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Mathematical model for transmission dynamics of novel COVID-19 with sensitivity analysis

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Abstract

In this study, we formulated a deterministic compartmental model of COVID-19 to describe the transmission dynamics of the disease. Stability theory of differential equations is used to study the qualitative behavior of the system. The basic reproduction number that represents the epidemic indicator is obtained by using next generation matrix. Both local and global stability of the disease free equilibrium and endemic equilibrium point of the model equation was established. The results show that, if the basic reproduction number is less than one then the solution converges to the disease free steady state and the disease free equilibrium is asymptotically stable. The endemic states are considered to exist when the basic reproduction number for each disease is greater than one. Numerical simulation carried out on the model revealed that an increase in level of transmission rate among individuals has an effect on reducing the prevalence of COVID-19 and COVID-19 disease. Furthermore, sensitivity analysis of the model equation was performed on the key parameters to find out their relative significance and potential impact on the transmission dynamics of COVID-19.

Keywords: Model, Global Stability, Sensitivity, Reproduction Number
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1 Introduction

The disease caused by an infection with SARS-CoV-2 is called COVID-19, which stands for coronavirus disease 2019. Coronaviruses are enveloped, positive-sense single-stranded RNA viruses with a nucleocapsid of helical symmetry. Coronaviruses have widely been known to cause respiratory and intestinal infections in humans after the outbreak of "severe acute respiratory syndrome (SARS)" in Guangdong, China [9]. It was first detected in Wuhan, China, in December 2019. On 30 January 2020, the WHO Director-General declared that the current outbreak constituted a public health emergency of international concern [12].

A lot of studies suggest that corona viruses, including preliminary information on the COVID-19 virus may persist on the surfaces for a few hours or up-to several days. COVID-19 may not initially cause any symptoms for some people; it may show symptoms after 2 days or up to 2 weeks. Some common symptoms that have been specifically

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linked to COVID-19 include; shortness of breath, having a cough that gets more severe over time, a low-grade fever that gradually increases in temperature [11]. Once the virus develops in people, coronaviruses can be spread from person to person through respiratory droplets. There's currently no treatment specifically approved for COVID-19, and no cure for an infection, although treatments and vaccines are currently under study. The best way to prevent the spread of infection is to avoid or limit contact with people who are showing symptoms of COVID-19 or any respiratory infection [7].

Globally, coronavirus disease 2019 (COVID-19) Situation Report –72 [13] shows that 1133758 confirmed and 62784 deaths, in Western Pacific Region 111396 confirmed and 3838 deaths, in European Region 621407 confirmed and 46416 deaths, in South-East Asia Region 7816 confirmed and 302 deaths, in Eastern Mediterranean Region 70293 confirmed and 3794 deaths, in Region of the Americas 315714 confirmed and 8187deaths and also in African Region 6420 confirmed and 236 deaths.Ethiopia's COVID-19 file as of April 24 has 1 cases (1 new case) with three deaths and 25recoveries. The total number of tests stands at 4,557. Active cases stand at 117 representing about 77% of recorded cases.

Many mathematical models have been used extensively in research into the epidemiology of COVID-19 to improve our understanding of the major contributing factors. A lot of authors developed a mathematical model to describe the dynamics of the disease that helped them to propose disease control mechanism and also described the transmission dynamics of the diseases. J. Jia et al [8] propose a dynamical model with seven compartments to describe the transmission of COVID-19 in China. In their study, they design detailed vaccination strategies for COVID-19 in different control phases and show the effectiveness of large scale vaccination. Furthermore, Chayu Yang et al. [14] model describes the multiple transmission pathways in the infection dynamics, and emphasizes the role of the environmental reservoir in the transmission and spread of this disease. The analytical and numerical results indicate that the coronavirus infection would remain endemic, which necessitates long-term disease prevention and intervention programs. A predictive simple mathematical model that can give us some idea of the fate of the virus, an indicative data and future projections to understand the further course this pandemic is proposed by Jyoti Bhola et al. [1]. This paper is used at regional level to manage the health care system in the present scenario. Several mathematical models are proposed to illustrate the transmission dynamics of the coronavirus infection. Eshetu Dadi et al. [3] proposed a mathematical model of COVID-19 and its transmission dynamics. They construct the dynamic models of the seven compartments and their results show that an increase in level of contact rate among individuals has an effect on reducing the prevalence of COVID-19 and COVID-19 disease. In this paper we modify the model developed by Eshetu Dadi al. [3], by adding the assumption individuals in Coronavirus class are recovered from disease.

2 Model Formulation and Description

In this section, we divide the model into six subcompartments. The total population $N(t)$ is divided into six subcompartments consisting of protected individuals who are protected against the disease over period of time (P), susceptible individuals who are vulnerable to the disease over a period of time (S), infectious individuals who are showing symptoms of corona virus (COVID-19) (I), quarantine individuals who are infectious and compulsory quarantine due to reduce the spread of COVID-19 and get treatment based on the patient's clinical condition (Q), coronavirus (COVID-19) individuals are at the chronic stage of corona virus (C) and recovered individuals who are recovered from the disease at a time t (R). The total human populations are represented as

$$N(t) = P(t) + S(t) + I(t) + Q(t) + C(t) + R(t) \tag{2.1}$$

Protected individuals are recruited into the population at a rate $\prod$ and decreased by natural death at a rate μ and by losing protection at a rate. Susceptible subcompartment is increased by losing protection of protected class at a rate η and from recovered subcompartment by losing immunity at a rate ψ. Susceptible individuals are acquiring COVID-19 infection with force of infection λ which is given by $\lambda = \frac{\beta I}{N}$, where β is the transmission. Infected individual are increased by the fraction of susceptible individual at a rate λ. Those individuals in the infected subcompartment can get treatment and join quarantined subcompartment with rate of φ and others join recovered subcompartment with rate α. Individuals who develop COVID-19 symptom in infected subcompartment join coronavirus subcompartment with rate ω. The quarantine subcompartment also increases with individuals who come frominfected subcompartment by getting treatment with a rate φ. Quarantine individuals who are recovered from the disease join the recovered subcompartment with rate γ and others join the coronavirus subcompartment with rate of θ. Corona virus subcompartment is increased by quarantine individuals who lose natural immunity at a rate θ and from infected subcompartment who developed symptom of COVID-19 with rate ω. Some individuals

in coronavirus subcompartment are recovered by natural immunity with rate δ. In all the subcompartment, μ is the natural death rate of individuals, but in the infectious compartment ξ is the disease induced death rate. All parameters in the model are positive. Depend on the basic assumptions the schematic diagram of the modified model can be given as in figure 1 below.

