A mathematical model of COVID-19 transmission dynamics with treatment and quarantine

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Abstract. Corona-virus disease (COVID-19) is caused by the novel-virus (SARS-COV2). This disease mainly targets human respiratory system. COVID-19 (Coronavirus) has affected day to day life and is slowing down the global economy. This pandemic has affected thousands of peoples, who are either sick or are being killed due to the spread of this disease. In this paper we developed an eight compartmental model with quarantine and treatment of COVID-19. After proposing the model, we analysed the qualitative behaviors of the model, like the disease free and endemic equilibrium points and their stability analysis. Moreover, we obtained the basic reproduction number using next-generation matrix method and we performed the sensitivity analysis to identify the most affecting parameters in terms of disease control and spreed. To investigate the detail effect of each major parameters, we performed numerical simulation. We obtained that using both quarantine and treatment is best way to combating COVID-19 in the community. Therefore, stakeholders and policy makers should work both quarantine and treatment simultaneously in combating the pandemic from the population.

Keywords: COVID-19, mathematical modeling, numerical simulation, stability analysis, quarantine and treatment.

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1. Introduction

Since the outbreak in Wuhan, China, December, 2019, coronavirus disease (COVID-19) caused by the novel coronavirus, has now become a global pandemic as declared by World Health Organization (WHO) [1] and the world is presently battling with it [1, 2, 3]. The most common symptoms of COVID-19 are fever, fatigue, and dry cough [1]. Some patients may have ache and discomfort, nasal congestion, runny nose, sore throat, or diarrhea [3]. Such symptoms occurs 2-14 days after exposure, most usually about 5 days [4].

The pandemic can be transmitted directly or indirectly from an infectious person to a healthy person through the eyes, nose, mouth, and sometimes through the ears through moisture content when coughing or sneezing [3]. According to the data reported by WHO (World Health Organization), on 13 August 2020, the reported laboratory confirmed that the number of affected humans reached more than 25.9 million including more than 0.86 million death cases and more than 18.2 million recovers are recorded [5]. The government of different countries have been implementing diverse control measures such as imposing strict, mandatory lockdowns other measures such as individuals maintaining individual social distancing, avoiding crowded events, imposing a maximum number on individuals in any religious and social, the use of face masks while in public, use of sanitizers in any contact many in the markets and etc [6, 7, 8] to mitigate the spread of this pandemic.

Mathematical models have long been used as tools in gaining insight into the dynamics of infectious diseases [9, 10]. Several mathematical models have already been formulated for the population dynamics of COVID-19 in several countries [4, 11, 12, 6, 13, 14]. From this studies, Tang et al. [15] considered, an SEIR-type mathematical model to estimate the transmission risk of COVID-19 and its implication. The study in [6] , formulated a model for novel coronavirus disease 2019 (COVID-19) in Lagos, Nigeria and shown the effect of control measures, specifically the common social distancing, use of face mask and case detection on the dynamics of COVID-19. Khan et.al,[16], formulated a fractional mathematical model for the dynamics of COVID-19 with quarantine and isolation. D.K Mamo [13], developed SHEIQRD coronavirus pandemic spread model. He Identified that isolation of exposed and infected individuals, reduction of transmission, and stay-at-home return rate can mitigate COVID-19 pandemic. In this study, we developed a model by incorporating the hospitalize/quarantine and home treatment subclasses as well as home quarantine subclasses.

2. Model description and formulation

In this study the total population, $N(t)$, at time, t is divided into eight subpopulations; Susceptible,$S(t)$,Stay-home susceptables, $S_h(t)$, Exposed, $E(t)$, Asymptomatic, $A(t)$, Infected, $I(t)$, home Treatment, $T(t)$, Hospitalized/quarantine,

$Q(t)$ and Recovered, $R(t)$. The Susceptible are recruited into the population at a constant rate, Π. It is assumed that β_1 and β_2 are the contact rate of susceptible individuals with asymptomatic and infected individuals respectively and they move to the exposed compartment. We also assumed that susceptible individuals stay at home at a rate of υ and at a rate of τ peoples move from stay at home for due to different reasons and susceptible to the pandemic. Finishing the incubation period, the exposed individuals becomes infected at a rate of γ. From this $\alpha\gamma$ proportion become asymptomatic and the rest $(1-\alpha)\gamma$ become infectious. Through diagnosis $\sigma\delta$ proportion asymptomatic individuals got positive and join quarantine/hospitalized. The rest $(1-\sigma)\delta$ proportion of asymptomatic individuals recover from the disease. Also from infected individuals, $c\varepsilon$ fraction of individuals move to hospitalized. The others are taking treatment at their home at a rate $(1-c)\varepsilon$. However, when the pandemic for the treated individuals become savior $\phi\rho$ fraction move the quarantine/hospitalized. The remaining fractions recovers with the home treatment. Infected individuals recover at a rate of ω and quarantine individuals recover from the pandemic a rate k. The asymptomatic, infectious, treated and quarantine individuals die due to the disease at a rate $\varrho_1, \varrho_2, \varrho_3, \varrho_4$ respectively. The whole population have an average death rate of μ. For more information, Table 2 shows the description of model parameters. The flow diagram of the model is shown in Figure 1 below. Therefore, based on the above asumptions, the model is governed by the

