 + \gamma_4 m_4))$$

$$n_5 = k\Lambda n_2 - \mu n_1$$

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Model Linearisation

The equilibrium points obtained enables us to describe some linear properties in terms of stability of the model. It is recall however that, the models obtained in equation 17 are nonlinear and thus requires a transformation to linearise the model. We then employ the Jacobian matrix which is a matrix of first order partial derivatives of the system of equations defined as

$$J_{ij} = \frac{\partial f_i}{\partial x_j} \qquad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 20$$

Where i runs from 1 to the number of equations in the system f, x the set of compartments and j running from 1 to the number of compartments in the population. Given X = {S, E, I, P, H, R} and the system of equations in equation (16), the Jacobian matrix is derived as

$$J = \begin{pmatrix} \beta(-(H+I+P)) - \mu & 0 & -\beta S & -\beta S & -\beta S & 0 \\ \beta(H+I+P) & -m_1 & \beta S & \beta S & \beta S & 0 \\ 0 & kp_1 & -m_2 & 0 & 0 & 0 \\ 0 & kp_2 & 0 & -m_3 & 0 & 0 \\ 0 & 0 & \gamma_2 & b & -m_4 & 0 \\ 0 & 0 & \gamma_4 & \gamma_1 & \gamma_3 & -\mu \end{pmatrix} \qquad \ldots\ldots\ldots\ldots\ldots 21$$

The Jacobian matrix is evaluated at the equilibrium points and based on the negativity or positivity of the eigenvalues, conclusions on the stability of the equilibrium points are drawn.

Disease Free Equilibrium

Given the average length of time a person spends in each compartment, the disease-free equilibrium point in equation is used to quantify the rate at which new infections are formed in the compartments. This threshold is known as the reproductive number, and it can be used to evaluate how sensitive it is to changes in the parameters that it is dependent on. The stability of the equilibrium point is also investigated.

The Basic Reproductive Number

The basic reproductive number, R0, is determined using the next generation approach. The basic reproductive number is defined as the average number of secondary infections created when infected individuals are introduced into the community When evaluating when an infection can spread and remain in a community, the basic reproductive number is usually employed as a criterion. The basic concept is to average the predicted number of new infectious illnesses across all of the likely infected types. Let G be a next-generation matrix, with the ijth element, gij representing the estimated number of secondary infectious diseases of type i caused by a single infected individual of type j. In other words, each element of the matrix G is a reproductive number, but one that accounts for who infects whom. The dominant

eigenvalue (spectral radius ρ) of $G = FV - 1$ i.e., R0 = ρ(G) determines R0. The vector of infected classes is defined as X with exposed, infectious, super-spreaders, and hospitalized being its elements.

X = {E, I, P, H} ……22

and Y as the vector of uninfected classes such as Susceptible and Recovered.

Z = {X, R} ……23

The matrix F is used to describe the rate of new infectious disease in different compartments, which are indicated by the elements of X, and is evaluated at a disease-free equilibrium, which is denoted by

$$F_{ij} = \frac{\partial \mathcal{F}_i}{\partial x_j}$$

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where F represents how new infections are formed in the infective compartments defined in X. From the system of equation in (16), our Fi's are defined as

$$\mathcal{F}_i = \begin{cases} \beta(I + H + P)S, i = 1 \\ 0, \text{otherwise} \end{cases}$$

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$$F = \begin{pmatrix} 0 & \beta S & \beta S & \beta S \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

ThusF is given by ……26

$$F|y_1^* = \begin{pmatrix} 0 & \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

…….27

Evaluating F at the equilibrium point yields

Now a new matrix V that determines the rate of infective transfer from one compartment to the next is also created as

$$V_{ij} = \frac{\partial \mathcal{V}_i}{\partial x_j}$$

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where Vi defines how infectives leave their corresponding compartments. Vi's are defined as follows

$$\mathcal{V}_1 = m_1 E$$
$$\mathcal{V}_2 = m_2 I - k p_1 E$$
$$\mathcal{V}_3 = m_3 P - k p_2 E$$
$$\mathcal{V}_4 = m_4 H - \gamma_2 I - b P$$

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The matrix V thus becomes

$$V = \begin{pmatrix} m_1 & 0 & 0 & 0 \\ -k\rho_1 & m_2 & 0 & 0 \\ -k\rho_2 & 0 & m_3 & 0 \\ 0 & -\gamma_2 & -b & m_4 \end{pmatrix}$$

……30

with inverse found as

$$V^{-1} = \begin{pmatrix} \frac{1}{m_1} & 0 & 0 & 0 \\ \frac{k\rho_1}{m_1 m_2} & \frac{1}{m_2} & 0 & 0 \\ \frac{k\rho_2}{m_1 m_3} & 0 & \frac{1}{m_3} & 0 \\ \frac{\gamma_2 k m_3 \rho_1 + b k m_2 \rho_2}{m_1 m_2 m_3 m_4} & \frac{\gamma_2}{m_2 m_4} & \frac{b}{m_3 m_4} & \frac{1}{m_4} \end{pmatrix}$$

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Thus G = FV-1 simplifies as

$$G = \begin{pmatrix} \frac{\beta\Lambda(\gamma_2 k m_3\rho_1 + bkm_2\rho_2)}{\mu m_1 m_2 m_3 m_4} + \frac{\beta k\Lambda\rho_1}{\mu m_1 m_2} + \frac{\beta k\Lambda\rho_2}{\mu m_1 m_3} & \frac{\beta\gamma_2\Lambda}{\mu m_2 m_4} + \frac{\beta\Lambda}{\mu m_2} & \frac{\beta b\Lambda}{\mu m_3 m_4} + \frac{\beta\Lambda}{\mu m_3} & \frac{\beta\Lambda}{\mu m_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

…….32

Thus, the reproductive number as defined as the spectral radius of the matrix J,which is the maximum of the eigen values yields

$$R_0 = \rho(G) = \frac{\beta k\Lambda\left(m_3\rho_1\left(\gamma_2 + m_4\right) + m_2\rho_2\left(\gamma_4 + m_4\right)\right)}{\mu m_1 m_2 m_3 m_4}$$

…….33

The Reproductive number devoid of substitutions and depending on the originalparameters of the model are obtained as

$$R_0 = \frac{A\beta k\left(p_2\left(\gamma_2 + \gamma_4 + \delta_1 + \mu\right)\left(b + \gamma_3 + \delta_3 + \mu\right) + p_1\left(\gamma_2 + \gamma_3 + \delta_3 + \mu\right)\left(b + \gamma_1 + \delta_2 + \mu\right)\right)}{\mu\left(\gamma_2 + \gamma_4 + \delta_1 + \mu\right)\left(\gamma_3 + \delta_3 + \mu\right)\left(kp_1 + kp_2 + \mu\right)\left(b + \gamma_1 + \delta_2 + \mu\right)}$$

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By the substitution equation, the reproductive number is simplified as

$$R_0 = \frac{k\Lambda n_2}{\mu n_1}$$

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which will be used for further calculations due to its simplified nature.

Sensitivity Analysis

The reproductive number's sensitivity analysis demonstrates how important each parameter is to the rate of subsequent infections. A sensitivity index is used to determine how sensitive this threshold is to its factors. When a parameter is changed, sensitivity indices let us to measure the relative change in the Reproductive number. This shift is quantified using the normalised forward sensitivity index. It calculates the ratio of the reproductive number's change in relation to changes in its parameters. The sensitivity index can be calculated using partial derivatives when the Reproductive number is a differentiable function of its parameters. The normalised

forward sensitivity index of R0 that depends on a parameter ρ in a differentiable way is defined by

$$\phi_{\rho}^{R_0} = \frac{\partial R_0}{\partial \rho}\frac{\rho}{|R_0|}$$

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From the reproductive number in equation, the threshold depends on the parameters

ρ =

Based on the definition in equation (36), the forward sensitivity indices of the Reproductive number in equation (35) with respect to the parameters in this set are given as

$$\{\Lambda, b, k, \mu, p_1, p_2, \gamma_1, \gamma_2, \gamma_3, \gamma_4, \delta_1, \delta_2, \delta_3, \beta\}.$$

$$\phi_{\Lambda}^{R_0} = 1$$

$$\phi_{\beta}^{R_0} = 1$$

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$$\phi_{\mu}^{R_0} = \mu\left(-\frac{1}{m_3} - \frac{1}{m_2} - \frac{1}{m_4} - \frac{1}{m_1}\right) + \frac{\mu\left(p_2\left(m_2 + m_4\right) + p_1\left(m_3 + m_4\right)\right)}{p_2 m_2 m_3 + p_1\left(\gamma_2 + m_4\right)\left(m_3\right)} - 1$$