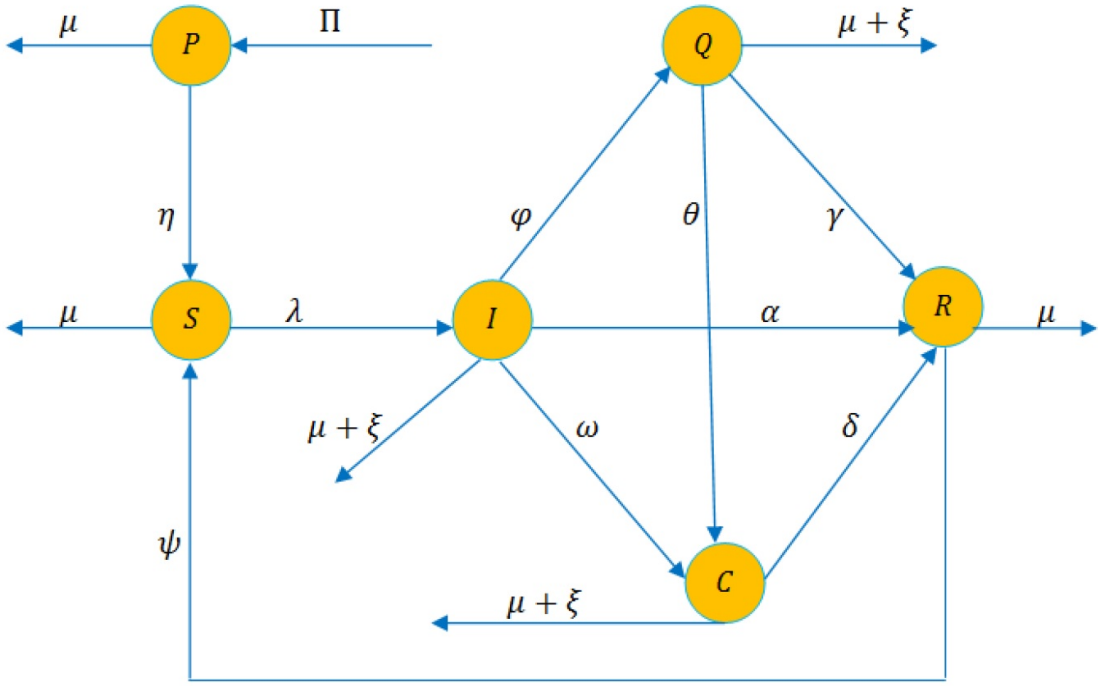

Figure 1: Schematic diagram of the model

Based on the model assumptions and the schematic diagram the model equations are formulated and given as follows:

$$\begin{aligned}
\frac{dP}{dt} &= \Pi - (\eta + \mu)P \\
\frac{dS}{dt} &= \eta P + \psi R - (\lambda + \mu)S \\
\frac{dI}{dt} &= \lambda S - (\varphi + \alpha + \omega + \mu + \xi)I \\
\frac{dQ}{dt} &= \varphi I - (\theta + \gamma + \mu + \xi)Q \\
\frac{dC}{dt} &= \omega I + \theta Q - (\delta + \mu + \xi)C \\
\frac{dR}{dt} &= \gamma Q + \alpha I + \delta C - (\psi + \mu)R
\end{aligned} \tag{2.2}$$

The non-negative initial conditions of the system of model equations (2.2) are denoted by $P(0) = P_0,\ S(0) = S_0,\ I(0) = I_0,\ Q(0) = Q_0,\ C(0) = C_0,\ R(0) = R_0$.

3 The Mode Analysis

3.1 Invariant Region

In this section, we get a region in which the solution of model equation (2.2) is bounded. To obtain this, first we considered the total population (N), where $N = P + S + I + Q + C + R$. Then, differentiating (N) both sides with respect to t leads

$$\frac{dN}{dt} = \frac{dP}{dt} + \frac{dS}{dt} + \frac{dI}{dt} + \frac{dQ}{dt} + \frac{dC}{dt} + \frac{dR}{dt}. \tag{3.1}$$

Substituting model equation (2.2) into equation (3.1), we can get

$$\frac{dN}{dt} = \Pi - \mu N - \xi(I + Q + C). \tag{3.2}$$

In the absence of mortality due to COVID-19 ($\xi = 0$), then equation (3.2) become

$$\frac{dN}{dt} \leq \Pi - \mu N. \tag{3.3}$$

Rearranging and integrating both sides of (3.3), we get

$$\int \frac{dN}{\Pi - \mu N} \leq \int dt \quad \text{if and only if} \quad \frac{-1}{\mu}\ln(\Pi - \mu N) \leq t + c_1, \text{where } c_1 \text{ is integration constant.}$$

Thus,

$$\ln(\Pi - \mu N) \geq -\mu t + c_2, \text{where } c_2 = -\mu c_1$$

and so,

$$(\Pi - \mu N) \geq ce^{-\mu t}, \text{where } c = e^{-c_2}.$$

Then, applying initial condition $N(0) = N_0$, we obtain $c = \Pi - \mu N_0$. Thus, $\Pi - \mu N \geq (\Pi - \mu N_0)e^{-\mu t}$. This implies that

$$N \leq \frac{\Pi}{\mu} - \left[\frac{\Pi - \mu N}{\mu}\right] e^{-\mu t}. \tag{3.4}$$

Further, it can be observed that $N(t) \longrightarrow (\Pi/\mu)$ as $t \longrightarrow \infty$. That is, the total population size $N(t)$ takes off from the value $N(0)$ at the initial time $t = 0$ and ends up with the bounded value (Π/μ) as the time t grows to infinity. Thus, it can be concluded that $N(t)$ is bounded as $0 \leq N(t) \leq (\Pi/\mu)$. Thus, the feasible solution set of the system equation of the model enters and remains in the region:

$$\Omega = \{(P, S, I, Q, C, R) \in \Re^6_+ : N \leq \Pi/\mu\}$$

Therefore, the model equation (2.2) is well posed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the basic model in the region Ω.

3.2 Existence of the solution

Lemma 3.1. (Existence) Solutions of the model equations (2.2) together with the initial conditions $P(0) > 0,\ S(0) > 0,\ I(0) > 0,\ Q(0) > 0,\ C(0) > 0,\ R(0) > 0$ exist in $\mathbb{R}^6_+$ i.e., the model variables $P(t), S(t), I(t), Q(t), C(t)$ and $R(t)$ exist for all t and will remain in $\mathbb{R}^6_+$.

Proof . The right hand sides of the system of equations (2.2) can be expressed as follows:

$$\begin{aligned}
f_1(P, S, I, Q, C, R) &= \Pi - (\eta + \mu)P \\
f_2(P, S, I, Q, C, R) &= \eta P + \psi R - (\lambda + \mu)S \\
f_3(P, S, I, Q, C, R) &= \lambda S - (\varphi + \alpha + \omega + \mu + \xi)I \\
f_4(P, S, I, Q, C, R) &= \varphi I - (\theta + \gamma + \mu + \xi)Q \\
f_5(P, S, I, Q, C, R) &= \omega I + \theta Q - (\delta + \mu + \xi)C \\
f_6(P, S, I, Q, C, R) &= \gamma Q + \alpha I + \delta C - (\psi + \mu)R
\end{aligned}$$

According to Derrick and Groosman theorem, let Ω denote the region $\Omega = \{(P, S, I, Q, C, R) \in \Re_+^6 : N \leq \Pi/\mu\}$. Then equations (2.2) have a unique solution if $(\partial f_i)/(\partial x_j),\ i, j = 1, 2, 3, 4, 5, 6$ are continuous and bounded in Ω. Here, $x_1 = P,\ x_2 = S,\ x_3 = I,\ x_4 = Q,\ x_5 = C$ and $x_6 = R$. The continuity and the boundedness are verified as here under Table 1:

