Int. J. Nonlinear Anal. Appl. 14 (2023) 10, 327–343
ISSN: 2008-6822 (electronic)
http://dx.doi.org/10.22075/ijnaa.2023.30025.4315

Mathematical modeling and analysis of the transmission dynamics of novel Corona virous (Covid-19) pandemic disease

Getachew Beyecha Batu[a], Eshetu Dadi Gurmu[b,*]

[a]Department of Mathematics, Arsi University, P.O.Box 193, Asella, Ethiopia

[b]Department of Mathematics, Samara University, P.O.Box 132, Samara, Ethiopia

(Communicated by Haydar Akca)

Abstract

In this paper, we have formulated a deterministic mathematical model of the novel corona virus to describe the dynamics of virus transmission in the community using a system of nonlinear ordinary differential equations. The invariant region of the solution, conditions for the positivity of the solution, existence of equilibrium points and their stabilities analysis, sensitivity analysis and numerical simulation of the model were determined. The system of a model equation has two equilibrium points, namely the disease-free equilibrium points where the disease does not exist and the endemic equilibrium points where the disease persists. Both local and global stability of the disease-free equilibrium and endemic equilibrium points of the model equation were established. The basic reproduction number that represents the epidemic indicator was obtained by using a next-generation matrix. The endemic states were considered to exist when the basic reproduction number was greater than one. Finally, our numerical findings were illustrated through computer simulations using MATLAB $R2015b$ with $ode45$ solver which shows the reliability of our model from the practical point of view. From our simulation results of the model, we came to realize that the number of infected people keeps decreasing if one carefully decreases the effective contact rate among protected and infectious individuals.

Keywords: Analysis, COVID-19, Reproduction Number, Model, Stability, Transmission Dynamics
2020 MSC:

1 Introduction

Modeling and simulation are important decision tools that can be useful to control human and animal diseases[2]. Coronavirus-19 pandemic disease is an infectious disease caused by a newly discovered coronavirus. It is a new strain that was discovered in 2019 and has not been previously identified in humans[8]. It is emerging in China in December 2019 and rapidly spread around China and many other countries[13]. On 30 January 2020, world health organization declared it to be a public health emergency of international concern [16]. This is a new virus and a completely new situation [14]. On February 11, 2020, the World Health Organization renamed the epidemic disease caused by 2019-nCoV as strain severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and announced a name for the new Coronavirus-19 disease "COVID-19" [15]. As of March 11, 2020, the disease has been confirmed in more than 118,000

*Corresponding author
Email addresses: gechobeyecha143@gmail.com (Getachew Beyecha Batu), eshetudadi2020@gmail.com (Eshetu Dadi Gurmu)

Received: February 2023 *Accepted:* May 2023

reported cases worldwide in 114 countries, more than 90% of cases occur in just four countries (two of which are China and the Republic of Korea - have significantly declining epidemics) and World Health Organization declared it to be a pandemic, the first one caused by a coronavirus [17]. As of April 1, 2020, 872,481 and 43,275 official cases and deaths have been recorded respectively, and there is no vaccine specifically designed for this virus, with proven effectiveness at the beginning of the outbreak. Several studies suggest that Coronaviruses, including preliminary information on the COVID-19 virus may persist on the surfaces for a few hours or up-to several days. The most common symptoms of COVID-19 are fever, cough, shortness of breath and difficulty breathing. The period within which the symptoms would appear is $2-14$ days. Its transmission from person to person is through respiratory droplets produced when an infected person coughs or sneezing and between people who are in close contact with each other [12]. The Most transmission is through respiratory droplets that we can inhale in close contact with each other [11]. It is not certain how long the virus that causes COVID-19 survives on the surfaces, but it seems to be have like other coronaviruses. In severe cases, the infection can cause pneumonia, severe acute respiratory syndrome, and even death. There is no specific treatment for the disease caused by COVID-19. However, many of the symptoms of the virus can be treated and therefore the treatment is based on the patient's clinical condition. The best ways that are recommended by World Health Organization to prevent the novel coronavirus (COVID-19) are, taking vaccine of covid-19, washing hands often with soap and water, if not available use hand sanitizer, avoid touching your eyes, nose, or mouth with unwashed hands, avoid contact with sick people, stay home when sick, and avoid close contact with others, cover your mouth/nose with a tissue or sleeve when coughing or sneezing and so on [12].

Currently, COVID19 is of great concern to researchers, governments and all peoples because of the high rate of spread of the infection and the large number of deaths that have occurred. Many authors have developed a mathematical model to illustrate the dynamics of the disease which helped to suggest a disease control mechanism and also described the transmission dynamics of coronavirus infection [10].

Chen et al [4] developed Bats-Hosts-Reservoir -People transmission network model for simulating the potential transmission from source of infection (probably bats) to human infection. Bats-Hosts-Reservoir network was hard to explore clearly and public concerns were focusing on the transmission from Huanan Seafood Wholesale Market (reservoir) to people, they simplified the model as Reservoir-People (RP) transmission network model. The model showed that the transmission of SARS-CoV-2 was higher than the Middle East respiratory syndrome in the Middle Eastern countries, similar to Severe Acute Respiratory Syndrome, but lower than Middle East respiratory syndrome in Republic of Korea.

In addition, the model of Chayu Yang and Jin Wang [18] describes the multiple routes of transmission in the infection dynamics and emphasizes the role of the environmental reservoir in the transmission and spread of this disease. Analytical and numerical results indicate that coronavirus infection would remain endemic, which necessitates long-term disease prevention and intervention programs. Li Y et al. [10] proposed a mathematical model, based on the transmission mechanism of COVID-19 in the population and the implemented prevention and control measures. They established the dynamic models of the six chambers and the time series models based on different mathematical formulas according to the variation law of the original data. E.D. Gurmu et al. [8] modify the model developed by Li Y et al. [10] by adding the asymptomatic compartment. In their paper they formulated a dynamical model of COVID-19 to describe the transmission dynamics of the disease. They established both local and global stability of the disease free and endemic equilibrium point of the model equation by using basic reproduction number. They performed the sensitivity analysis of the model equation on the key parameters to find out their relative significance and potential impact on the transmission dynamics of COVID-19.

In this paper we modify the model developed by E.D.Gurmu et al. [8]. Moreover, the future work of this paper will consider the fractional derivatives to COVID-19 model and its optimal control.

2 Model Description and Formulation

Mathematical modeling process requires the translation of a biological scenario into a mathematical problem. It begins with a clear description of the processes based on the modeler understanding of the system. The translation of a biological scenario into mathematical equations should be made with a specific goal or biological question in mind. Then the verbal description of the system is encoded in mathematical equations.

The total number of human population at a time t, denoted by $N(t)$, is divided into eight sub-classes consisting of:

- Protected individuals $P(t)$; individuals those that are protected against the disease over period of time at specific area not to vulnerable to the virus.

- Exposed individuals $E(t)$; individuals those that are in the incubation period of the novel corona viruse disease as long as 2 to 14 days.
- Infective individual in symptomatic phase $I(t)$; individuals those that are infected and infectious of novel corona virus (COVID-19) disease.
- Quarantine individuals $Q(t)$; are individual those that are infectious and compulsory quarantine due to reduce the spread of COVID-19 virus.
- Hospitalized individuals $H(t)$; individuals those that are infected and infectious with COVID-19 and under a symptoms treatment at designated healthcare facilities.
- Fatal individuals $F(t)$; individuals those that are in the intensive care unit class that leads to or ends to the death.
- Recovered individual $R(t)$; individuals those that recovered from COVID-19 at a time t due to symptoms treatment at a quarantine class, hospitalized class and fatal class of COVID-19 virus.
- Death individual $D(t)$; individuals those that do not recovered from COVID-19 and died.

Then the total population at a time t denoted by $N(t)$ is given by:

$$N(t) = P(t) + E(t) + Q(t) + I(t) + H(t) + F(t) + R(t) + D(t).$$

Thus the model assumed that: protected individuals are recruited into the population at a constant rate of π and decreased by losing protection at a rate λ, acquiring COVID-19 infection following effective contact with infectious individual, such that $\lambda = \frac{\beta[I(t)+Q(t)]}{N}$ is a force of infection. where β is an effective contact rate which is leading to infection.

Exposed individuals are generated by losing protection of protected individuals at a rate λ and decreased by joining quarantine subclass at a rate ϕ and infected subclass at a rate ψ. Infected individuals are generated by the fraction of exposed individuals at a rate ψ and decreased by joining the hospitalized subclass to get symptoms treatment at a rate δ, fatal subclass due to the severity of the disease at rate φ and died due to COVID-19 at a rate ζ.

