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A Mathematical Model and Analysis for the COVID-19 Infection

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Abstract: The dreaded COVID-19 is a communicable respiratory disease caused by a new strain of coronavirus that causes illness in humans. A study of the transmission dynamics of the disease is essential in the control and elimination of the disease. In this research work, we made some assumptions and employed a deterministic SEIR model in the study of the transmission dynamics of the novel coronavirus disease. A mathematical analysis is performed on the model. This analysis includes the positivity of solutions of the model, boundedness of solution, equilibrium points, basic reproduction number, stability and sensitivity analysis. The effects of some sensitive parameters of the basic reproduction number of the COVID-19 disease are made visible in the numerical solutions of the disease model. These simulations which can be employed as a guide in the control and elimination of the disease shows that individual's compliance to government's laws on the use of facemask and social distancing is a major successful tool to be positively embraced in the fight against this human enemy.

Keywords: COVID-19, SEIR, Model, Stability, Equilibrium, Simulations

Introduction

Over the course of time, deadly diseases have ravaged the entire world at different points of human existence and most of these deadly diseases have caused mankind to live in an ocean of untold suffering and want (Balkhair, 2020). WHO (2020a) stated that over the past decade, more than 19 infectious agents have caused several disease outbreaks and epidemics. A disease pandemic is a form of epidemic which eats into the human population affecting very large number of humans (Muthu, 2005). Samal (2014) revealed that the infectious nature of these diseases makes them a pandemic, not merely because of the fact that they kill people. The newest deadly disease is the coronavirus disease, COVID-19 which is caused by SARS-COV-2, and COVID-19 was declared a public health emergency of international concern on January 30th, 2020 and a pandemic on March 11th, 2020 (Balkhair, 2020). World Health Organisation (2020) revealed that as of 9th April, 2020, COVID-19 has already occupied a dreaded position as one of the worst pandemics in the history of man with over 1.39 million infections in 177 countries and over 85000 deaths globally. COVID-19 was first spotted in a sea food market in Wuhan, China in December, 2019. Imperial College COVID-19 Response Team (2020) opined that proactive containment and suppression measures like contact tracing, travel restrictions, isolation of confirmed cases, case finding, social distancing, closure of institutions, cancelation of large-scale public gatherings, lockdown measures, etc, are viable options to employ in the effective management and control of the COVID-19 pandemic. WHO (2020b) also stated that it is important to note that disease-induced immunity has not been scientifically established for COVID-19 and as such, the use of such "immunity passport" may increase the risk of continued transmission. Mathematical epidemiology has helped scientists to address the problems created by infectious diseases (Hamer, 1906, Kernmack *et al*., 1927, May and Anderson, 1979, Hethcote, 2000, Thieme, 2003).

One of the most common ways to use rates of change is the setting up equation showing the relationships which exist between unknown functions and their rates of change with respect to one or more independent variables. These equations are called differential equations. Meng (2020) proposed the method steps of establishing an ordinary differential equation model, and combined the practical exploration of the application of ordinary differential equations in mathematical modelling. Ordinary differential equations have also been applied to population prediction models (Xiaohua and Min, 2013).

The overall goal of mathematical models is to achieve the inequality R_0<1, where R_0 is a threshold representing

the average number of new cases resulting from one infectious individual in an entirely susceptible population. With R_0<1, the stability of the disease-free equilibrium is guaranteed either in a local or a global sense (Diekmann *et al*, 1990, Perasso, 2018). Different research works have been done on the COVID-19 pandemic and over 5300 publications have been recorded in the database of publications on COVID-19 of the world health organisation (Eikenberry *et al*, 2020). Several works have dealt with the transmission dynamics of COVID-19 disease using SIR and SEIR model, and many research works predicted a decline in secondary infections when all precautionary measures are observed globally. (Zeb *et al*., 2019, Jia *et al*., 2020, Kucharski *et al*., 2020, Prem *et al*., 2020).

In this research work, we build a deterministic model for the COVID-19 disease based on some assumptions. Based on the disease status of individuals, six mutually exclusive compartments are considered. These compartments include the susceptible compartment, the exposed compartment, the asymptomatic infectious compartment, the symptomatic infectious compartment, the hospitalised compartment, and the recovered compartment, which we denote by *S, E, I_A, I_S, H, R* respectively. The positivity and boundedness of solution is considered. We also analyse the equilibrium points and their stability, the basic reproduction number. This paper also considers the sensitivity of the parameters of the basic reproduction number, and some numerical simulations of the model where the effects of two parameters χ and σ (where χ is the rate of transmission and σ is the average compliance level of susceptible individuals to government laws on the use of facemask and the social distancing rule) on the infectious compartments sizes are studied.

Materials and Methods

We formulate a deterministic *SEIR* model of non-linear ordinary differential equations for the coronal virus disease also known as COVID-19. The model divides the population of individuals into six compartments: the susceptible individuals, the exposed individuals, the asymptomatic infectious individuals, the symptomatic infectious individuals, the hospitalised individuals, and the recovered individuals denoted by *S, E, I_A, I_S, H, R* respectively. Basic mathematical analyses are performed on the model. These analyses include establishing the positivity of solution, the invariant region, the disease-free equilibrium, the disease-endemic equilibrium, the basic reproduction number, the stability of the equilibrium points, bifurcation and sensitivity analysis. The Mathematica programming software is employed to perform some simulations on the model while varying some parameter values of the model.

Model Formulation

The per capita recruitment rate is Λ and it is assumed to occur only in the susceptible class. Susceptible individuals (*S*) become exposed at the rate $(1-\sigma)\varpi$ where ϖ is the force of infection given by $\varpi = \frac{\chi\left(I_A + \varsigma_1 I_S + \varsigma_2 H\right)}{N}$ and $0 \le \sigma \le 1$ is the average compliance level of susceptible individuals to government laws on the use of facemask and the social distancing rule. The rate of transmission is given by $\chi = k\tau$, where k is the contact rate, and τ is the probability that a contact is effective enough to cause infection. $0 \le \varsigma 1 \le 1$, $0 \le \varsigma 2 \le 1$ $0 \le \varsigma_2 \le$ are the transmission coefficients of the symptomatic infectious (I_S) and hospitalised individuals (*H*). The exposed individuals (*E*) become infectious at the rate γ, with p proportion becoming asymptomatic and (1-p) proportion becoming symptomatic. The symptomatic class (I_S) is also increased by the asymptomatic individuals (I_A) at the rate ψ. It is assumed that individuals who are tested and confirmed positive are hospitalised, hence the hospitalised compartment is increased by individuals from the exposed class, the asymptomatic class, and the symptomatic class at the rate ϕ. The recovery rate for infected individuals not hospitalised is ξ, while the recovery rate for the hospitalised individuals receiving treatment is ζ. The disease-induced death rate is η and it occurs only in the infectious classes. We assume there is no disease-induced immunity, hence, after some time, some recovered individuals move back into the susceptible class at the rate δ. The natural death rate is μ and it occurs in all compartments. The model flow diagram is given in Fig. 1:

$$
\begin{cases}
\frac{dS(t)}{dt} = \Lambda + \delta R(t) - \left((1-\sigma)\varpi + \mu\right)S(t) \\
\frac{dE(t)}{dt} = (1-\sigma)\varpi S(t) - \left(\gamma + \phi + \xi + \mu\right)E(t) \\
\frac{dI_A(t)}{dt} = p\gamma E(t) - \left(\psi + \phi + \xi + \eta + \mu\right)I_A(t) \\
\frac{dI_s(t)}{dt} = \left(1-p\right)\gamma E(t) + \psi I_A(t) - \left(\phi + \xi + \eta + \mu\right)I_s(t) \\
\frac{dH(t)}{dt} = \phi\left(I_A(t) + I_s(t) + E(t)\right) - \left(\zeta + \eta + \mu\right)H(t) \\
\frac{dR(t)}{dt} = \xi\left(E(t) + I_A(t) + I_S(t)\right) + \zeta H(t) - (\delta + \mu)R(t) \\
\varpi = \frac{\chi\left(I_A + \varsigma_1 I_S + \varsigma_2 H\right)}{N}, \chi = k\tau, \\
N(t) = S(t) + E(t) + I_A(t) + I_S(t) + H(t) + R(t), \\
S(0) = S_0, \quad E(0) = E_0, \quad I_A(0) = I_{A_0}, \\
I_s(0) = I_{s_0}, \quad H(0) = H_0, \quad R(0) = R_0.
\end{cases}
\tag{1}
$$

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Assumptions of the Model

The model is based on the following assumptions.

1. Per-capita recruitment occurs only in the susceptible compartment
2. Disease-induced death only occurs in the infectious compartments
3. Individuals leave the hospitalised compartment if and only if they have been discharged or they die
4. Testing rate influences hospitalisation rate, hence hospitalization rate is the same for the exposed, the asymptomatic and the symptomatic classes
5. Recovery rate differs for individuals hospitalized and not hospitalized
6. There is no disease-induced immunity

Descriptions of Variables and Parameters

The descriptions of all variables and parameters of the model are clearly presented here as Table 1.