Table 1: Continuity and boundedness of the model solution

$\lvert(\partial f_1)/(\partial P)\rvert = \lvert-(\eta+\mu)\rvert < \infty$	$\lvert(\partial f_2)/(\partial P)\rvert = \lvert\eta\rvert < \infty$
$\lvert(\partial f_1)/(\partial S)\rvert = 0 < \infty$	$\lvert(\partial f_2)/(\partial S)\rvert = \lvert-(\lambda+\mu)\rvert < \infty$
$\lvert(\partial f_1)/(\partial I)\rvert = 0 < \infty$	$\lvert(\partial f_2)/(\partial I)\rvert = \lvert(-\beta S/N)\rvert < \infty$
$\lvert(\partial f_1)/(\partial Q)\rvert = 0 < \infty$	$\lvert(\partial f_2)/(\partial Q)\rvert = 0 < \infty$
$\lvert(\partial f_1)/(\partial C)\rvert = 0 < \infty$	$\lvert(\partial f_2)/(\partial C)\rvert = 0 < \infty$
$\lvert(\partial f_1)/(\partial R)\rvert = 0 < \infty.$	$\lvert(\partial f_2)/(\partial R)\rvert = \lvert\psi\rvert < \infty.$
$\lvert(\partial f_3)/(\partial P)\rvert = 0 < \infty$	$\lvert(\partial f_4)/(\partial P)\rvert = 0 < \infty$
$\lvert(\partial f_3)/(\partial S)\rvert = \lvert\lambda\rvert < \infty$	$\lvert(\partial f_4)/(\partial S)\rvert = 0 < \infty$
$\lvert(\partial f_3)/(\partial I)\rvert = \lvert-(\varphi+\alpha+\omega+\mu+\xi)\rvert < \infty$	$\lvert(\partial f_4)/(\partial I)\rvert = \lvert\varphi\rvert < \infty$
$\lvert(\partial f_3)/(\partial Q)\rvert = 0 < \infty$	$\lvert(\partial f_4)/(\partial Q)\rvert = \lvert-(\theta+\gamma+\mu+\xi)\rvert < \infty$
$\lvert(\partial f_3)/(\partial C)\rvert = 0 < \infty$	$\lvert(\partial f_4)/(\partial C)\rvert = 0 < \infty$
$\lvert(\partial f_3)/(\partial R)\rvert = 0 < \infty.$	$\lvert(\partial f_4)/(\partial R)\rvert = 0 < \infty.$
$\lvert(\partial f_5)/(\partial P)\rvert = 0 < \infty$	$\lvert(\partial f_6)/(\partial P)\rvert = 0 < \infty$
$\lvert(\partial f_5)/(\partial S)\rvert = 0 < \infty$	$\lvert(\partial f_6)/(\partial S)\rvert = 0 < \infty$
$\lvert(\partial f_5)/(\partial I)\rvert = \lvert\omega\rvert < \infty$	$\lvert(\partial f_6)/(\partial I)\rvert = \lvert\alpha\rvert < \infty$
$\lvert(\partial f_5)/(\partial Q)\rvert = \lvert\theta\rvert < \infty$	$\lvert(\partial f_6)/(\partial Q)\rvert = \lvert\gamma\rvert < \infty$
$\lvert(\partial f_5)/(\partial C)\rvert = \lvert-(\delta+\mu+\xi)\rvert < \infty$	$\lvert(\partial f_6)/(\partial C)\rvert = \lvert\delta\rvert < \infty$
$\lvert(\partial f_5)/(\partial R)\rvert = 0 < \infty.$	$\lvert(\partial f_6)/(\partial R)\rvert = \lvert-(\psi+\mu)\rvert < \infty.$

Thus, all the partial derivatives $(\partial f_i)/(\partial x_j),\ i, j = 1, 2, 3, 4, 5, 6$ exist, continuous and bounded in Ω. Hence, by Derrick and Groosman theorem, a solution for the model (2.2) exists and is unique. □

3.3 Positivity of the solution

The solution of the system remains positive at any point in time t, if the initial values of all the variables are positive.

Lemma 3.2. Let $\Omega = \{(P, S, I, Q, C, R) \in \mathbb{R}_+^6; P_0 > 0,\ S_0 > 0,\ I_0 > 0,\ Q_0 > 0,\ C_0 > 0,\ R_0 > 0\}$; then the solutions of $\{P, S, I, Q, C, R\}$ are positive for all $t \geq 0$.

Proof . Positivity is verified separately for each of the model $P(t), S(t), I(t), Q(t), C(t)$ and $R(t)$.

Positivity of $P(t)$: From model equation (2.2) we have, $\frac{dP}{dt} = \Pi - (\eta+\mu)P$, eliminating the positive terms (Π) we get, $\frac{dP}{dt} \geq -(\eta+\mu)P$, using variables separable method we get, $\frac{dP}{P} \geq -(\eta+\mu)dt$, and integrating both side we can get,

$$\int \frac{dP}{P} \geq -\int (\eta+\mu)dt.$$

Then, $\ln P \geq -(\eta+\mu)t + c_3$, where c_3 is the integration constant. Thus, $P(t) \geq P_0 e^{-(\eta+\mu)t}, P_0 = e^{c_3}$ and $e^{-(\eta+\mu)t} \geq 0$, for all $t \geq 0$. Hence, it can be concluded that $P(t) \geq 0$.

Positivity of $S(t)$: From model equation (2.2), we have, $\frac{dS}{dt} = \eta P + \psi R - (\lambda+\mu)S$, eliminating the positive terms $(\eta P + \psi R)$ we get, $\frac{dS}{dt} \geq -(\lambda+\mu)S$, using variables separable method we get, $\frac{dS}{S} \geq -(\lambda+\mu)dt$, integrating both side we can get,

$$\int \frac{dS}{S} \geq -\int (\lambda+\mu)dt.$$

Then, $\ln S \geq -(\lambda+\mu)t + c_4$, where c_4 is the integration constant. Thus, $S(t) \geq S_0 e^{-(\lambda+\mu)t},\ S_0 = e^{c_4}$ and $e^{-(\lambda+\mu)t} \geq 0$, for all $t \geq 0$. Hence, it can be concluded that $S(t) \geq 0$.

Positivity of $I(t)$: From model equation (2.2), we have, $\frac{dI}{dt} = \lambda S - (\varphi+\alpha+\omega+\mu+\xi)I$, eliminating the positive terms (λS) we get, $\frac{dI}{dt} \geq -(\varphi+\alpha+\omega+\mu+\xi)I$, using variables separable method we get, $\frac{dI}{I} \geq -(\varphi+\alpha+\omega+\mu+\xi)dt$,

integrating both side we can get,

$$\int \frac{dI}{I} \geq -\int (\varphi + \alpha + \omega + \mu + \xi)dt.$$

Then, $\ln I \geq -(\varphi + \alpha + \omega + \mu + \xi)t + c_5$, where c_5 is the integration constant. Thus, $I(t) \geq I_0 e^{-(\varphi+\alpha+\omega+\mu+\xi)t}$, $I_0 = e^{c_5}$ and $e^{-(\varphi+\alpha+\omega+\mu+\xi)t} \geq 0$, for all $t \geq 0$. Hence, it can be concluded that $I(t) \geq 0$.

Positivity of $Q(t)$: From model equation (2.2), we have, $\frac{dQ}{dt} = \varphi I - (\theta + \gamma + \mu + \xi)Q$, eliminating the positive terms (φI) we get, $\frac{dQ}{dt} \geq -(\theta + \gamma + \mu + \xi)Q$, using variables separable method we get, $\frac{dQ}{Q} \geq -(\theta + \gamma + \mu + \xi)dt$, integrating both side we can get,

$$\int \frac{dQ}{Q} \geq -\int (\theta + \gamma + \mu + \xi)dt.$$

Then, $\ln Q \geq -(\theta + \gamma + \mu + \xi)t + c_6$, where c_6 is the integration constant. So, $Q(t) \geq Q_0 e^{-(\theta+\gamma+\mu+\xi)t}$, $Q_0 = e^{c_6}$ and $e^{-(\theta+\gamma+\mu+\xi)t} \geq 0$, for all $t \geq 0$. Hence, it can be concluded that $Q(t) \geq 0$.

