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Mathematical Model of Novel COVID-19 and Its Transmission Dynamics

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Abstract. In this paper, we formulated a dynamical model of COVID-19 to describe the transmission dynamics of the disease. The well possedness of the formulated model equations was proved. Both local and global stability of the disease free equilibrium and endemic equilibrium point of the model equation was established using basic reproduction number. The results show that, if the basic reproduction number is less than one then the solution converges to the disease free steady state i.e. 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 contact 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.

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1. Introduction

Coronavirus-19 disease (COVID-19) is an infectious disease caused by a newly discovered Coronavirus. The COVID-19 is a novel Coronavirus that was first reported to the world health organization country office in China on 31 December 2019 [17]. The outbreak was declared a public health emergence of international concern on 30 January 2020. On 11 February 2020, WHO announced a name for the new Coronavirus-19 disease "COVID-19" [19].

Several 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. The most common symptoms of Coronavirus-19 (COVID-19) are fever, cough, and shortness of breath and breathing difficulties. In more severe cases infection can cause pneumonia, sever acute respiratory syndrome and even death. The period within which the symptoms would appear is 2-14 days. It is transmitted from person to person via respiratory droplets produced when an infected person coughs or sneezes and between people who are in close

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close contact with one another with in about 6 feet [6]. There is no specific treatment for disease caused by Coronavirus-19 (COVID-19). However, many of the symptoms can be treated and therefore treatment based on the patient's clinical condition. No Pharmaceutical product has yet been shown to be safe and effective for the treatment of COVID-19. The best ways that are recommended to prevent the novel coronavirus (COVID-19) are 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 people who are sick, stay home while you are sick and avoid close contact with others, stay at home even you are not sick, cover your mouth/nose with a tissue or sleeve when coughing or sneezing and so on [8].

Coronavirus disease 2019 (COVID-19) Situation Report-72 [18] shows that globally 823,626confirmed and 40,598 deaths, in Western Pacific Region 106,422 confirmed and 3,701 deaths, in European Region 464,212 confirmed and 30,089 deaths, in South-East Asia Region 5175 confirmed and 195 deaths, in Eastern Mediterranean Region 54,281 confirmed and 3115 deaths, in Region of the Americas 188,751 confirmed and 3,400 deaths and also in African Region 4073 confirmed and 91 deaths. Furthermore, Figure 1 illustrates that the countries, territories or areas with reported confirmed cases of COVID-19, 1 April 2020.

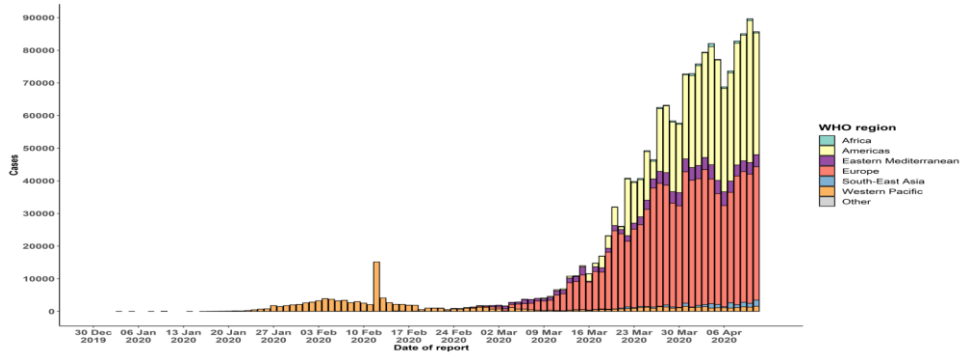

Figure 1. Epidemic curve of confirmed COVID-19 [18].

Now there are more than 15,000 confirmed cases of Coronavirus across the continent, with a number of African countries imposing a range of prevention and containment measures against the spread of the pandemic. According to the latest data by the John Hopkins University and Africa Center for Disease Control on COVID-19 in Africa, the break down remains fluid as countries confirm cases and when the whole of Africa has rising cases with only two countries holding out as of April 14. Ethiopia's COVID-19 file as of April 14 has 82 cases (eight new cases) with three deaths and 14 recoveries. The total number of tests stands at 4,557. Active cases stand at 63 representing about 77% of recorded cases [18].

The mathematical model is used as an important tool for better understanding of the infectious disease, studying the approximations, and effects of the parameters and predicting the behavior of the problem in a specific period of time as well as showing the connectivity of theories and observations using the system of equations with state variables and parameters [1]. Many mathematical models have been formulated by different scholars [3,13] to study the transmission dynamics COVID-19. Chen et al [5] developed a Bats-Hosts-Reservoir - People transmission network model for simulating the potential transmission from the infection source (probably be bats) to the 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 East countries, similar to severe acute respiratory syndrome, but lower than MERS in the Republic of Korea. Effect of delay in diagnosis on transmission dynamics of COVID-19 was also discussed in [15]. Sensitivity analyses and numerical simulations reveal that, improving the proportion of timely diagnosis and shortening the waiting time for diagnosis cannot eliminate COVID-19 but can effectively decrease the basic reproduction number, significantly reduce the transmission risk, and effectively prevent the endemic of COVID-19, e.g., shorten the peak time and reduce the peak value of new confirmed cases and new infection, decrease the cumulative number of confirmed cases and total infection. Furthermore, Chayu Yang and Jin Wang [16] 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 lot of authors developed a mathematical model to illustrate the dynamics of the disease that helped them to suggest disease control mechanism and also described the transmission dynamics of the Coronavirus infection. Li Y et al. [14] 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 establish the time series models based on different mathematical formulas according to the variation law of the original data. In this paper we modify the model developed by Li Y et al. [14], by adding the asymptomatic compartment.

2. Model description and formulation

The total population at a time$t$, denoted by $N(t)$, is divided into sub-classes consisting of Protected individuals (P); are the individual who are protected against the disease over period of time, Susceptible individual (S); individual those who are vulnerable to the disease over a period of time, Infective individual in asymptomatic phase (I_a); are individual who are not showing symptoms of Coronavirus (COVID-19), Infective individual in symptomatic phase(I_s); are individuals who are showing symptoms of Coronavirus (COVID-19), Quarantine individual (Q); are individual who are infectious and compulsory quarantine due to reduce the spread of COVID-19 and get treatment based on the patient's clinical condition. Coronavirus (COVID-19) individuals (C); are individuals who are at the chronic stage of Coronavirus and recovered individual (R);individual those who are recovered from the disease at a time *t*. Then the total population at a time t denoted by $N(t)$ is given by:

$$N(t) = P(t) + S(t) + I_a(t) + I_s(t) + Q(t) + C(t) + R(t) \quad (1)$$

Thus, the model assumed that; protected individuals are recruited into the population at a rate Π and decreased by natural death at a rate μ and by losing protection at a rate δ. Susceptible individuals are increased by losing protection of protected class at a rate δ and from recovered class by losing immunity at a rate θ. Also, susceptible individuals are decreased by natural death at a rate μ. Susceptible individuals are acquiring COVID-19 infection with effective force of infection λ which is given by $\lambda = \frac{\beta(I_s+qI_a)}{N}$, where β is the effective contact rate and q is the transmission coefficient for asymptomatic individuals. If $q > 1$, then asymptomatic individuals infect the susceptible individuals more likely than the symptomatic individuals. If $q < 1$, then the infective symptomatic individuals have a good chancetoinfect the susceptible individuals than asymptomatic

individuals and if $q = 1$, then both asymptomaticand symptomatic individuals have equal chance to infect the susceptible individuals.