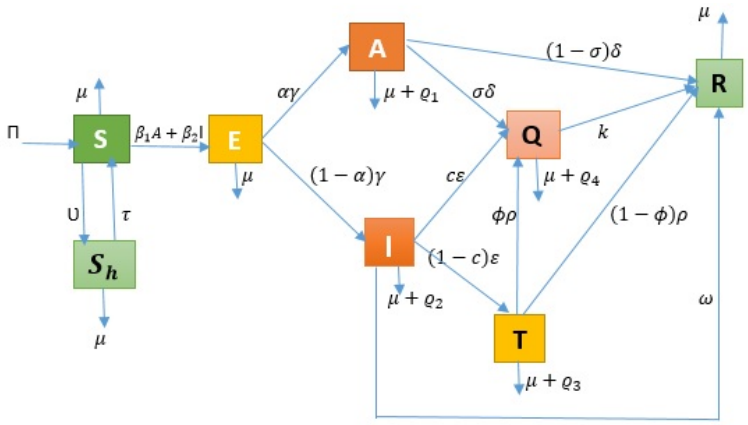


Figure 1: Compartmental flow diagram of the pandemic COVID 19 transmission

following system of differential equation:

$$
(1)\qquad \begin{cases} \dfrac{dS}{dt} = \Pi + \tau S_h - (\beta_1 A + \beta_2 I)\, S - (\upsilon + \mu) S, \\ \dfrac{dS_h}{dt} = \upsilon\, S - (\tau + \mu)\, S_h, \\ \dfrac{dE}{dt} = (\beta_1 A + \beta_2 I)\, S - (\gamma + \mu)\, E, \\ \dfrac{dA}{dt} = \alpha\, \gamma\, E - (\varrho_1 + \delta + \mu)\, A, \\ \dfrac{dI}{dt} = (1-\alpha)\, \gamma\, E - (\varrho_2 + \omega + \varepsilon + \mu)\, I, \\ \dfrac{dT}{dt} = (1-c)\, \varepsilon\, I - (\varrho_3 + \rho + \mu)\, T, \\ \dfrac{dQ}{dt} = \sigma\, \delta\, A + c\varepsilon I + \phi\rho T - (\varrho_4 + k + \mu)\, Q, \\ \dfrac{dR}{dt} = \omega I + (1-\sigma)\, \delta\, A + kQ + (1-\phi)\rho\, T - \mu\, R, \end{cases}
$$

with the initial condition

$$
(2)\qquad S(0) = S_0 \geq 0,\ E(0) = E_0 \geq 0\ \ I(0) = I_0 \geq 0,\ R(0) = R_0 \geq 0.
$$

3. Model analysis

3.1 Invariant region

In this section, a region in which solutions of the model are uniformlly bounded is the proper subset of $\Omega \in \mathcal{R}^8_+$. The total population at any time t is given by $N = S + S_h + E + A + I + T + Q + R$ and $\frac{dN}{dt} = \Pi - \varrho_1 A - \varrho_2 I - \varrho_3 T - \varrho_4 Q - \mu N$. In the absence of mortality due to COVID-19 pandemic, it becomes

$$
(3)\qquad \frac{dN}{dt} \leq \Pi - \mu N.
$$

Solving equation (3), we obtain $0 \leq N \leq \frac{\Pi}{\mu}$. Therefore, the feasible solution set of the system in equation (1) is the region given by:

$$
(4)\qquad \Omega = \left\{ (S, S_h, E, A, I, T, Q, R) \in \mathcal{R}^8_+ : N \leq \frac{\Pi}{\mu} \right\}.
$$

3.2 Positivity of solutions

Theorem 3.1. *If the initial conditions of the model are nonnegative in the feasible set* Ω*, then the solution set* $(S(t), S_h(t), E(t), A(t), I(t), Q(t), T(t), R(t))$ *of system* (1) *is positive for future time* $t \geq 0$.