The population of quarantined individuals are generated by isolating rate ϕ of exposed individuals and decreased by recovering rate from the quarantined sub class at a rate θ and further decreased by the failure of symptoms treatment in the quarantined class at a rate ω.

The population of hospitalized individuals are increased by the failure of symptoms treatment in the quarantined subclass at the rate ω and from the infected subclass whose sickness are hard at a rate δ and decreased by success of symptoms treatment in the hospitals at a rate α and join the recovered subclass and further decrease at a rate γ and join fatal subclass, at a rateε and joined death subclass and died due to COVID-19 at a rate ζ. The population of recovered subclass are generated by recovering rate at θ, α and η due to symptoms treatment at quarantine , hospital and fatal class respectively. The population of fatal subclass are generated by the failure of symptoms treatment in the hospitals at a rate γ ,due to the hardness of the disease at rate φ in the infected subclass and decreased by the success of symptoms treatment at fatal subclass at a rate η and join recovered sublass, failure of symptoms treatment at fatal subclass at a rate ϵ and join the death subclass. The population of death subclasses are generated by the failure of symptoms treatment in the hospital subclass at a rate ε fatal sublass at a rate ϵ. All types of cells suffer natural mortality at a rate μ. All parameters value used in the model are assumed to be non-negative. Up on including the basic assumption the schematic diagram of the modified model is as follows:

Based on the model assumptions, the notations of variables, parameters and the schematic diagram, the model equations are formulated and given as follows:

$$\begin{cases} \frac{dP}{dt} = \pi - (\mu + \lambda)P, \\ \frac{dE}{dt} = \lambda P - (\mu + \psi + \phi)E, \\ \frac{dI}{dt} = \psi E - (\mu + \delta + \varphi + \zeta)I, \\ \frac{dQ}{dt} = \phi E - (\mu + \theta + \omega)Q, \\ \frac{dH}{dt} = \omega Q + \delta I - (\mu + \alpha + \gamma + \varepsilon + \zeta)H, \\ \frac{dF}{dt} = \varphi I + \gamma H - (\mu + \epsilon + \zeta + \eta)F, \\ \frac{dR}{dt} = \theta Q + \alpha H + \eta F - \mu R, \\ \frac{dD}{dt} = \varepsilon H + \epsilon F - \mu D. \end{cases} \tag{2.1}$$

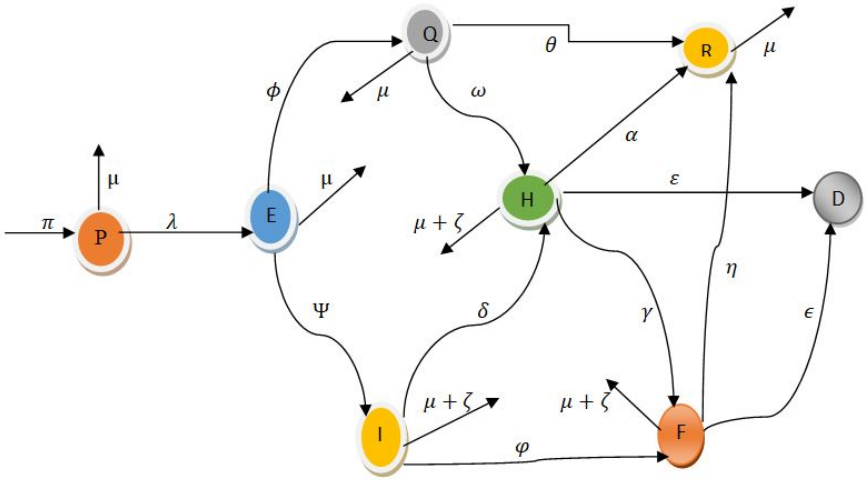


Figure 1: Schematic diagram of the model

The non-negative initial conditions of the system of model equations (2.1) are denoted by $P(0) > 0, E(0) \geq 0, I(0) \geq 0, Q(0) \geq 0, H(0) \geq 0, F(0) \geq 0, R(0) \geq 0, D(0) \geq 0$.

3 Qualitative Model Analysis

3.1 Positive Invariance

To determine the positive invariance we shall investigate that all the state variables of system (2.1) are non-negative for all time t with its initial conditions. To prove positivity of the model, we state the following theorem.

Theorem 3.1. All the solution of $(P(t), E(t), Q(t), I(t), H(t), F(t), R(t), D(t))$ of the system (2.1) are non-negative for all time t with initial conditions

$$(P(0), E(0), Q(0), I(0), H(0), F(0), R(0), D(0)) \in \mathbf{R}^8_+ > 0 \quad \forall t > 0.$$

Then the system (2.1) is positively invariant and attracting with in $\mathbf{R}^8_+$.

Proof . Positivity of the model variables are shown separately for each of the model variables $P(t)$, $E(t)$, $Q(t)$, $I(t)$, $H(t)$, $F(t)$, $R(t)$ and $D(t)$.

Positivity of P(t): From system (2.1), the model equation of $\frac{dP}{dt}$ is given by $\frac{dP}{dt} = \pi - (\mu + \lambda)P$ which can be expressed without loss of generality, after eliminating the positive terms π, as an inequality $\frac{dP}{dt} \geq -(\mu + \lambda)P$.

Using the separable method of variables and applying integration, the solution of the above differentially inequality can be obtained as $P_0 e^{(-\mu t - \frac{\beta}{N}\int (I+Q)dt)}$. Recall that an exponential function is always positive regardless of the sign of the exponent. Hence, it can be concluded that

$$P(t) = P_0 e^{(-\mu t - \beta \int \frac{(I+Q)}{N} dt)} \geq 0.$$

Similarly, solving each equations in the system of differential equation of the model, we obtain the solution in an exponential function form as:

$$\begin{aligned} E(t) &= E_0 e^{-(\mu+\eta)t} \geq 0 & I(t) &= I_0 e^{-(\mu+\delta+\varphi+\zeta)t} \geq 0 \\ Q(t) &= Q_0 e^{-(\mu+\theta+\omega)t} \geq 0 & H(t) &= H_0 e^{-(\mu+\alpha+\zeta+\varepsilon+\gamma+)t} \geq 0 \\ R(t) &= R_0 e^{-\mu t} \geq 0 & F(t) &= F_0 e^{-(\mu+\epsilon+\zeta+\eta)t} \geq 0 \\ D(t) &= D_0 e^{-\mu t} \geq 0 \end{aligned}$$

Since all exponential function is always non-negative irrespective of the sign of the exponent, it can be concluded that all the solutions of model equations are non-negative. Thus, all the solution trajectories of the model variables,

$$(P(t), E(t), Q(t), I(t),\ H(t), F(t), R(t), D(t))$$

that represent the population sizes of various types of cells are non-negative quantities and will remain in $\mathbf{R}^8_+$ for all $t \geq 0$. □

3.2 Boundedness

Now we start with the theorem which assure that the solutions of the system (2.1) is bounded with non-negative initial values conditions.

Theorem 3.2. The solution of system (2.1) with the initial condotion which initiate in $\mathbf{R}^8_+$ are uniformly bounded in the invariant region Ω.

Proof . To prove the boundedness, it suffices to show that

$$N(t) = P(t) + E(t) + Q(t) + I(t) + H(t) + F(t) + R(t) + D(t) \quad \text{is bounded.}$$

Clearly, $N(t) \geq 0$, for all $t \geq 0$ as all the initial conditions are non-negative. The rate of change of the total population by adding all the equations considered in (2.1) is:

$$\frac{dN}{dt} = \pi - \mu N(t) - \zeta(I + F + H) \leq \pi - \mu N(t)$$

This implies that

$$N(t) \leq \frac{\pi}{\mu} + (N(0) - \frac{\pi}{\mu})e^{-\mu t} \leq \max(\frac{\pi}{\mu},\ N(0)).$$

which is bounded as it is bounded below up at any time. Thus, accordingly, we obtain the following positively invariant bounded region

$$\Omega = \{(P, E, Q, I, H, F, R, D) \in \mathbf{R}^8_+\}, \text{ such that } P(t) + E(t) + Q(t) + I(t) + F(t) + R(t) + D(t) \leq \tfrac{\pi}{\mu}\}.$$

Therefore, all the solutions trajectories initiating in R^8_+ will enter Ω with finite time. □

We note that the study of positivity and boundedness enables us that the population will be nonnegative and below up at any time in our model.

4 Steady State of The Model

Steady state are time-idependent solutions of equations of the system (2.1) that satisfying:

$$\frac{dP}{dt} = \frac{dE}{dt} = \frac{dQ}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dF}{dt} = \frac{dR}{dt} = \frac{dD}{dt} = 0.$$

There are two steady states: The disease free equilibrium and endemic equilibrium points.