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$$\phi_{k}^{R_0} = \frac{\mu}{k\left(p_1 + p_2\right) + \mu}$$

$$\phi_{\delta_1}^{R_0} = -\frac{\delta_1 m_3 p_1\left(\gamma_2 + m_4\right)}{m_2\left(m_3 p_1\left(\gamma_2 + m_4\right) + m_2 m_3 p_2\right)}$$

$$\phi_{\delta_2}^{R_0} = -\frac{\delta_2 m_2 m_3 p_2}{m_3\left(m_3 p_1\left(\gamma_2 + m_4\right) + m_2 m_3 p_2\right)}$$

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…….39

$$\phi_{\delta_3}^{R_0} = -\frac{\delta_3\left(2bm\_p_2 + \gamma_2 m_2 p_1\right)}{m_4\left(m_2 p_1\left(\gamma_2 + m_4\right) + m_2 p_2\left(\gamma_2 + m_4\right)\right)}$$

$$\phi_{b}^{R_0} = \frac{bm_2 p_2\left(\gamma_1 - \gamma_3 + \delta_2 - \delta_3\right)}{m_2\left(m_2 p_1\left(\gamma_2 + m_4\right) + m_2 m_3 p_2\right)}$$

$$\phi_{\gamma_1}^{R_0} = -\frac{\gamma_1 m_2 m_3 p_2}{m_3\left(m_2 p_2\left(b + m_4\right) + m_3 p_1\left(\gamma_2 + m_4\right)\right)}$$

$$\phi_{\gamma_2}^{R_0} = \frac{\gamma_2 m_3 p_1\left(-\gamma_3 + \gamma_4 + \delta_1 - \delta_3\right)}{m_2\left(m_2 p_2\left(b + m_4\right) + m_3 p_1\left(\gamma_2 + m_4\right)\right)}$$

$$\phi_{\gamma_3}^{R_0} = -\frac{\gamma_3\left(bm_2 p_2 + \gamma_2 m_3 p_1\right)}{m_4\left(m_2 p_1\left(\gamma_2 + m_4\right) + m_2 m_3 p_2\right)}$$

$$\phi_{\gamma_4}^{R_0} = -\frac{\gamma_4 m_3 p_1\left(\gamma_2 + m_4\right)}{m_2\left(m_2 p_2\left(b + m_4\right) + m_3 p_1\left(\gamma_2 + m_4\right)\right)}$$

…….40

$$\phi_{p_1}^{R_0} = \frac{p_1\left(-km_2 p_2\left(b + m_3\right) - kp_1 m_3\left(\gamma_2 + m_4\right) + m_3 m_1\left(\gamma_2 + m_4\right)\right)}{m_1\left(m_2 p_2\left(b + m_4\right) + m_2 p_1\left(\gamma_2 + m_4\right)\right)}$$

……41

$$\phi_{p_2}^{R_0} = \frac{\left(m_2 p_2\left(b + m_4\right) + m_3 p_1\left(\gamma_2 + m_4\right)\right)\left(m_2 p_2\left(-kp_2\left(b + m_4\right) + m_1\left(b + m_4\right) - km_3 p_1\left(\gamma_2 + m_4\right)\right)\right)}{m_1}$$

…...42

Global Stability Analysis

The analysis is done based on the direct method of the Lyapunov stability analysis approach. The system of equation (16) can be represented as

Y = f (Y) ……..43

which has the disease-free equilibrium point also represented as y*. Note that

Y = {S, E, I, P, H, R}. A Lyapunov function V (Y) which is continuously differentiable in the neighbourhood of U can be constructed a for equation 43 satisfying the following conditions.

1. V (Y) > 0, ¥Y £ U
2. V (y*) = 0

A Lyapunov function is constructed for our system of equations as

$$V(Y) = f^T(Y) f(Y)$$

$$= \left(bP - Hm_4 + \gamma_2 J\right)^2 + \left(\beta S(H + J + P) - m_1 W\right)^2 + \left(\gamma_3 H + \gamma_4 J + \gamma_1 P - \mu R\right)^2$$

$$+ \left(-\beta S(H + J + P) + \lambda - \mu S\right)^2 + \left(kp_1 W - Jm_2\right)^2 + \left(kp_2 W - m_3 P\right)^2$$

……44

Clearly, we can see that V > 0 and that evaluating it at the disease-free equilibrium also yields 0. Thus, for the disease-free equilibrium point to be globally stable

$$\frac{dV}{dt} \leqslant 0$$

……45

From equation 44, we have,

$$\dot{V}(Y) = \dot{f}^T(Y) f(Y) + f^T(Y) \dot{f}(Y)$$

$$\dot{f}(Y) = \frac{\partial f}{\partial Y} \frac{dY}{dt} = J(Y) f(Y)$$

$$\dot{V}(Y) = \dot{f}^T(Y) \left[J^T(Y) + J(Y) \right] \dot{f}(Y)$$

…….46

where J (Y) is the Jacobian matrix as computed in equation 21.

Local Stability Analysis

The eigenvalues of the Jacobian matrix in equation (21) at the disease-free equilibrium can be used to calculate the local stability. The matrix columns with negative eigenvalues that were discovered by inspection have already been removed. At the disease-free equilibrium point (y^1*) evaluating the Jacobian matrix yields:

$$J_{(E,I,P,H)}|y_1^* = \begin{pmatrix} -m_1 & \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} \\ kp_1 & -m_2 & 0 & 0 \\ kp_2 & 0 & -m_3 & 0 \\ 0 & \gamma_2 & b & -m_4 \end{pmatrix}$$

……...47

We then find the eigenvalues (Li, i = 1…4) of the matrix in equation (47) by solving | J -H |= 0 which yields the characteristic polynomial.

$$l^4 + A_1 l^3 + B_1 l^2 + C_1 l + D_1 = 0$$

……48

Which gives

$$A_1 = m_1 + m_2 + m_3 + m_4$$

$$B_1 = -\frac{2A\beta kp_1}{\mu} + m_1m_2m_3m_2 + m_4m_2 + m_1m_3 + m_1m_4 + m_3m_4$$

$$C_1 = -\frac{Ab\beta kp_1}{\mu} - \frac{A\beta km_3p_1}{\mu} - \frac{A\beta km_2p_1}{\mu} - \frac{2A\beta km_4p_1}{\mu} - \frac{A\beta\gamma_2kp_1}{\mu} + m_1m_2m_3 + m_1m_4m_3 + m_2m_4m_3 + m_1m_2m_4$$

$$D_1 = -\frac{Ab\beta km_2p_1}{\mu} - \frac{A\beta km_2m_4p_1}{\mu} - \frac{A\beta km_3m_4p_1}{\mu} + m_1m_2m_3m_4 - \frac{A\beta\gamma_2km_3p_1}{\mu} \quad \text{……...49}$$

To determine the stability conditions for the disease-free equilibrium point, the Routh-Hurwitz criterion is applied. If the following requirements are met, the disease-free equilibrium is locally asymptotically stable.

$$A_1 > 0$$

$$A_1B_1 - C_1 > 0$$

$$A_1B_1C_1 - A_1^2D_1 - C_1^2 > 0$$

$$D_1 > 0$$

…….50

Stability Analysis

Evaluating the Jacobian matrix at the endemic equilibrium point (y*) yields:

$$J_{(S,E,I,P,H)}|y_2^* = \begin{pmatrix} -\beta(h_2^* + i_2^* + p_2^*) - \mu & 0 & -\beta s_2^* & -\beta s_2^* & -\beta s_2^* \\ \beta(h_2^* + i_2^* + p_2^*) & -m_1 & \beta s_2^* & \beta s_2^* & \beta s_2^* \\ 0 & kp_1 & -m_2 & 0 & 0 \\ 0 & kp_2 & 0 & -m_3 & 0 \\ 0 & 0 & \gamma_2 & b & -m_4 \end{pmatrix}$$