Proof. We let $\tau = sup\{t > 0 : S_0(\zeta) \geq 0,\ S_{h0}(\zeta) \geq 0,\ E_0(\zeta) \geq 0, A_0(\zeta) \geq 0, I_0(\zeta) \geq 0, T_0(\zeta) \geq 0, Q_0(\zeta) \geq 0, R_0(\zeta) \geq 0$ for all $\zeta \in [0,t]\}$. Since $S_0(t) \geq 0,\ S_{h0}(t) \geq 0,\ E_0(t) \geq 0,\ A_0(t) \geq 0, I_0(t) \geq 0, T_0(t) \geq 0, Q_0(t) \geq 0, R_0(t) \geq 0$ then $\tau > 0$. If $\tau < \infty$, then automaticaly $S_0(t)$ or $S_{h0}(t)$ or $E_0(t)$ or $A_0(t)$ or $I_0(t)$ or $T_0(t)$ or $Q_0(t)$ or $R_0(t)$ is equal to zero at τ. Taking the first equation of the model (1)

$$\frac{dS}{dt} = \Pi - (\beta_1 A + \beta_2 I)\, S - (\upsilon + \mu)S. \tag{5}$$

Then, using the variation of constants formula the solution of equation (5) at τ is given by:

$$S(\tau) = S(0)\exp\left[-\int_0^\tau \left((\beta_1 A + \beta_2 I)\, S + (\upsilon + \mu)S\right)(S)dS\right] + \int_0^\tau \Pi.\exp\left[-\int_S^\tau \left((\beta_1 A + \beta_2 I)\, S + (\upsilon + \mu)S\right)(\zeta)d\zeta\right] dS > 0.$$

Moreover, since all the variables are positive in $[0,\tau]$, hence, $S(\tau) > 0$. It can be shown in a similar way that $S_h(\tau) > 0, E(\tau) > 0, A(\tau) > 0\ I(\tau) > 0, T(\tau) > 0, Q(\tau) > 0$ and $R(\tau) > 0$. Which is a contradiction. Hence, $\tau = \infty$. Therefore, all the solution sets are positive for $t \geq 0$. □

3.3 COVID-19 Free Equilibrium Point (CFEP)

COVID-19 free equilibrium point is the state at which the infection is not present in the population and note that it has been eradicated. In the case of COVID 19 free the compartments $E = I = A = 0$. Hence, equating zero for the remaining equations in (1) leads the COVID-19 free equilibrium point and given by:

$$E_0 = (\frac{\pi}{\mu}, \frac{\upsilon\pi}{\mu(\gamma + \mu)}, 0, 0, 0, 0, 0, 0). \tag{6}$$

3.4 Basic reproduction number

To analyze the stability of the equilibrium points, the basic reproduction number $\mathcal{R}_0$ of the model is important. It is obtained using the next-generation matrix method [17, 18]. The first step is rewrite the model equations, starting with newly infective classes:

$$\begin{cases} \frac{dE}{dt} = (\beta_1 A + \beta_2 I)\, S - (\gamma + \mu)\, E, \\ \frac{dA}{dt} = \alpha\, \gamma\, E - (\varrho_1 + \delta + \mu)\, A, \\ \frac{dI}{dt} = (1-\alpha)\, \gamma\, E - (\varrho_2 + \omega + \varepsilon + \mu)\, I, \\ \frac{dT}{dt} = (1-c)\, \varepsilon\, I - (\varrho_3 + \rho + \mu)\, T, \\ \frac{dQ}{dt} = \sigma\, \delta\, A + c\varepsilon I + \phi\rho T - (\varrho_4 + k + \mu)\, Q, \\ \frac{dR}{dt} = \omega I + (1-\sigma)\, \delta\, A + kQ + (1-\phi)\rho\, T - \mu\, R. \end{cases} \tag{7}$$

Then, by the principle of next-generation matrix, the Jacobian matrices at DFE is given by

$$\mathcal{F} = \begin{pmatrix} 0 & \frac{\beta_1\Pi}{\mu} & \frac{\beta_2\Pi}{\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} \gamma+\mu & 0 & 0 & 0 & 0 & 0 \\ -\alpha\,\gamma & \psi_1 & 0 & 0 & 0 & 0 \\ -(1-\alpha)\,\gamma & 0 & \psi_2 & 0 & 0 & 0 \\ 0 & 0 & -(1-c)\,\epsilon & \psi_3 & 0 & 0 \\ 0 & -\sigma\,\delta & -c\epsilon & 0 & \psi_4 & 0 \\ 0 & -(1-\sigma)\,\delta & -\omega & -\phi\,\rho & -k & \mu \end{pmatrix},$$

where

$$\psi_1 = \varrho_1 + \delta + \mu, \psi_2 = \varrho_2 + \omega + \epsilon + \mu,$$
$$\psi_3 = \varrho_3 + \theta + \mu + \varphi + \epsilon + \mu, \psi_4 = \varrho_4 + k + \rho + \mu.$$