Asymptomatic individuals are increased from susceptible subclass at a rate $(1-\eta)\lambda(t)$ and decreased by developing symptoms of COVID-19 at a rate ψ. Those individuals in the asymptomatic subclass can get treatment and join quarantined subclass with rate of γ. Symptomatic individuals are increased by the fraction of susceptible individual at a rate $\eta\lambda(t)$ and those which come from asymptomatic subclass by developing symptoms of COVID-19 at a rate ψ. Those individuals in the symptomatic subclass can get treatment and join quarantined subclass with rate of φ. The quarantine subclass also increases with individuals who come from asymptomatic class and symptomatic subclass by getting treatment with a rate γandφrespectively. Individuals who recovered from the disease join the recovered subclass with rate $\alpha$and others join the Coronavirus subclass with rate of ϕ. Coronavirus subclass is increased by quarantine individuals who lose natural immunity at a rate ϕ. In all the sub-classes, μ is the natural death rate of individuals, but in the infectious class ξ is the disease induced death rate. All parameters in the model are positive. Upon including the basic assumptions, the schematic diagram of the modified model can be given as in Figure 2 below.

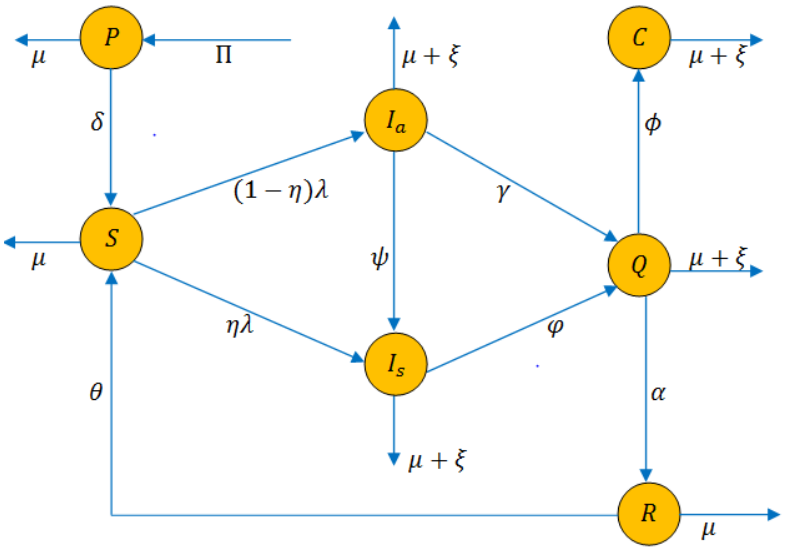

Figure 2. Schematic diagram of the model.

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

$$\begin{aligned}
\frac{dP(t)}{dt} &= \Pi - (\delta + \mu)P \\
\frac{dS(t)}{dt} &= \delta P + \theta R - (\lambda + \mu)S \\
\frac{dI_a(t)}{dt} &= (1-\eta)\lambda S - (\psi + \gamma + \mu + \xi)I_a \\
\frac{dI_s(t)}{dt} &= \eta\lambda S + \psi I_a - (\varphi + \mu + \xi)I_s \\
\frac{dQ(t)}{dt} &= \gamma I_a + \varphi I_s - (\phi + \alpha + \mu + \xi)Q
\end{aligned} \tag{2}$$

$$\frac{dC(t)}{dt} = \phi Q - (\mu + \xi)C$$
$$\frac{dR(t)}{dt} = \alpha Q - (\mu + \theta)R$$

The non-negative initial conditions of the system of model equations (2) are denoted by $P(0) > 0, S(0) > 0, I_a(0) \geq 0,\ I_s(0) \geq 0,\ Q(0) \geq 0,\ C(0) \geq 0,\ R(0) \geq 0.$

3. Mathematical analysis of the model

3.1 *Invariant Region*

In the model equation (2) that governs human population; all the variables and parameters used in the model equation are non-negative. We consider a biologically-feasible region

$$\Omega = \{(P, S, I_a, I_s, Q, R, C) \in R_+^7 : N \leq \frac{\Pi}{\mu}\}.$$

We adhere to the following steps to show the positive invariance of that are all the solution of model equation (2) that initiate in Ω remains in the region Ω and is bounded in Ω. We have the total population from (1);

$$N(t) = P(t) + S(t) + I_a(t) + I_s(t) + Q(t) + C(t) + R(t)$$

The rate of change of the total population by adding all the equations considered in Equation (2) is given by

$$\frac{dN}{dt} = \Pi - \mu N - \xi(I_a(t) + I_s(t) + Q(t) + C(t)) \leq \Pi - \mu N.$$

Notice that $\frac{dN}{dt}$ is bounded by $\Pi - \mu N$. By using standard comparison theorem as in [10] it can be shown that

$$0 \leq N(t) \leq \frac{\Pi}{\mu} + (N_0 - \frac{\Pi}{\mu})e^{-(\mu t)} \tag{3}$$

As $t \to \infty$ in equation (3), the population size $N \to \Pi/\mu$ which implies that $0 \leq N \leq \Pi/\mu$. Thus the feasible solution set of the model equation remain in the region $\Omega = \{(P, S, I_a, I_s, Q, R, C) \in R_+^7 : N \leq \Pi/\mu\}$. Therefore, the basic model is wellposed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the basic model in region Ω.

3.2 *Positivity of the solution of the model*

It is also necessary to prove that all the variables of the model (2) are non-negative; so that the solution of the system with non-negative initial conditions remains positive for all $t > 0$. The following lemma describe this fact.

Lemma 3.1 *If* $P(0) \geq 0,\ S(0) \geq 0, I_a(0) \geq 0, I_s(0) \geq 0,\ Q(0) \geq 0,\ C(0) \geq 0,\ R(0) \geq 0$ *the solution of* $P(t),\ S(t),\ I_a(t),\ I_s(t),\ Q(t),\ C(t),\ R(t)$ *in the system* (2) *is non-negative for all* $t \geq 0$.

Proof We shall prove this lemma using a contradiction by assuming that the total population $N(t) \neq 0$ for all $t \geq 0$. We assume that there exists the first time $t_1, t_2, t_3, t_4, t_5, t_6, t_7$ respectively such that:

*Positivity of P(t):*Assume that $P(t_1) = 0,\ \frac{dP(t_1)}{dt} < 0,\ S(0) \geq 0,\ I_a(0) \geq 0,\ I_s(0) \geq 0,$

$Q(0) \geq 0,\ C(0) \geq 0,\ R(0) \geq 0,\ 0 \leq t \leq t_1$. Here,

$$\frac{dP(t_1)}{dt} < 0 \Rightarrow \frac{dP(t_1)}{dt}|_{t=t_1} = \Pi - (\mu + \delta)P(t_1) = \Pi \leq 0,$$

which is contradiction as, $\Pi > 0$. Hence it can be concluded that $P(t) \geq 0,\ \forall t \geq 0$.