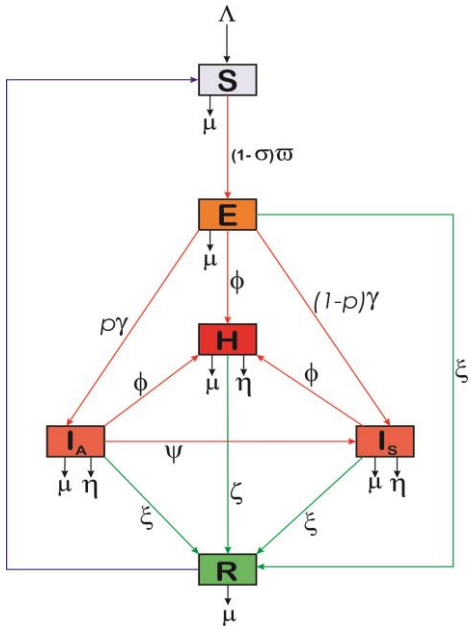

Fig. 1: Schematic diagram

Table 1: Variables and Parameters

Variables	Description
S(t)	Susceptible individuals at time t
E(t)	Exposed individuals at time t
IA(t)	Asymptomatic infectious individuals at time t
IS(t)	Symptomatic infectious individuals at time t
H(t)	Hospitalised individuals at time t
R(t)	Recovered individuals at time t
	Parameters description
Λ	Per capita recruitment rate into the susceptible compartment
ω	Force of infection of the susceptible individuals
σ	Compliance level of susceptible individuals to government's laws on the use of facemask and social distancing, measured on a scale of 0-1
γ	Rate at which exposed individuals become infectious
p	Proportion of exposed individuals that becomes infectious but asymptomatic
ϕ	Rate at which infected individuals become hospitalised
ξ	Recovery rate of exposed and infectious individuals
ζ	Recovery rate of the hospitalised individuals
χ	Rate of transmission
k	Contact rate
τ	Probability that a contact is effective enough to cause infection
ζ_1	Transmission coefficient of the symptomatic individuals
ζ2	Transmission coefficient of the hospitalised individuals
ψ	Rate at which asymptomatic individuals become symptomatic
δ	Rate at which recovered individuals become susceptible
η	Disease-induced death rate
μ	Natural death rate

Initial Values S(0) = 206,011,565, E(0) = 28, 074, IA(0) = 21,000, IS(0) = 17,000 H(0) = 10,943, R(0) = 43,998

Results

In this section, we present our results starting with the basic results such as non-negativity of solution, where we establish that the solution of the model is non-negative for all values of time, t. The invariant region and boundedness of solution is also presented in this section. Boundedness of solution ensures that the size of each compartment, and hence the total population size, is bounded by a positive constant, K.

Non-Negativity of Solution

We want to verify that the solution of the sys. 1 is non-negative for all values of t. Let us consider the following theorem.

Theorem 1 (Positivity of Solution)

Suppose $\Gamma = \{(S,E,I_A,I_S,H,R) \in \mathbb{R}^6 : S(0)>0, E(0)>0, I_A(0)>0, I_S(0)>0, H(0)>0, R(0)>0\}$, *then the solution set* $\{S, E, I, R\}$ *is positive for all* $t \geq 0$.

Proof

Let us consider the first equation of system (1):

$$\frac{dS(t)}{dt} = \Lambda + \delta R(t) - \big((1-\sigma)\varpi + \mu\big)S(t).$$

Observe that:

$$\frac{dS(t)}{dt} \geq -\big((1-\sigma)\varpi + \mu\big)S(t)$$

By separation of variables, and applying the initial condition $S(0) = S_0 \geq 0$, we obtain:

$$S(t) \geq S_0 e^{-\big((1-\sigma)\varpi+\mu\big)t} \geq 0 \quad (2)$$

Similarly:

$$E(t)\geq 0, I_A(t)\geq 0, I_s(t)\geq 0, H(t)\geq 0, R(t)\geq 0 \; \forall t \geq 0.$$

Thus, the solution of the model (1) is positive for all values of $t \geq 0$. This completes the proof.

Invariant Region and Boundedness of Solution

Another way to establish that the sys. 1 is well-posed is to study the invariant region in which the solution to the sys. 1 is biologically relevant. Consider the following theorem.

Theorem 2: The set

$$\Gamma = \{(S,E,I_A,I_S,H,R) \in \mathbb{R}^6_+ : 0 \leq S+E+I_A+I_S+H+R = N \leq \Lambda/\mu\} \quad (3)$$

Is positively-invariant for the model 1.

Proof

Consider the total population $N(t)$ of individuals at time t, given by:

$$N(t) = S(t) + E(t) + I_A(t) + I_s(t) + H(t) + R(t)$$

Observe that:

$$N(t) = \Lambda - \eta\big(I_A(t) + I_s(t) + H(t)\big)$$
$$-\mu N(t) \leq \Lambda - \mu N(t)$$

By separation of variables, we obtain:

$$N(t) \leq \frac{\Lambda}{\mu} - \frac{c_3 e^{-\mu t}}{\mu}$$

Taking limit as $t \to \infty$, we obtain:

$$N(t) \leq \frac{\Lambda}{\mu} \quad (4)$$

Equation 4 is referred to as the threshold population level. Therefore, the feasible solution set of system (1) enters and remains in the region: $\Gamma = \left\{(S,E,I_A,I_S,H,R) \in \mathbb{R}^6_+ : 0 \leq S+E+I_A(t)+I_S(t)+H(t)+R = N \leq \frac{\Lambda}{\mu}\right\}$. If the population is higher than the threshold population level, the population of individuals reduces asymptotically to the carrying capacity. If $N \leq \frac{\Lambda}{\mu}$, then the solution of the sys. 1 remains in Γ for all t > 0. Therefore, the region Γ is positively invariant. This completes the proof.

Disease-Free Equilibrium

The point where the human population is free from the dreaded COVID-19 is worth analysing. This is the Disease-Free Equilibrium (DFE) point of the model (1). It is denoted by $\mathbb{E}_0$. We obtain $\mathbb{E}_0$ by solving system (1) with the right hand side equated to zero and letting $E = I_A = I_S = H = R = 0$ Thus, we obtain:

$$\mathbb{E}_0 = \left(S^0, E^0, I_A^0, I_S^0, H^0, R^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right) \tag{5}$$

Theorem 3:

The model (1) admits a unique DFE.

Proof

Substituting E_0 into the system (2) shows that all the derivatives are equal to zero, hence, the DFE is unique. This proves the theorem.

Basic Reproduction Number

We now turn our attention to the average number of secondary infections caused by a single infectious COVID-19 patient within an entirely susceptible population during his/her infective period. This number which is referred to as the basic reproduction R_0 is a very important dimensionless quantity in mathematical epidemiology. The next generation matrix due to is employed in obtaining R_0. Let us consider the infected compartments $X(t) = (E(t)), I_A(t), I_S(t), H(t))$ in the form $X'(t) = \mathcal{F}(t) - v(t)$ where:

$$\mathcal{F} = \begin{bmatrix} (1-\sigma)\varpi S(t) \\ 0 \\ 0 \\ 0 \end{bmatrix} and$$

$$v = \begin{bmatrix} (\gamma+\phi+\xi+\mu)E(t) \\ -p\gamma E(t) + (\psi+\phi+\xi+\eta+\mu)I_A(t) \\ -(1-p)\gamma E(t) - \psi I_A(t) + (\phi+\xi+\eta+\mu)I_S(t) \\ -\phi\left(I_A(t) + I_S(t) + E(t)\right) + (\zeta+\eta+\mu)H(t) \end{bmatrix}.$$

Evaluating the Jacobian of the matrices $\mathcal{F}$ and $\mathcal{V}$, respectively, at the disease-free equilibrium we obtain:

$$F = \begin{bmatrix} 0 & (1-\sigma)\chi\varsigma_1 & \chi(1-\sigma)\varsigma\chi(1-\sigma) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, and$$

$$V = \begin{bmatrix} \gamma+\phi+\xi+\mu & 0 & 0 & 0 \\ -p\gamma & \psi+\phi+\xi+\eta+\mu & 0 & 0 \\ -(1-p)\gamma & -\psi & \phi+\xi+\eta+\mu & 0 \\ -\phi & -\phi & -\phi & \zeta+\eta+\mu \end{bmatrix}$$

$$\therefore V^{-1} = \begin{bmatrix} \frac{1}{\gamma+\mu+\xi+\phi} & 0 & 0 & 0 \\ \frac{p\gamma}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)} & \frac{1}{\eta+\mu+\xi+\phi+\psi} & 0 & 0 \\ \frac{\gamma(\psi-(p-1)(\eta+\mu+\xi+\phi))}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi)(\mu+\mu+\xi+\phi+\psi)} & \frac{\psi}{(\eta+\mu+\xi+\phi+\psi)} & \frac{1}{\eta+\mu+\xi+\phi} & 0 \\ \frac{\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi)} & \frac{\phi}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi+\psi)} & \frac{\phi}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)} & \frac{1}{\zeta+\eta+\mu} \end{bmatrix}$$

and hence:

$$FV^{-1} = \begin{bmatrix} k_1 & k_2 & k_3 & k_4 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, where\, k_1 = \frac{\frac{p\gamma(\sigma-1)\chi}{\eta+\mu+\xi+\phi+\psi} + \frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)\chi(1-\sigma)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)} - \frac{\gamma\varsigma_1\left((p-1)(\eta+\mu+\xi+\phi)(\sigma-1)\right)}{(\eta+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)}}{\gamma+\mu+\xi+\phi},$$

$$k_2 = \frac{\chi-\sigma\chi}{\eta+\mu+\xi+\phi+\psi} + \frac{\left(\frac{\zeta_2\phi}{\zeta+\eta+\mu} + \frac{\zeta_2\psi}{\eta+\mu+\xi+\phi+\psi}\right)\chi(1-\sigma)}{\eta+\mu+\xi+\phi}, k_3 = \frac{\left((\zeta+\eta+\mu)\zeta_1 + \zeta_2\phi\right)\chi(1-\sigma)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}, k_4 = \frac{\zeta_2\chi(1-\sigma)}{\zeta+\eta+\mu}.$$