Therefore, the basic reproduction number is the spectral radius of the next-generation matrix $\mathcal{F}\mathcal{V}^{-1}$, is given us

$$\mathcal{R}_0 = \frac{((1-\alpha)\,(\delta+\varrho_1+\mu)\,\beta_2 + (\epsilon+\omega+\varrho_2+\mu)\,\alpha\,\beta_1)\,\gamma\,\Pi}{\mu\,(\gamma+\mu+\upsilon)\,(\delta+\varrho_1+\mu)\,(\epsilon+\omega+\varrho_2+\mu)}. \tag{8}$$

Which is a threshold parameter that represents the average number of infection caused by one infectious individual when introduced in the susceptible population [17] in its infectious life time.

3.5 Local stability of DFEP

Theorem 3.2. *The DFEP point is locally asymptotically stable if* $\mathcal{R}_0 < 1$ *and unstable if* $\mathcal{R}_0 > 1$.

Proof. The Jacobian matrix, evaluated at the disease-free equilibrium E_0, we get:

$$J = \begin{pmatrix} -\mu-\upsilon & \tau & 0 & -\frac{\beta_1(\tau+\mu)\Pi}{(\tau+\mu+\upsilon)\mu} & -\frac{\beta_2(\tau+\mu)\Pi}{(\tau+\mu+\upsilon)\mu} & 0 & 0 & 0 \\ \upsilon & -\tau-\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma-\mu & \frac{\beta_1(\tau+\mu)\Pi}{(\tau+\mu+\upsilon)\mu} & \frac{\beta_2(\tau+\mu)\Pi}{(\tau+\mu+\upsilon)\mu} & 0 & 0 & 0 \\ 0 & 0 & \alpha\,\gamma & -\psi_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\alpha)\,\gamma & 0 & -\psi_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & (1-c)\,\epsilon & -\psi_3 & 0 & 0 \\ 0 & 0 & 0 & \sigma\,\delta & c\epsilon & \phi\,\rho & -\psi_4 & 0 \\ 0 & 0 & 0 & (1-\sigma)\,\delta & \omega & (1-\phi)\,\rho & k & -\mu \end{pmatrix},$$

where

$$\psi_1 = \varrho_1 + \delta + \mu, \psi_2 = \varrho_2 + \omega + \epsilon + \mu,$$
$$\psi_3 = \varrho_3 + \theta + \mu + \varphi + \epsilon + \mu, \psi_4 = \varrho_4 + k + \rho + \mu.$$

The first five eigenvalues are listed as:

$$-\mu, -(\tau + \mu), -\psi_3, -\psi_4, -\mu.$$

The other eigenvalues are obtained from the characteristic polynomial:

$$\mathcal{P}(\lambda) = \lambda^3 + \varphi_1\lambda^2 + \varphi_2\lambda + \varphi_3 = 0. \tag{9}$$

where

$$\varphi_1 = \psi_1 + \psi_2 + \gamma + \mu,$$
$$\varphi_2 = \frac{-\Pi\,\alpha\,\gamma\,\beta_1 + (1-\alpha)\Pi\,\gamma\,\beta_2 + \gamma\,\mu\,\psi_1 + \gamma\,\mu\,\psi_2 + \psi_1\mu^2 + \psi_2\mu^2 + \psi_2\mu\,\psi_1}{\mu},$$
$$\varphi_3 = -\frac{\Pi\,\alpha\,\gamma\,\beta_1\psi_2 - \Pi\,\alpha\,\gamma\,\beta_2\psi_1 + \Pi\,\gamma\,\beta_2\psi_1 - \gamma\,\mu\,\psi_1\psi_2 - \mu^2\psi_1\psi_2}{\mu}.$$

To check the positivity of the eigenvalues, We used Routh-Hurwitz criteria and by this principle equation (9) has strictly negative real root iff $\psi_1 > 0$, $\psi_2 > 0$ and $\psi_3 > 0$. Clearly we see that $\psi_1 > 0$ and $\psi_2 > 0$ because they are the sum of positive parameters. Then taking the third equation,

$$\psi_3 = (\varepsilon + \rho + \mu)\,(\delta + \mu)\,[1 - \mathcal{R}_0] > 0$$

Hence the DFEP is locally asymptotically stable if $\mathcal{R}_0 < 1$. □

3.6 Global stability of DFEP

In this section, we investigate global asymptotic stability of the disease free equilibrium using the theorem of Castillo-Chavez [19, 14]. We rewrite model in equation (1) as:

$$\begin{cases} \frac{dZ}{dt} = F(Z, Y), \\ \frac{dY}{dt} = G(Z, Y), G(Z, 0) = 0, \end{cases} \tag{10}$$

where $Z{=}(S, S_h, R) \in \mathcal{R}^3$ denotes uninfected populations and $Y = (E, A, I, T, Q)$ $\in \mathcal{R}^5$ denotes the infected population. $E_0 = (Z^*, 0)$ represents the DFEP of this system. List two conditions as:

(i) For $\frac{dZ}{dt} = F(Z, 0), Z^*$ is globally asymptotically stable.