…...51

The polynomial the characteristic then yields

$$A_2 = \beta s_2^* \left(h_2^* + i_2^* + p_2^*\right) + \mu + m_1 + m_2 + m_3 + m_4$$

$$B_2 = \beta m_1 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_2 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_3 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_4 \left(h_2^* + i_2^* + p_2^*\right) + \mu m_1 + \beta \mathrm{kp}_1 s_2^* + \beta \mathrm{kp}_2 s_2^* + \mu m_2 + \mu m_3 + \mu m_4 + m_2 m_1 + m_3 m_1 + m_4 m_1 + m_2 m_3 + m_2 m_4 + m_3 m_4$$

$$C_2 = \beta m_1 m_2 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_3 m_2 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_4 m_2 \left(h_2^* + i_2^* + p_2^*\right) - \mu m_3 m_4 + \beta m_1 m_4 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_3 m_4 \left(h_2^* + i_2^* + p_2^*\right) - \beta \mathrm{kp}_2 m_2 s_2^* - \beta \mathrm{kp}_1 m_3 s_2^* - \beta \mathrm{kp}_1 m_4 s_2^* - \beta \gamma_2 \mathrm{kp}_1 s_2^* - \beta \gamma_4 \mathrm{kp}_2 s_2^* - \beta \mathrm{kp}_1 \mu s_2^* - \beta \mathrm{kp}_2 \mu s_2^* + \mu m_1 m_2 + \mu m_3 m_2 + \mu m_4 m_2 + \mu m_1 m_3 + \beta m_1 m_3 \left(h_2^* + i_2^* + p_2^*\right) - \beta \mathrm{kp}_2 m_4 s_2^* - m_1 m_3 m_2 - m_1 m_4 m_2 - m_3 m_4 m_2 - m_1 m_3 m_4 + \mu m_1 m_4 \quad (3.7.3)$$

$$D_2 = \beta m_1 m_2 m_3 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_1 m_4 m_3 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_2 m_4 m_3 \left(h_2^* + i_2^* + p_2^*\right) + \beta m_1 m_2 m_4 \left(h_2^* + i_2^* + p_2^*\right) - \beta \gamma_2 \mathrm{kp}_1 m_3 s_2^* - \beta \gamma_4 \mathrm{kp}_2 m_2 s_2^* - \beta \mathrm{kp}_1 \mu m_3 s_2^* - \beta \mathrm{kp}_2 \mu m_2 s_2^* - \beta \mathrm{kp}_1 \mu m_4 s_2^* - \beta \mathrm{kp}_2 \mu m_4 s_2^* - \beta \mathrm{kp}_1 m_4 m_3 s_2^* - \beta \mathrm{kp}_2 m_2 m_4 s_2^* - \beta \gamma_2 \mathrm{kp}_1 \mu s_2^* + \mu m_1 m_2 m_4 - \beta \gamma_4 \mathrm{kp}_2 \mu s_2^* + \mu m_1 m_2 m_3 + \mu m_1 m_4 m_3 + \mu m_2 m_4 m_3 + m_1 m_2 m_4 m_3$$

$$K_2 = \mu m_1 m_2 m_3 m_4 + \beta m_1 m_2 m_3 m_4 \left(h_2^* + i_2^* + p_2^*\right) - \beta \gamma_2 \mathrm{kp}_1 \mu m_3 s_2^* + \beta \gamma_4 \mathrm{kp}_2 \mu m_2 s_2^* + \beta \mathrm{kp}_2 \mu m_2 m_4 s_2^* + \beta \mathrm{kp}_1 \mu m_3 m_4 s_2^* \quad \ldots 52$$

The stability conditions of the endemic equilibrium thus become

$$A_2 > 0$$

$$A_2 B_2 - C_2 \mu^2 > 0$$

$$A_2 B_2 C_2 - A_2^2 D_2 - C_2^2 \mu^2 + A_2 K_2 \mu^2 > 0$$

$$A_2 \left(A_2 D_2^2 + B_2^2 K_2 - B_2 C_2 D_2\right) + \mu^2 \left(2 A_2 D_2 K_2 + B_2 C_2 K_2 - C_2^2 D_2\right) - K_2^2 \mu^4 > 0$$

$$K_2 > 0 \quad \ldots 53$$

The Optimal Control Problem

Optimal control analysis is added to the mathematical model in equation (16). Based on the results of the sensitivity analysis, optimal control theory is applied to the pandemic novel COVID-19 mathematical model in equation (refmodmodeleqns), as well as other infectious models, in an attempt to limit the impact of novel COVID-19 on the population. Two time-dependent control variables U1(t) and U1(t) were included to the mathematical model in the following tactics to reduce the propagation of the COVID-19 pandemic virus. following the differential equation (16). The primary goal of incorporating the control strategy into the mathematical model is to determine the optimal levels of intervention strategy required to reduce the spread of the COVID-19 pandemic virus in the human population. As a result, the

number of symptomatic, asymptomatic, and hospitalised individuals responsible for the spread of COVID-19 in the population is minimised at the lowest possible cost. Where the objective functional subject to the differential equation is obtained as,

$$\mathcal{J}(u_1, u_2) = \int_0^{t_f} \left[w_1 I + w_2 P + w_3 H + \frac{1}{2}\left(w_4 u_1^2 + w_5 u_2^2\right) \right] dt$$

…….54

Hamiltonian And Optimality

To determine the necessary conditions for optimal control, Pontryangins' maximum principle (Pontryagin, 1987) is applied. The preceding equations are transformed into a problem of minimizing the pointwise Hamiltonian (H) with respect to (U1, U2) by applying this principle.

$$\begin{aligned}\mathcal{H} = {} & w_1 I + w_2 P + w_3 H + \frac{1}{2}\left[w_4 u_1^2 + w_5 u_2^2\right] \\ & + \lambda_1 \left[\Lambda - (1-u_1)\beta (I + H + P) S - \mu S\right] \\ & + \lambda_2 \left[(1-u_1)\beta (I + H + P) S - m_1 E\right] \\ & + \lambda_3 \left[k p_1 E - m_2 I\right] + \lambda_4 \left[k p_2 E - m_3 P\right] \\ & + \lambda_5 \left[\gamma_2 I + bP - (m_4 + u_2) H\right] \\ & + \lambda_6 \left[\gamma_1 P + (u_2 + \gamma_3) H + \gamma_4 I - \mu R\right]\end{aligned}$$

…….55

Where □i, I, ……, 6 represent the adjoint variables associated with S; E; I; P; H; R the state variables of the model above. The adjoint equation and the transversality conditions are typical Pontryagins Maximum principle results. The Hamiltonian is differentiated in terms of the states variables S, E, I, P, H, and R, and thus the adjoint system:

$$\frac{\lambda_1}{dt} = -\frac{\partial \mathcal{H}}{\partial S}$$
$$= \lambda_1[(1-u_1)\beta(I+H+P)+\mu] - \lambda_2[(1-u_1)\beta(I+H+P)]$$

$$\frac{\lambda_2}{dt} = -\frac{\partial \mathcal{H}}{\partial E}$$
$$= \lambda_2 m_1 - \lambda_3 k p_1 - k p_2 \lambda_4$$

$$\frac{\lambda_3}{dt} = -\frac{\partial \mathcal{H}}{\partial I}$$
$$= -w_1 + \lambda_1[(1-u_1)\beta S] - \lambda_2[(1-u_1)\beta S] + \lambda_3 m_2 - \lambda_5\gamma_2 - \lambda_6\gamma_4$$

$$\frac{\lambda_4}{dt} = -\frac{\partial \mathcal{H}}{\partial P}$$
$$= -w_2 + \lambda_1[(1-u_1)\beta S] - \lambda_2[(1-u_1)\beta S] + \lambda_4 m_3 - \lambda_5 b - \lambda_6\gamma_1$$

$$\frac{\lambda_5}{dt} = -\frac{\partial \mathcal{H}}{\partial H}$$
….56
$$= \lambda_5(m_4+u_2) + \beta\lambda_1 S(1-u_1) - \beta\lambda_2 S(1-u_1) - \lambda_6(\gamma_3+u_2) - w_3$$

$$\frac{\lambda_6}{dt} = -\frac{\partial \mathcal{H}}{\partial R}$$
$$= \lambda_6\mu$$
….57

Similarly, by following the methodology of Pontryagin's Maximum Principle (1987), the characterization of optimal controls (U*1, U*2) that is based on the conditions

$$\frac{\partial H}{\partial u_i} = 0$$

……...58

Which gives,

$$u_1^* = \frac{\beta\lambda_2 S(H+I+P) - \beta\lambda_1 S(H+I+P)}{w_4}$$

$$u_2^* = \frac{H\lambda_5 - H\lambda_6}{w_5}$$

..…59

and optimality conditions are given by

$$u_1^* = \max\{0, \min(1, \theta_1)\}$$

$$u_2^* = \max\{0, \min(1, \theta_1)\}$$

$$\theta_1 = \frac{\beta\lambda_2 S(H+I+P) - \beta\lambda_1 S(H+I+P)}{w_4}$$

$$\theta_2 = \frac{H\lambda_5 - H\lambda_6}{w_5}$$

……60

Presentation of results

Model of parameter estimations

Coronavirus data was collected locally from the Ghana health services website, which aided in the estimation of disease-related parameters. For certain parameters that could not be estimated due to lack of data, these values were assumed based on similar works reported in the literature. The least square approach was used to estimate most of the parameters as summarised in Table1 below.