Positivity of $C(t)$: From model equation (2.2) we have, $\frac{dC}{dt} = \omega I + \theta Q - (\delta + \mu + \xi)C$, eliminating the positive terms $(\omega I + \theta Q)$ we get, $\frac{dC}{dt} \geq -(\delta + \mu + \xi)C$, using variables separable method we get, $\frac{dC}{C} \geq -(\delta + \mu + \xi)dt$, integrating both side we can get,

$$\int \frac{dC}{C} \geq -\int (\delta + \mu + \xi)dt.$$

Then, $\ln C \geq -(\delta + \mu + \xi)t + c_7$, where c_7 is the integration constant. Thus, $C(t) \geq C_0 e^{-(\delta+\mu+\xi)t}$, $C_0 = e^{c_7}$ and $e^{-(\delta+\mu+\xi)t} \geq 0$, for all $t \geq 0$. Hence, it can be concluded that $C(t) \geq 0$.

Positivity of $R(t)$: From model equation (2.2), we have, $\frac{dR}{dt} = \gamma Q + \alpha I + \delta C - (\psi + \mu)R$, eliminating the positive terms $(\gamma Q + \alpha I + \delta C)$ we get, $\frac{dR}{dt} \geq -(\psi + \mu)R$, using variables separable method we get, $\frac{dR}{R} \geq -(\psi + \mu)dt$, integrating both side we can get,

$$\int \frac{dR}{R} \geq -\int (\psi + \mu)dt.$$

Then, $\ln R \geq -(\psi + \mu)t + c_8$, where c_8 is the integration constant. Therefore, $R(t) \geq R_0 e^{-(\psi+\mu)t}$, $R_0 = e^{c_8}$ and $e^{-(\psi+\mu)t} \geq 0$, for all $t \geq 0$. Hence, it can be concluded that $R(t) \geq 0$.

Therefore, the model variables $P(t), S(t), I(t), Q(t), C(t)$ and $R(t)$ representing population sizes of various types of cells are positive quantities and will remain in $\mathbb{R}^6_+$ for all t. □

3.4 The Disease Free Equilibrium (DFE)

We obtained the disease free equilibrium of model equation (2.2) by equating the right hand side of model (2.2) to zero, evaluating it at $I = Q = C = R = 0$ and solving for the non infected variables we get, $\Pi - (\eta + \mu)P = 0$. Then, $P = \frac{\Pi}{(\eta+\mu)}$. This implies that $\eta\left[\frac{\Pi}{(\eta+\mu)}\right] + \psi R - (\lambda + \mu)S = 0$ and so we have, $S = \frac{\Pi\eta}{\mu(\eta+\mu)}$.

Therefore, the disease-free equilibrium point of the model equation in (2.1)–(3.4) above is given by:

$$E_0 = \{P^0, S^0, I^0, Q^0, C^0, R^0\} = \left\{\left[\frac{\Pi}{(\eta + \mu)}\right], \left[\frac{\Pi\eta}{\mu(\eta + \mu)}\right], 0, 0, 0, 0\right\}.$$

3.5 The Basic Reproduction Number ($\mathfrak{R}_0$)

The basic reproductive number $\mathfrak{R}_0$ can be determined using the next generation matrix as in [5]. In this method, $\mathfrak{R}_0$is defined as the largest eigenvalue of the next generation matrix. The formulation of this matrix involves classification of all compartments of the model in to two classes: infected and non-infected. That is, the basic reproduction number cannot be determined from the structure of the mathematical model alone but depends on the definition of infected and uninfected compartments. The model equations are rewritten starting with newly infective classes:

$$\begin{aligned}
\frac{dI}{dt} &= \lambda S - (\varphi + \alpha + \omega + \mu + \xi)I \\
\frac{dQ}{dt} &= \varphi I - (\theta + \gamma + \mu + \xi)Q \\
\frac{dC}{dt} &= \omega I + \theta Q - (\delta + \mu + \xi)C
\end{aligned} \tag{3.5}$$

Then by the principle of next-generation matrix, we obtained

$$f_i = \begin{bmatrix} \beta IS/N \\ 0 \\ 0 \end{bmatrix} \text{ and } v_i = \begin{bmatrix} (\varphi+\alpha+\omega+\mu+\xi)I \\ -\varphi I + (\theta+\gamma+\mu+\xi)Q \\ -\omega I - \theta Q + (\delta+\mu+\xi)C \end{bmatrix} \tag{3.6}$$

The Jacobian matrices of f_i and v_i evaluated at DFE are given by F and V, respectively, such that

$$F = \begin{bmatrix} \beta & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} a & 0 & 0 \\ -\varphi & b & 0 \\ -\omega & 0 & c \end{bmatrix} \tag{3.7}$$

It can be verified that the matrix V is non-singular as its determinant $det[V] = abc$ is non-zero and after some algebraic computations its inverse matrix is constructed as

$$V^{-1} = \begin{bmatrix} \frac{1}{a} & 0 & 0 \\ \frac{\varphi}{ab} & \frac{1}{b} & 0 \\ \frac{\varphi\theta+\omega b}{abc} & \frac{\theta}{bc} & \frac{1}{c} \end{bmatrix}$$

The product of the matrices F and V^{-1} can be computed as:

$$FV^{-1} = \begin{bmatrix} \beta & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{a} & 0 & 0 \\ \frac{\varphi}{ab} & \frac{1}{b} & 0 \\ \frac{\varphi\theta+\omega b}{abc} & \frac{\theta}{bc} & \frac{1}{c} \end{bmatrix} = \begin{bmatrix} \frac{\beta}{a} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

Now it is possible to calculate the eigenvalue to determine the basic reproduction number $\Re_0$ by taking the spectral radius of the matrix FV^{-1}. Thus, the eigenvalues are computed by evaluating $det[FV^{-1} - \chi I] = 0$ or equivalently solving

$$\begin{vmatrix} \frac{\beta}{a} - \chi & 0 & 0 \\ 0 & -\chi & 0 \\ 0 & 0 & -\chi \end{vmatrix} = 0.$$

This implies that

$$\chi^2 \left[\frac{\beta}{a} - \chi\right] = 0,$$

and so,

$$\chi_1 = \left[\frac{\beta}{a}\right], \chi_2 = \chi_3 = 0.$$

However, the dominant eigenvalue here is $\chi_1 = \left[\frac{\beta}{a}\right]$ and is the spectral radius as the threshold value or the basic reproductive number. Thus, it can be concluded that the reproduction number of the model is $\Re_0 = \left[\frac{\beta}{(\varphi+\alpha+\omega+\mu+\xi)}\right]$, where $a = (\varphi+\alpha+\omega+\mu+\xi)$, $b = (\theta+\gamma+\mu+\xi)$, $c = (\delta+\mu+\xi)$.

3.6 Local Stability of Disease Free Equilibrium

Theorem 3.3. The disease free equilibrium point E_0 of the system (1) is locally asymptotically stable if $\Re_0 < 1$ and unstable if $\Re_0 > 1$.