(ii) $\frac{dY}{dt} = D_Y G(Z, 0)Y, -\hat{G}(Z, Y), \hat{G}(Z, Y) \geq 0$ for all $(Z, Y) \in \Omega$.

If DFEP satisfies the above two conditions, we conclude that E_0 is globally asymptotically stable and according to Castillo-Chavez [19] and the following theorem holds.

Theorem 3.3. *The equilibrium point $E_0 = (Z^*, 0)$ of the system (10) is globally asymptotically stable if $\mathcal{R}_0 < 1$ and the conditions (i) and (ii) are satisfied.*

Proof. We start the proof by defining new variables and dividing the system into subsystems. $Z = (S, R, Q)$ and $Y = (E, A)$. From equation (10) we have two functions $G(Z, Y)$ and $F(Z, Y)$ given by:

$$F(X,Y) = \begin{pmatrix} \Pi + \varphi S_h - (\beta_1 A + \beta_2 I)\, S - (\upsilon + \mu) S \\ \upsilon\, S - (\tau + \mu)\, S_h \\ \omega I + (1-\sigma)\,\delta\, A + kQ + (1-\phi)\rho\, T - \mu\, R \end{pmatrix}$$

and

$$G(Z,Y) = \begin{pmatrix} (\beta_1 A + \beta_2 I)\, S - (\gamma + \mu)\, E \\ \alpha\, \gamma\, E - (\varrho_1 + \delta + \mu)\, A \\ (1-\alpha)\, \gamma\, E - (\varrho_2 + \omega + \varepsilon + \mu)\, I \\ (1-c)\, \varepsilon\, I - (\varrho_3 + \rho + \mu)\, T \\ \sigma\, \delta\, A + c\varepsilon I + \phi\rho T - (\varrho_4 + k + \mu)\, Q \end{pmatrix}.$$

Now, we consider the reduced system $\frac{dZ}{dt} = F(Z, 0)$ from condition (i)

$$\begin{cases} \frac{dS}{dt} = \Pi + \tau S_h - (\upsilon + \mu) S, \\ \frac{dS_h}{dt} = \upsilon\, S - (\tau + \mu)\, S_h, \\ \frac{dR}{dt} = -\mu R. \end{cases} \tag{11}$$

We note that this asymptomatic dynamics is independent of the initial conditions in Ω , therefore the convergence of the solutions of the reduced system equation (11) is global in Ω. We compute

$$G(Z,Y) = D_Y G(Z^*, 0) Y - \hat{G}(Z;Y)$$

and show that $\hat{G}(Z;Y) \geq 0$. Now,

$$D_Y G(Z^*, 0) = \begin{pmatrix} -\gamma - \mu & \frac{\beta_1(\tau+\mu)\Pi}{(\tau+\mu+\upsilon)\mu} & \frac{\beta_2(\tau+\mu)\Pi}{(\tau+\mu+\upsilon)\mu} & 0 & 0 \\ \alpha\, \gamma & -\varrho_1 - \delta - \mu & 0 & 0 & 0 \\ (1-\alpha)\, \gamma & 0 & -\varrho_2 - \varphi - \omega - \epsilon - \mu & 0 & 0 \\ 0 & 0 & (1-c)\, \epsilon & -\varrho_3 - \rho - \mu & 0 \\ 0 & \sigma\, \delta & c\epsilon & \phi\, \rho & -\varrho_4 - k - \mu \end{pmatrix}.$$

And, we get

$$\hat{G}(X,Y) = \begin{pmatrix} \left(\frac{\Pi}{\mu} - S\right)(\beta_1 A + \beta_2 I) \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Here, since $\frac{\Pi}{\mu} = S^0 \geq S$, Hence, it is clear that $\hat{G}(Z,Y) \geq 0$ for all (Z,Y) $\in \Omega$. Therefore, by LaSalle's invariance principle [20] this proves that DFE is globally asymptotically stable for $\mathcal{R}_0 < 1$. From this result, we can say that the model exhabits forward bifurication. In other words, for $\mathcal{R}_0 < 1$ the DFEP and EEP does not co-exist. □