Parameter	Values	Source
δ_1	0.00081	Estimated
δ_2	0.000405	Estimated
δ_3	0.006885	Estimated
p_1	0.99	Estimated
p_2	0.01	Estimated
β	0.0025	Estimated
α	0.0000787596	Estimated
b	0.000014	Estimated
Λ	0.0000787596	
b	0.000014	Estimated
μ	0.0000199016	
k	0.0714	
γ_1	0.000127504	Estimated
γ_2	0.002884	Estimated
γ_3	0.00216757	Estimated
γ_4	0.000255008	Estimated

Table1: list of parameters for the model

The initial values were chosen to help represent the true essence of the coronavirus dynamics in Ghana. The country initially reported two cases in which these people were quarantined. We assumed that, the individuals made contact with some other susceptible individuals in the population thereby moving them into the exposed class. Our initial choice of exposed individuals was then chosen as 12 with the idea that each of the individuals made contact with a minimum of 6 Susceptible individuals. The table below also shows the selection of initial conditions. Random choices of initial cases were also used to explain the dynamics of the disease in the Ghanaian population under different scenarios.

Compartment	Size	Population Proportion
Susceptible	30419986	0.999999539777
Exposed	12	$3.94477318 \times 10^{-07}$
Infected	0	0
Super Spreaders	0	0
Quarantined	2	6.5746×10^{-08}
Recovered	0	0

Table 2: Shows the choice of initial condition

Mathematical model for describing the dynamics of the virus

The system of equations obtained is solved numerically using MATLAB and the ode45 tool. The dynamics of the novel coronavirus in Ghana is simulated over a 2000-day period. With the given parameters, the initial conditions presented in the Table 2 and three other random initial values, the following images illustrates the daily infection dynamics of the novel coronavirus disease in the Ghanaian population.

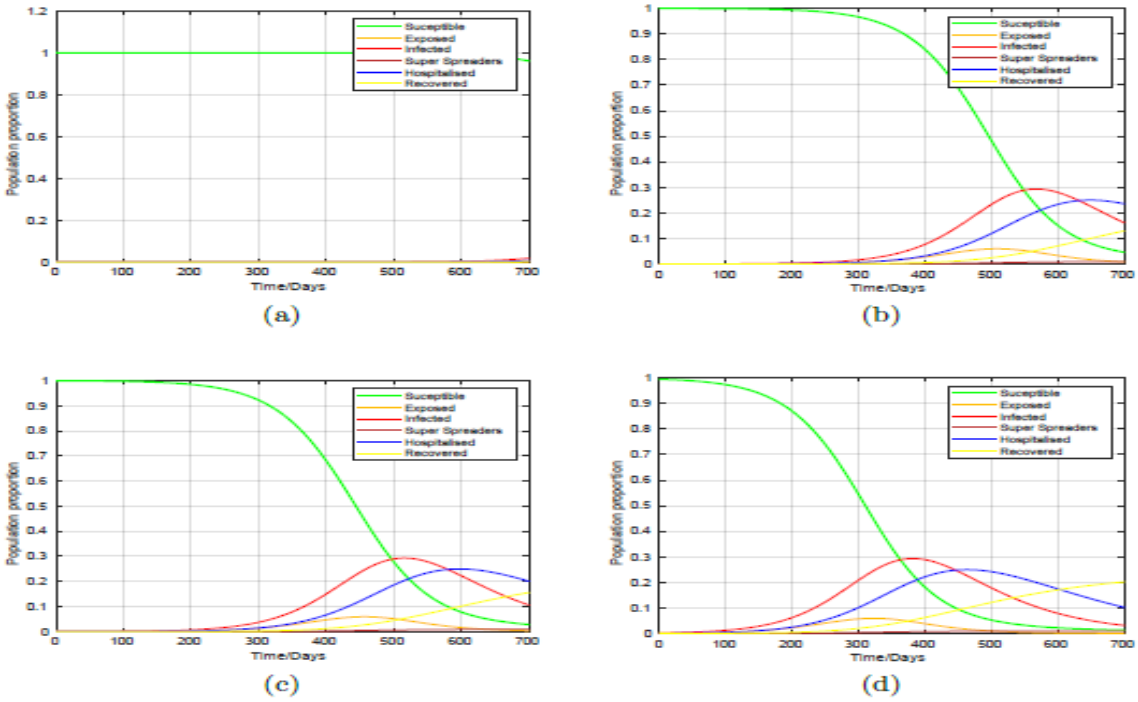


Figure 1: showing dynamics of the disease under different initial conditions

From the various scenarios presented in the figure (1), it is evident that the likelihood of the disease spreading and at a faster rate depends on the size of the classes with infections. Having relatively larger values in the infected, superspreaders and hospitalised classes cause the susceptible class to decline at a faster rate.

Analysis of the Disease-Free Equilibrium Point

The disease-free equilibrium point, as the name implies, is the point at which the population is thought to have no record of the novel coronavirus disease. This means there are no exposed people, sick people, or quarantined people. The reproductive Number, which specifies the average number of secondary generated infection cases in the Ghanaian population, is another parameter of interest. This can be simplified by considering new infection cases recruited from the infectious and quarantined groups, assuming interactions are made with such individuals.

Sensitivity Analysis

We investigate the effects of changes in this set of parameters on the reproductive number by evaluating their sensitivity indices on the parameter values of the model. Recall the sensitivity indices of these parameters to the reproductive number has been derived which when evaluated on the values of our estimated parameters yield the following results.

Parameter(ρ)	Sensitivity Index($\gamma_\rho^{R_0}$)
Λ	1
β	1
b	-0.00118303
k	0.000278656
γ_1	-0.0114906
γ_2	-0.460661
γ_3	-0.0547059
γ_4	-0.0609717
μ	-1
p_1	0.0407684
p_2	0.041047
δ_1	-0.193669
δ_2	-0.0364984
δ_3	-0.173767

The negativity or positivity of the sensitivity index values implies an inverse or direct relationship between the parameter and the reproductive number respectively. We visualise the sensitivity indices of each of the parameters and their corresponding effects on the reproductive number explaining the significant of each of the calculated sensitivity indexes

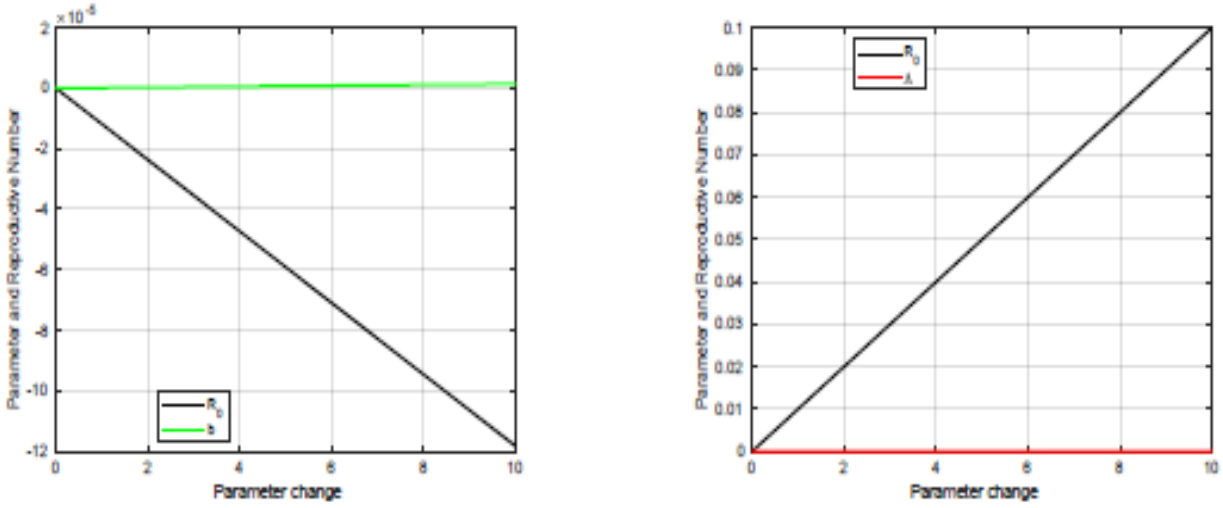


Figure 3: Sensitivity analysis of parameter b and Λ

For the parameter b which represents the rate at which Superspreaders are hospitalised, there exists an indirect relationship between this parameter value and the reproductive number. An increase in the b parameter causes a corresponding decrease in the reproductive number. From the sensitivity index value of -0.00118, the implication is that when the value of the parameter increases by 100%, it causes a 0.118% decrease in the reproductive number. For the Λ parameter which represents recruitment rate, there exist a direct relationship between this parameter and the reproductive number, where the sensitivity index of 1 implies that a 100% change in the value of this parameter causes a 100% change in the reproductive number.