Using $\left|FV^{(-1)} - \lambda I\right| = 0$ we obtain the following eigenvalues for the matrix FV^{-1}:

$$\lambda_1 = \lambda_2 = \lambda_3 = 0, and$$

$$\lambda_4 = \frac{\frac{\varsigma_2\chi\phi(1-\sigma)(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)} - \frac{p\gamma(\sigma-1)\chi}{\eta+\mu+\xi+\phi+\psi} - \frac{\varsigma_1\chi\gamma\left((p-1)(\eta+\mu+\xi+\phi)-\psi\right)}{(\eta+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)}}{\gamma+\mu+\xi+\phi}$$

It follows that the spectral radius and hence the basic reproduction (R_0) number is given by:

$$R_0 = \frac{p\gamma(1-\sigma)\chi}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)} + \frac{\varsigma_1\chi\gamma(1-\sigma)\left((1-p)(\eta+\mu+\xi+\phi)+\psi\right)}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)(\eta+\mu+\xi+\phi)} + \frac{\varsigma_2\phi\chi(1-\sigma)(\gamma+\mu+\xi+\phi)}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi)(\zeta+\eta+\mu)}. \tag{6}$$

It is clearly seen that the basic reproduction number (R_0) above is sponsored by a combination of three sets of

individuals. The first term shows that when an infectious individual is introduced into an entirely susceptible population, susceptible individuals become exposed and a proportion of these exposed individuals become infectious but asymptomatic. The total time spent by this proportion both in the exposed and asymptomatic compartments is given by $\frac{1}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)}$ and the combined rate is $p\gamma(1-\sigma)\chi$. The second term accounts for susceptible individuals who become exposed, infectious without symptoms but later developed symptoms of the disease. The total time spent by this proportion is $\frac{1}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)(\eta+\mu+\xi+\phi)}$ and the combined rate is $\varsigma_1\chi\gamma(1-\sigma)\big((1-p)(\eta+\mu+\xi+\phi)+\psi\big)$. The third term accounts for individuals who become exposed, then infectious with symptoms of the COVID-19 disease, and are later hospitalised. The total time spent by this proportion is $\frac{1}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+)(\varsigma+\eta+\mu)}$ and the combined rate is $\zeta_2\phi\chi(1-\sigma)(\gamma+\eta+\mu+\xi+\phi)$.

Endemic Equilibrium

We now uncover the equilibrium point where the disease is persistent in the population. This is the endemic equilibrium point $\mathbb{E}_*$of the model (1). To obtain this point, we equate the system (1) to zero and solve the resulting steady state system. For simplicity, we set:

$$s=\frac{S}{N},e=\frac{E}{N},i_A=\frac{I_A}{N},i_s=\frac{I_s}{N},h=\frac{H}{N},\gamma=\frac{R}{N}$$

and note that $\varpi_e=\chi\left(i_A^*+\varsigma_1 i_s^*+\varsigma_2 h^*\right)$.Thus, we obtain $E_*=\left(s^*,e^*,i_A^*,i_S^*,h^*,r^*\right)$ as given below:

$$\begin{cases} s^*=ai_{s_e} \\ e^*=-\dfrac{(\eta+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)}{(-1+p)\lambda(\eta+\mu+\xi+\phi)-\gamma\psi}i_s^* \\ i_A^*=-\dfrac{p(\eta+\mu+\xi+\phi)}{(-1+p)(\eta+\mu+\xi+\phi)-\psi}i_s^* \\ i_s^*=i_s^* \\ h^*=-\dfrac{\phi(\gamma+\eta+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)}{\gamma(\varsigma+\eta+\mu)\big((-1+p)(\eta+\mu+\xi+\phi)-\psi\big)}i_s^* \\ r^*=-\dfrac{(\gamma+\eta+\mu+\xi+\phi)\big((\varsigma+\eta+\mu)\xi+\varsigma\phi\big)(\eta+\mu+\xi+\phi+\psi)}{\gamma(\varsigma+\mu)(\varsigma+\eta+\mu)\big((-1+p)(\eta+\mu+\xi+\phi)-\psi\big)}i_s^* \end{cases} \tag{7}$$

where:

$$a=\Big(\big(-\gamma-\mu-\xi-\phi\big)\big(-\eta-\mu-\xi-\phi\big)\chi\big(-\eta-\mu-\xi-\phi-\psi\big)p\gamma\big(\zeta+\eta+\mu\big)\big(-1+\zeta_1\big)\big(\eta+\mu+\xi+\phi\big)-\big(\gamma\big(\zeta+\eta+\mu\big)\big)\zeta 1+\gamma\zeta_2\phi+\zeta_2\phi(\eta+\mu+\xi+\phi)\Big)$$
$$\big(\eta+\mu+\xi+\phi+\psi\big)\big)\big)/(R_0\gamma(\zeta+\eta+\mu)(\gamma+\eta+\mu)(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi)((-1+p)(\eta+\mu+\xi+\phi)-\psi\omega).$$
$$i_s^*=-(R_0\gamma\Lambda(-\delta-\mu)(-\zeta-\eta-\mu)((-1+p)(\eta+\mu+\xi+\phi)-\psi\omega)/(-p\gamma\mu(\delta+\mu)(\zeta+\eta+\mu)(-1+\zeta_1)(\eta+\mu+\xi+\phi)\chi+(\eta+\mu+\xi+\phi+\psi)$$
$$(\eta+\mu+\xi+\phi+\psi)(\gamma\mu(\delta+\mu)((\zeta+\eta+\mu)((\zeta+\eta+\mu)\zeta_1+\zeta_2\phi)\chi+R_0\gamma(\delta(\eta+\mu)(\zeta+\eta+\mu)+(\zeta+\eta+\mu+\phi)+\mu\big(\zeta+\eta+\mu\big)\big(\eta+\mu+\xi+\phi\big)\big)\omega+$$
$$(\eta+\mu+\xi+\phi)(\eta+\mu+\xi+\phi)(\mu(\delta+\mu)\zeta_2\phi\chi+R_0(\delta\mu(\zeta+\eta+\mu)+\delta(\eta+\mu)\phi+\mu(\zeta+\eta+\mu)(\mu+\xi+\phi))).$$

Stability Analysis

We now examine the stability of the equilibrium points. Here, we consider the local stability of the DFE and the global stability of DEE. They are presented in the theorems below.

Theorem 4

The DFE is locally asymptotically stable if $R_0 < 1$. It is unstable whenever $R_0 > 1$.

Proof

Consider and evaluate the Jacobian matrix for the coupled system of Eq. (1) at the disease-free equilibrium. This Jacobian matrix is denoted by $J_{\mathbb{E}0}$ and is given below:

$$J_{\mathbb{E}_0}=\begin{bmatrix} -\mu & 0 & (-1+\sigma)\chi & (-1+\sigma)\varsigma_1\chi & (-1+\sigma)\xi_2\chi & \delta \\ 0 & -\gamma-\mu-\xi-\phi & -(-1+\sigma)\chi & -(-1+\sigma)\varsigma_1\chi & -(-1+\sigma)\xi_2\chi & 0 \\ 0 & p\gamma & -\eta-\mu-\xi-\phi-\phi & 0 & 0 & 0 \\ 0 & \gamma-p\gamma & \psi & -\gamma-\mu-\xi-\phi & 0 & 0 \\ 0 & \phi & \phi & \phi & -(\varsigma+\eta+\mu) & 0 \\ 0 & \xi & \xi & \xi & \xi & -(\delta+\mu) \end{bmatrix}$$

Observe that:

$$trace(J_{\mathbb{E}e})=-\big(\gamma+\delta+\zeta+3\eta+6\mu+3\xi+3\phi+\psi\big)<0.$$
$$det(J_{\mathbb{E}0})=-\big(-1+R_0\big)\mu(\delta+\mu)\big(\zeta+\eta+\mu\big)\big(\gamma+\mu+\xi+\phi\big)$$
$$\big(\eta+\mu+\xi+\phi\big)\big(\eta+\mu+\xi+\phi+\psi\big)$$

Noting that, from the expression of R_0 given in Eq. 6:

$$\chi=-\frac{R_0(\gamma+\mu+\xi+\phi)}{(-1+\sigma)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma^2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}-\frac{p\gamma(-1+\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)}. \tag{8}$$

Now, using the condition that det (J_{E0}) > 0, we obtain:

$$R_0<1.$$

Therefore, the DFE ($\mathbb{E}_0$) is locally asymptotically stable whenever $R_0<1$. This completes the proof.

Theorem 5

The DFE is globally asymptotically stable if $R_0 \le 1$.