4.1 The Disease Free Equilibrium Points(E_0)

Disease-free equilibrium points are steady-state solutions in which there is no disease in the population. In the absence of the disease this implies that,

$$E(t) = I(t) = Q(t) = H(t) = R(t) = F(t) = D(t) = 0$$

and the equilibrium points required that the right hand side of the model equations set equal to zero. Thus, the disease-free equilibrium of the model equations in (2.1) above is given by

$$E_0 = \{\tfrac{\pi}{\mu}, 0, 0, 0, 0, 0, 0, 0\}.$$

The local stability of the disease-free equilibrium points of the model can be established using the basic reproduction number. The basic reproduction number is denoted by R_0 and it is defined as the average number of secondary infections caused by a single infected individuals in a population of purely susceptible. It is computed using next-generation matrix defined in [6]. In this method R_0 is defined as the largest eigenvalue of the next generation matrix. Using the notation as in [6] for the model system (2.1)the associated matrices F_i and V_i for the new infectious terms and the remaining transition terms are respectively given by:

$$F_i = \begin{bmatrix} \frac{\beta(\ I+Q)P}{N} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad V_i = \begin{bmatrix} aE \\ -\psi E + bI \\ -\phi E + cQ \\ -\Omega Q - \delta I + dH \\ -\varphi I - \gamma H + eF \\ -\varepsilon H - eF + \mu D \end{bmatrix}$$

where, $a = \mu + \psi + \phi, b = \delta + \varphi + \mu + \zeta, c = \mu + \theta + \omega, d = \mu + \zeta + \alpha + \varepsilon + \gamma, e = \mu + \zeta + \epsilon + \eta$. The Jacobian matrices F and V of F_i and V_i with respect to the disease free equilibrium point E_0 takes the form respectively as

$$F(E_0) = \begin{bmatrix} 0 & \beta & \beta & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and,} \quad V(E_0) = \begin{bmatrix} a & 0 & 0 & 0 & 0 & 0 \\ -\psi & b & 0 & 0 & 0 & 0 \\ -\phi & 0 & c & 0 & 0 & 0 \\ 0 & -\delta & -\omega & d & 0 & 0 \\ 0 & -\varphi & 0 & -\gamma & e & 0 \\ 0 & 0 & 0 & -\varepsilon & -e & \mu \end{bmatrix}$$

Then after some algebraic computations the inverse of the matrix $V(E_0)$ is constructed as follows:

$$[V(E_0)]^{-1} = \begin{bmatrix} \frac{1}{a} & 0 & 0 & 0 & 0 & 0 \\ \frac{\psi}{ab} & \frac{1}{b} & 0 & 0 & 0 & 0 \\ \frac{\phi}{ac} & 0 & \frac{1}{c} & 0 & 0 & 0 \\ \frac{c\delta\psi + b\phi\omega}{abcd} & \frac{\delta}{bd} & \frac{\omega}{cd} & \frac{1}{d} & 0 & 0 \\ \frac{c\gamma\delta\psi + cd\psi\varphi + b\gamma\phi\omega}{bcde} & \frac{\gamma\delta + d\varphi}{bde} & \frac{\gamma\omega}{cde} & \frac{\gamma}{de} & \frac{1}{e} & 0 \\ \frac{-c\gamma\delta\psi + c\delta\zeta\psi - cd\varphi\psi - b\gamma\phi\omega + b\zeta\phi\omega}{bcd\mu} & \frac{-\gamma\delta + \delta\zeta - \delta\varphi}{bd\mu} & \frac{-\gamma\omega + \zeta\omega}{cd\mu} & \frac{-\gamma + \zeta}{d\mu} & \frac{-1}{\mu} & \frac{1}{\mu} \end{bmatrix}$$

The product of the matrices $F(E_0)$ and $[V(E_0)]^{-1}$ which is the next generation matrix can be computed as

$$[F(E_0)][V(E_0)]^{-1} = \begin{bmatrix} \frac{\beta(b\phi + c\psi)}{abc} & \frac{\beta}{b} & \frac{\beta}{c} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

Now it is possible to calculate the eigenvalues to determine the basic reproduction number R_0 by taking the spectral radius of the matrix $[F(E_0)][V(E_0)]^{-1}$. Thus, the eigenvalues are computed by evaluating $det[[F(E_0)][V(E_0)]^{-1} - \lambda I] = 0$ or equivalently solving

$$\begin{vmatrix} \frac{\beta(b\phi + c\psi)}{abc} - \lambda & \frac{\beta}{b} & \frac{\beta}{c} & 0 & 0 & 0 \\ 0 & -\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & -\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & -\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -\lambda \end{vmatrix} = 0$$

It reduces to the sixth power equation for λ as $\lambda^5[\frac{\beta(b\phi + c\psi)}{abc} - \lambda] = 0$ giving the six eigenvalues as $\lambda_1 = 0$, $\lambda_2 = 0$, $\lambda_3 = 0$,$\lambda_4 = 0$,$\lambda_5 = 0$,$\lambda_6 = \frac{\beta(b\phi + c\psi)}{abc}$. However, the dominant eigenvalue here is $\lambda_6 = \frac{\beta(b\phi + c\psi)}{abc}$ and it is the spectral radius as the threshold value or the basic reproduction number. Thus, it can be concluded that the basic reproduction number of the model is

$$R_0 = \frac{\beta(b\phi + c\psi)}{abc},$$

where, $a = \mu + \psi + \phi, \quad b = \delta + \varphi + \mu + \zeta, \quad c = \mu + \theta + \omega$. We note that the basic reproduction number R_0 is an important epidimiological parameters used to understand and predict the spread of an infection. For example, $R_0 > 1$ means that each infected individuals will infect at least one individual in average and the disease may remain in the population forever.

4.1.1 Local Stability of Disease Free Equilibrium Points (E_0)

To find the local stability of E_0, the Jacobian of the model equations evaluated at E_0 is used. Now, the stability analysis of disease free equilibrium points is conducted and the results are presented in the form of theorems and proofs as follows:

Theorem 4.1. The disease free equilibrium points $E_0 = \{\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0, 0\}$ of the system (2.1) is locally asymptotically stable if $R_0 < 1$.

Proof . Consider the right hand side expressions of the equations (2.1) as functions so as to find the Jacobian matrix as follows:

$$\begin{cases} \frac{dP}{dt} = \pi - (\mu + \lambda)P = f_1, \\ \frac{dE}{dt} = P - (\mu + \psi + \phi)E = f_2, \\ \frac{dI}{dt} = \psi E - (\mu + \delta + \varphi + \zeta)I = f_3, \\ \frac{dQ}{dt} = \phi E - (\mu + \theta + \omega)Q = f_4, \\ \frac{dH}{dt} = \omega Q + \delta I - (\mu + \alpha + \gamma + \varepsilon + \zeta)H = f_5, , \\ \frac{dF}{dt} = \varphi I + \gamma H - (\mu + \epsilon + \zeta + \eta)F = f_6, \\ \frac{dR}{dt} = \theta Q + \phi H + \eta F - \mu R = f_7, \\ \frac{dD}{dt} = \varepsilon H + \epsilon F - \mu D = f_8, . \end{cases} \tag{4.1}$$

The Jacobian (J) of the system 4.1 is;

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial P} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial Q} & \frac{\partial f_1}{\partial H} & \frac{\partial f_1}{\partial F} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial D} \\ \frac{\partial f_1}{\partial P} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial Q} & \frac{\partial f_2}{\partial H} & \frac{\partial f_2}{\partial F} & \frac{\partial f_2}{\partial R} & \frac{\partial f_2}{\partial D} \\ \frac{\partial f_3}{\partial P} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial Q} & \frac{\partial f_3}{\partial H} & \frac{\partial f_3}{\partial F} & \frac{\partial f_3}{\partial R} & \frac{\partial f_3}{\partial D} \\ \frac{\partial f_4}{\partial P} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial Q} & \frac{\partial f_4}{\partial H} & \frac{\partial f_4}{\partial F} & \frac{\partial f_4}{\partial R} & \frac{\partial f_4}{\partial D} \\ \frac{\partial f_5}{\partial P} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial Q} & \frac{\partial f_5}{\partial H} & \frac{\partial f_5}{\partial F} & \frac{\partial f_5}{\partial R} & \frac{\partial f_5}{\partial D} \\ \frac{\partial f_1}{\partial P} & \frac{\partial f_6}{\partial E} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial Q} & \frac{\partial f_6}{\partial H} & \frac{\partial f_6}{\partial F} & \frac{\partial f_6}{\partial R} & \frac{\partial f_6}{\partial D} \\ \frac{\partial f_1}{\partial P} & \frac{\partial f_7}{\partial E} & \frac{\partial f_7}{\partial I} & \frac{\partial f_7}{\partial Q} & \frac{\partial f_7}{\partial H} & \frac{\partial f_7}{\partial F} & \frac{\partial f_7}{\partial R} & \frac{\partial f_7}{\partial D} \\ \frac{\partial f_8}{\partial P} & \frac{\partial f_8}{\partial E} & \frac{\partial f_8}{\partial I} & \frac{\partial f_8}{\partial Q} & \frac{\partial f_8}{\partial H} & \frac{\partial f_8}{\partial F} & \frac{\partial f_8}{\partial R} & \frac{\partial f_8}{\partial D} \end{bmatrix} \tag{4.2}$$