3.7 The endemic equilibrium point (EEP)

For endemic equilibrium point of the model we denote it by E^* and $E^* = (S^*, S_h^*, E^*, A^*, I^*, T^*, Q^*, R^*) \geq 0$. The COVID-19 pandemic model has a unique endemic equilibrium and it can be obtained by equating each equation of the model equal to zero. i.e

$$\frac{dS}{dt} = \frac{dS_h}{dt} = \frac{dA}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dQ}{dt} = \frac{dR}{dt} = 0.$$

Then, we obtain

$$\text{(12)} \quad \begin{cases} S^* = \frac{(\gamma+\mu)[\delta(\varepsilon+\omega+\varrho_2+\mu)+\mu(\omega+\varrho_1+\varrho_2+\mu)+\varepsilon(\varrho_1+\mu)+\varrho_1(\omega+\varrho_2)]}{\gamma[(1-\alpha)(\delta+\varrho_1+\mu)\beta_2+(\varepsilon+\omega+\varrho_2+\mu)\alpha\beta_1]} \\ S_h^* = \frac{\upsilon(\gamma+\mu)[\delta(\varepsilon+\omega+\varphi+\varrho_2+\mu)+\mu(\omega+\varrho_1+\varrho_2+\mu)+\varepsilon(\varrho_1+\mu)+\varrho_1(\omega+\varrho_2)]}{\gamma(\tau+\mu)[(1-\alpha)(\delta+\varrho_1+\mu)\beta_2+(\varepsilon+\omega+\varrho_2+\mu)\alpha\beta_1]} \\ E^* = \frac{\xi_1}{\gamma\xi_2} \\ A^* = \frac{\alpha\xi_1}{(\varrho_1+\delta+\mu)\xi_2} \\ I^* = \frac{(1-\alpha)\xi_1}{(\varrho_2+\omega+\varepsilon+\mu)\xi_2} \\ T^* = \frac{(\alpha-1)(c-1)\varepsilon\xi_1}{(\varrho_2+\omega+\varepsilon+\mu)(\rho+\varrho_3+\mu)\xi_2} \\ Q^* = \frac{\sigma\delta A^*+c\varepsilon I^*+\phi\rho T^*}{\varrho_4+k+\mu} \\ R^* = \frac{\omega I^*+(1-\alpha)\delta A^*+kQ^*+(1-\phi)\rho T^*}{\mu}, \end{cases}$$

where

$$\begin{aligned} \xi_1 &= (\delta+\varrho_1+\mu)(\alpha-1)\Pi\gamma\beta_2 - (\varepsilon+\omega+\varrho_2+\mu)\Pi\alpha\gamma\beta_1 \\ &\quad + \mu(\gamma+\mu)[\delta(\varepsilon+\omega+\varrho_2+\mu)+\varepsilon(\varrho_1+\mu)+\mu(\omega+\varrho_1+\varrho_2+\mu) \\ &\quad + \varrho_1(\omega+\varphi+\varrho_2)], \\ \xi_2 &= (\gamma+\mu)[\delta\beta_2(\alpha-1)-\alpha\varepsilon\beta_1] - (\omega+\varrho_2+\mu)(\gamma+\mu)\alpha\beta_1 \\ &\quad + (\alpha-1)(\gamma+\mu)(\varrho_1+\mu)\beta_2. \end{aligned}$$

3.8 Sensitivity analysis

We used the normalized forward sensitivity index definition to go through sensitivity analysis on the basic parameters [21] as done in [22, 23]. The Normalized forward sensitivity index of a variable, $\mathcal{R}_0$, that depends differentiably on a parameter, p, is defined as: $\Lambda_p^{\mathcal{R}_0} = \frac{\partial\mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}$ for p represents all the basic parameters. Here, we have $\mathcal{R}_0 = \frac{((1-\alpha)(\delta+\varrho_1+\mu)\beta_2+(\epsilon+\omega+\varrho_2+\mu)\alpha\,\beta_1)\gamma\,\Pi}{\mu(\gamma+\mu+\upsilon)(\delta+\varrho_1+\mu)(\epsilon+\omega+\varrho_2+\mu)}$. For the

sensitivity index of $\mathcal{R}_0$ to the parameters:

$$\Lambda_{\beta_1}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_1} \times \frac{\beta_1}{\mathcal{R}_0} = \frac{(\epsilon + \omega + \varrho_2 + \mu)\,\alpha\,\beta_1}{(1-\alpha)\,(\delta + \varrho_1 + \mu)\,\beta_2 + (\epsilon + \omega + \varrho_2 + \mu)\,\alpha\,\beta_1} > 0,$$

$$\Lambda_{\beta_2}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_2} \times \frac{\beta_2}{\mathcal{R}_0} = \frac{(1-\alpha)\,(\delta + \varrho_1 + \mu)\,\beta_2}{(1-\alpha)\,(\delta + \varrho_1 + \mu)\,\beta_2 + (\epsilon + \omega + \varrho_2 + \mu)\,\alpha\,\beta_1} > 0,$$

$$\Lambda_{\alpha}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \alpha} \times \frac{\alpha}{\mathcal{R}_0} = \frac{\alpha\,\left(-(\delta + \varrho_1 + \mu)\,\beta_2 + (\epsilon + \omega + \varrho_2 + \mu)\,\beta_1\right)}{(1-\alpha)\,(\delta + \varrho_1 + \mu)\,\beta_2 + (\epsilon + \omega + \varrho_2 + \mu)\,\alpha\,\beta_1} < 0.$$