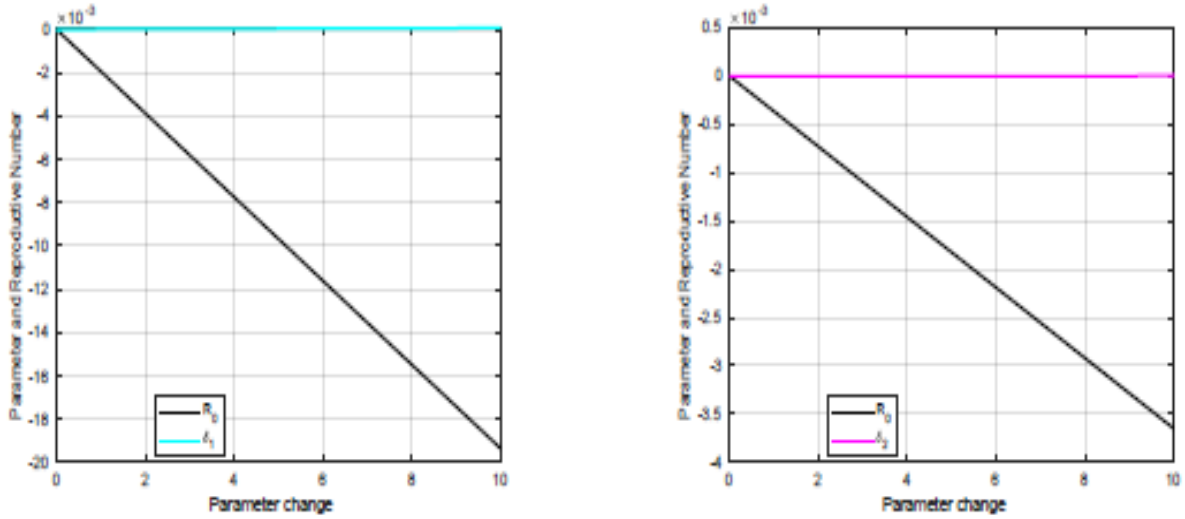


Figure 4: Sensitivity analysis of parameters Ə1 and Ə 2

Ə 1 represents the rate at which infectives dies out of the population and has an indirect relation with the reproductive number. The sensitivity index of - 0:1937 implies that increasing the value of this parameter by 100% causes a 19.37% decrease in the reproductive number. Ə 2 on the other hand, represents the disease induced death rate of the superspreaders class.

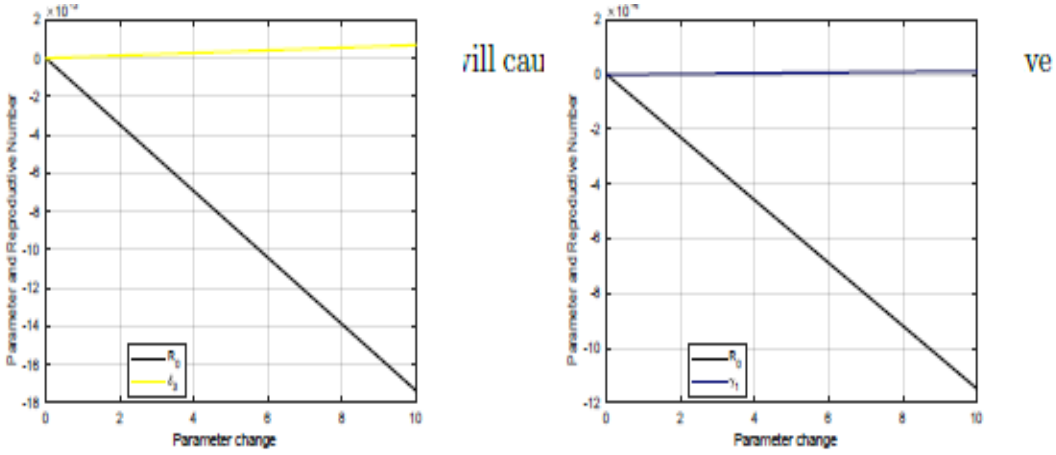


Figure 5: Sensitivity analysis of parameters Ə3 and □1

Ə 3 also represents the rate at which hospitalised individuals die due to the disease and similarly has an inverse relationship with the reproductive number. The sensitivity index value of this parameter, -0.17368, suggests that increasing this parameter by 100% will decrease the reproductive number by 17:368%. For □ 1 which represents the recovery rate of superspreaders has a sensitivity index of _0:0115 which implies the parameter is inversely related to the reproductive number. Increasing this parameter value by 100% will decrease the reproductive number by 1:15%.

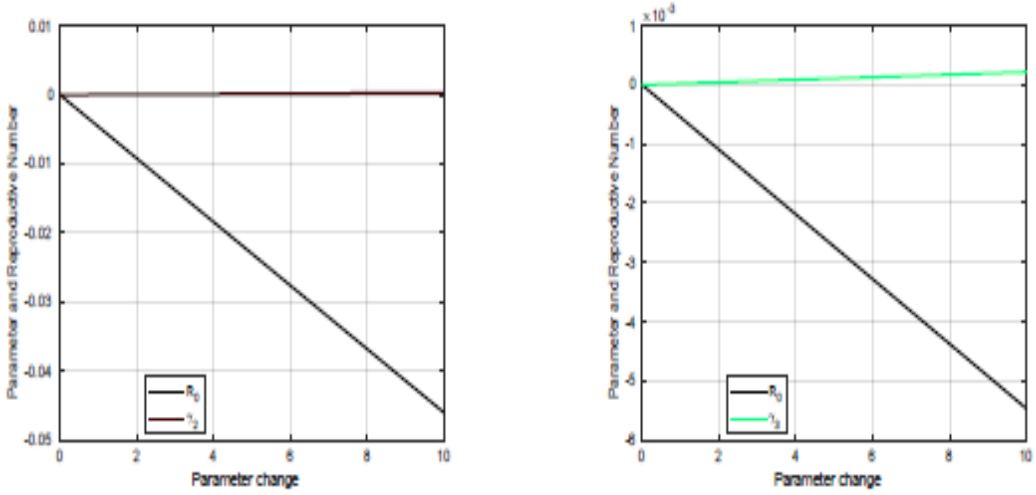


Figure 6: Sensitivity analysis of parameters □ 2 and □ 3

For □ 2 which represents the rate at which infectives get hospitalised, increasing this parameter value by 100% will cause a 46:07% reduction in the reproductive number. Increasing □ 3 which represents the recovery rate of hospitalised individuals will cause a 5:47% reduction in the reproductive number.