Now, the Jacobian matrix of $(f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)$ with respect to (P, E, I, Q, H, F, R, D) at the disease free equilibrium points E_0 reduces to

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -\beta & -\beta & 0 & 0 & 0 & 0 \\ 0 & -a & \beta & \beta & 0 & 0 & 0 & 0 \\ 0 & \psi & -b & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi & 0 & -c & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & \omega & -d & 0 & 0 & 0 \\ 0 & 0 & \varphi & 0 & \gamma & -e & 0 & 0 \\ 0 & 0 & 0 & \theta & \alpha & \eta & -\mu & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & \epsilon & 0 & -\mu \end{bmatrix}$$

where $a = \mu + \psi + \phi, b = \mu + \delta + \varphi + \zeta, c = \mu + \theta + \omega, d = \mu + \alpha + \gamma + \varepsilon + \zeta, e = \mu + \epsilon + \zeta + \eta$. Now, the eigenvalues of $J(E_0)$ are required to be found. Then, the characteristic equation $det[J(E_0) - \lambda I] = 0$ is simplified and found as

follows:

$$\begin{vmatrix} -\mu-\lambda & 0 & -\beta & -\beta & 0 & 0 & 0 & 0 \\ 0 & -a-\lambda & \beta & \beta & 0 & 0 & 0 & 0 \\ 0 & \psi & -b-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi & 0 & -c-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & \omega & -d-\lambda & 0 & 0 & 0 \\ 0 & 0 & \varphi & 0 & \gamma & -e-\lambda & 0 & 0 \\ 0 & 0 & 0 & \theta & \alpha & \eta & -\mu-\lambda & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & \epsilon & 0 & -\mu-\lambda \end{vmatrix} = 0$$

The characteristic equation of the jacobian matrix $det[J(E_0) - \lambda I] = 0$ is

$$(\lambda+d)(\lambda+e)(\lambda+\mu)^3[\lambda^3 + (a+b+c)\lambda^2 + (ab+ac+bc-\beta(\phi+\psi))\lambda + abc - \beta(\phi b + \psi c)] = 0.$$

Then,

$$(\lambda+d)(\lambda+e)(\lambda+\mu)^3[\lambda^3 + (a+b+c)\lambda^2 + (ab+ac+bc-\beta(\phi+\psi))\lambda + abc(1-R_0)] = 0.$$

This implies that

$$(\lambda+d)(\lambda+e)(\lambda+\mu)^3[\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3] = 0$$

which implies that

$$(\lambda+d)(\lambda+e)(\lambda+\mu)^3 = 0 \qquad \text{or} \qquad \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0.$$

From $(\lambda+d)(\lambda+e)(\lambda+\mu)^3 = 0$, we obtained that

$$\lambda_1 = -d, \lambda_2 = -e, \lambda_3 = -\mu, \lambda_4 = -\mu, \lambda_5 = -\mu.$$

Which implies that the obtained eigenvalues are real and negative. To determine the sign of the rest eigenvalues of polynomial $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$. We use the Routh Hurtzi cretaria such that

(i) $a_1 > 0$ implies that $a+b+c > 0$, since all parameters are nonnegative.

(ii) $a_2 > 0$ implies that $ab+ac+bc-\beta(\phi+\psi) > 0$ and so, $ab+ac+bc > \beta(\phi+\psi)$.

(ii) $a_3 > 0$ implies that $abc(1-R_0) > 0$ and so, $R_1 < 1$.

From this we conclude that all the required eigenvalues are negative and the Routh Hurtzi creterias are satisfied. Thus the disease free equilibrium points are locally asympitotically stable if $R_0 < 1$. □

4.1.2 Global Stability of The Disease Free Equilibrium Points

Theorem 4.2. The disease free equilibrium point is globally asymptotically stable if $R_0 < 1$.

Proof . To investigate the global stability of the disease free equilibrium points we used technique implemented by Castillo-Chavez and Song [3]. The point $U = (X^0, 0) = \{\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0, 0\}$ to be globally asymptotically stable for the model provided that $R_0 < 1$ and the following condition must be met.

$H_1 : \frac{dX}{dt} = F(X^*, 0), X^*$ is globally asymptotically stable.

$H_2 : G(X,Z) = AZ - \hat{G}(X,Z), \hat{G}(X,Z) \geq 0$ for $(X,Z) \in \Omega$
where $A = D_Z G(U, 0)$ is a Metzler matrix (the off diagonal elements of A are non-negative) and G is the region where the model make biologically sense. First the model equation (2.1) can be re-written as

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

where, $X = (P,R)$ stands for the uninfected population and $Z = (E,I,Q,H,F,D)$ also stands for the infected population. From system (2.1), we get

$$F(X,Z) = \begin{bmatrix} \pi - (\mu+\lambda)P \\ \theta Q + \alpha H + \eta F - \mu R \end{bmatrix} \qquad \text{and} \qquad G(X,Z) = \begin{bmatrix} \lambda P - (\mu+\psi+\phi)E \\ \psi E - (\mu+\delta+\varphi+\zeta)I \\ \phi E - (\mu+\theta+\omega)Q \\ \omega Q + \delta I - (\mu+\zeta+\alpha+\varepsilon+\gamma)H \\ \varphi I + \gamma H - (\mu+\zeta+\epsilon+\eta)F \\ \varepsilon H + \epsilon F - \mu D \end{bmatrix}$$

The compartmental model 2.1 stated in condition (H_1) can be expressed in the reduced system as

$$\frac{dX}{dt_{Z=0}} = \begin{bmatrix} \pi - \mu P \\ -\mu R \end{bmatrix} \tag{4.3}$$

Analytically solving equation (4.3) above it is obvious that $\frac{dX}{dt_{z=0}} = 0$, implies that, $\frac{dX}{dt_{z=0}} = F(X^*, 0) = \{\frac{\pi}{\mu}, 0\}$. Thus the point $\{\frac{\pi}{\mu}, 0\}$ is the global asymptotic point. Hence, X^* is globally asymptotically stable for $\frac{dX}{dt} = F(X^*, 0)$ and the first condition (H_1) holds for the system 2.1. Now for the second condition the matrices A for the model system 2.1 can be expressed from the equation for infected compartments in the model as.

$$A = \begin{bmatrix} -(\mu+\psi+\phi) & \beta & \beta & 0 & 0 & 0 \\ \psi & -(\mu+\delta+\varphi+\zeta) & 0 & 0 & 0 & 0 \\ \phi & 0 & -(\mu+\theta+\omega) & 0 & 0 & 0 \\ 0 & \delta & \omega & -(\mu+\zeta+\alpha+\varepsilon+\gamma) & 0 & 0 \\ 0 & \varphi & 0 & \gamma & -(\mu+\zeta+\epsilon+\eta) & 0 \\ 0 & 0 & 0 & \varepsilon & \epsilon & -\mu \end{bmatrix}$$

and the matrix $\hat{G}(X, Z)$ can be written as, $\hat{G}(X, Z) = AZ - G(X, Z)$, which is:

$$\hat{G}(X,Z) = \begin{bmatrix} \hat{G}_1(X,Z) \\ \hat{G}_2(X,Z) \\ \hat{G}_3(X,Z) \\ \hat{G}_4(X,Z) \\ \hat{G}_5(X,Z) \\ \hat{G}_6(X,Z) \end{bmatrix} = \begin{bmatrix} \beta(I+Q)(1-\frac{P}{N}) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

Then the matrix A is a Metzler- matrix since all its off diagonal elements are non-negative and $\hat{G}(X, Z) \geq 0$ in the region Ω and the condition (H_2) holds. Since the two conditions (H_1) and (H_2) holds, the disease free steady state E_0 of the model (2.1) is globally symptotically stable in the region Ω for $R_0 < 1$. □