Similarly, we can work for the other parameters. The sensitivity indices of the basic reproductive number with respect to main parameters are found in Table 1.

Table 1: Sensitivity indecies table.

Parameter symbol	**Sensitivity indecies**
β_1	+ve
β_2	+ve
γ	+ve
σ_1	-ve
σ_2	-ve
k	-ve
ε	-ve
δ	-ve
ω	-ve
μ	-ve

4. Numerical simulations

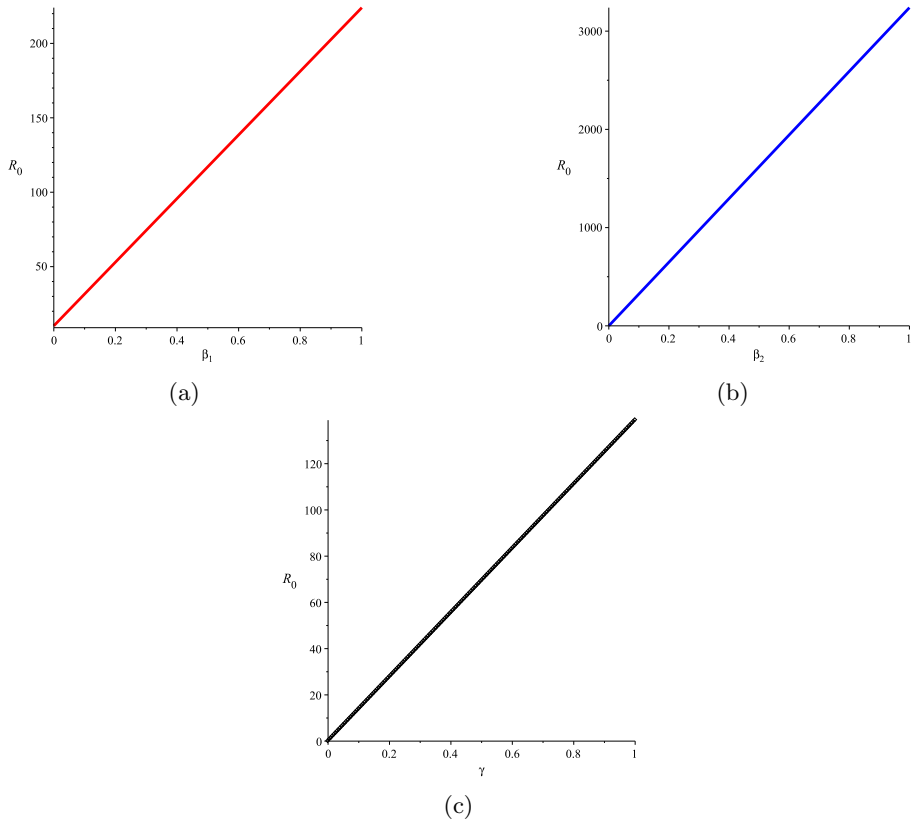
Analytic studies cannot be complete without numerical verification of the results. In this section, we present computer simulation of some solutions of the system (1). Besides verification of our analytical outcomes, these numerical simulations are very significant from practical point of view. To illustrate the results, we used parameter values in the Table 2 .

From Figure 2, we find the positve indices parameters. These parameters ($\beta_1,\ \beta_2,\ \ and\ \ \gamma$) show that they have great impact on expanding the disease in the community if their values are increasing. This is because that the basic reproduction number increases as their values increase, so that the average number of secondary cases of infection increases in the community. Therfore, stakeholders should take action to decrease the effect of the pandemic.