Positivity of $S(t)$: Assume that $S(t_1) = 0,\ \frac{dS(t_2)}{dt} < 0, P(0) \geq 0, I_a(0) \geq 0,\ I_s(0) \geq 0,\ Q(0) \geq 0,\ C(0) \geq 0,\ R(0) \geq 0,\ 0 \leq t \leq 0$. Here,

$$\begin{aligned}\frac{dS(t_2)}{dt} < 0 \Rightarrow \frac{dS(t_2)}{dt}|_{t=t_2} &= \delta P(t_2) + \theta R(t_2) - (\mu + \lambda)S(t_2) \\ &= \delta P(t_2) + \theta R(t_2) \leq 0\end{aligned}$$

which is a contradiction as $P(t),\ R(t) > 0$. Hence it can be concluded that $S(t) \geq 0,\ \forall t \geq 0$.

Positivity of $I_a(t)$: Assume that $I_a(t_3) = 0,\ \frac{dI_a(t_3)}{dt} < 0\ , P(0) \geq 0\ , S(0) \geq 0, I_s(0) \geq 0,\ Q(0) \geq 0,\ C(0) \geq 0,\ R(0) \geq 0, 0 \leq t \leq t_3$. Here,

$$\begin{aligned}\frac{dI_a(t_3)}{dt} < 0 \Rightarrow \frac{dI_a(t_3)}{dt}|_{t=t_3} &= (1-\eta)\lambda S(t_3) - (\psi + \gamma + \mu + \xi)I_a(t_3) \\ &= (1-\eta)\lambda S(t_3) < 0\end{aligned}$$

which is a contradiction as $S(t) > 0$. Hence it can be concluded that $I_a(t) \geq 0, \forall t \geq 0$.

Positivity of $I_s(t)$:Assume that $I_s(t_4) = 0, \frac{dI_s(t_4)}{dt} < 0\ , P(0) \geq 0\ , S(0) \geq 0, I_a(0) \geq 0,\ Q(0) \geq 0,\ C(0) \geq 0,\ R(0) \geq 0, 0 \leq t \leq t_4$. Here,

$$\begin{aligned}\frac{dI_s(t_4)}{dt} < 0 \Rightarrow \frac{dI_a(t_4)}{dt}|_{t=t_4} &= \eta\lambda S(t_4) + \psi I_a(t_4) - (\varphi + \mu + \xi + \gamma)I_s(t_4) \\ &= \eta\lambda S(t_4) + \psi I_a(t_4) < 0\end{aligned}$$

which is a contradiction as $\eta\lambda S(t_4) + \psi I_a(t_4) > 0$. Hence it can be concluded that $I_s(t) \geq 0, \forall t \geq 0$.

Positivity of $Q(t)$: Assume that $(t_5) = 0,\ \frac{dQ(t_5)}{dt} < 0\ , P(0) \geq 0\ , S(0) \geq 0, I_a(0) \geq 0, I_s(0) \geq 0,\ C(0) \geq 0,\ R(0) \geq 0, 0 \leq t \leq t_5$. Here,

$$\begin{aligned}\frac{dQ(t_5)}{dt} < 0 \Rightarrow \frac{dQ(t_5)}{dt}|_{t=t_5} &= \gamma I_a(t_5) + \varphi I_s(t_5) - (\phi + \alpha + \mu + \xi)Q(t_5) \\ &= \gamma I_a(t_5) + \varphi I_s(t_5) < 0\end{aligned}$$

which is a contradiction as $\gamma I_a(t_5) + \varphi I_s(t_5) > 0$. Hence it can be concluded that $Q(t) \geq 0, \forall t \geq 0$.

Positivity of $R(t)$: Assume that $R(t_6) = 0,\ \frac{dR(t_6)}{dt} < 0, P(0) \geq 0, S(0) \geq 0, I_a(0) \geq 0, I_s(0) \geq 0,\ C(0) \geq 0,\ Q(0) \geq 0, 0 \leq t \leq t_6$. Here,

$$\begin{aligned}\frac{dR(t_6)}{dt} < 0 \Rightarrow \frac{dR(t_6)}{dt}|_{t=t_6} &= \alpha Q(t_6) - (\theta + \mu)R(t_6) \\ &= \alpha Q(t_6) \leq 0\end{aligned}$$

which is a contradiction as $\alpha Q(t_6) > 0$. Hence it can be concluded that $R(t) \geq 0,\ \forall t \geq 0$.

Positivity of $C(t)$: Assume that $C(t_7) = 0$, $\frac{dC(t_7)}{dt} < 0, P(0) \geq 0, S(0) \geq 0, I_a(0) \geq 0, I_s(0) \geq 0$, $R(0) \geq 0$, $Q(0) \geq 0, 0 \leq t \leq t_7$. Here,

$$\frac{dC(t_7)}{dt} < 0 \Rightarrow \frac{dC(t_7)}{dt}|_{t=t_7} = \phi Q(t_7) - (\mu + \xi)C(t_7)$$
$$= \phi Q(t_7) \leq 0$$

which is a contradiction as $\phi Q(t_7) \geq 0$. Hence it can be concluded that $C(t) \geq 0, \forall t \geq 0$. Thus, the solutions of $P(t)$, $S(t)$, $I_a(t)$, $I_s(t)$, $Q(t)$, $C(t)$, $R(t)$ in the system (2) remain positive for all $t > 0$. ■

3.3 *Disease free equilibrium points*

Disease free equilibrium points are steady state solutions where there is no disease in the population. In the absence of the disease this implies that $I_a(t) = I_s(t) = Q(t) = R(t) = C(t) = 0$ and the equilibrium points require that the right hand side of the model equation set equal to zero. These requirements reflect in reducing the model equations (2) as

$$\begin{cases} \Pi - (\delta + \mu)\mathrm{P} = 0 \\ \delta p + \theta R - (\lambda + \mu)\mathrm{S} = 0 \end{cases}$$

Then solving for $P$and S we obtain: $P^0 = \frac{\Pi}{\mu+\delta}$ and $S^0 = \frac{\delta\Pi}{\mu(\mu+\delta)}$. Thus, the disease-free equilibrium point of the model equation in (2) above is given by

$$E_0 = \{P^0, S^0, {I_a}^0, {I_s}^0, Q^0, R^0, C^0\} = \left\{\frac{\Pi}{\mu+\delta}, \frac{\delta\Pi}{\mu(\mu+\delta)}, 0, 0, 0, 0, 0\right\} \quad (4)$$

3.4 *Basic reproduction number*

The basic reproduction number is denoted by $\Re_0$ and it is defined as the expected number of people getting secondary infection among the whole susceptible population. It is computed using next-generation matrix defined in [7]. In this method $\Re_0$ is defined as the largest eigenvalue of the next generation matrix. Using the notation as in [7] for the model system (2) the associated matrices F and V for the new infectious terms and the remaining transition terms are respectively given by:

$$f_i = \begin{bmatrix} \frac{[\beta(I_s+qI_a)S]}{N} \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad v_i = \begin{bmatrix} (\mu+\xi+\psi+\gamma)I_a \\ -\psi I_a + (\mu+\xi+\varphi)I_s \\ -\gamma I_a - \varphi I_s + (\mu+\xi+\phi+\alpha)Q \\ -\phi Q + (\mu+\xi)C \end{bmatrix}.$$

The Jacobian matrices of f_i and v_i at the disease free equilibrium point E_0 take the form respectively as

$$F(E_0) = \begin{bmatrix} \frac{[\beta\delta q(\mu+\delta)]}{\mu+\delta} & \frac{\beta\delta}{b(\mu+\delta)} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V(E_0) = \begin{bmatrix} a & 0 & 0 & 0 \\ -\psi & b & 0 & 0 \\ -\gamma & -\varphi & c & 0 \\ 0 & 0 & -\phi & d \end{bmatrix},$$

where $a = \mu + \xi + \psi + \gamma$, $b = \mu + \xi + \varphi$, $c = \xi + \phi + \alpha$, $d = \mu + \xi$.