4.2 Endemic Equilibrium Point (E_1)

The endemic equilibrium point $E_1 = \{P^*, E^*, I^*, Q^*, H^*, F^*, R^*, D^*, \}$ is a steady-state solution where the disease persists in the population. The endemic equilibrium point is obtained by setting rates of changes of variables with respect to time in model equations (2.1) to zero. The model equations take the solved form for the state variables in terms of parameters after some alebraic operation which is the solution of the system 2.1) and obtain the following solution as follows;

$$\begin{cases} P^* = \frac{N^*}{R_0} \\ E^* = \frac{\pi}{a}(1 - \frac{1}{R_0}) \\ Q^* = \frac{\phi\pi}{ac}(1 - \frac{1}{R_0}) \\ I^* = \frac{\psi\pi}{ab}(1 - \frac{1}{R_0}) \\ H^* = \frac{\pi}{abcd}(\omega\phi b + \delta\psi c)(1 - \frac{1}{R_0}) \\ R^* = \frac{\pi}{\mu abcde}[\theta\phi bde + \alpha e(\omega\phi b + \delta\psi c) + \eta\varphi\psi cd + \eta\gamma(\omega\phi b + \delta\psi c)](1 - \frac{1}{R_0}) \\ F^* = \frac{\pi}{abcde}[\varphi\psi cd + \gamma(\omega\phi b + \delta\psi c)](1 - \frac{1}{R_0}) \\ D^* = \frac{\pi}{\mu abcde}[\varepsilon\omega be + \delta\psi ce + \varphi\psi\epsilon cd + \gamma\epsilon(\omega\phi b + \delta\psi c)](1 - \frac{1}{R_0}) \end{cases} \tag{4.4}$$

where $a = \mu + \psi + \phi,\ b = \mu + \delta + \varphi + \zeta,\ c = \mu + \theta + \omega,\ d = \mu + \zeta + \alpha + \varepsilon + \gamma,\ e = \mu + \zeta + \epsilon + \eta$. From this solution of the model, it can be seen that positive unique endemic equilibrium point exists when $R_0 > 1$.

4.2.1 Stability Analysis of Endemic Equilibrium

In the presence of the infectious disease, the model populations have a unique endemic steady state. To find the local stability of endemic steady state, the Jacobian of the model equations evaluated at E_1 is used. Now, the stability analysis of endemic steady state is conducted and the results are presented in the form of theorems as follows:

Theorem 4.3. The endemic equilibrium point, E_1 of the system (2.1) is locally asymptotically stable if $R_1 > 1$.

Proof . The jacobian of (4.2) at the endemic equilibrium point 4.4 are:

$$J(E_1) = \begin{bmatrix} -\frac{\pi R_0}{N^*}(1-\frac{1}{R_0}) & 0 & \frac{-\beta}{R_0} & \frac{-\beta}{R_0} & 0 & 0 & 0 & 0 \\ 0 & -a & \frac{\beta}{R_0} & \frac{\beta}{R_0} & 0 & 0 & 0 & 0 \\ 0 & \psi & -b & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi & 0 & -c & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta & \omega & -d & 0 & 0 & 0 \\ 0 & 0 & \varphi & 0 & \gamma & -e & 0 & 0 \\ 0 & 0 & 0 & \theta & \alpha & \eta & -\mu & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & \epsilon & 0 & -\mu \end{bmatrix}$$

Now, the eigenvalues of $J(E_1)$ are required to be found. The characteristic equation $det[J(E_1)-\lambda I] = 0$ is expanded and simplified as follows:

$$(d+\lambda)(e+\lambda)(\mu+\lambda)^2(N^*R_0\lambda - \pi R_0 + \pi R_0^2)(R_0\lambda^3 + aR_0\lambda^2 + bR_0\lambda^2 + cR_0\lambda^2 + abR_0\lambda + acR_0\lambda + bcR_0\lambda + abc + abcR_0 - \beta\lambda\phi - \beta\psi\lambda - b\phi\beta - c\beta\psi) = 0.$$

Then, we have

$$(d+\lambda)(e+\lambda)(\mu+\lambda)^2R_0(N^*\lambda - \pi + \pi R_0)(R_0\lambda^3 + aR_0\lambda^2 + bR_0\lambda^2 + cR_0\lambda^2 + abR_0\lambda + acR_0\lambda + bcR_0\lambda - \beta\lambda\phi - \beta\psi\lambda + abc + abc(R_0 - \frac{\beta(b\phi+c\psi)}{abc}) = 0$$

which implies that

$$(d+\lambda)(e+\lambda)(\mu+\lambda)^2R_0(N^*\lambda - \pi + \pi R_0) = 0$$

or

$$R_0\lambda^3 + R_0(a+b+c)\lambda^2 + (abR_0 + acR_0 + bcR_0 - \beta\phi - \beta\psi)\lambda + abc = 0,$$

where $R_0 = \frac{\beta(b\phi+c\psi)}{abc}$. From $(d+\lambda)(e+\lambda)(\mu+\lambda)^2R_0(N^*\lambda - \pi + \pi R_0) = 0$, the eigenvalues of the endemic equilibrium points are $\lambda_1 = -d$, $\lambda_2 = -e$, $\lambda_3 = -\mu$, $\lambda_4 = -\mu$, $\lambda_5 = -\frac{\pi}{N^*}(R_0 - 1)$, which imply that $R_0 > 1$ and from the polynomial function

$$R_0\lambda^3 + R_0(a+b+c)\lambda^2 + (abR_0 + acR_0 + bcR_0 - \beta\phi - \beta\psi)\lambda + abc = 0,$$

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + abc = 0, \tag{4.5}$$

where $a_0 = \frac{\beta(b\phi+c\psi)}{abc}, a_1 = \frac{\beta(b\phi+c\psi)}{abc}(a+b+c), a_2 = \frac{\beta(b\phi+c\psi)}{abc}(ab+ac+bc) - \beta\phi - \beta\psi, a_3 = abc$. Then the eigenvalues of the characteristic equation (4.5) will be negative if it fulfill the Routh-Hurwitz criteria [9] that are $a_i > 0$ for $i = 0, 1, 2, 3$ and $a_1a_2 > a_3$.

According to the Routh - Hurwitz criteria it follows that all eigenvalues of the characteristic equation (4.5) has negative real part. So, we obtained (i) $a_0 > 0 \Rightarrow \frac{\beta(b\phi+c\psi)}{abc} > 0$ Since all the parameters in the models are nonnegative.

(ii) $a_1 > 0 \Rightarrow \frac{\beta(b\phi+c\psi)}{abc}(a+b+c) > 0$ as all parameters in the models are nonnegative.

(iii) $a_2 > 0 \Leftrightarrow \frac{\beta(b\phi+c\psi)}{abc}(ab+ac+bc) > \beta\phi + \beta\psi$

(iv) $a_3 > 0 \Rightarrow abc > 0$ and $a_1a_2 > a_3$.

The Routh Hurwitz stability criterion is accomplished so if $R_0 > 1$ all the eigenvalues from the linear equilibrium system are negative which generate the endemic equilibrium point of disease locally stability asymptotic. As a result of this prove to be the case that point of any of the endemic equilibrium disease asymptotic stability it means that the disease spread to other individuals. Therefore, it is concluded that the endemic equilibrium point E_1 of the system of differential equations (2.1) is locally asymptotically stable if $R_0 > 1$. □

Theorem 4.4. The endemic equilibrium point of the model equation (2.1) is globally asymptotically stable if $R_0 > 1$.