Proof

To prove the global asymptotic stability of the DFE, we use the method of Lyapunov functions. Let us define the Lyapunov function L as:

$$L = \frac{1}{\left((1-\sigma)\varpi+\mu\right)s}S + \frac{1}{\left(\gamma+\phi+\xi+\mu\right)}E + \frac{1}{\left(\psi+\phi+\xi+\eta+\mu\right)}I_A + \frac{1}{\left(\phi+\xi+\eta+\mu\right)}I_S + \frac{1}{\left(\zeta+\eta+\mu\right)}H + \frac{1}{\left(\zeta+\eta+\mu\right)} + \frac{1}{\left(\delta+\mu\right)}R.$$

Then:

$$\frac{dL}{dt} = \frac{1}{\left((1-\sigma)\varpi+\mu\right)}\frac{dS}{dt} + \frac{1}{\left(\gamma+\phi+\xi+\mu\right)}\frac{dE}{dt} + \frac{1}{\left(\phi+\xi+\eta+\mu\right)}\frac{dI_S}{dt} + \frac{1}{\left(\zeta+\eta+\mu\right)} + \frac{dH}{dt} + \frac{1}{\left(\delta+\mu\right)}\frac{dR}{dt}$$

$$\Rightarrow \frac{dL}{dt} = \frac{\Lambda+\delta R}{\left((1-\sigma)\varpi+\mu\right)} - S + \frac{(1-\sigma)\varpi S}{\left(\gamma+\phi+\xi+\mu\right)} - E + \frac{p\gamma E}{\left(\psi+\phi+\xi+\eta+\mu\right)} - I_A + \frac{(1-p)\gamma E+\psi I_A}{\left(\phi+\xi+\eta+\mu\right)} - I_S + \frac{\phi\left(I_A+I_S+E\right)}{\left(\zeta+\eta+\mu\right)} - H + \frac{\xi\left(E+I_A+I_S\right)+\xi H(t)}{\left(\delta+\mu\right)} - R$$

$$\Rightarrow \frac{dL}{dt} = \frac{\Lambda}{\left((1-\sigma)\varpi+\mu\right)} - S + \left(\frac{p\gamma}{\left(\psi+\phi+\xi+\eta+\mu\right)} + \frac{(1-p)\gamma}{\left(\phi+\xi+\eta+\mu\right)} + \frac{\phi}{\left(\zeta+\eta+\mu\right)} + \frac{\zeta}{\left(\delta+\mu\right)} - 1\right)E + \left(\frac{\chi(1-\sigma)s}{N\left(\gamma+\phi+\xi+\mu\right)} + \frac{\psi}{\left(\phi+\xi+\eta+\mu\right)} + \frac{\phi}{\left(\zeta+\eta+\mu\right)} + \frac{\xi}{\left(\delta+\mu\right)} - 1\right)$$

$$I_A + \left(\frac{\chi\varsigma_1(1-\sigma)\delta}{N\left(\gamma+\phi+\xi+\mu\right)} + \frac{\phi}{\left(\zeta+\eta+\mu\right)} + \frac{\xi}{\left(\delta+\mu\right)} - 1\right)I_S + \left(\frac{(1-\sigma)\chi\varsigma_2 s}{N\left(\gamma+\phi+\xi+\mu\right)} + \frac{\zeta}{\left(\delta+\mu\right)} - 1\right)H + \left(\frac{\delta}{\left((1-\sigma)\varpi+\mu\right)} - 1\right)R$$

$$\Rightarrow \frac{dL}{dt} \le \frac{\Lambda}{\mu}\left(\frac{\mu}{\left((1-\sigma)\varpi+\mu\right)} - 1\right) + \left(\frac{p\gamma}{\left(\psi+\phi+\xi+\eta+\mu\right)} + \frac{(1-p)\gamma}{\left(\phi+\xi+\eta+\mu\right)} + \frac{\phi}{\left(\zeta+\eta+\mu\right)} + \frac{\xi}{\left(\delta+\mu\right)} - 1\right)E + \left(\frac{\chi(1-\sigma)\Lambda}{\mu N\left(\gamma+\phi+\xi+\mu\right)} + \frac{\psi}{\left(\phi+\xi+\eta+\mu\right)} + \frac{\phi}{\left(\zeta+\eta+\mu\right)} + \frac{\xi}{\left(\delta+\mu\right)} - 1\right)$$

$$I_A + \left(\frac{\chi\varsigma_1(1-\sigma)\Lambda}{\mu N\left(\gamma+\phi+\xi+\mu\right)} + \frac{\phi}{\left(\zeta+\eta+\mu\right)} + \frac{\xi}{\left(\delta+\mu\right)} - 1\right)I_S + \left(\frac{(1-\sigma)\chi\varsigma_2\Lambda}{\mu N\left(\gamma+\phi+\xi+\mu\right)} + \frac{\xi}{\left(\delta+\mu\right)} - 1\right)H + \left(\frac{\delta}{\left((1-\sigma)\varpi+\mu\right)} - 1\right)R$$

$$\Rightarrow \frac{dL}{dt} \le \frac{\Lambda}{\mu}\left(R_0 - 1\right) + \left(R_0 - 1\right)E + \left(R_0 - 1\right)I_A + \left(R_0 - 1\right)I_S + \left(R_0 - 1\right)H + \left(R_0 - 1\right)R$$

$$\Rightarrow \frac{dL}{dt} \le \left(R_0 - 1\right)\left(\frac{\Lambda}{\mu} + E + I_A + I_S + H + R\right)$$

So $\frac{dL}{dt} \le 0$ if $R_0 < 1$. Observe that $\frac{dL}{dt} = 0$ if the disease free equilibrium $\left(\frac{\Lambda}{\mu}, 0, 0, 0, 00\right)$. It follows immediately by the principles of [28] that the disease-free equilibrium is globally asymptotically stable if $R_0 \le 1$.

Theorem 6

If $R_0 > 1$, the endemic equilibrium $\mathbb{E}_e$ of the system (1) is globally asymptotically stable.

Proof

In investigating the nature of R_0 for the global stability of the disease endemic equilibrium, we employ the method of Lyapunov functions. Define:

$$L(S^*, E^*, I_A^*, I_S^*, H, R^*) = \left(S - S^* - S^* ln\frac{S}{S^*}\right) + \left(E - E^* - E^* ln\frac{E}{E^*}\right) + \left(I_A - I_A^* - I_A^* ln\frac{I_A}{I_A^*}\right)$$
$$+ \left(I_S - I_S^* - I_S^* ln\frac{I_S}{I_S^*}\right) + \left(H - H - H ln\frac{H}{H^*}\right) + \left(R - R^* - R^* ln\frac{R}{R^*}\right)$$

Calculating the derivative of L along the solution of (1), we obtained:

$$\frac{dL}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{E^*}{E}\right)\frac{dE}{dt} + \left(1 - \frac{I_A^*}{I_A}\right)\frac{dI}{dt}$$
$$+ \left(1 - \frac{I_S^*}{I_S}\right)\frac{dI}{dt}\left(1 - \frac{H^*}{H}\right) + \frac{dH}{dt} + \left(1 - \frac{R^*}{R}\right)\frac{dR}{dt} \therefore \frac{dL}{dt} = A - B \quad (9)$$

where:

$$A = \Lambda\delta R + \left((1-\sigma)\varpi+\mu\right)S^* + (1-\sigma)\varpi S + \left(\gamma+\phi+\xi+\mu\right)E^*$$
$$+ p\gamma E + \left(\psi+\phi+\xi+\eta+\mu\right)I_A^* + (1-p)$$
$$\gamma E + \psi I_A + \left(\phi+\xi+\eta+\mu\right)I_S^* + \phi\left(I_A+I_S+E\right)$$
$$+ \left(\zeta+\eta+\mu\right)H^* + \xi\left(E+I_A+I_S\right) + \zeta H + \left(\delta+\mu\right)R^*$$

$$B + \left((1-\sigma)\varpi+\mu\right)S + \frac{\Lambda S^*}{S} + \frac{\delta RS^*}{S} + \left(\gamma+\phi+\xi+\mu\right)$$

$$E + \frac{(1-\sigma)\varpi SE^*}{E} + \left(\psi+\phi+\xi+\eta+\mu\right)I_A + \frac{p\gamma EI_A^*}{I_A} + \phi + \xi +$$

$$\eta+\mu)I_S + \frac{(1-p)\gamma EI_S^*}{I_S} + \frac{\Psi I_A I_S^*}{I_S} + \left(\zeta+\eta+\mu\right)$$

$$H + \frac{\phi(I_A+I_S-E)H^*}{H} + \left(\delta+\mu\right)R + \frac{\xi\left(E+I_A+I_A\right)R^*}{R} + \frac{\zeta HR^*}{R}$$

Observe that:

i. $\frac{dL}{dt} = 0 iff\ S = S^*, E = E^*, I_A = I_A^*, I_S = I_S^*, H = H^*, R = R^*,$

ii. $\frac{dL}{dt} \le 0 if\ A \le B$

It follows that the singleton set $\mathbb{E}^*$ which is the endemic equilibrium of the system (1) is the largest compact invariant set in $\left\{\left(S, E, I_A, I_S, H, R\right) \in \Gamma : \frac{dL}{dt} = 0\right\}$. Therefore, $\mathbb{E}^*$ is globally asymptotically stable in Γ if $A \le B$ [2].