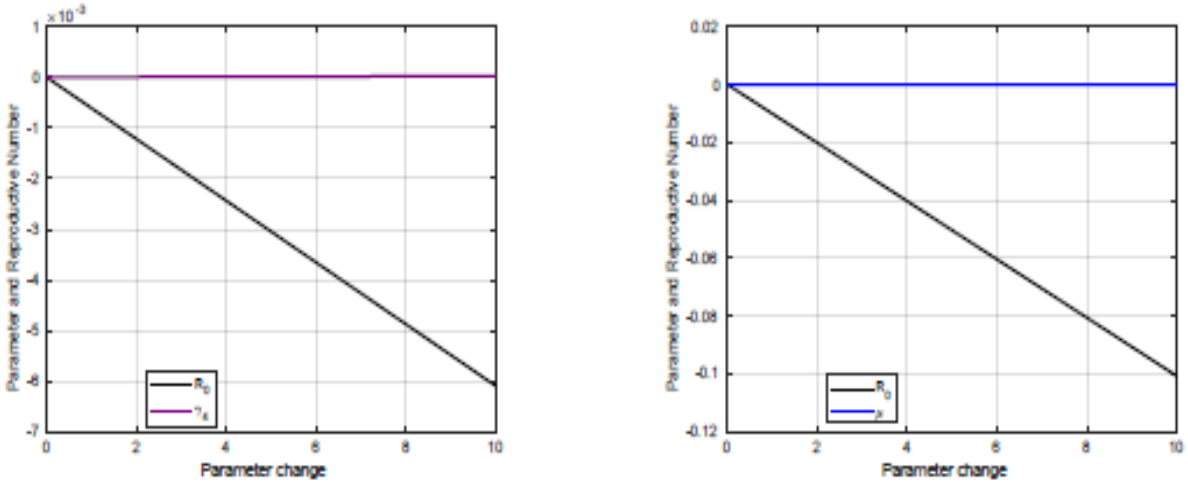


Figure 7: Sensitivity analysis of parameters □ 4 and μ

□ 4 represents the recovery rate of infectives and the sensitivity index for this parameter suggests that increasing the value of the parameter will reduce the reproductive number by 6:10%. For μ which represents the natural death rate, a 100% increase in this parameter will cause a 100:007% decrease in the reproductive number. p1 and p2 represents the proportion of the exposed individuals that become infectives and superspreaders respectively. A 100% increase in the proportion of exposed individuals becoming infectives will cause 4:07% increase in the reproductive number whereas a similar increase in the proportion of exposed who become superspreaders will increase the reproductive number by 4:105%.

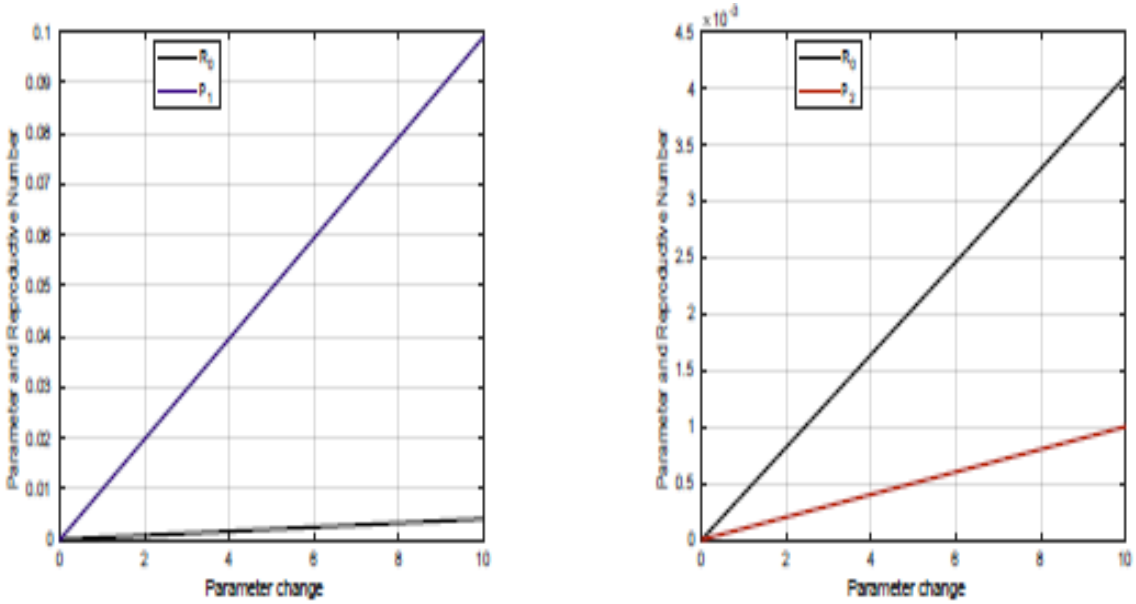


Figure 8: Sensitivity analysis of parameters p1 and p2

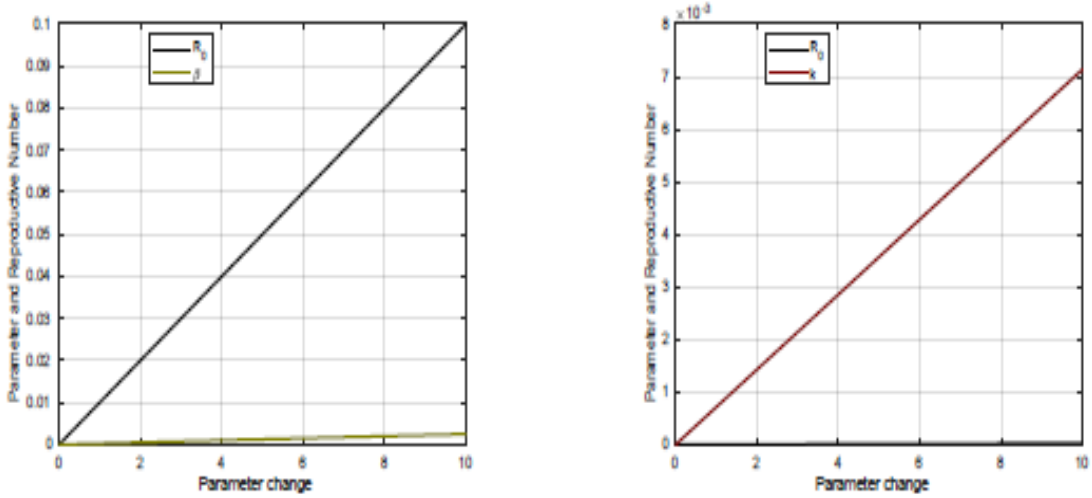
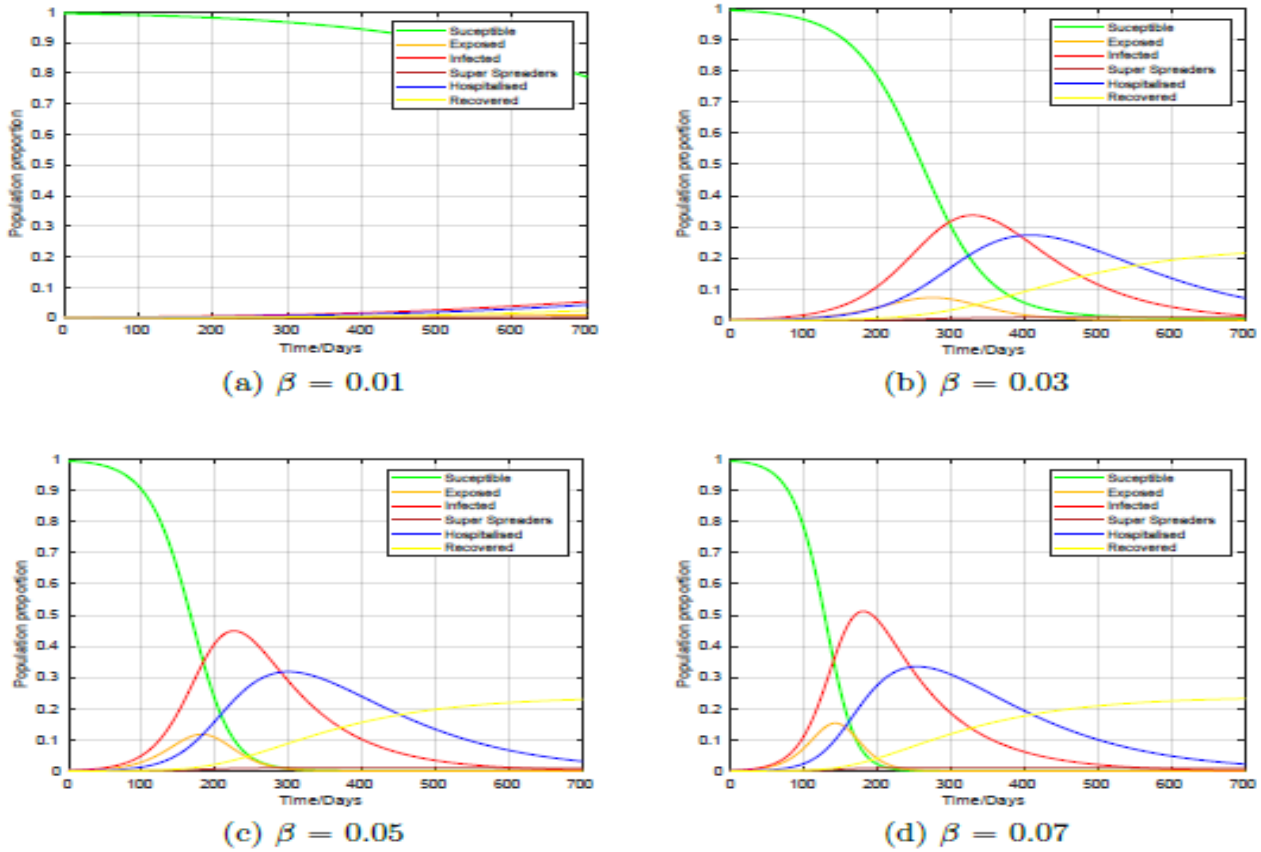


Figure 9: Sensitivity analysis of parameters β and Ķ

The contact rate parameter β will cause a 100% increase in the reproductive number if its value increases by 100% whereas the infection rate parameter Ķ will cause a 0:02% change in the reproductive number if its value increases by 100%. Obviously, the parameters that causes increases in the population generally causes increase in the reproductive number whiles those that cause decreases in the population also decreases the reproductive number. Thus, the contact rate (β) parameter is very sensitive to increasing secondary infections while the hospitalisation of infected individuals (2) also significant reduced

secondary infections. We chose different values of β and □ 2 and see how they affects the respective compartments. For the parameter β, it is observed that small contact rates are associated with slower decline in the susceptible class.