Proof . To proof this theorem first we obtain the Jacobian matrix of model equation (2.2) at the disease free equilibrium E_0 as follows:

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

Now, the eigenvalues of $J(E_0)$ are required to be found. The characteristic equation $det[J(E_0) - \chi I] = 0$ is expanded and simplified as follows:

$$\begin{vmatrix} -d-\chi & 0 & 0 & 0 & 0 & 0 \\ \eta & -\mu-\chi & 0 & 0 & 0 & \psi \\ 0 & 0 & -a-\chi & 0 & 0 & 0 \\ 0 & 0 & \varphi & -b-\chi & 0 & 0 \\ 0 & 0 & \omega & \theta & -c-\chi & 0 \\ 0 & 0 & \alpha & \gamma & \delta & -e-\chi \end{vmatrix} = 0. \tag{3.8}$$

From the Jacobian matrix of (3.8), we obtained a characteristic polynomial:

$$(-d-\chi)(-\mu-\chi)(-a-\chi)(-b-\chi)(-c-\chi)(-e-\chi) = 0. \tag{3.9}$$

Thus, from equation (3.9) clearly we see that;

$$\chi_1 = -d,\ \chi_2 = -\mu,\ \chi_3 = -a,\ \chi_4 = -b,\ \chi_5 = -c,\ \chi_6 = -e.$$

It can be observed that the eigenvalues $\chi_1, \chi_2, \chi_3, \chi_4, \chi_5$ and χ_6 are absolutely negative quantities. Therefore, it is concluded that the DFE E_0 of the system of differential equations (2.2) is locally asymptotically stable if $\Re_0 < 1$ and unstable if $\Re_0 > 1$. □

3.7 Global Stability of Disease Free Equilibrium

The global stability of disease free equilibrium was implemented by Castillo-Chavez and Song technique [2]. The model equation (2.2) can be re-written as

$$\begin{aligned} dX/dt &= F(X, Z) \\ dZ/dt &= G(X, Z),\ G(X, 0) = 0 \end{aligned}$$

Where, X stands for the uninfected population, that is $X = (P, S, R)$ and Z also stands for the infected population, that is $Z = (I, Q, C)$. The disease free equilibrium point of the model is denoted by $U = (X^*, 0)$. The point $U = (X^*, 0)$ to be globally asymptotically stable equilibrium for the model provided that $\Re_0 < 1$ and the following conditions must be met:

(H_1). For $dX/dt = F(X, 0)$, X^* is globally asymptotically stable.

(H_2). $G(X, Z) = AZ - \tilde{G}(X, Z),\ \tilde{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$.

Where $A = D_Z G(U, 0)$ is a Metzler matrix i.e. the off diagonal elements of A are non-negative and G is the region where the model makes biologically sense.

If the model (1) met the above two criteria, then the following theorem holds.

Theorem 3.4. The point $U = (X^*, 0)$ is globally asymptotically stable equilibrium provided that $\Re_0 < 1$ and the condition (H_1) and (H_2) are satisfied.

Proof . From system (1) we can get $F(X, Z)$ and $G(X, Z)$;

$$F(X, Z) = \begin{bmatrix} \Pi - (\eta + \mu)P \\ \eta P + \psi R - (\lambda + \mu)S \\ \gamma Q + \alpha I + \delta C - (\psi + \mu)R \end{bmatrix} \text{ and } G(X, Z) = \begin{bmatrix} \lambda S - (\varphi + \alpha + \omega + \mu + \xi)I \\ \varphi I - (\theta + \gamma + \mu + \xi)Q \\ \omega I + \theta Q - (\delta + \mu + \xi)C \end{bmatrix}$$

Consider the reduced system

$$\frac{dX}{dt}_{|Z=0} = \begin{bmatrix} \Pi - (\eta + \mu)P \\ \eta P - \mu S \\ 0 \end{bmatrix} \tag{3.10}$$

From equation (3.10) above it is obvious that $X^* = \left\{\frac{\Pi}{\eta+\mu}, \frac{\eta\Pi}{\mu(\eta+\mu)}, 0\right\}$ is the global asymptotic point. This can be verified from the solution, namely $P = \frac{\Pi}{\eta+\mu} + \left[P(0) - \frac{\Pi}{\eta+\mu}\right] e^{-\mu t}$, $S = \frac{\eta\Pi}{\mu(\eta+\mu)} + \left[S(0) - \frac{\eta\Pi}{\mu(\eta+\mu)}\right] e^{-\mu t}$. As $t \longrightarrow \infty$ the solution $P \longrightarrow \frac{\Pi}{\eta+\mu}$ and $S \longrightarrow \frac{\eta\Pi}{\mu(\eta+\mu)}$ implying that the global convergence of (3.10) in Ω. From the equation for infected compartments in the model we have:

$$A = \begin{bmatrix} -(\varphi+\alpha+\omega+\mu+\xi) & 0 & 0 \\ \varphi & -(\theta+\gamma+\mu+\xi) & 0 \\ \omega & \theta & -(\delta+\mu+\xi) \end{bmatrix}$$

Since A is Metzler matrix, i.e. all of diagonal elements are nonnegative. Then, $G(X,Z)$ can be written as, $G(X,Z) = AZ - \tilde{G}(X,Z)$, where

$$\tilde{G}(X,Z) = \begin{bmatrix} -\lambda S \\ 0 \\ 0 \end{bmatrix} = \begin{bmatrix} \tilde{G}_1(X,Z) \\ \tilde{G}_2(X,Z) \\ \tilde{G}_3(X,Z) \end{bmatrix} \tag{3.11}$$

In equation (3.11) $\tilde{G}_1(X,Z) < 0$, that means the second condition (H_2) is not satisfied. Therefore $U = (X^*, 0)$ may not be globally asymptotically stable for $\Re_0 < 1$. □

3.8 Endemic Equilibrium Points

The endemic equilibrium points are $E_1 = \{P^*, S^*, I^*, Q^*, C^*, R^*\}$ 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 (2.2) to zero. That is, setting $\frac{dP}{dt} = \frac{dS}{dt} = \frac{dI_s}{dt} = \frac{dI_a}{dt} = \frac{dQ}{dt} = \frac{dC}{dt} = \frac{dR}{dt} = 0$ the model equations take the form as solved for state variables interms of the parameters after some algebraic operation and obtain the following;

$$\begin{aligned}
P^* &= \frac{\Pi}{(\eta+\mu)} \\
S^* &= \frac{\eta P^* + \psi R^*}{(\lambda^*+\mu)} \\
I^* &= \frac{b^2 c\lambda^* \eta P^*(\psi+\mu)}{[ab^2c(\psi+\mu) - bc\psi\gamma\varphi - b^2c\psi\alpha - b\psi\delta\omega - \psi\delta\theta\varphi]\lambda^* + ab^2\mu(\psi+\mu\mu)} \\
Q^* &= \frac{\varphi I^*}{b} \\
C^* &= \frac{\omega I^* + \theta Q^*}{c} \\
R^* &= \frac{\gamma Q^* + \alpha I^* + \delta C^*}{(\psi+\mu)}
\end{aligned}$$

Therefore $E_1 = \{P^*, S^*, I^*, Q^*, C^*, R^*\}$ is an endemic equilibrium point such that $P^* > 0,\ S^* > 0,\ I^* > 0,\ Q^* > 0,\ C^* > 0,\ R^* > 0$ exist.

3.9 Local Stability of Endemic Equilibrium

Theorem 3.5. he unique endemic equilibrium of model equation (2.2) is locally asymptotically stable if $\Re_0 > 1$.