Sensitivity Analysis

Sensitivity analysis shows which parameter has high impact on R_0. The approach used by Kizito and Tumwiine (2018) is employed here to compute the sensitivity indices of the parameters The normalized forward sensitivity index of R_0 that depends differentiability index on a parameter υ, is defined as:

$$\zeta_{\upsilon}^{R_0} = \frac{\partial R_0}{\partial_{\upsilon}} \times \frac{\upsilon}{R_0} \tag{10}$$

From Eq. 6 and 10, we obtain the following results:

$$\zeta_{\chi}^{R_0} = 1 > 0;$$

$$\zeta_{\sigma}^{R_0} = -\frac{\sigma}{1-\sigma} < 0;$$

$$\zeta_{\phi}^{R_0} = \frac{b_1}{b_2} < 0;$$

$$\zeta_{\mu}^{R_0} = \frac{b_3}{b_4} < 0;$$

$$\zeta_{p}^{R_0} = \frac{p\gamma(1-\varsigma_1)}{(\eta+\mu+\xi+\phi+\psi)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)} > 0;$$

$$\zeta_{\gamma}^{R_0} = \frac{\gamma\left(\frac{(\zeta+\eta+\mu)(\mu+\xi)\varsigma_1+((\zeta+\eta+\mu)\varsigma_1-\eta\varsigma_2)\phi}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p(1-\varsigma_1)(\mu+\xi+\phi+\psi)}{\eta+\mu+\xi+\phi+\psi}\right)}{(\gamma+\mu+\xi+\phi)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)} > 0;$$

$$\zeta_{\varsigma_1}^{R_0} = \frac{\gamma(1-\sigma)\varsigma_1\left((1-p)(\eta+\mu+\xi+\phi)+\psi\right)}{(1-\sigma)(\eta+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)} > 0;$$

$$\zeta_{\psi}^{R_0} = -\frac{p\gamma(\zeta+\eta+\mu)(1-\varsigma_1)(\eta+\mu+\xi+\phi)\psi}{b_0} < 0;$$

$$\zeta_{\varsigma_2}^{R_0} = \frac{(1-\sigma)\varsigma_2\phi(\gamma+\gamma+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(1-\sigma)(\eta+\mu+\xi+\phi)\left(\frac{\gamma\varsigma_1}{(\eta+\mu+\xi+\phi)}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)} > 0;$$

$$\zeta_{\eta}^{R_0} = -\frac{\eta\left(\frac{\gamma\varsigma_1}{(\eta+\mu+\xi+\phi)^2}+\frac{\varsigma_2\phi\left((\eta+\mu+\xi+\phi)\right)^2+\gamma(\zeta+2\eta+2\mu+\xi+\phi)}{(\zeta+\eta+\mu)^2+(\eta+\mu+\xi+\phi)^2}+\frac{p\gamma(1-\varsigma_1)}{(\eta+\mu+\xi+\phi+\psi)^2}\right)}{(\gamma+\mu+\xi+\phi)^2\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)} < 0;$$

$$\zeta_{\zeta}^{R_0} = -\frac{\zeta(1-\sigma)\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)^2(1-\sigma)(\gamma+\mu+\xi+\phi)^2(\eta+\mu+\xi+\phi)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)} < 0;$$

$$\zeta_{\xi}^{R_0} = \frac{\xi\left(\frac{\varsigma_2\phi\left(\gamma^2+(\eta+\mu+\xi+\phi)^2+\gamma(\eta+2(\mu+\xi+\phi))\right)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)^2}+\gamma\left(\frac{\varsigma_1(\gamma+\eta+2(\eta+\xi+\phi))}{(\eta+\mu+\xi+\phi)^2}+\frac{p-p\varsigma_1}{\eta+\mu+\xi+\phi+\psi}\right)\right)}{(\gamma+\mu+\xi+\phi)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right)} < 0.$$

where:

$$b_0 = (\eta+\mu+\xi+\phi+\psi)\left(p\gamma(\zeta+\eta+\mu)(1-\varsigma_1)(\eta+\mu+\xi+\phi)+\left(\gamma(\zeta+\eta+\mu)\varsigma_1+\gamma\varsigma_2\phi+\varsigma_2\phi\right)(\eta+\mu+\xi+\phi)\right)(\eta+\mu+\xi+\phi+\psi)),$$

$$b_1 = \phi\left(\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma}{\eta+\mu+\xi+\phi+\psi}+(\gamma+\mu+\xi+\phi)\left(\frac{\gamma(\zeta+\eta+\mu)\varsigma_1-\gamma(\eta+\mu+\xi)\varsigma_2-\varsigma_2(\eta+\mu+\xi+\phi)^2}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)^2}+\frac{p\gamma(1-\varsigma_1)}{(\eta+\mu+\xi+\phi+\psi)^2}\right)+\gamma\varsigma_1\left(\frac{1}{\eta+\mu+\xi+\phi}-\frac{p}{\eta+\mu+\xi+\phi+\psi}\right)\right),$$

$$b_2 = (\gamma+\mu+\xi+\phi)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right),$$

$$b_3 = \mu\left(\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma}{\eta+\mu+\xi+\phi+\psi}+(\gamma+\mu+\xi+\phi)\left(\frac{\gamma\varsigma_1}{(\eta+\mu+\xi+\phi)^2}+\frac{\varsigma_2\phi\left((\eta+\mu+\xi+\phi)^2+\gamma(\zeta+2\eta+2\mu+\xi+\phi)\right)}{(\zeta+\eta+\mu)^2(\eta+\mu+\xi+\phi)^2}+\frac{p\gamma(1-\varsigma_1)}{(\eta+\mu+\xi+\phi+\psi)^2}\right)+\gamma\varsigma_1\left(\frac{1}{\eta+\mu+\xi+\phi}-\frac{p}{\eta+\mu+\xi+\phi+\psi}\right)\right),$$

$$b_3 = (\gamma+\mu+\xi+\phi)^3\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi)}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi)}+\frac{p\gamma(1-\varsigma_1)}{\eta+\mu+\xi+\phi+\psi}\right).$$

We observe that χ, p, γ, ζ_1 and ζ_2 are the most sensitive parameters of the basic reproduction number R_0. Increasing the values of these parameters (especially the value of χ whose sensitivity index is +1), while keeping the other parameters fixed, increases the value of R_0. Also keeping the values of χ, p, γ, ζ_1 and ζ_2 fixed while increasing the values of the other parameters of R_0, decreases the value of R_0.

Bifurcation Analysis

In this section, we shall employ the centre manifold theorem contained in the theorem below. The parameter values in Table 2 are also substituted, when necessary, in order to simplify expressions.

Theorem (Castillo-Chavez and Song, 2004):

Consider the following general system of ODEs with a parameter ϕ:

$$\frac{dy}{dt} = f(y,\varphi),\ f:\mathbb{R}^n\times\mathbb{R} and\ f\in C^2(\mathbb{R}^n\times\mathbb{R}), \tag{12}$$

where, 0 is an equilibrium point of the system (that is, $f(0,\phi)\equiv 0\forall\phi$) and assume.

$$AI: A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right) is \quad the \quad linearization$$

matrix of matrix of the system (12) around the equilibrium point 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts;

A2: Matrix A has a right eigenvector w and a left vector v (each corresponding to the zero eigenvalue).

Let f_k be the k[th] component of f and:

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0),$$

The local dynamics of the system (1.18) around 0 is totally determined by the signs of a and b:

i. $a>0$, $b>0$ when $\phi<0$ with $|\phi|\le 1,0$ is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0<\phi\ll 1,0$ is unstable and there exists a negative and locally asymptotically stable equilibrium
ii. $a<0, b<0\cdot when\ \phi<0\ with\ |\phi|\ll 1,0$ is unstable; when $0<\phi\ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium
iii. $a>0, b<0\cdot when\ \phi<0\ with\ |\phi|\ll 1,0$ is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0<\phi\ll 1,0$ is stable, and a positive unstable equilibrium appears
iv. $a<0, b<0\cdot when\ \phi$ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.
v. Particularly, if a > 0 and b > 0 then a backward bifurcation occurs at $\phi=0$

Now, let $S=x_1, E=x_2, I_A=x_3, I_s=x_4, H=x_5, R=x_6$. Then we can write system (1) as:

$$\begin{cases} \frac{dx_1}{dt} = \Lambda+\delta x_6-\left((1-\sigma)\varpi+\mu\right)x_1 \\ \frac{dx_2}{dt} = (1-\sigma)\varpi x_1-\left(\gamma+\phi+\xi+\mu\right)x_2 \\ \frac{dx_3}{dt} = p\gamma x_2-\left(\psi+\phi+\xi+\eta+\mu\right)x_3 \\ \frac{dx_4}{dt} = (1-p)\gamma x_2+\psi x_3-\left(\phi+\xi+\eta+\mu\right)x_4 \\ \frac{dx_5}{dt} = \phi\left(x_3+x_4+x_2\right)-(\zeta+\eta+\mu)x_5 \\ \frac{dx_6}{dt} = \xi\left(x_2+x_3+x_4\right)+\zeta x_5-\left(\delta+\mu\right)x_6 \end{cases}$$