(a) $\beta = 0.01$ (b) $\beta = 0.03$

(c) $\beta = 0.05$ (d) $\beta = 0.07$

Figure 10: showing dynamics of the disease given variations of parameter β

In the figure (10) shows that increasing the contact rate with all other parameters held constant, significant increases the infection class and larger proportions of the population get the virus over a very short period of time. Infections increased drastically from 5% to almost 50% as the parameter was increased from 0:01 to 0:07. For the parameter □ 2, increases in this parameter are known to significantly reduce infections.

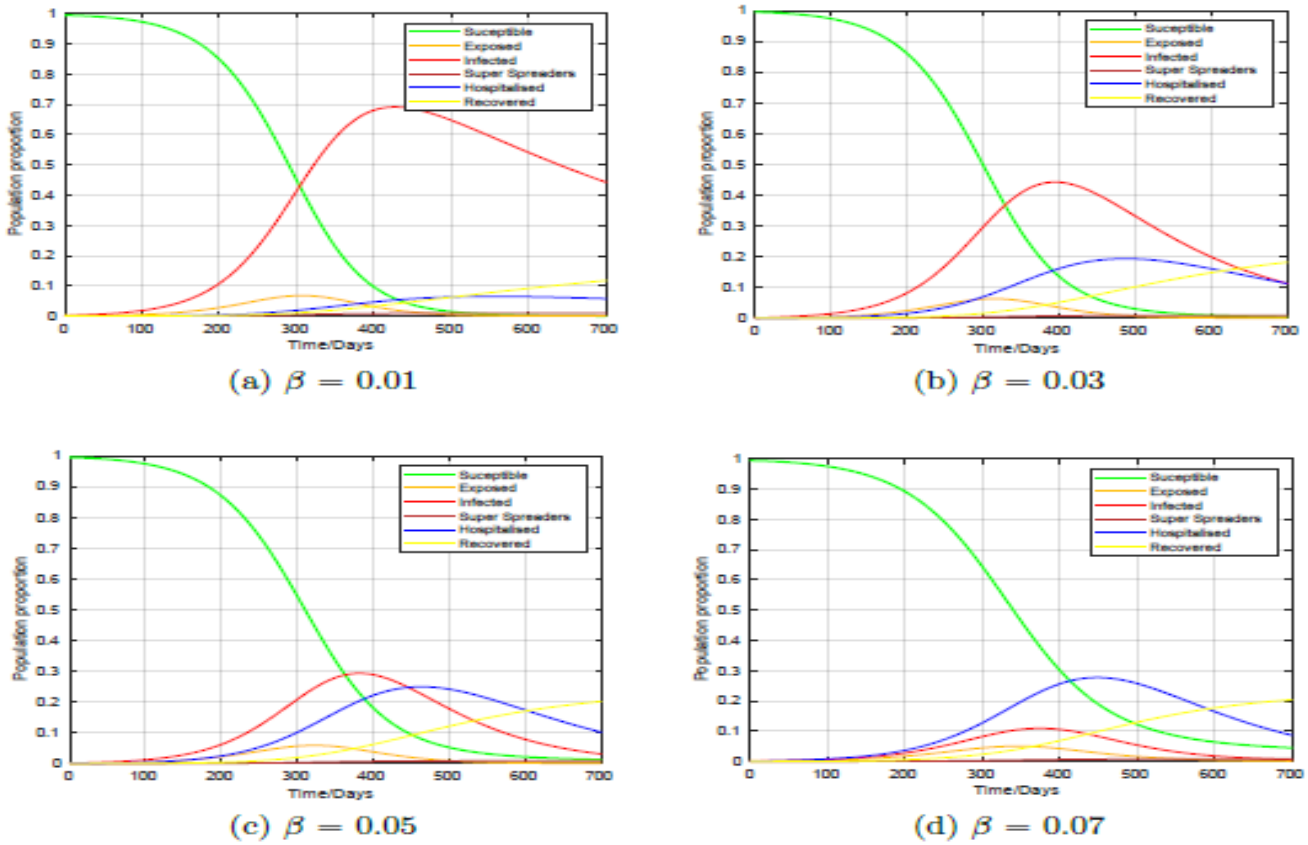


Figure 11: Figure showing dynamics of the disease given variations of parameter β

From the scenarios presented in figure (11) it is observed that, with all other parameters kept constant; the infections compartment is reduced significantly from 70% to 10% as □ 2 increases from 0:001 to 0:03.

Optimal Control

The mathematical model established was extended to optimal control, with two control techniques implemented: u1 and u2. u1 represented the prevention strategy aimed at preventing the pandemic virus from infecting symptomatic, asymptomatic, and hospitalised humans, while u2 represented the management strategy aimed at ensuring prompt recovery, preventing deaths, and reducing complications caused by the COVID-19 pandemic virus in hospitalised individuals. The impact of the two tactics on the transmission of the COVID-19 virus in Ghana's population is investigated numerically.

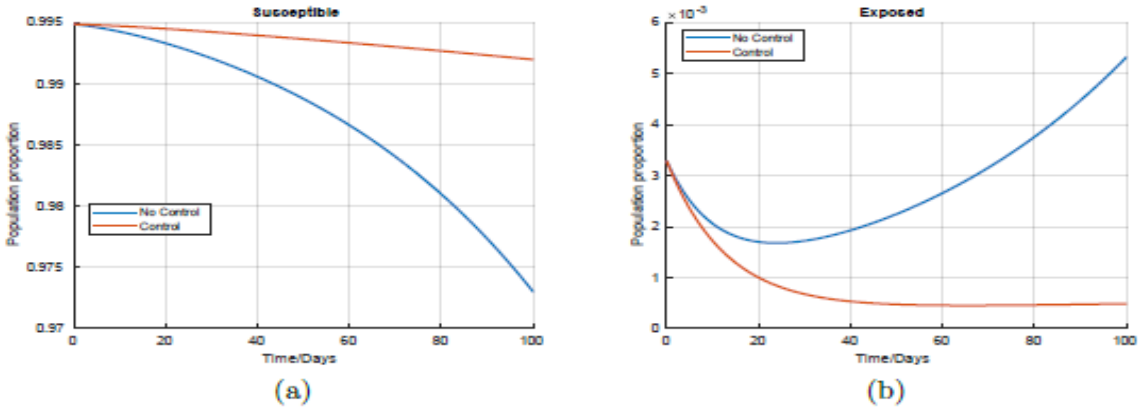
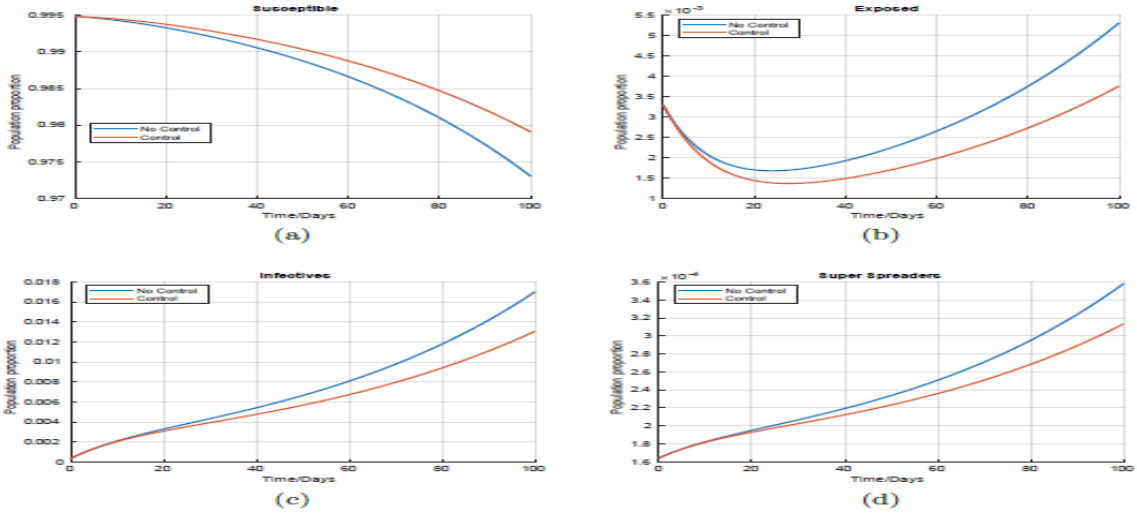
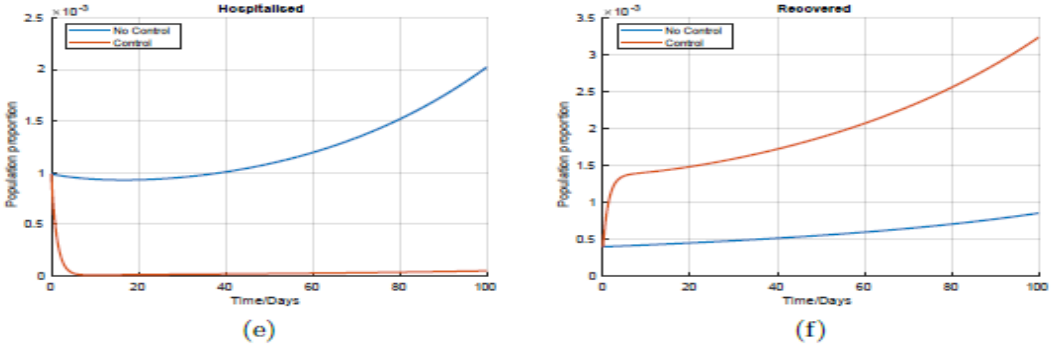