It can be verified that the matrix $V(E_0)$ is non-singular as its determinant $det[V(E_0)] = abcd$ is non-zero and after some algebraic computations its inverse matrix is constructed as

$$[V(E_0)]^{-1} = \begin{bmatrix} \frac{1}{a} & 0 & 0 & 0 \\ \frac{\psi}{ab} & \frac{1}{b} & 0 & 0 \\ \frac{\phi\varphi + b\gamma}{abc} & \frac{\varphi}{bc} & \frac{1}{c} & 0 \\ \frac{(\phi\varphi + b\gamma)\phi}{abcd} & \frac{\varphi\phi}{bcd} & \frac{\phi}{cd} & \frac{1}{d} \end{bmatrix}.$$

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

$$[F(E_0)][V(E_0)]^{-1} = \begin{bmatrix} \frac{\beta\delta(bq+\psi)}{ab(\mu+\delta)} & \frac{\beta\delta}{b(\mu+\delta)} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Now it is possible to calculate the eigenvalue to determine the basic reproduction number $\Re_0$ by taking the spectral radius of the matrix $[F(E_0)][V(E_0)]^{-1}$. Thus, the eigenvalues are computed by evaluating $det[\,[F(E_0)][V(E_0)]^{-1} - \lambda I] = 0$ or equivalently solving

$$\begin{vmatrix} \frac{\beta\delta(bq+\psi)}{ab(\mu+\delta)} - \lambda & \frac{\beta\delta}{b(\mu+\delta)} & 0 & 0 \\ 0 & -\lambda & 0 & 0 \\ 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & -\lambda \end{vmatrix} = 0.$$

It reduces to the fourth power equation for λ as $\lambda^3(\lambda - \frac{\beta\delta(bq+\psi)}{ab(\mu+\delta)}) = 0$ giving the four eigenvalues as

$$\lambda_1 = 0, \lambda_2 = 0, \lambda_3 = 0, \lambda_4 = \frac{\beta\delta(bq+\psi)}{ab(\mu+\delta)}.$$

However, the largest eigenvalue here is $\lambda_4 = \frac{\beta\delta(bq+\psi)}{ab(\mu+\delta)}$ 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

$$\Re_0 = \frac{\beta\delta(bq+\psi)}{ab(\mu+\delta)}.$$

3.5 *Local stability of the disease free equilibrium*

In absence of the infectious disease, the model populations have a unique disease free steady state E_0. To find the local stability of E_0, the Jacobian of the model equations evaluated at DEF E_0 is used. Also, to determine the global stability at E_0 Metzler Method is used. It is already shown that the DFE of model (2) is given by

$$E_0 = \{P^0, S^0, {I_a}^0, {I_s}^0, Q^0, R^0, C^0\} = \{\frac{\Pi}{\mu+\delta}, \frac{\delta\Pi}{\mu(\mu+\delta)}, 0, 0, 0, 0, 0\}$$

Now, the stability analysis of DEF is conducted and the results are presented in the form of theorems and proofs as follows:

Theorem 3.1 *The DFE* E_0 *of the system* (2) *is locally asymptotically stable if* $\Re_0 < 1$ *and unstable if* $\Re_0 > 1$.

Proof Consider the right hand side expressions of the equations (2) as functions so as to find the Jacobian matrix as follows:

$$\frac{dP(t)}{dt} = \Pi - (\delta + \mu)P = f_1(P, S, I_a, I_s, Q, R, C)$$
$$\frac{dS(t)}{dt} = \delta p + \theta R - (\lambda + \mu)S = f_2(P, S, I_a, I_s, Q, R, C)$$
$$\frac{dI_a(t)}{dt} = (1-\eta)\lambda S - (\psi + \gamma + \mu + \xi)I_a = f_3(P, S, I_a, I_s, Q, R, C)$$
$$\frac{dI_s(t)}{dt} = \eta\lambda S + \psi I_a - (\varphi + \mu + \xi + \gamma)I_s = f_4(P, S, I_a, I_s, Q, R, C)$$
$$\frac{dQ(t)}{dt} = \gamma I_a + \varphi I_s - (\phi + \alpha + \mu + \xi)Q = f_5(P, S, I_a, I_s, Q, R, C)$$
$$\frac{dC(t)}{dt} = \phi Q - (\mu + \xi)C = f_7(P, S, I_a, I_s, Q, R, C)$$
$$\frac{dR(t)}{dt} = \alpha Q - (\mu + \theta)R = f_6(P, S, I_a, I_s, Q, R, C)$$

Now the Jacobian matrix of the $f_1,\ f_2,\ f_3,\ f_4,\ f_5,\ f_6,\ f_7$ with respect to $(P,\ S, I_a,\ I_s,\ Q,\ R,\ C)$ is given by:

$$J = \begin{bmatrix} -\mu-\delta & 0 & 0 & 0 & 0 & 0 & 0 \\ \delta & \frac{-[\mu+\beta(I_s+qI_a)]}{N} & \frac{-\beta qS}{N} & \frac{-\beta S}{N} & 0 & \theta & 0 \\ 0 & \frac{(1-\eta)[\beta(I_s+qI_a)]}{N} & \left(\frac{(1-\eta)\beta qS}{N}\right) - a & \frac{-(1-\eta)[\beta S]}{N} & 0 & 0 & 0 \\ 0 & \frac{\eta\beta(I_s+qI_a)}{N} & \frac{\eta\beta qS}{N} & \left(\frac{\eta\beta S}{N}\right) - b & 0 & 0 & 0 \\ 0 & 0 & \gamma & \varphi & -c & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -d & 0 \\ 0 & 0 & 0 & 0 & \phi & 0 & e \end{bmatrix} \quad (5)$$

Therefore, the Jacobian matrix J of model at the disease free equilibrium E_0 reduces to

$$J(E_0) = \begin{bmatrix} -\mu-\delta & 0 & 0 & 0 & 0 & 0 & 0 \\ \delta & -\mu & \frac{-\beta q\delta}{\mu+\delta} & \frac{-\beta\delta}{\mu+\delta} & 0 & \theta & 0 \\ 0 & 0 & \frac{(1-\eta)\beta\delta q}{\mu+\delta} - a & \frac{-(1-\eta)\beta S}{\mu+\delta} & 0 & 0 & 0 \\ 0 & 0 & \frac{\eta\beta\delta q}{\mu+\delta} & \left(\frac{\eta\beta\delta}{\mu+\delta}\right) - b & 0 & 0 & 0 \\ 0 & 0 & \gamma & \varphi & -c & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -d & 0 \\ 0 & 0 & 0 & 0 & \phi & 0 & -e \end{bmatrix}$$