Proof . Substituting endemic equilibrium point $E_1 = (P^*, S^*, I^*, Q^*, C^*, R^*)$ into force of infection λ^*, we get

$$\lambda^* = = \frac{\beta b^2 c\lambda^* P^*(\psi+\mu)}{\lambda^*[ab^2c(\psi+\mu) - bc\psi\gamma\varphi - b^2c\psi\alpha - b\psi\delta\omega - \psi\delta\theta\varphi] + ab^2c\mu(\psi+\mu)}$$

Thus,

$$\lambda^*[ab^2c(\psi+\mu) - bc\psi\gamma\varphi - b^2c\psi\alpha - b\psi\delta\omega - \psi\delta\theta\varphi] + abc\mu(\psi+\mu) = \Re_0 ab^2c\lambda^* P^*(\psi+\mu).$$

This implies that

$$\lambda^*[ab^2c(\psi+\mu)-bc\psi\gamma\varphi-b^2c\psi\alpha-b\psi\delta\omega-\psi\delta\theta\varphi]+[abc\mu(\psi+\mu)]\left[1-\frac{\Re_0\Pi b}{(\eta+\mu)}\right]=0.$$

This shows that the non-zero (positive endemic) equilibrium point of the model equation satisfy

$$D_1\lambda^*+D_2=0, \tag{3.12}$$

where $D_1=[ab^2c(\psi+\mu)-bc\psi\gamma\varphi-b^2c\psi\alpha-b\psi\delta\omega-\psi\delta\theta\varphi]$ and

$$D_2=abc\mu(\psi+\mu)\left[1-\frac{\Re_0\Pi b}{(\eta+\mu)}\right]$$

It is clear that $D_1>0$ and $D_2<0$ when $\Re_0>1$ and $\Re_0>\frac{1}{\Pi b}$. Thus the linear system (3.12) has a unique positive solution, given by $\lambda^*=\frac{-D_2}{D_1}$ whenever $\Re_0>1$.

Now, to show its local stability analysis, equation (3.11) gives a fixed point problem of the form;

$$f(\lambda^*)=\lambda^*[ab^2c(\psi+\mu)-bc\psi\gamma\varphi-b^2c\psi\alpha-b\psi\delta\omega-\psi\delta\theta\varphi]+[abc\mu(\psi+\mu)]\left[1-\frac{\Re_0\Pi b}{(\eta+\mu)}\right]$$

Then, derivatives of $f(\lambda^*)$ become;

$$f'(\lambda^*)=[ab^2c(\psi+\mu)-bc\psi\gamma\varphi-b^2c\psi\alpha-b\psi\delta\omega-\psi\delta\theta\varphi]+[abc\mu(\psi+\mu)]$$

Evaluating $f'(\lambda^*)$ at $\lambda^*=-D_2/D_1$ gives:

$$f'(-D_2/D_1)=\frac{1}{\Re_0}\left[\beta b^2c(\psi+\mu)-\frac{\beta[bc\psi\gamma\varphi+b^2c\psi\alpha+b\psi\delta\omega+\psi\delta\theta\varphi]}{a}\right]$$

It is clear that

$$|f'(\lambda^*)|<1 \text{ at } \lambda^*=-D_2/D_1, \text{ whenever } \Re_0>1$$

Therefore, the unique endemic equilibrium is locally asymptotically stable if $\Re_0>1$. □

3.10 Global Stability of Endemic Equilibrium

Theorem 3.6. The endemic equilibrium point of the model equation (2.2) is globally asymptotically stable whenever $\Re_0>1$.

Proof . To prove the global asymptotic stability of the endemic equilibrium we use the method of Lyapunov functions. Define

$$\begin{aligned}L(P^*,S^*,I^*,Q^*,C^*,R^*)=&\left[P-P^*-P^*\ln\left(\frac{P^*}{P}\right)\right]+\left[S-S^*-S^*\ln\left(\frac{S^*}{S}\right)\right]+\left[I-I^*-I^*\ln\left(\frac{I^*}{U}\right)\right]\\&+\left[Q-Q^*-Q^*\ln\left(\frac{Q^*}{Q}\right)\right]+\left[C-C^*-C^*\ln\left(\frac{C^*}{C}\right)\right]\\&+\left[R-R^*-R^*\ln\left(\frac{R^*}{R}\right)\right]\end{aligned}$$

By direct calculating the derivative of L along the solution (1) we have

$$
\begin{aligned}
\frac{dL}{dt} &= \left[\frac{P-P^*}{P}\right]\frac{dP}{dt} + \left[\frac{S-S^*}{S}\right]\frac{dS}{dt} + \left[\frac{I-I^*}{I}\right]\frac{dI}{dt} + \left[\frac{Q-Q^*}{Q}\right]\frac{dQ}{dt} + \left[\frac{C-C^*}{C}\right]\frac{dC}{dt} + \left[\frac{R-R^*}{R}\right]\frac{dR}{dt} \\
&= \left[\frac{P-P^*}{P}\right][\Pi-(\eta+\mu)P] + \left[\frac{S-S^*}{S}\right][\eta P+\psi R-(\lambda+\mu)S] \\
&\quad + \left[\frac{I-I^*}{I}\right][\lambda S-(\varphi+\alpha+\omega+\mu+\xi)I] + \left[\frac{Q-Q^*}{Q}\right][\varphi I-(\theta+\gamma+\mu+\xi)Q] \\
&\quad + \left[\frac{C-C^*}{C}\right][\omega I+\theta Q-(\delta+\mu+\xi)C] + \left[\frac{R-R^*}{R}\right][\gamma Q+\alpha I+\delta C-(\psi+\mu)R] \\
&= \left[1-\frac{P^*}{P}\right][\Pi-(\eta+\mu)P] + \left[1-\frac{S^*}{S}\right][\eta P+\psi R-(\lambda+\mu)S] \\
&\quad + \left[1-\frac{I^*}{I}\right][\lambda S-(\varphi+\alpha+\omega+\mu+\xi)I] + \left[1-\frac{Q^*}{Q}\right][\varphi I-(\theta+\gamma+\mu+\xi)Q] \\
&\quad + \left[1-\frac{C^*}{C}\right][\omega I+\theta Q-(\delta+\mu+\xi)C] + \left[1-\frac{R^*}{R}\right][\gamma Q+\alpha I+\delta C-(\psi+\mu)R], \\
&= [\Pi+\eta P^*+\lambda S^*+(\varphi+\alpha+\omega)I^*+(\theta+\gamma)Q^*+\delta C^*+\psi R^*+(N^*-N)\mu+\xi[(I^*+Q^*+C^*)-(I+Q+C)]] \\
&\quad - \left[\Pi\left(\frac{P^*}{P}\right)+(\eta P+\psi R)\left(\frac{S^*}{S}\right)+\lambda S\left(\frac{I^*}{I}\right)+\varphi I\left(\frac{Q^*}{Q}\right)+(\omega I+\theta Q)\left(\frac{C^*}{C}\right)+(\gamma Q+\alpha I+\delta C)\left(\frac{R^*}{R}\right)\right]
\end{aligned}
$$