Proof . To show the result we define the following Lypunov function as follows

$$L(P^*,E^*,I^*,Q^*,H^*,F^*,R^*,D^*)=\left[P-P^*-P^*\ln(\frac{P}{P^*})\right]+\left[E-E^*-E^*\ln(\frac{E}{E^*})\right]+\left[I-I^*-I^*\ln(\frac{I}{I^*})\right]$$
$$+\left[Q-Q^*-Q^*\ln(\frac{Q}{Q^*})\right]+\left[H-H^*-H^*\ln(\frac{H}{H^*})\right]+\left[F-F^*-F^*\ln(\frac{F}{F^*})\right]$$
$$+\left[R-R^*-R^*\ln(\frac{R}{R^*})\right]+\left[D-D^*-D^*\ln(\frac{D}{D^*})\right].$$

By taking the derivative of L with respect to t:

$$\begin{aligned}\frac{dL}{dt}=&(1-\frac{p^*}{p})\frac{dp}{dt}+(1-\frac{E^*}{E})\frac{dE}{dt}+(1-\frac{I^*}{I})\frac{dI}{dt}+(1-\frac{Q^*}{Q})\frac{dQ}{dt}+(1-\frac{H^*}{H})\frac{dH}{dt}+(1-\frac{F^*}{F})\frac{dF}{dt}+(1-\frac{D^*}{D})\frac{dD}{dt}\\
=&(1-\frac{p^*}{p})(\pi-(\mu+\lambda)P)+(1-\frac{E^*}{E})(\lambda P-aE)+(1-\frac{I^*}{I})(\psi E-bI)+(1-\frac{Q^*}{Q})(\phi-cQ)\\
&+(1-\frac{H^*}{H})(\Omega Q+\delta I-dH)+(1-\frac{F^*}{F})(\varphi I+\gamma H-eF)+(1-\frac{R^*}{R})(\theta Q+\phi H+\eta F-\mu R)\\
&+(1-\frac{D^*}{D})(\varepsilon H+\epsilon F-\mu D)\\
=&\pi+\lambda p+\psi E+\phi E+\omega Q+\delta I+\varphi I+\gamma H+\theta Q+\alpha H+\eta F+\mu R+\varepsilon H+\epsilon F+(\mu+\lambda)P^*+aE^*+bI^*+cQ^*\\
&+dH^*+eF^*+\mu D^*-\left[(\mu+\lambda)P+aE+bI+cQ+dH+eF+\mu R+\mu D+\pi\frac{P^*}{P}+\gamma H\frac{F^*}{F}+\psi E\frac{I^*}{I}\right.\\
&\left.+\phi E\frac{Q^*}{Q}+\omega Q\frac{H^*}{H}+\delta I\frac{H^*}{H}+\varphi I\frac{F^*}{F}+\gamma H\frac{F^*}{F}+\theta Q\frac{R^*}{R}+\alpha H\frac{R^*}{R}+\eta F\frac{R^*}{R}+\varepsilon H\frac{D^*}{D}+\epsilon F\frac{D^*}{D}\right].\end{aligned}$$

After some simplification and rearrangement we obtain;

$$\begin{aligned}\frac{dL}{dt}=&\pi+(\mu+\lambda)P^*+aE^*+bI^*+cQ^*+dH^*+eF^*+\mu D^*-[\mu(P+E+I+Q+H+F+D)+\zeta(I+H+F)\\
&+\pi\frac{P^*}{P}+\lambda P\frac{E^*}{E}+\psi E\frac{I^*}{I}+\phi E\frac{Q^*}{Q}+(\omega Q+\delta)\frac{H^*}{H}+(\gamma H+\varphi I+\gamma H)\frac{F^*}{F}+(\theta Q+\alpha H+\eta F)\frac{R^*}{R}\\
&+(\varepsilon H+\epsilon F)\frac{D^*}{D}]\\
=&M-K\end{aligned}$$

where

$$M=\pi+(\mu+\lambda)P^*+aE^*+bI^*+cQ^*+dH^*+eF^*+\mu D^*$$

and

$$K=\left[\mu(P+E+I+Q+H+F+D)+\zeta(I+H+F)+\pi\frac{P^*}{P}+\lambda P\frac{E^*}{E}+\psi E\frac{I^*}{I}+\phi E\frac{Q^*}{Q}+(\omega Q+\delta)\frac{H^*}{H}\right.$$
$$\left.+(\gamma H+\varphi I+\gamma H)\frac{F^*}{F}+(\theta Q+\alpha H+\eta F)\frac{R^*}{R}+(\varepsilon H+\epsilon F)\frac{D^*}{D}\right].$$

Now , $\frac{dL}{dt}=M-K<0$, if $M<K$. Thus if $M<K$ then $\frac{dL}{dt}<0$. Nothing that $\frac{dL}{dt}=0$ if and only if $P=P^*,E=E^*,Q=Q^*,I=I^*,H=H^*,F=F^*,R=R^*,D=D^*$. Thus, the largest compact invariant set in $\{(P^*,E^*,Q^*,I^*,H^*,F^*,F^*,D^*)\in\Omega;\frac{dL}{dt}=0\}$ is a singleton E_1 is the endemic equilibrium point of the system (2.1). By LaSalle's invariant principle [9], it implies that E_1 is globally asymptotically stable in Ω if $M<k$ and $R_0>1$. □

4.3 Sensitivity Analysis

Sensitivity analysis allows us to assess the impact that changes in a certain parameter will have on the model and it can help someone to determine which parameters are the key drivers of a model's results. To investigate which

parameters have high impact on the R_0, we apply the approach presented in [5]. For instance, the normalized forward sensitivity index on R_0 , which depends differentially on a parameter P, as defined in [7] as

$$\Upsilon_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0}. \tag{4.6}$$

The explicit expression of R_0 is given by

$$R_0 = \frac{\beta(b\phi+c\psi)}{abc} = \frac{\beta(\phi(\delta+\varphi+\mu+\zeta)+\psi(\mu+\theta+\omega))}{(\mu+\psi+\phi)(\delta+\varphi+\mu+\zeta)(\mu+\theta+\omega)}.$$

Since R_0 depends only on nine parameters, we derive the analytical expression for its sensitivity to each parameters using the normalized forward sensitivity index as in [5] by taking the values of the paramters from table 2 below and computed as follows:

$$\Upsilon_\beta^{(R_0)} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1,$$

$$\Upsilon_\phi^{(R_0)} = \frac{\partial R_0}{\partial \phi} \times \frac{\phi}{R_0} = \frac{\phi\big(\delta(\mu+\psi)+\zeta(\mu+\psi)-\theta\psi+\mu^2+\mu\varphi+\varphi\psi-\psi\omega\big)}{(\mu+\psi+\phi)(\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega))},$$

$$\Upsilon_\psi^{(R_0)} = \frac{\psi\big(-\phi(\delta+\zeta+\varphi)+\theta(\mu+\phi)+\mu^2+\omega(\mu+\phi)\big)}{(\mu+\psi+\phi)(\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega))},$$

$$\Upsilon_\varphi^{(R_0)} = \frac{\partial R_0}{\partial \varphi} \times \frac{\varphi}{R_0} = -\frac{\varphi\psi(\theta+\mu+\omega)}{(\delta+\zeta+\mu+\varphi)(\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega))},$$

$$\Upsilon_\delta^{(R_0)} = \frac{\partial R_0}{\partial \delta} \times \frac{\delta}{R_0} = -\frac{\delta\psi(\theta+\mu+\omega)}{(\delta+\zeta+\mu+\varphi)(\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega))},$$

$$\Upsilon_\zeta^{(R_0)} = \frac{\partial R_0}{\partial \zeta}\zeta \times \frac{\zeta}{R_0} = -\frac{\zeta\psi(\theta+\mu+\omega)}{(\delta+\zeta+\mu+\varphi)(\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega))},$$

$$\Upsilon_\theta^{(R_0)} = \frac{\partial R_0}{\partial \theta} \times \frac{\theta}{R_0} = -\frac{\theta\phi(\delta+\zeta+\mu+\varphi)}{(\theta+\mu+\omega)(\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega))},$$

$$\Upsilon_\omega^{(R_0)} = \frac{\partial R_0}{\partial \omega} \times \frac{\omega}{\omega}R_0 = -\frac{\omega\phi(\delta+\zeta+\mu+\varphi)}{(\theta+\mu+\omega)(\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega))}.$$

$$\Upsilon_\mu^{(R_0)} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} = \mu\left(\frac{\psi+\phi}{\phi(\delta+\zeta+\mu+\varphi)+\psi(\theta+\mu+\omega)} - \frac{1}{\delta+\zeta+\mu+\varphi} - \frac{1}{\theta+\mu+\omega} - \frac{1}{\mu+\psi+\phi}\right)$$

Table 1: The Values of Sensitivity indices

Parameter Symbol	Sensitivity indices
β	1
ϕ	0.194
ψ	0.151
η	-0.210
ω	-0.240
φ	-0.253
θ	-0.345
μ	-0.540
δ	-0.603

Those parameters that have positive indices i.e. β, ϕ and ψ show that they have great impact on expanding the disease in the community if their values are increasing due to the reason that the average number of secondary cases of infection increases in the community. Furthermore, those parameters in which their sensitivity indices are negative i.e. $\eta, \omega, \varphi, \theta, \mu$ and δ have an influence of minimizing the burden of the disease in the community as their values increase. And also, as their values increase, the basic reproduction number decreases, which leads to minimize the endemicity of the disease in the community.