In more concise form, (11) is written as:

$$\frac{dx}{dt} = F(x)$$

where, $x=(x_1,x_2,x_3,x_4), F=(f_1,f_2,f_3,f_4)$. We recall the Jacobian matrix ($J_{\mathbb{E}0}$) of the system (1) at the disease free equilibrium:

$$J_{\mathbb{E}_0}=\begin{pmatrix} -\mu & 0 & (-1+\sigma)\chi & (-1+\sigma)\varsigma_1\chi & (-1+\sigma)\xi_2\chi & \delta \\ 0 & -\gamma-\mu-\xi-\phi & -(-1+\sigma)\chi & -(-1+\sigma)\varsigma_1\chi & -(-1+\sigma)\xi_2\chi & 0 \\ 0 & p\gamma & -\eta-\mu-\xi-\phi-\phi & 0 & 0 & 0 \\ 0 & \gamma-p\gamma & \psi & -\gamma-\mu-\xi-\phi & 0 & 0 \\ 0 & \phi & \phi & \phi & -(\varsigma+\eta+\mu) & 0 \\ 0 & \xi & \xi & \xi & \xi & -(\delta+\mu) \end{pmatrix}$$

Let us take χ as the bifurcation parameter. Observe that from Eq. (8), with R_0 = 1, we have the critical value of χ^* given by:

$$\chi^* = -\frac{(\gamma+\mu+\xi+\phi)}{(-1+\sigma)\left(\frac{\gamma\varsigma_1}{\eta+\mu+\xi+\phi}+\frac{\varsigma_2\phi(\gamma+\eta+\mu+\xi+\phi}{(\zeta+\eta+\mu)(\eta+\mu+\xi+\phi+\Psi}-\frac{p\gamma(-1+\zeta 1)}{\eta+\mu+\xi+\phi+\psi}\right)} \tag{14}$$

From the characteristic equation of $J_{\mathbb{E}0}$ given by $\left|J_{E_0}-\lambda_1\right|=0$, we obtain the eigenvalues:

$$\lambda_1 = -0.499466, \lambda_2 = -0.243136,$$
$$\lambda_3 = -0.160792, \lambda_4 = -0.003555,$$
$$\lambda_5 = -0.000034,$$
$$\lambda_6 = -3.331512703558856 \times 10^{-17} \cong 0$$

Hence, 0 is a simple eigenvalue of $J_{\mathbb{E}0}$ and other eigenvalues have negative real parts. We now solve for the right eigenvector $(g_1, g_2, g_3, g_4, g_5, g_6)^T$ of the Jacobian matrix (JE_0).

$$\begin{pmatrix} -\mu & 0 & (-1+\sigma)\chi & (-1+\sigma)\varsigma_1\chi & (-1+\sigma)\varsigma_2\chi & \delta \\ 0 & -\gamma-\mu-\xi-\phi & -(-1+\sigma)\chi & -(-1+\sigma)\varsigma_1\chi & -(-1+\sigma)\varsigma_2\chi & 0 \\ 0 & p\gamma & -\eta-\mu-\xi-\phi-\psi & 0 & 0 & 0 \\ 0 & \gamma-p\gamma & \psi & -\eta-\mu-\xi-\phi & 0 & 0 \\ 0 & \phi & \phi & \phi & -(\varsigma+\eta+\mu) & 0 \\ 0 & \xi & \xi & \xi & \zeta & -(\delta+\mu) \end{pmatrix}$$
$$\begin{pmatrix} g_1 \\ g_2 \\ g_3 \\ g_4 \\ g_5 \\ g_6 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Solving the system, we obtained:

$$g_2 = g_2 > 0$$
$$g_3 = 0.3401250329581882 g_2$$
$$g_4 0.7014911672925501 g_2$$
$$g_5 = 1.4130787654005668 g_2$$
$$g_6 = 37.13412317260833 g_2$$
$$g_1 = -5310.96330321312 g_2$$

Similarly, solving for the left eigenvector (h_1, h_2, h_3, h_4, h_5, h_6), we obtained:

$$h_1 = h_6 = 0, h_2 = h_2 > 0,$$
$$h_5 = 0.5963375639770276 h_2,$$
$$h_4 = 0.9857481094090845 h_2,$$
$$h_3 = 1.7031307539191602 h_2.$$

We now compute $a$and b. From the formula:

$$a = \sum_{k,i,j=1}^{4} h_k g_i g_i \frac{\partial^2 f_k}{\partial x_i \partial x_j}(s_0, 0, 0, 0),$$
$$b = \sum_{k,i,j=1}^{4} h_k g_i \frac{\partial^2 f_k}{\partial x_i \partial \chi}(s_0, 0, 0, 0),$$

and considering only the non-zero components (h_2, h_3, h_4, h_5) of the left eigenvectors, we obtained:

$$a = -0.023055651043759232 g_2{}^2 h_2 < 0,$$
$$b = -2529.779752835934 g_2 h_2 < 0.$$

Since a< 0 and b< 0 when χ< 0 with $|\phi|$ << 1, E_0 is unstable; when 0 < χ << 1, E_0 s locally asymptotically stable, and there exists a negative unstable equilibrium Observe from Fig. 2, the bifurcation plot (the infectious compartment against the basic reproduction number). It is seen that the system exhibits forward bifurcation, and the disease endemic equilibrium is globally asymptotically stable when R_0> 1. We also see an unstable negative endemic equilibrium with R_0< 1.

Numerical Simulations

Here, we carry numerical simulations in order to study the progression of the infection over a course of two years by varying the rate of transmission (χ) and the compliance level (σ) of susceptible individuals to government's laws on the use of facemask and social distancing. The parameter values used for these simulations are presented in Table 2 below and the simulations are shown in Fig. 3-8.

Simulation with χ = 0.151725 *and* σ = 0.7.

In simulating the model with χ = 0.151725 and σ 0.7, we observed 96% increase in the population of the hospitalised compartment within the first six months, which thereafter, begins to drop; but for a period of fifty months, this compartment is reduced by 82%. This is shown in Fig. 3. We also noticed a 9% increase in the population of the symptomatic infectious individuals for the first two months, and for a period of fifty months, this compartment is reduced by 95%. Figure 3 also shows 97 and 98% drops in the population of the exposed class and the asymptomatic infectious class respectively, for the said period of time.

Simulation with χ = 0.3 and σ = 0.7

In this simulation with an increased rate of transmission, we observed that for the first eight months, there is a 123% increase in population of the hospitalised compartment, and for a whole period of fifty months, the compartment is reduced by 9% only. This is shown in Fig. 4. There is an 11% increase in the symptomatic infectious compartment for first three months. Thereafter, a 74% drop in the population size is observed for the said period of 50 months. This simulation also revealed that 93% and 76% of individuals leave the exposed compartment and asymptomatic infectious compartment for the said period of time.

Simulation with $\chi = 0.05$ and $\sigma = 0.7$

Figure 5 shows the simulation of the system with a small rate of transmission,χ. In this simulation, we observed that the hospitalised compartment increased by 83% within the first five months and for a period of fifty months, it has dropped by 94%. For a period of two months, the symptomatic infectious compartment increased by 2%, and for the period of fifty months, it has dropped by 99%. For the first ten months, we noticed a remarkable drop in the exposed compartment and the asymptomatic compartments, and by the end of the fifty months period, they have been lowered by over 99.9% of their population sizes.

Simulation with $\chi = 0.151725$ and $\sigma = 0.35$

Figure 6 shows the simulation of the system with a lowered compliance level, σ, of susceptible individuals to government's laws on the use of facemask and social distancing. The simulation shows that for a period of thirteen months, the hospitalised population has increased by 142%, and the compartment is reduced by only 55% at the end of the fifty-month period. The simulation reveals that the symptomatic compartment increased by 18% in the first three months, and for the said period of fifty months, it only reduced by 53%. We also observed that for the first ten months, only 64 and 36% of individuals left the exposed compartment and the asymptomatic compartment respectively, and these compartments are reduced by only 82 and 47% at the end of the fifty-month period.

Simulation with $\chi = 0.151725$ and $\sigma = 0.9$

In simulating the system with a high level of compliance, σ, of susceptible individuals to government's laws on the use of facemask and social distancing, we observed that within the first five months, the hospitalised compartment increased by 82%, and with a fast rate, this compartment is reduced by 98% within the said period of fifty months. This is shown in Fig. 7. The simulation shows that within a period of twenty months a remarkable result is achieved for the exposed compartment and the asymptomatic compartment, and for a period of forty months, over 99% of individuals have exited the exposed, asymptomatic and symptomatic compartments.

Simulation with $\chi = 0.05$ and $\sigma = 0.9$

In simulating the system with a very low rate of transmission and a very high level of compliance to government's laws on social distancing and the use of face mask, we observed that within the first two months and five months, the symptomatic infectious compartment and the hospitalised compartment only increased by 5 and 74% respectively. Thereafter, they begin to drop at a very fast rate. We observed a remarkable reduction of over 99% in the number of exposed individuals and asymptomatic individuals, within the first twenty-two months. The symptomatic class has also been reduced by over 99% in a period of thirty-five months; and the hospitalised compartment has been reduced by over 99% within the said period of fifty months.