Thus collecting positive and negative terms together we obtain

$$\frac{dL}{dt} = Q - K.$$

here,

$$Q = [\Pi+\eta P^*+\lambda S^*+(\varphi+\alpha+\omega)I^*+(\theta+\gamma)Q^*+\delta C^*+\psi R^*+(N^*-N)\mu+\xi[(I^*+Q^*+C^*)-(I+Q+C)]]$$

and

$$K = \left[\Pi\left(\frac{P^*}{P}\right)+(\eta P+\psi R)\left(\frac{S^*}{S}\right)+\lambda S\left(\frac{I^*}{I}\right)+\varphi I\left(\frac{Q^*}{Q}\right)+(\omega I+\theta Q)\left(\frac{C^*}{C}\right)+(\gamma Q+\alpha I+\delta C)\left(\frac{R^*}{R}\right)\right].$$

Thus if $Q < K$, then $\frac{dL}{dt} \leq 0$. Noting that $\frac{dL}{dt} = 0$ if and only if $P = P^*, S = S^*, I = I^*, Q = Q^*, C = C^*, R = R^*$. Therefore, the largest compact invariant set in $\left\{(P^*, S^*, I^*, Q^*, C^*, R^*) \in \Omega : \frac{dL}{dt} = 0\right\}$ is the singleton E_1 is the endemic equilibrium of the system (1). By LaSalle's invariant principle [10], it implies that E_1 is globally asymptotically stable in Ω if $Q < K$. □

4 Sensitivity Analysis

We carried out sensitivity analysis in order to determine the relative significance of model parameters on disease transmission. The analysis will enable us to find out parameters that have high impact on the basic reproduction number and which should be targeted by intervention strategies. We perform sensitivity analysis by calculating the sensitivity indices of the basic reproduction number $\Re_0$ in order to determine whether COVID-19 can be spread in the population or not. These indices tell us how crucial each parameter is on the transmission of the COVID-19. To investigate which parameters in the model system (1) have high impact on the $\Re_0$, we apply the approach presented by [6]. Following Eshetu and Koya [6], we present the normalized forward sensitivity indices of $\Re_0$ with respect to model parameter values $\mu = 0.02, \beta = 0.67, \varphi = 0.001, \alpha = 0.054, \omega = 0.064, \xi = 0.00001$. The explicit expression of $\Re_0$is given by:

$$\Re_0 = \frac{\beta}{(\varphi+\alpha+\omega+\mu+\xi}$$

Since $\Re_0$ depends only on nine parameters, we derive an analytical expression for its sensitivity to each parameter using the normalized forward sensitivity index as follows in Table 2 [6].

Table 2: Sensitivity index and indices Table.

Parameter Symbol	Sensitivity index	Sensitivity indices
β	$Y_{\beta}^{\Re_0} = [\partial \Re_0/\beta] \times [\beta/\Re_0]$	1
ξ	$Y_{\xi}^{\Re_0} = [\partial \Re_0/\xi] \times [\xi/\Re_0]$	-0.0000719
φ	$Y_{\varphi}^{\Re_0} = [\partial \Re_0/\varphi] \times [\varphi/\Re_0]$	-0.00719
μ	$Y_{\mu}^{\Re_0} = [\partial \Re_0/\mu] \times [\mu/\Re_0]$	-0.14387
α	$Y_{\alpha}^{\Re_0} = [\partial \Re_0/\alpha] \times [\alpha/\Re_0]$	-0.38846
ω	$Y_{\omega}^{\Re_0} = [\partial \Re_0/\omega] \times [\omega/\Re_0]$	-0.460

The sensitivity indices of the basic reproductive number with respect to main parameters are arranged orderly in Table 2. The parameters are arranged from the most sensitive one to the least sensitive one. The parameter that have positive indices i.e. β show that it has great impact on expanding the disease in the community if its value is increasing. Due to the reason that the basic reproduction number increases as transmission values increases, it means 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. $\xi, \varphi, \mu, \alpha$ and ω have an influence of minimizing the burden of the disease in the community as their values increase while the others are left constant. And also as their values increase, the basic reproduction number decreases, which leads to minimizing then endemicity of the disease in the community.

5 Numerical Simulation

In this section, numerical simulation study of model equations (3.1) are carried out using the software MATLAB R2015b 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 population of Ethiopia (2020 and Historical) [15] or assumed on the basis of reality. Using the parameter values given in Table 3 and the initial conditions $P(0) = 86326278, S(0) = 1000000, I(0) = 7450, Q(0) = 8000, C(0) = 117, R(0) = 25$ in the model equations (3.1) a simulation study is conducted and the results are given in the following Figures.

Table 3: Parameter values

Parameter	Value	Source
Π	0.0005	Assumed
η	0.0004	Assumed
β	0.067	Assumed
ψ	0.0023	Assumed
γ	0.0002	Assumed
φ	0.001	Assumed
α	0.054	Assumed
θ	0.001	Assumed
ω	0.064	Assumed
δ	0.003	Assumed
μ	0.02	[4]
ξ	0.00001	Assumed

From Figure 2 we see that the protected individuals decrease due to more number of protected individuals join susceptible compartment and converges to disease free equilibrium. Similarly, Figure 3 illustrated that the susceptible individual decreases due to more number of infectious individuals. However, Figure 4 show that the infected individual increases firstly as the consequence of some number of susceptible individuals joined the infected subcompartment but decline because of some infected individuals joined quarantine compartment. Moreover, Figure 5 shows that the number of quarantine individuals increases in the beginning as a result of infectious from infected individuals enters it and decreases due to recovery. Also, Figure 6 shows that the number of coronavirus individuals increases in the beginning as a result of some number from quarantine and infected compartment enters it and decreases due to death rate. In addition to this figure 7 increases initially as number of quarantine, infected, and coronavirus individuals are recovered and decreases finally as result of losing natural immunity. Finally, Figure 8, Figure 9 and Figure 10

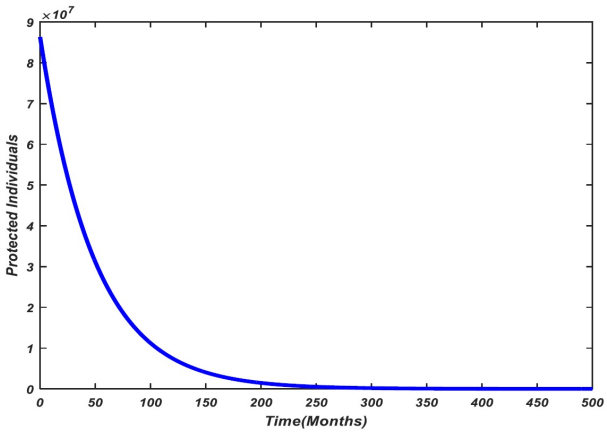

Figure 2: Transmission dynamics of Protected Individuals.