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

$$\begin{vmatrix} -\mu-\delta-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ \delta & -\mu-\lambda & \frac{-\beta q\delta}{\mu+\delta} & \frac{-\beta\delta}{\mu+\delta} & 0 & \theta & 0 \\ 0 & 0 & \frac{(1-\eta)\beta\delta q}{\mu+\delta}-a-\lambda & \frac{-(1-\eta)\beta S}{\mu+\delta} & 0 & 0 & 0 \\ 0 & 0 & \frac{\eta\beta\delta q}{\mu+\delta} & \frac{\eta\beta\delta}{\mu+\delta}-b-\lambda & 0 & 0 & 0 \\ 0 & 0 & \gamma & \varphi & -c-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -d-\lambda & 0 \\ 0 & 0 & 0 & 0 & \phi & 0 & -e-\lambda \end{vmatrix} = 0$$

$$\Rightarrow (-\mu-\delta-\lambda)(-\mu-\lambda)\left(\frac{(1-\eta)\beta\delta q}{\mu+\delta}-a-\lambda\right)\left(\frac{\eta\beta\delta}{\mu+\delta}-b-\lambda\right)(-c-\lambda)(-d-\lambda)(-e-\lambda)=0$$

Thus, the seven eigenvalues of the matrix are determined as

$$\lambda_1 = -\mu-\delta,$$
$$\lambda_2 = -\mu,$$
$$\lambda_3 = \frac{(1-\eta)\beta\delta q}{\mu+\delta}-a,$$
$$\lambda_4 = \frac{\eta\beta\delta}{\mu+\delta}-b,$$
$$\lambda_5 = -c,$$
$$\lambda_6 = -d,$$
$$\lambda_7 = -e.$$

where $a=\mu+\xi+\psi+\gamma, b=\mu+\xi+\varphi,\ c=\xi+\phi+\alpha,\ d=\mu+\xi,\ e=\mu+\theta.$

It can be observed that all the eigenvalues $\lambda_1,\ \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ and λ_7 are absolutely negative quantities. Therefore, it is concluded that the DFE E_0 of the system of differential equations (2) is locally asymptotically stable if $\Re_0 < 1$ and unstable if $\Re_0 > 1$. ■

3.6 *Global stability of disease free equilibrium*

To investigate the global stability of the disease free equilibrium points we used technique implemented by Castillo-Chavez and Song [4]. First the model equation (2) can be re-written as

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

where, X stands for the uninfected population, that is $X=(P,S,R)$ and Z also stands for the infected population, that is $Z=(I_a, I_s,\ Q, C)$. 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:

$H_1: \frac{dX}{dt} = F(X^*,0),\ X^*$ 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. If the model (2) met the above two criteria then the following theorem holds.

Theorem 3.2 *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 (2), we can get $F(X,Z)$ and $G(X,Z)$;

$$F(X,Z) = \begin{bmatrix} \Pi - (\delta+\mu)P \\ \delta P + \theta R - (\lambda+\mu)S \\ \alpha Q - (\mu+\xi)R \end{bmatrix} \text{ and } G(X,Z) = \begin{bmatrix} (1-\eta)\lambda S - (\mu+\xi+\psi+\gamma)I_a \\ \psi I_a - (\mu+\xi+\varphi)I_s \\ \gamma I_a + \varphi I_s - (\mu+\xi+\phi+\alpha)Q \\ \phi Q - (\mu+\xi)C \end{bmatrix}.$$

Consider the reduced system

$$\frac{dX}{dt_{Z=0}} = \begin{bmatrix} \Pi - (\delta+\mu)P \\ \delta P - \mu S \\ 0 \end{bmatrix} \tag{6}$$

From equation (6) above it is obvious that $X^* = \left\{\frac{\Pi}{\mu+\delta}, \frac{\delta\Pi}{\mu(\mu+\delta)}, 0\right\}$ is the global asymptotic point. This can be verified from the solution, namely $P = \frac{\Pi}{\mu+\delta} + [P(0) - \frac{\Pi}{\mu+\delta}]e^{-\mu t}$, $S = \frac{\delta\Pi}{\mu(\mu+\delta)} + \left[S(0) - \frac{\delta\Pi}{\mu(\mu+\delta)}\right]e^{-\mu t}$. As $t \to \infty$ the solution $P \to \frac{\Pi}{\mu+\delta}$ and $S \to \frac{\delta\Pi}{\mu(\mu+\delta)}$ implying that the global convergence of (6) in Ω. From the equation for infected compartments in the model we have

$$J(E_0) = \begin{bmatrix} -[a - \frac{(1-\eta)\beta\delta q}{\mu+\delta}] & \frac{-(1-\eta)\beta S}{\mu+\delta} & 0 & 0 \\ \frac{\eta\beta\delta q}{\mu+\delta} & -[b - \frac{\eta\beta\delta}{\mu+\delta}] & 0 & 0 \\ \gamma & \varphi & -c & 0 \\ 0 & 0 & \alpha & -e \end{bmatrix}.$$

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

$$\hat{G}(X,Z) = \begin{bmatrix} [\beta(I_s + qI_a)(1-\eta)][\frac{\delta}{\mu+\delta} - \frac{S}{N}] \\ [\beta\eta(I_s + qI_a)][\frac{\delta}{\mu+\delta} - \frac{S}{N}] - \psi I_a \\ 0 \\ 0 \end{bmatrix} = \begin{bmatrix} \hat{G}_1(X,Z) \\ \hat{G}_2(X,Z) \\ \hat{G}_3(X,Z) \\ \hat{G}_4(X,Z) \end{bmatrix}.$$

It follows that $\hat{G}_1(X,Z) \geq 0$, $\hat{G}_2(X,Z) \geq 0$, $\hat{G}_3(X,Z) = 0, \hat{G}_4(X,Z) = 0$. Thus, the condition (H_1) and (H_2) are satisfied and we conclude that *U* is globally asymptotically stable for $\Re_0 < 1$. ■