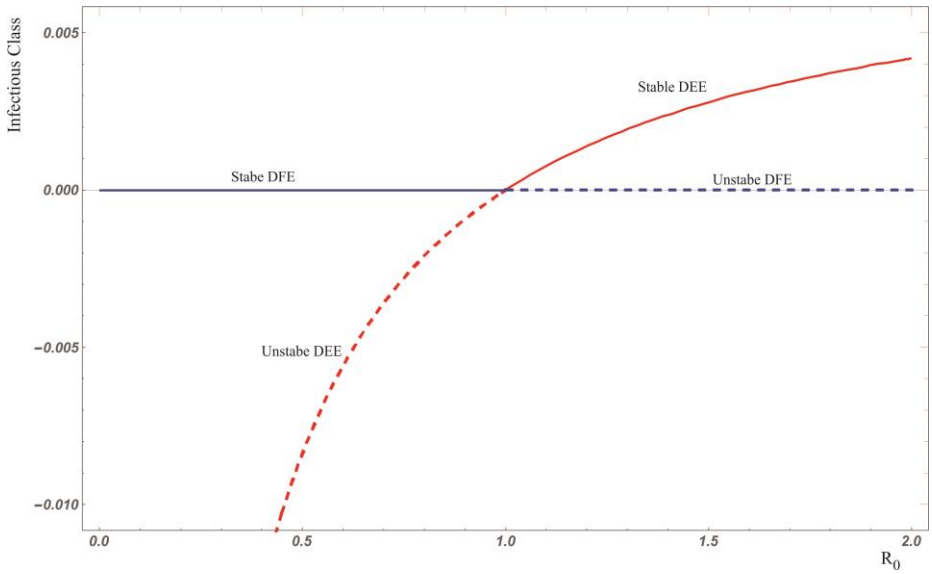

Fig. 2: Bifurcation plot (forward bifurcation)

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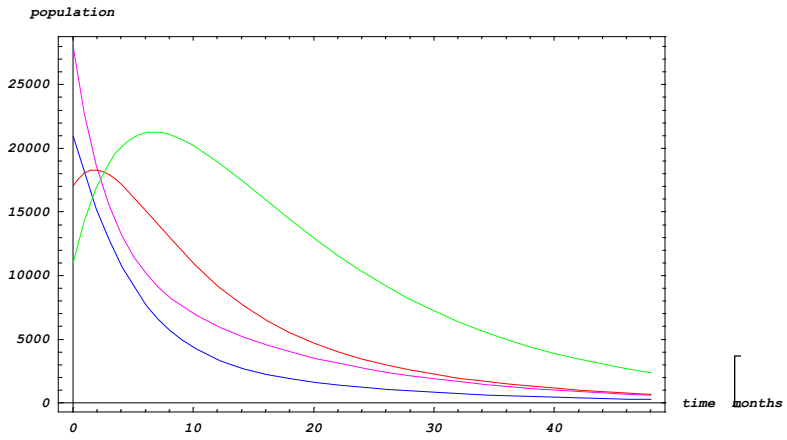

Fig. 3: Simulation with $\chi = 0.151725$ and $\sigma = 0.7$

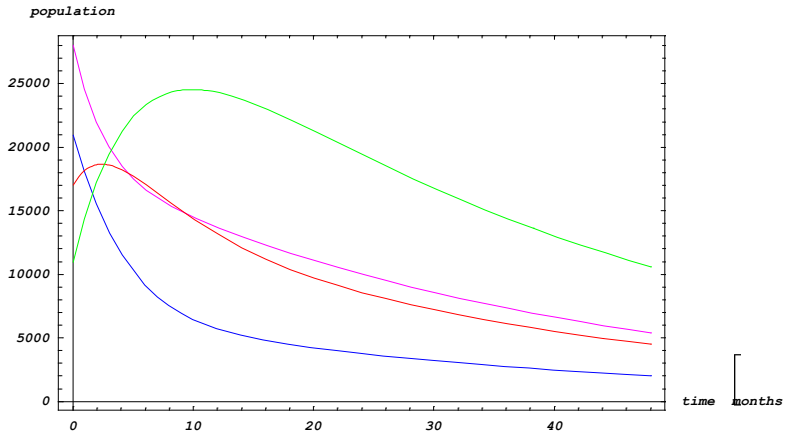

Fig. 4: Simulation with $\chi = 0.3$ and $\sigma = 0.7$

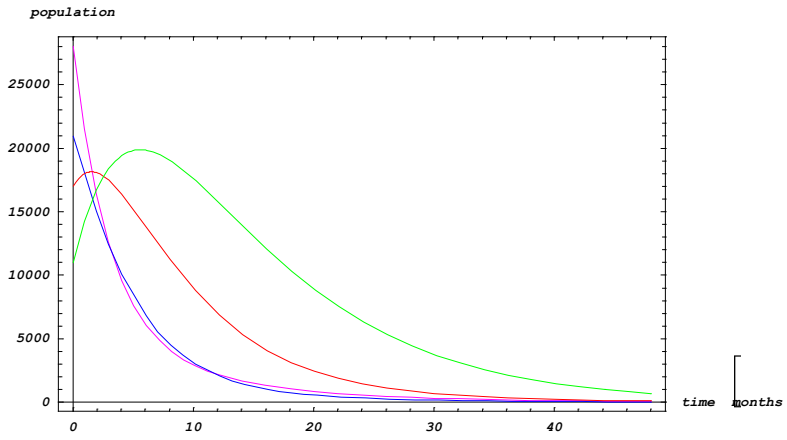

Fig. 5: Simulation with $\chi = 0.05$ and $\sigma = 0.7$

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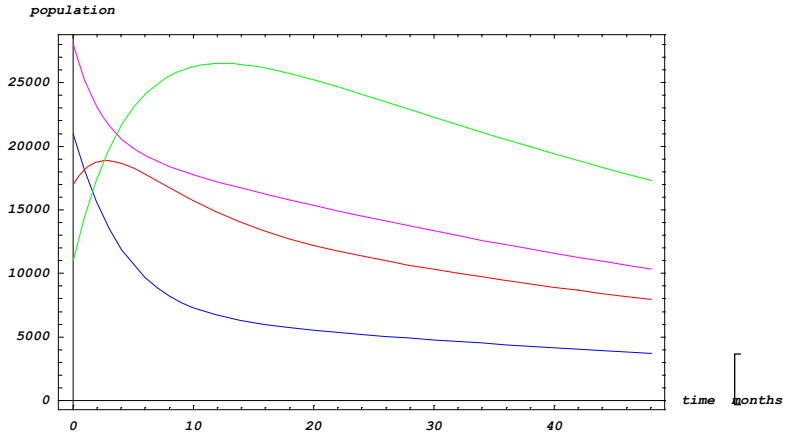

Fig. 6: Simulation with $\chi = 0.151725$ and $\sigma = 0.35$

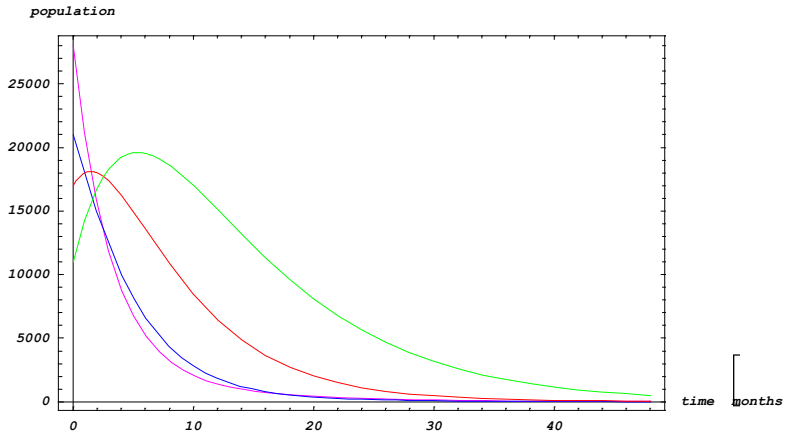

Fig. 7: Simulation with $\chi = 0.151725$ and $\sigma = 0.9$

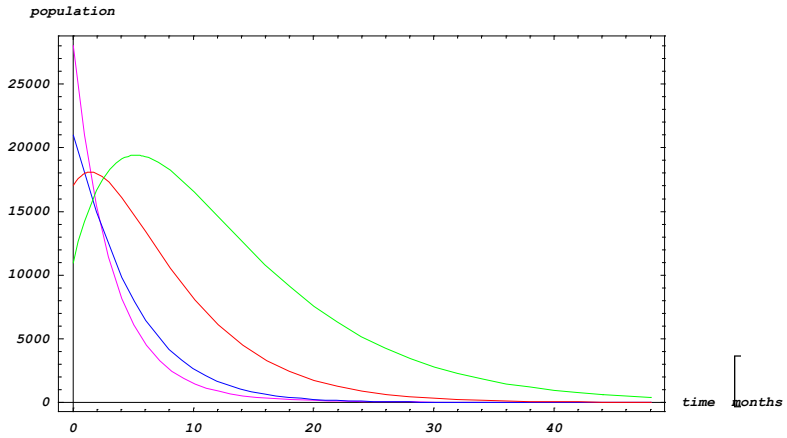

Fig. 8: Simulation of system (1) with $\chi = 0.05$ and $\sigma = 0.9$

Table 2: Values of variables and parameters

Parameter symbol	*Value (day^{-1})*	*Source*
Λ	0.037269	Assumed
δ	0.003521	Estimated
σ	0.700000	Assumed
μ	0.000034	
γ	0.196078	Lauer *et al.* (2019; Li *et al.*, 2020)
k	0.433500	
τ	0.350000	Assumed
p	0.500000	Ferguson *et al.* (2020)
ϕ	0.080000	
χ	0.151725	Estimated
ξ	0.035210	Rabajante, (2020)
ψ	0.100000	Assumed
η	0.073000	
ζ	0.042550	Rabajante (2020)
ζ_1	0.400000	Assumed
ζ_2	0.200000	Assumed