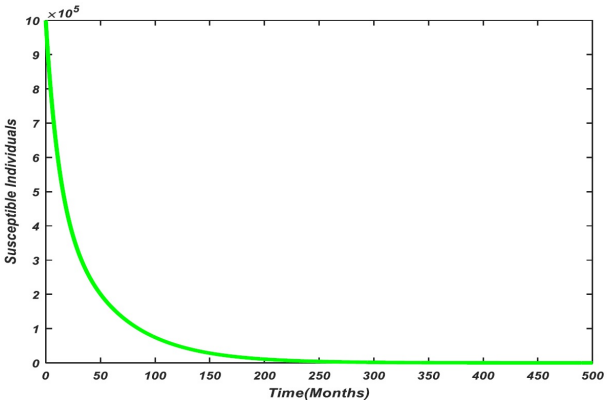

Figure 3: Transmission dynamics of Susceptible Individuals

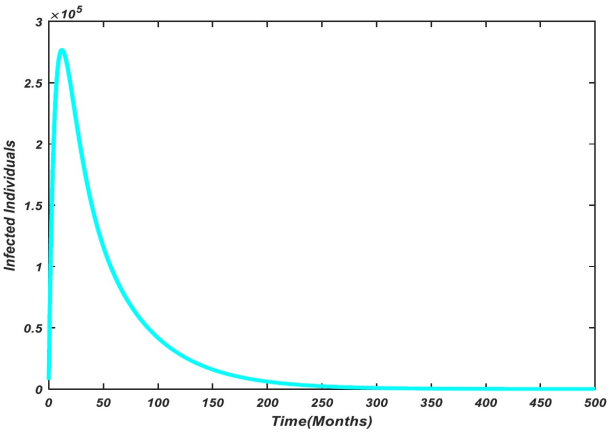

Figure 4: Transmission dynamics of Infected Individuals

indicating that transmission rate have an effect on reducing the disease from community. An increase in level of transmission rate among individuals has an effect on reducing the prevalence of COVID-19 and COVID-19 disease.

6 Conclusion and Recommendation

In this paper, a deterministic mathematical model of COVID-19 was established. Both qualitative and numerical analysis of the model was done. We have shown that there exists a feasible region where the model is well posed and biologically meaningful in which a unique disease free equilibrium point exists. The steady state points were obtained

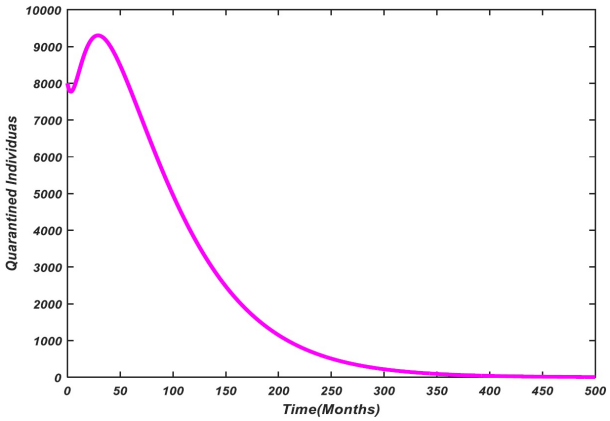

Figure 5: Transmission dynamics of Quarantined Individuals

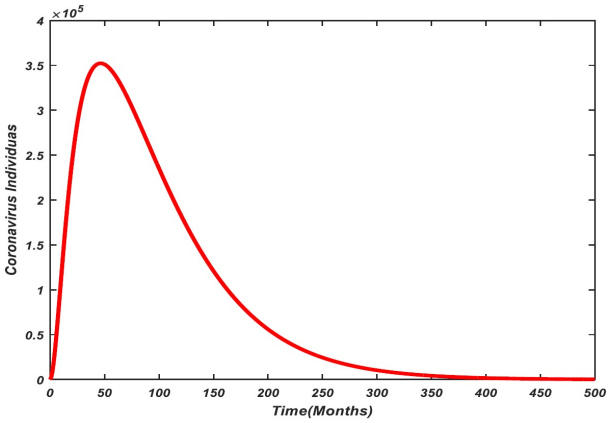

Figure 6: Transmission dynamics of Coronavirus Individuals

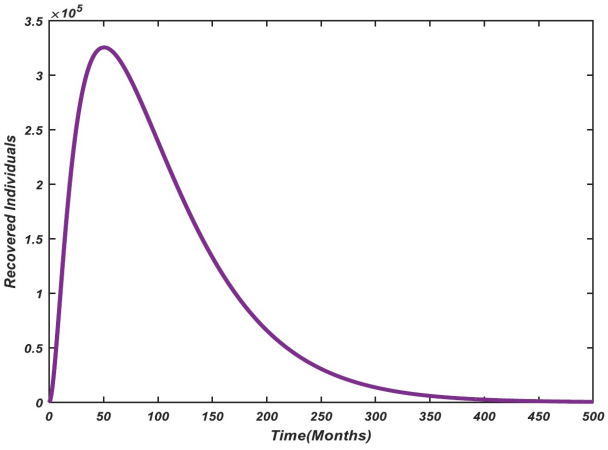

Figure 7: Transmission dynamics of Recovered Individuals

and their local and global stability conditions were investigated. And also, the solution of the model equation is numerically supplemented and sensitivity analysis of the model is analyzed to determine which parameter has high impact on the transmission of diseases. Although eradication of COVID-19 infection remains a challenge in the world, but from results of this study we recommend that, the government should introduce education programmers on the importance of voluntary and routinely screening on COVID-19 infection. Also, there is need to increase the number of hospitals to deal with COVID-19 infection and to screen more person.

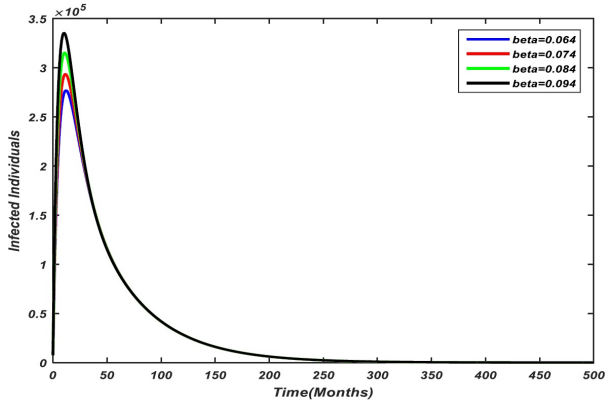

Figure 8: Effect of varying transmission rate on infected individuals.

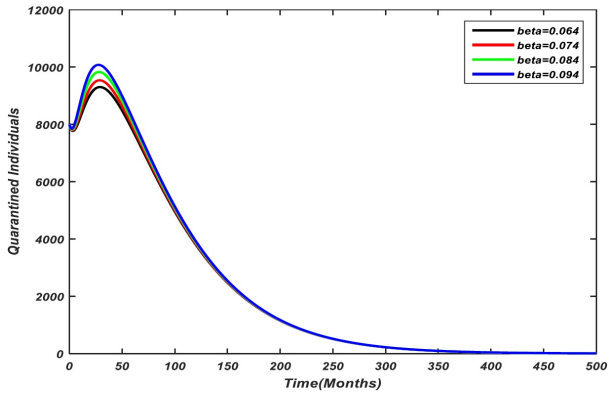

Figure 9: Effect of varying transmission rate on quarantined individuals.

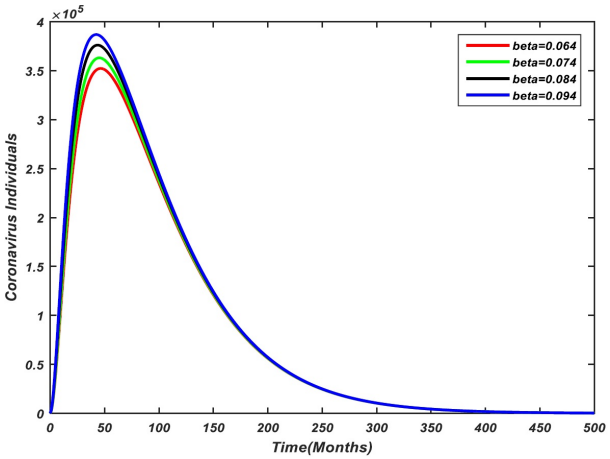

Figure 10: Effect of varying transmission rate on coronavirus individuals.

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