Figure 12: Effect of control strategy u1 on the dynamics of COVID-19 in Ghana

Figure (12), shows the effect of the preventive strategy u1 on the dynamics of COVID-19 in the Ghanaian population. It was observed that in figure 12a the control strategy was not effective on the susceptible class. The results from figure 12b shows that the control strategy was effective in bringing down the exposed population. From the numerical result depicted in figure 12c, it is clearly shown that the control measure was effective in bringing down the infectious class. It was observed that when the control strategy was activated on the super-spreaders class in figure 12d, the control strategy was effective in bringing down the super-spreaders' population. From the numerical result depicted in figure 12e shows that the control measure was highly effective in reducing the hospitalised patients. From the numerical simulation result of figure 12f, the result shows that the activation of the control measure had a positive reduction on the recovered class.

However, we consider the case where the management strategy was implemented at the absence of the preventive strategy.





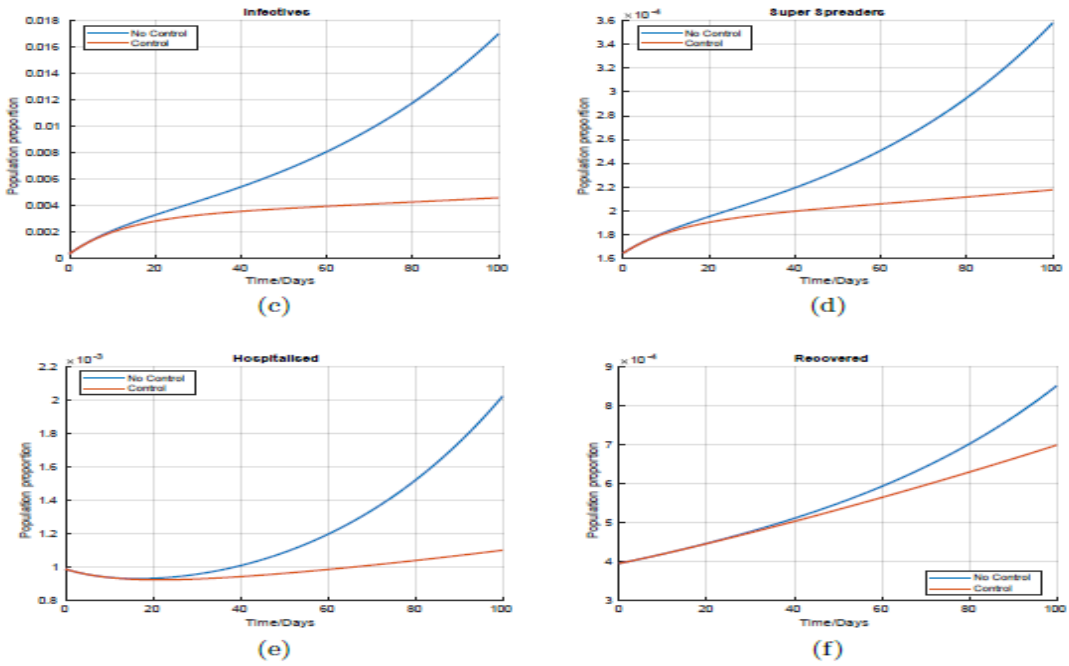


Figure 13: Effect of control strategy u2 on the dynamics of COVID-19 in Ghana

Figure (13), shows the effective of the management strategy u2 on the dynamics of COVID-19 in the Ghanaian population. The strategy focused on effective healthcare management. From the numerical simulation results depicted in figure 13a shows that the control method was not effective on the susceptible class. The result from figure 13b shows that the control measure was very effective in bringing down the exposed class. From the numerical simulation result depicted in figure13c shows that activation of the control strategy was effectivein bringing down the infectious class in the host population. The result from figure 13d shows that the control method was highly effective in reducing the super spreaders class in the host population. From the numerical result depicted in 4.13e shows clearly that the control strategy was very effective in reducing the hospitalised patients. The numerical result from figure 13f shows that the control method was not effective on recovered class in the host population. Lastly, we considered the combination of the preventive and management strategies and how they effect the spread of the virus in the Ghanaian population and the results was that, the combined effect of control strategies from the graph indicated that, the control strategies was not effective on the susceptible. However, the activation of all the control strategies was highly effective in bringing down the exposed class in the host of population.

Conclusion

The purpose of mathematical model is not to make unconditional claims about the consequences of interventions, but reveal the relation between assumptions and outcome" (Weinstein, 2003). In this study, a compartmental ordinary differential equation model to understand the transmission dynamics of COVID-19 was presented. The study incorporated the super spreaders individuals as well as hospitalised individuals into the SEIR model to have the six compartmental model SEIPHR. The study analysed the mathematical model to suggest strategies for effective prevention and control of the epidemic. The study estimated the reproduction number for Ghana outbreak. The reproduction number was estimated using the related parameters. The estimated value of the reproduction number (R0) was 3.42634. When we take the upper bound of the estimated reproduction number which approximate to 4. This means that an infected

person was capable of spreading the disease to 4 persons. The disease outbreak dies out if and the disease is endemic if R0 >1. From the sensitivity analysis, the study revealed clearly that, the most important parameter is the contact rate (β) while the hospitalization of infected individuals (also significantly reduced secondary infections. This clearly demonstrate that reduced contact rate would decline the transmission dynamics of COVID-19 disease. The study further conducted stability analysis. The study revealed that long time term solution of the dynamical system is asymptotically stable, which clearly shows that, in the long run close to 11% of the Ghanaian population is expected to be susceptible to the disease with majority of the population in the recovered compartment. The analysis of the endemic equilibrium points by the study revealed that majority of the population was projected to be in the recovered compartment in the long term, with just around 11%, and still susceptible to the disease. Finally, the study performed optimal control analysis. Pontryagin's maximal principle was used to prove the existence of optimal control controls and characterisation. Different simulation instances were also compared in order to find the optimum control tactics. Using Pontryagin's maximum principle, the model was also modified to include time-dependent optimal control variables (preventive and management strategies) to investigate their impacts in minimizing the impact of COVID-19 in the population. Although the management control method outperformed the preventive control measure in terms of reducing disease transmission dynamics, the combined effort of the two controls had a substantial influence on lowering the number of infectious individuals in the population. The study can deduce from the above optimum control analysis that a low transmission rate and a high recovery rate are required to effectively overcome the disease's spread in the host population.

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