Discussion

This study has considered a deterministic model and analysis for the covid-19 infection. The model divides the population into six mutually-exclusive compartments, which are the susceptible compartment, the exposed compartment, the asymptomatic infectious compartment, the symptomatic infectious compartment, the hospitalised compartment, and the recovered compartment denoted by S, E, I_A, I_S, H, R respectively.We have established that all solutions of the model are positive. This is necessary for the biological relevance of the model. We have also shown that the total population is bounded. These results were obtained when establishing the qualitative behaviour of the model. We also obtained the disease-free equilibrium and the endemic equilibrium of the model. The basic reproduction number, (R_0), of the covid-19 disease was obtained via the next generation matrix, and the expression for R_0consists of three terms; the first term shows that when an infectious individual is introduced into an entirely susceptible population, susceptible individuals become exposed and a proportion of these exposed individuals become infectious but asymptomatic. The total time spent by this proportion both in the exposed and asymptomatic compartments is given by $\frac{1}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)}$ and the combined rate is $p\gamma(1\text{-}\sigma)\chi$. The second term accounts for susceptible individuals who become exposed, infectious without symptoms but later developed symptoms of the disease. The total time spent by this proportion is $\frac{1}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi+\psi)(\eta+\mu+\xi+\phi)}$ and the combined rate is $\zeta 1\chi\gamma(1-\sigma)\big((1-p)(\eta+\mu+\xi+\phi)+\zeta\big)$. The third term accounts for individuals who become exposed, then infectious with symptoms of the COVID-19 disease, and are later hospitalised. The total time spent by this proportion is $\frac{1}{(\gamma+\mu+\xi+\phi)(\eta+\mu+\xi+\phi)(\zeta+\eta+\mu)}$ and the combined rate is $\zeta 2\chi\gamma(1-\sigma)(\gamma+\eta+\mu+\xi+\phi)$. The stability analysis was carried out and it was shown that the disease-free equilibrium is globally asymptotically stable if $R_0 \leq 1$ and the endemic equilibrium is globally asymptotically stable if $R_0 \leq 1$.The sensitivity analysis revealed that the rate of transmission (χ),the proportion (p) of exposed individuals that becomes infectious but asymptomatic, the rate (γ) at which exposed individuals become infectious, the transmission coefficient (ς_1) of the symptomatic individuals, and the transmission coefficient (ς_2) of the hospitalised individuals are the most sensitive parameters of the basic reproduction number R_0.

Conclusion

The bifurcation analysis done in this work via the centre manifold theory, revealed that the system exhibits a forward bifurcation. Numerical simulations were carried out, first on the endemic equilibrium, for the validation of the result of the bifurcation analysis; secondly, simulations were carried out on the model, using different combinations of values of the rate of transmission (χ) and the compliance level (σ) of susceptible individuals to government's laws on the use of facemask and social distancing. It was observed that when $\chi < 0.1$ and $\sigma > 0.8$, the number of infected individuals are greatly reduced at a very fast rate. Thus, in order to curtail and possibly eliminate the COVID-19 disease from a given population, the rate of

transmission of the disease must be minimized (since $\chi = k\tau$, this can be achieved by reducing the contact rate, k, with infected individuals and surfaces), and individuals in the population must adhere strictly to government's laws social distancing and the use of facemask.

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Author's Contributions

Tsetimi Jonathan: Original draft, Supervision, Conceptualization, Writing.

Marcus Ifeanyi Ossaiugbo: Design, Writing, Data Acquisition and Analysis.

Augustine Atonuje: Writing, Data Acquisition and Interpretation.

Ethics

There are no ethical violations in this work in line with university and international standards.

References

Balkhair, A. A. (2020). COVID-19 pandemic: A new chapter in the history of infectious diseases. Oman medical journal, 35(2), e123. doi.org/10.5001/omj.2020.41

Castillo-Chavez, C., & Song, B. (2004). Dynamical models of tuberculosis and their applications. Mathematical Biosciences & Engineering, 1(2), 361. doi.org/10.3934/mbe.2004.1.361

Diekmann, O., Heesterbeek, J.A.P. and Metz, J.A.J. (1990) On the Definition and Computation of Basic Reproduction Ratio R0 in Models for Infectious Diseases in Heterogeneous Populations. Journal of Mathematical Biology, 28, 365-382. https://doi.org/10.1007/BF00178324.

Eikenberry, S. E., Mancuso, M., Iboi, E., Phan, T., Eikenberry, K., Kuang, Y., ... & Gumel, A. B. (2020). To mask or not to mask: Modeling the potential for face mask use by the general public to curtail the COVID-19 pandemic. Infectious disease modelling, 5, 293-308. doi.org/10.1016/j.idm.2020.04.001

Ferguson, N. M., Laydon, D., Nedjati-Gilani, G., Imai, N., Ainslie, K., Baguelin, M., ... & Ghani, A. C. (2020). Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand. doi.org/10.25561/77482

Hamer, W. H. (1906). The milroy lectures on epidemic diseases in england: The evidence of variability and of persistency of type; delivered before the royal college of physicians of london, march 1st, 6th, and 8th, 1906. Bedford Press.

Hethcote, H. W. (2000). The mathematics of infectious diseases. SIAM review, 42(4), 599-653. doi.org/10.1137/S0036144500371907

Jia, J., Ding, J., Liu, S., Liao, G., Li, J., Duan, B., ... & Zhang, R. (2020). Modeling the control of COVID-19: Impact of policy interventions and meteorological factors. arXiv preprint arXiv:2003.02985. doi.org/10.48550/arXiv.2003.02985

Kizito, M., &Tumwiine, J. (2018). A mathematical model of treatment and vaccination interventions of pneumococcal pneumonia infection dynamics. Journal of Applied Mathematics, 2018. doi.org/10.1155/2018/2539465

Kucharski, A. J., Russell, T. W., Diamond, C., Liu, Y., Edmunds, J., Funk, S., ... & Flasche, S. (2020). Early dynamics of transmission and control of COVID-19: a mathematical modelling study. The lancet infectious diseases, 20(5), 553-558. doi.org/10.1016/S1473-3099(20)30144-4

Lauer, S. A., Grantz, K. H., Bi, Q., Jones, F. K., Zheng, Q., Meredith, H. R., ... & Lessler, J. (2020). The incubation period of coronavirus disease 2019 (COVID-19) from publicly reported confirmed cases: estimation and application. Annals of internal medicine, 172(9), 577-582. doi.org/10.7326/M20-0504 doi.org/10.1016/j.mbs.2018.04.002

Li, R., Pei, S., Chen, B., Song, Y., Zhang, T., Yang, W., & Shaman, J. (2020). Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV-2). Science, 368(6490), 489-493. doi.org/10.1126/science.abb3221

May, R. M., & Anderson, R. M. (1979). Population biology of infectious diseases: Part II. Nature, 280(5722), 455-461. doi.org/10.1038/280455a0

Meng, L. (2020) "Applications of Ordinary Differential Equations in Mathematical Modeling," International Journal of New Developments in Engineering and Society, Vol. 4, Issue 2, pp. 102-107, doi.org/10.25236/IJNDES.040220, 2020

Muthu, V. K. (2005). A Short Book of Public Health. Jaypee Brothers Medical Publishers (P) Ltd, New Delhi. ISBN-10: 9789351522287.

Perasso, A. (2018). An introduction to the basic reproduction number in mathematical epidemiology. ESAIM: Proceedings and Surveys, 62, 123-138. doi.org/10.1051/proc/201862123

Prem, K., Liu, Y., Russell, T. W., Kucharski, A. J., Eggo, R. M., Davies, N., ... & Klepac, P. (2020). The effect of control strategies to reduce social mixing on outcomes of the COVID-19 epidemic in Wuhan, China: a modelling study. The Lancet Public Health, 5(5), e261-e270. doi.org/10.1016/S2468-2667(20)30073-6

Rabajante, J. F. (2020). Insights from early mathematical models of 2019-nCoV acute respiratory disease (COVID-19) dynamics. arXiv preprint.doi.org/10.48550/arXiv.2002.05296

Samal, J. (2014). A historical exploration of pandemics of some selected diseases in the world. International Journal of Health Sciences and Research, 4(2), 165-169. https://citeseerx.ist.psu.edu/viewdoc/download?doi=10.1.1.656.6545&rep=rep1&type=pdf

Thieme, H. R. (2003). Princeton series in theoretical and computational biology. In Mathematics in Population Biology. Princeton: Princeton University Press. https://www.degruyter.com/serial/puppstcb-b/html?lang=de

WHO. (2020a). Coronavirus disease 2019 (COVID-19) Situation Report – 80.

WHO. (2020b). Disease outbreaks by year. https://www.who.int/csr/don/archive/year/en/.

Xiaohua, H., & Min, Y. (2013) "Based on ODE and ARIAMA Modeling for the population of China," Technology and Investment, Vol. 4, No. 1B. doi.org/10.4236/ti.2013.41B006, 2013

Zeb, A., Alzahrani, E., Erturk, V. S., & Zaman, G. (2020). Mathematical model for coronavirus disease 2019 (COVID-19) containing isolation class. BioMed research international, 2020. doi.org/10.1155/2020/3452402