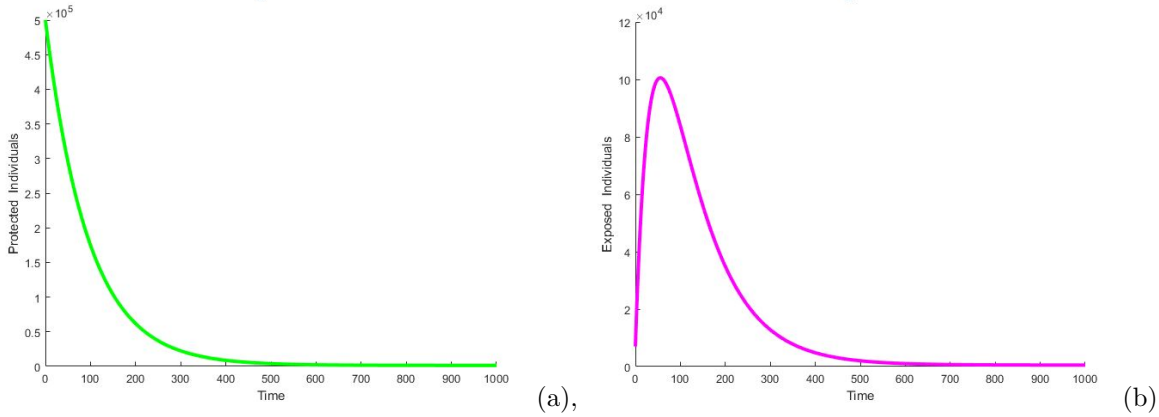
5 Numerical Simulation

In this section, numerical simulation study of model equations 2.1 are carried out using the software $MATLABR2015b$ with $ODE45$ solver. To conduct the study, a set of physically meaningful values are assigned to the model parameters. These values are either taken from literature review or assumed on the basis of reality.

Table 2: Parameter Values for Covid-19 Model

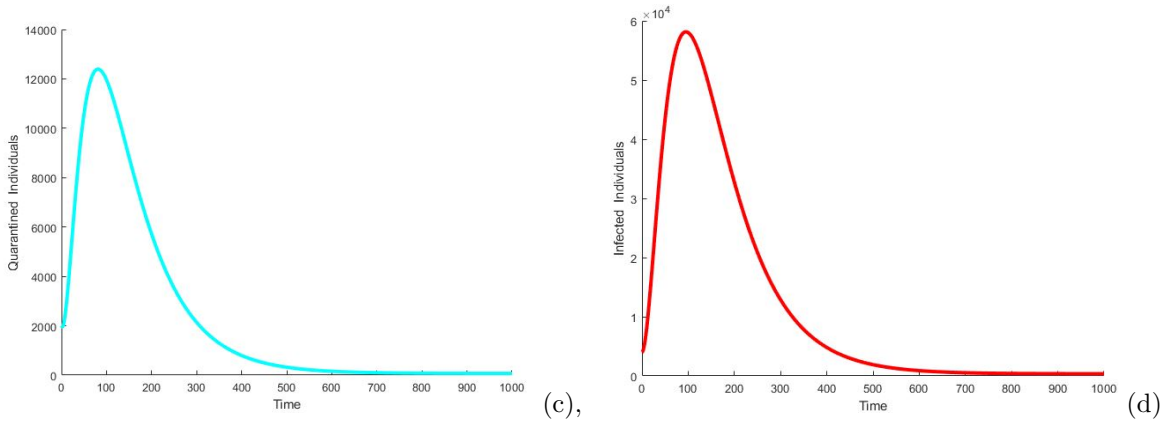
Parameters	Value	Source
π	150	Assumed
μ	0.020	[8]
β	0.07600	Assumed
φ	0.0260	[8]
ϕ	0.0140	Assumed
γ	0.024	[8]
η	0.012	Assumed
δ	0.062	Assumed
ε	0.012	Assumed
ζ	0.0216	Assumed
α	0.016	Assumed
ϵ	0.156	Assumed
ψ	0.024	Assumed
θ	0.046	[8]
ω	0.032	Assumed

Using the parameter values given in table 2 and the initial conditions $P(0) = 500000$, $E(0) = 17000$, $I(0) = 4000$, $Q(0) = 2000$, $H(0) = 1000$, $R(0) = 6000$, $D(0) = 5000$ in the model equations 2.1. A simulation study is conducted and the results are given in the following figure below.



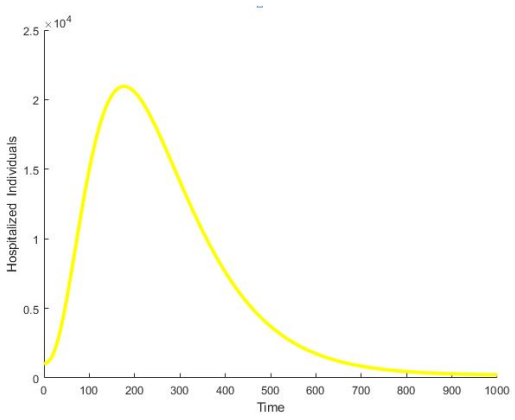
(a), (b)

Figure 2: Dynamics of protected and exposed individuals respectively

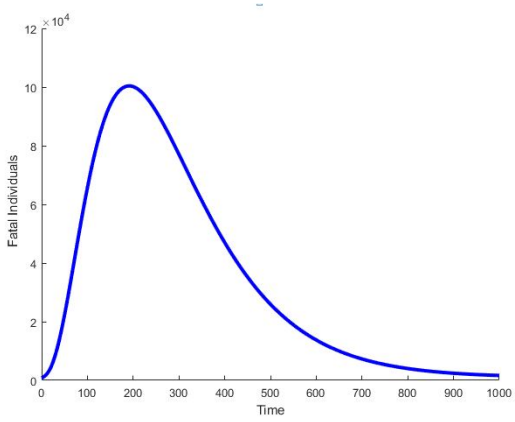


(c), (d)

Figure 3: Dynamics of quarantined and infected individuals respectively

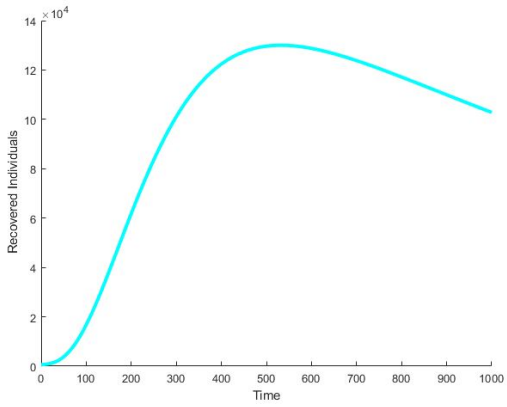


(e),



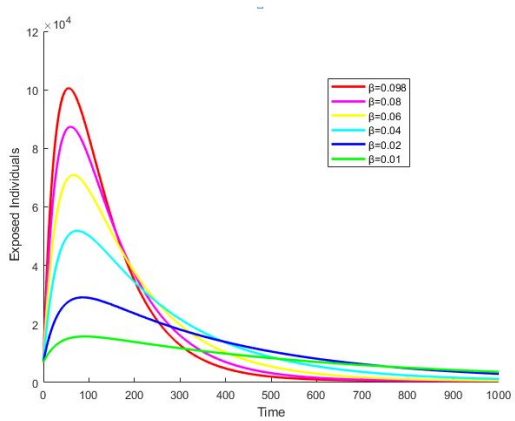
(f)

Figure 4: Dynamics of hospitalized and fatal individuals Respectively

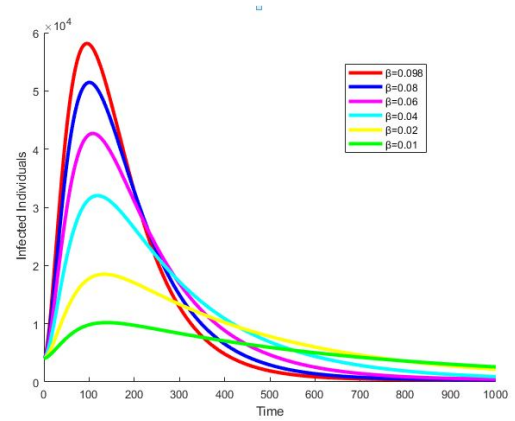


(g)