3.7 *Endemic equilibrium points*

The endemic equilibrium points are $E_1 = \{P^*, S^*, I_s^*, I_a^*, Q^*, R^*, C^*\}$ 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) to zero. That is, setting $\frac{dP}{dt} = \frac{dS}{dt} = \frac{dI_s}{dt} = \frac{dI_a}{dt} = \frac{dQ}{dt} = \frac{dR}{dt} = \frac{dC}{dt} = 0$ then we obtain the following

$$P^* = \frac{\Pi}{\mu+\delta},$$

$$S^* = \frac{ab\Pi}{[\mu\beta(a\eta+(\psi+bq)(1-\eta))]},$$

$$I_a{}^* = \frac{bcd[ab\Pi(\mu+\delta)-\delta\beta\Pi(a\eta+(\psi+bq)(1-\eta))](1-\eta)}{\beta(\mu+\delta)(a\eta+(\psi+bq)(1-\eta))[\theta\alpha[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))]-abcd]},$$

$$I_s{}^* = \frac{bcd[ab\Pi(\mu+\delta)-\delta\beta\Pi(a\eta+(\psi+bq)(1-\eta))][a\eta+\psi(1-\eta)}{\beta(\mu+\delta)(a\eta+(\psi+bq)(1-\eta))[\theta\alpha[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))]-abcd]},$$

$$Q^* = \frac{d[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))][ab\Pi(\mu+\delta)-\delta\beta\Pi(a\eta+(\psi+bq)(1-\eta))]}{[\beta(\mu+\delta)(a\eta+(\psi+bq)(1-\eta))][\theta\alpha[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))]-abcd]},$$

$$R^* = \frac{\alpha[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))][ab\Pi(\mu+\delta)-\delta\beta\Pi(a\eta+(\psi+bq)(1-\eta))]}{[\beta(\mu+\delta)(a\eta+(\psi+bq)(1-\eta))][\theta\alpha[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))]-abcd]},$$

$$C^* = \frac{\phi[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))][ab\Pi(\mu+\delta)-\delta\beta\Pi(a\eta+(\psi+bq)(1-\eta))]}{[\beta e(\mu+\delta)(a\eta+(\psi+bq)(1-\eta))][\theta\alpha[\gamma b(1-\eta)+\varphi(a\eta+\psi(1-\eta))]-abcd]}.$$

Here, $a = \mu + \xi + \psi + \gamma, b = \mu + \xi + \varphi,\ c = \xi + \phi + \alpha\ ,\ d = \mu + \xi,\ e = \mu + \theta.$

3.8 *Local stability of endemic equilibrium points*

Theorem 3.3 *The Endemic Equilibrium point* E_1 *of the system* (2) *is locally asymptotically stable if* $R_0 > 1$.

Proof To find the local stability of E_1, the Jacobian of the model equations evaluated at E_1 is used. The Jacobian of (5) at the endemic equilibrium point (7) are

$J(E_1) =$

$$\begin{bmatrix} -\mu-\delta & 0 & 0 & 0 & 0 & 0 & 0 \\ \delta & -[\mu+\frac{\beta\mu}{\Pi}(qI_a{}^*+I_s{}^*)] & \frac{-abq}{a\eta+(\psi+bq)(1-\eta)} & \frac{-ab}{a\eta+(\psi+bq)(1-\eta)} & 0 & \theta & 0 \\ 0 & \frac{(1-\eta)\beta\mu}{\Pi}(qI_a{}^*+I_s{}^*) & \frac{abq(1-\eta)}{a\eta+(\psi+bq)(1-\eta)}-a & \frac{ab(1-\eta)}{a\eta+(\psi+bq)(1-\eta)} & 0 & 0 & 0 \\ 0 & \frac{\eta\beta\mu}{\Pi}(qI_a{}^*+I_s{}^*) & \frac{abq\eta}{a\eta+(\psi+bq)(1-\eta)} & \frac{ab\eta}{a\eta+(\psi+bq)(1-\eta)}-b & 0 & 0 & 0 \\ 0 & 0 & \gamma & \varphi & -c & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -d & 0 \\ 0 & 0 & 0 & 0 & \phi & 0 & -e \end{bmatrix}.$$

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

$$P(\lambda) = (-\mu-\delta-\lambda)\left(-\left(\mu+\frac{\beta\mu}{\Pi}(qI_a{}^*+I_s{}^*)\right)-\lambda\right)\left(\frac{abq(1-\eta)}{a\eta+(\psi+bq)(1-\eta)}-a-\lambda\right)$$

$$\left(\frac{ab\eta}{a\eta+(\psi+bq)(1-\eta)}-b-\lambda\right)(-c-\lambda)(-d-\lambda)(-e-\lambda) = 0$$

Thus, the eigenvalues of the endemic equilibrium points are:

$$\lambda_1 = -\mu-\delta\ ,$$

$$\lambda_2 = -(\mu+\frac{\beta\mu}{\Pi}(qI_a{}^*+I_s{}^*)),$$

$$\lambda_3 = \frac{abq(1-\eta)}{a\eta+(\psi+bq)(1-\eta)}-a,$$

$$\lambda_4 = \frac{ab\eta}{a\eta+(\psi+bq)(1-\eta)}-b,$$

$$\lambda_5 = -c,$$
$$\lambda_6 = -d,$$
$$\lambda_7 = -e,$$

where

$$I_a{}^* = \frac{bcd[ab\Pi(\mu+\delta) - \delta\beta\Pi(a\eta + (\psi+bq)(1-\eta))](1-\eta)}{\beta(\mu+\delta)(a\eta + (\psi+bq)(1-\eta))[\theta\alpha[\gamma b(1-\eta) + \varphi(a\eta + \psi(1-\eta))] - abcd]},$$

$$I_s{}^* = \frac{bcd[ab\Pi(\mu+\delta) - \delta\beta\Pi(a\eta + (\psi+bq)(1-\eta))][a\eta + \psi(1-\eta)}{\beta(\mu+\delta)(a\eta + (\psi+bq)(1-\eta))[\theta\alpha[\gamma b(1-\eta) + \varphi(a\eta + \psi(1-\eta))] - abcd]}.$$

It can be observed that all the eigenvalues λ_1, λ_2, λ_3, λ_4, λ_5, λ_6, λ_7 are absolutely negative quantities. Therefore, it is concluded that the endemic equilibrium points E_1 of the system of differential equations (2) is locally asymptotically stable if $\Re_0 > 1$.

5. Sensitivity analysis

Sensitivity indices allow us to measure the relative change in a variable when a parameter changes [9]. If the model is simple, it may be possible to differentiate the outcome with respect to each parameter in turn. The derivatives are the rate of change of predictions with respect to the parameters [2]. This work adopts the normalized forward sensitivity index to conduct the sensitivity analysis. The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of the relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivative. For instance, the normalized forward sensitivity index on $\Re_0$, which depends differentially on a parameter M, as defined in [9]

$$\mathrm{Y}_M^{\Re_0} = \frac{\partial \Re_0}{\partial M} \times \frac{M}{\Re_0}. \tag{8}$$

The parameter values displayed in a table 2 below are taken as the baseline values and they are used to evaluate the sensitivity indices of some parameters which are responsible for the transmission dynamics of COVID-19 infectious disease to four places of decimal in relation to the effective reproduction number $\Re_0$ using equation (8) above as a guide, the result of which is presented in table 1 below.