Figure 5: Dynamics of recovered Individuals

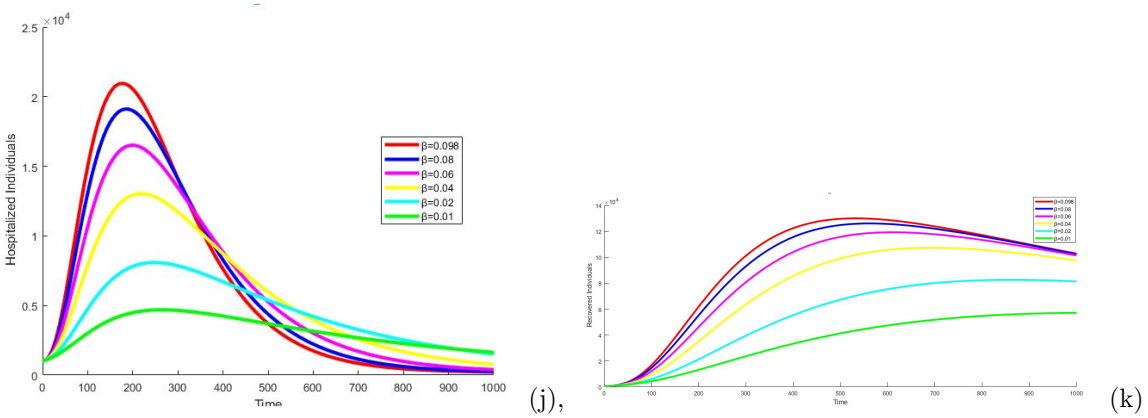


(h),



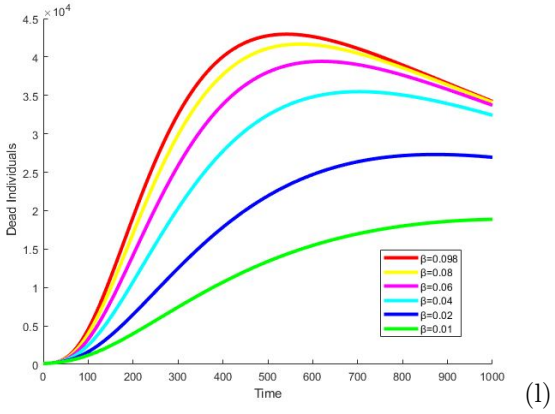
(i)

Figure 6: Effect of varying contact rate on exposed and infected individuals respectively.



(j), (k)

Figure 7: Effect of varying contact rate on hospitalized and fatal individuals respectively.



(l)

Figure 8: Effect of varying contact rate on Death individuals.

Figure(2a) shows that protected individuals decreases due to loss of protection at a rate λ and more number of protected individuals join exposed class and converges to disease free equilibrium points. Like wise Figure(2b) shows that exposed individuals increase firstly as a result of some protected individual joins exposed class because of effective contact with an infectious individuals and decrease due to it join the quarantine and infected classes. Also figure(3c) shows that quarantine individuals increase firstly as a consequence of some exposed individual join quarantine class at a rate and decrease due to it join hospitalized and recovered classes. Similarly Figure (3d) shows that infected individuals increase firstly as a result of some exposed individual joins infected class and decrease due to it join hospitalized and fatal classes . Figure (4e) shows that hospitalized individuals increase firstly as a result of some quarantined and infected individual join it and decrease due to it join recovered,fatal and death classes. Figure (4f), shows that fatal individuals increase firstly as a result of some infected and hospitalized individual joins it and decrease due to it join reovered and death classes and figure (5g) shows that recovered individuals icreass due to quarantine, hospitalized and fatal classes join to it and decrease only due to natural death. Finally, Figure (6h), (6i), (7j), (7k) and (8l) shows that contact rate has an effect on reducing COVID-19 viruse from the community. Thus decreasing the level of effective contact rate among individuals has an effect on reducing the prevalence of COVID-19 pandemic disease from the community.

6 Conclusions and Recommendations

In this study, we have formulated a special model on the transmission dynamics of COVID-19 which contains eight compartments. Moreover, the existence, positivity and boundedness of the formulated model are verified to illustrate that the model is biologically meaningful and mathematically well posed. In particular, the stability analyses of the model were investigated using the basic reproduction number. Also, the solution of the formulated model equation is numerically integrated and the sensitivity analysisof the model is analyzed to determine which parameter has a high impact on the transmission dynamics of the diseases. Numerical simulations of the model show that each parameters in the model has an effect on the model variables and when the effective contact rate is increasing the disease transmission in the community is increasing and when the effective contact rate is decreasing the disease transmission in the community is decreasing. Although, eradication of COVID-19 infection remains a challenge on the world, but from results of this study we recommend that, the government should introduce education programmers on the importance of voluntary and routinely quarantined on COVID-19 infection. Also, there is need to increase the number of hospitals to deal with COVID-19 infection and to quarantine more individuals with infection to reduce the transmission. Moreover, the future work of this paper will consider the fractional derivatives to COVID-19 model and its optimal control.

References

[1] S. Abdulrahman, N.I. Akinwande, O.B. Awojoyogbe, and U.Y. Abubakar, *Sensitivity analysis of the parameters of a mathematical model of hepatitis b virus transmission*, Univer. J. Appl. Math. 1 (2013), no. 4, 230–241.

[2] M. Anderson. *Mathematical biology of infectious diseases: Part 1* Nature **280** (1979), 361–367.

[3] C. Castillo-Chavez, Z. Feng, and W. Huang, *On the computation of Ro and its role on global stability*, Math. Approach. Emerg. Reemerg. Infec. **1** (2002), 229.

[4] T.-M. Chen, J.R., Q.-P. Wang, Z.-Y. Zhao, J.-A. Cui, and L. Yin, *A mathematical model for simulating the phase-based transmissibility of a novel coronavirus*, Infectious Diseases of Poverty **9** (2020), no. 1, 1–8.

[5] N. Chitnis, J.M. Hyman, and J.M. Cushing, *Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model*, Bull. Math. Bio. **70** (2008), no. 5, 1272.

[6] O. Diekmann, J.A.P. Heesterbeek, and J.A. Metz, *On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations*, J. Math. Bio. **28** (1990), no. 4, 365–382.

[7] S. Edward and N. Nyerere, *A mathematical model for the dynamics of cholera with control measures*, Appl. Comput. Math. **4** (2015), no. 2, 53–63.

[8] E.D. Gurmua, G.B. Batu, and M.S. Wameko, *Mathematical model of novel covid-19 and its transmission dynamics*, Int. J. Math. Modell. Comput. **10** (2020), no. 2, 141–159.

[9] J.P. La Salle, *The stability of dynamical systems*, Society for Industrial and Applied Mathematics, 1976.

[10] Y. Li, B. Wang, R. Peng, C. Zhou, Y. Zhan, Z. Liu, X. Jiang, and B, Zhao, *Mathematical modeling and epidemic prediction of COVID-19 and its significance to epidemic prevention and control measures*, Ann. Infect. Disease Epidemio. **5** (2020), no. 1, 1052.

[11] W. Ming, J.V. Huang, and C.J.P. Zhang, *Breaking down of healthcare system: Mathematical modelling for controlling the novel coronavirus (2019-nCoV) outbreak in Wuhan, China*, BioRxiv (2020). doi: 10.1101/2020.01.27.922443

[12] Coronavirus: Common Symptoms, Preventive Measures, and How to Diagnose It; https://www. caringlyyours.com/coronavirus/, Caringly Yours. Retrieved **January 28, 2020**

[13] C. Wang, P.W. Horby, F.G. Hayden, and G.F. Gao, *A novel coronavirus outbreak of global health concern*, Lancet **395** (2020), 470–473.

[14] L. Wang, Y. Wang, Y. Chen, and Q. Qin. *Unique epidemiological and clinical features of the emerging 2019 novel coronavirus pneumonia (covid-19) implicate special control measures*, J. Med. Virology **92** (2020), no. 6, 568–576.

[15] World Health Organization. *Naming the coronavirus disease (COVID-19) and the virus that causes it.* https://www.who.int/emergencies/diseases/novel-coronavirus-2019/technical-guidance/naming-the-coronavirus-disease-(covid-2019)-and-the-virus-that-causes-it, **February (2020)**.

[16] World Health Organization. Statement on the second meeting of the international health regulations (2005). emergency committee regarding the outbreak of novel coronavirus (2019-ncov). https://www.who.int/news-room/detail/30-01-2020-statement-on-the-second-meeting-of-the-international-health-regulations-(2005)-emergency-committee-regarding-the-outbreak-of-novel-coronavirus-(2019-ncov), **January (2020)**.

[17] World Health Organization. *Director-general's opening remarks at the media briefing on COVID-19 - 11 march 2020*. https://www.who.int/dg/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-covid-19-11-March-2020, March **2020**.

[18] C. Yang and J. Wang, *A mathematical model for the novel coronavirus epidemic in Wuhan, China*, MBE **17** (2020), no. 3, 2708–2724.