$$\mathrm{Y}_\beta^{\Re_0} = \frac{\partial \Re_0}{\partial \beta} \times \frac{\beta}{\Re_0} = 1,$$
$$\mathrm{Y}_q^{\Re_0} = \frac{\partial \Re_0}{\partial q} \times \frac{q}{\Re_0} = 1 - \delta,$$
$$\mathrm{Y}_\mu^{\Re_0} = \frac{\partial \Re_0}{\partial \mu} \times \frac{\mu}{\Re_0} =$$
$$\frac{[q(\mu+\xi+\psi+\gamma)(\mu+\xi+\varphi)(\mu+\delta) - [q(\mu+\xi+\varphi)+\psi][(\mu+\xi+\varphi)(\mu+\delta)+(\mu+\xi+\psi+\gamma)(2\mu+\xi+\varphi+\delta)]]\mu}{[(\mu+\xi+\varphi)(\mu+\delta)(q(\mu+\xi+\varphi)+\psi)]},$$
$$\mathrm{Y}_\xi^{\Re_0} = \frac{\partial \Re_0}{\partial \xi} \times \frac{\xi}{\Re_0} = \frac{[q(\mu+\xi+\psi+\gamma)(\mu+\xi+\varphi) - [q(\mu+\xi+\varphi)+\psi][2\mu+2\xi+\varphi+\psi+\gamma]]\xi}{[(\mu+\xi+\psi+\gamma)(\mu+\xi+\varphi)(q(\mu+\xi+\varphi)+\psi)]},$$
$$\mathrm{Y}_\varphi^{\Re_0} = \frac{\partial \Re_0}{\partial \varphi} \times \frac{\varphi}{\Re_0} = \frac{-(\psi\varphi)}{[(\mu+\xi+\varphi)(q(\mu+\xi+\varphi)+\psi)]},$$

$$Y_{\psi}^{\Re_0} = \frac{\partial \Re_0}{\partial \psi} \times \frac{\psi}{\Re_0} = \frac{[(\mu+\xi+\gamma)-q(\mu+\xi+\varphi)]q}{[(\mu+\xi+\psi+\gamma)(q(\mu+\xi+\varphi)+\psi)]},$$

$$Y_{\gamma}^{\Re_0} = \frac{\partial \Re_0}{\partial \gamma} \times \frac{\gamma}{\Re_0} = \frac{-\gamma}{\mu+\xi+\psi+\gamma}.$$

Table 1. Sensitivity index and indices Table.

Parameter Symbol	Sensitivity indices
β	+1
δ	0.9996
ψ	0.1895
q	0.0906
ξ	-0.0002
γ	-0.0134
μ	-0.1098
φ	-0.6928

Those parameters that have positive indices i.e. β, δ, ψ and $q$show that they have great impact on expanding the disease in the community if their values are increasing. Due to the reason that the basic reproduction number increases as their values increase, 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. ξ, γ, μ and φ 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.

6. Numerical simulation

In this section, numerical simulation study of model equations (2) 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) [12] or assumed on the basis of reality. Using the parameter values given in Table 1 and the initial conditions $P(0) = 86326278, S(0) = 1000000,\ I_a(0) = 10000, I_s = 4557,\ Q(0) = 8000, C(0) = 63,\ R(0) = 14$ in the model equations (2) a simulation study is conducted and the results are given in the following Figures.

Table 2. Parameter values.

Parameter	Value	Source
Π	0.0005	Assumed
δ	0.0004	Assumed
η	0.067	Assumed
ψ	0.054	Assumed
γ	0.001	Assumed
φ	0.064	Assumed
ϕ	0.001	Assumed
α	0.0002	Assumed
θ	0.0023	Assumed
μ	0.02	[11]
ξ	0.00001	Assumed

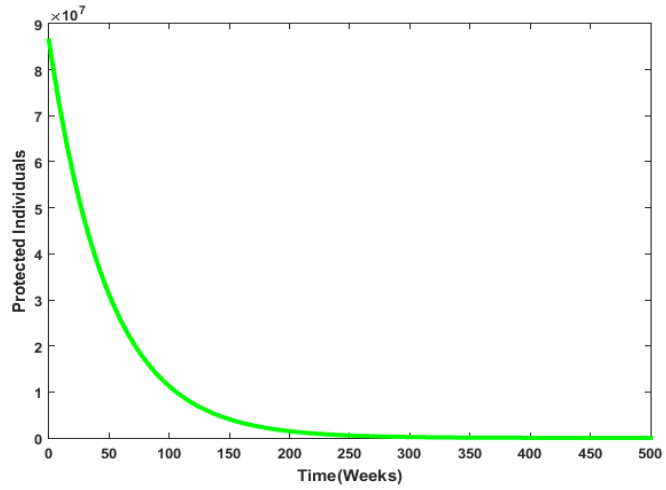

Figure 3. Dynamics of Protected Individuals.

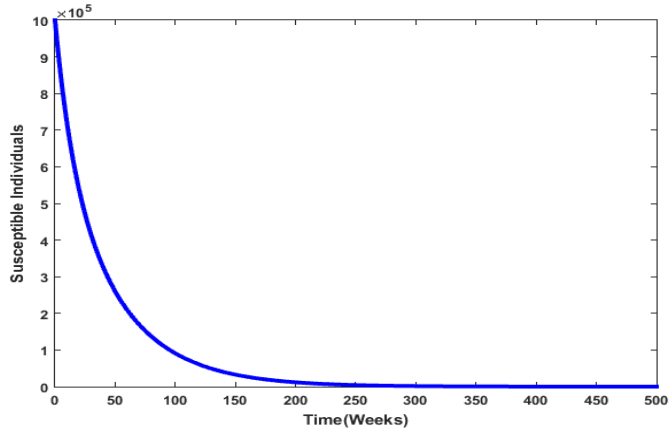

Figure 4. Dynamics of Susceptible Individuals.

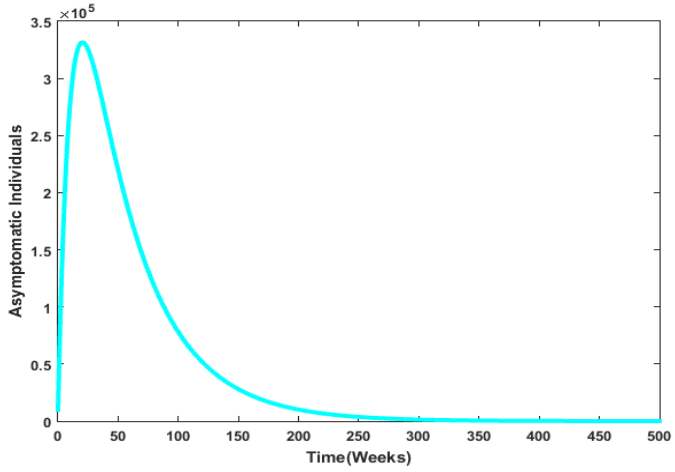

Figure 5. Dynamics of Asymptomatic Individuals.

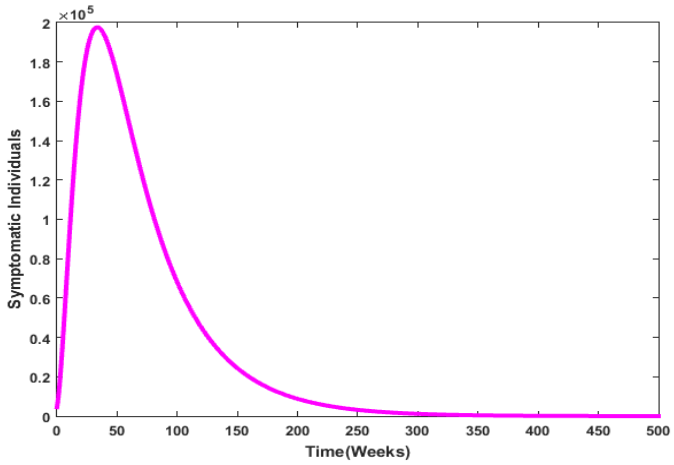

Figure 6. Dynamics of Symptomatic Individuals.

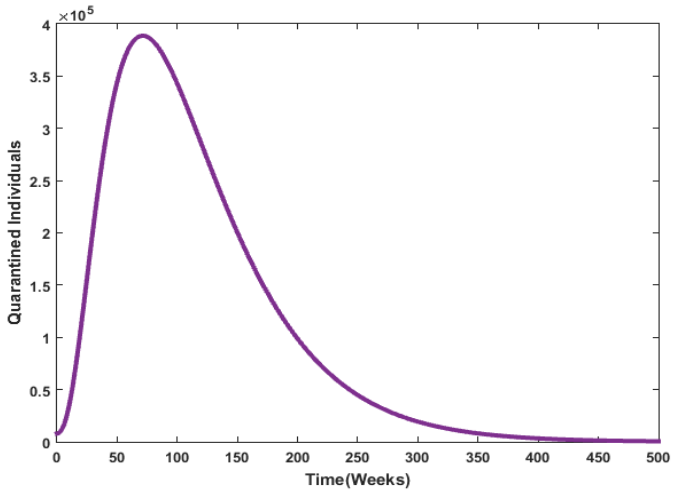

Figure 7. Dynamics of Quarantined Individuals.

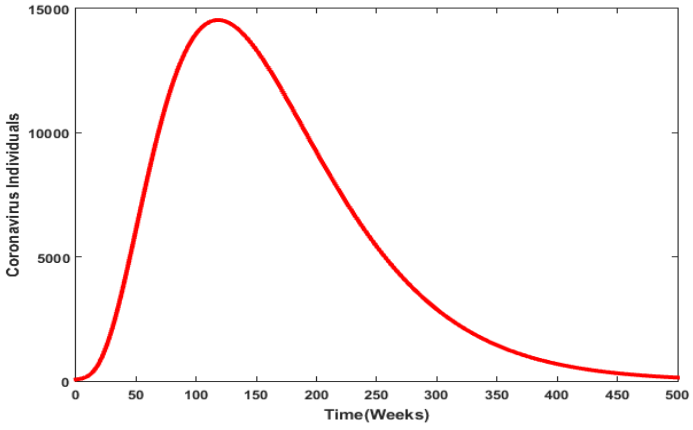

Figure 8. Dynamics of Coronavirus Individuals.

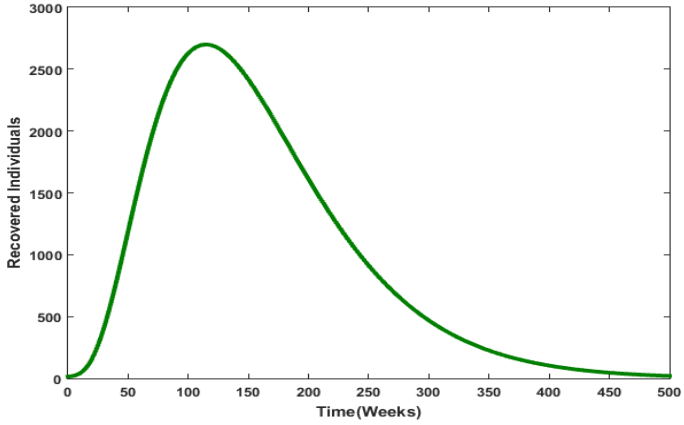

Figure 9. Dynamics of Recovered Individuals.

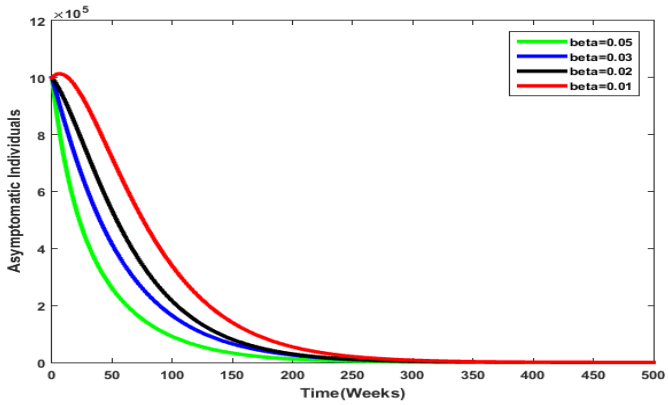

Figure 10. Effect of Varying contact rate on asymptomatic individuals.

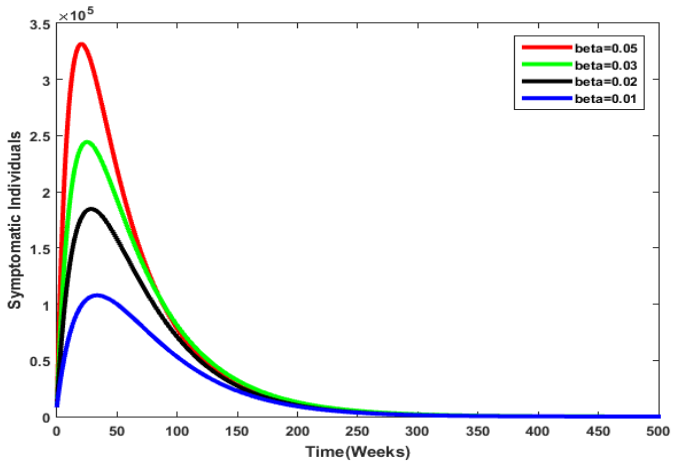

Figure 11. Effect of Varying contact rate on symptomatic individuals.

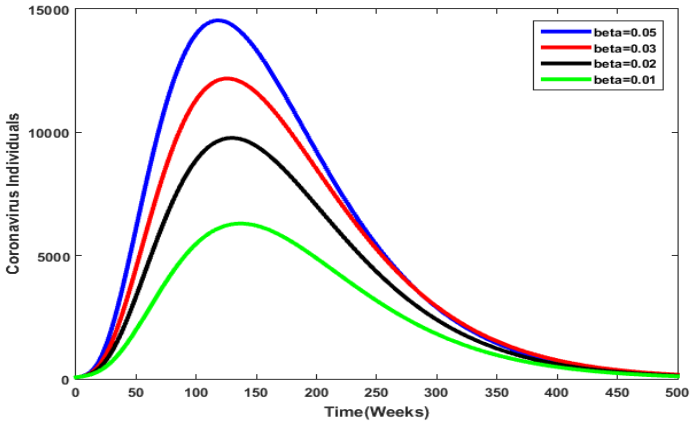

Figure 12. Effect of Varying contact rate on Coronavirus individuals.

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

Finally, Figure 10, Figure 11 and Figure 12 indicating that contact rate has an effect on reducing the disease from community. An increase in level of contact rate among individuals has an effect on reducing the prevalence of COVID-19 and COVID-19 disease.

7. Conclusion and recommendation

In this study, we formulated a mathematical model on the transmission dynamics COVID-19. Moreover, existence, positivity and boundedness of the formulated model are verified to illustrate that the model is biologically meaningful and mathematically wellposed. In particular, the stability analyses of the model were investigated using the basic reproduction number. 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 individuals with infection.

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