Figure 3, shows those parameters in which their sensitivity indices are negative ($\delta, \omega, \varepsilon, k,\ \ and\ \ \mu$) and the increment of the parameters have an effect of minimizing the burden of the disease in the community. Therefore, research advice

Table 2: Description of parameters of the model (1)

Parameter	**Description**	Value	Source
Π	Ricuirement rate of individuals	150	Assumed
β_1	Transmission rate from asymptomatic to susceptible individuals	0.00000115	[16]
β_2	Transmission rate from infected to susceptible individuals	0.003	[16]
ρ	Individuals who leave from treatment subpopulation	0.2	[16]
δ	Proportion of exposed individuals leaving the compartment	0.2	[16]
ε	Individuals who leave leave from the infected subpopulation	0.001	[16]
τ	Proportion of exposed individuals who join infected compartment	0.07	[13, 24]
υ	Proportion of exposed individuals who join infected compartment	0.005	[13]
μ	Natural death rate the population	0.016	[13]
k	Recovery rate of individuals under quarantine	0.2	[16]
ϱ_1	Induced death rate of asymptomatic individuals	0.002	Assumed
ϱ_2	Induced death rate of infected individuals	0.0002	[16, 24]
ϱ_3	Induced death rate of individuals under treatment	0.0303	Assumed
ϱ_4	Induced death rate of individuals under quarantine	0.0103	[16]
γ	Exposed individuals that become infectious	0.143	[16, 24]
ϕ	Proportion of individuals under treatment who join quarantine	0.3	[16]
c	Proportion of infected individuals who join quarantine	0.5	[16]
ω	Fraction of infected individuals that are immune	0.00023	[16]
σ	Fraction of asymptomatic individuals that are immune	0.01	[16]
α	Fraction of exposed individuals that become asymptomatic	0.1	[16]



(a) (b)

(c)

Figure 2: The positive indices parametres

for stakeholders to work on increasing negative indices parameters to fight the pandemic persistence.

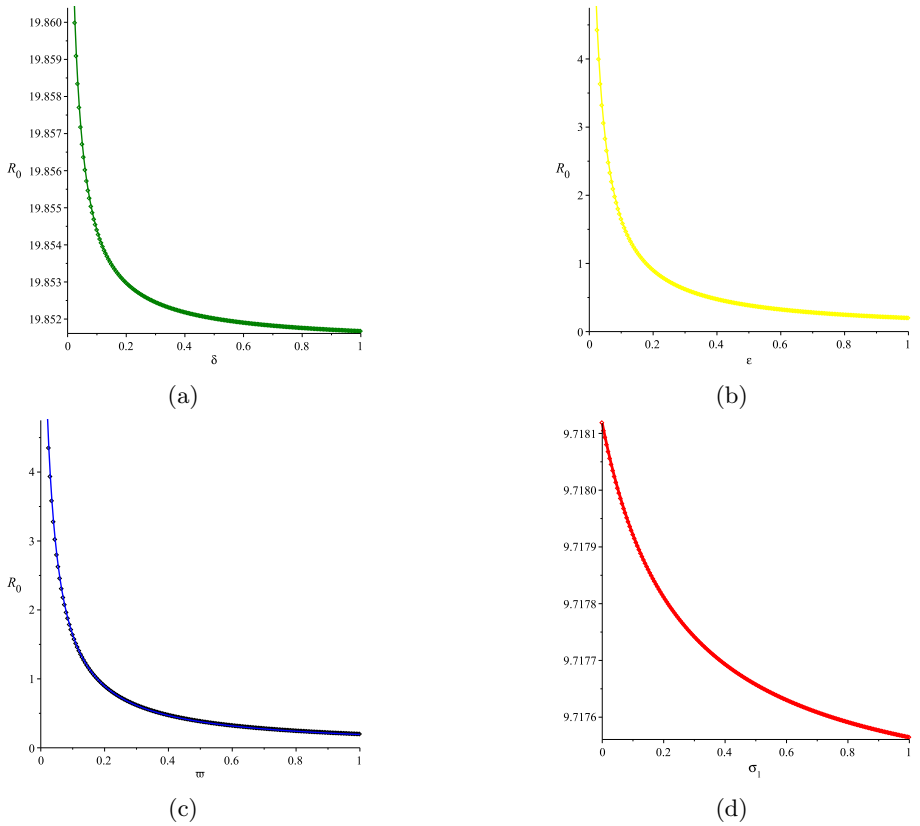


Figure 3: The negative indices parameters

4.1 Impact of γ on infected population

From Figure 4, as we incease the rate of the number of exposed population to infected and asymptomatic stage increases the number of total infected individuals in the population. Thus, the closing of government offices fully or partially was an important decision to control the spread of the pandemic.

4.2 Impact of hospitalizing and treatment (ε) on infected population

As we see from the Figure 5, by increasing the value of ε, the number of infected people is decreasing due to an increase number of hospitalize/quarantine and treantment of infectives at home. This is due to the reason that infectious individuals plays an important role in the infection generation, and therefore, the people should use every control mechanisms and should be educated to avoid the interaction with such people and ready for testing. Therefore, the government should work testing and diagnosis to reduce the infectious number from the population by quarantine/ hospitalize or and treantment of infectives at home.

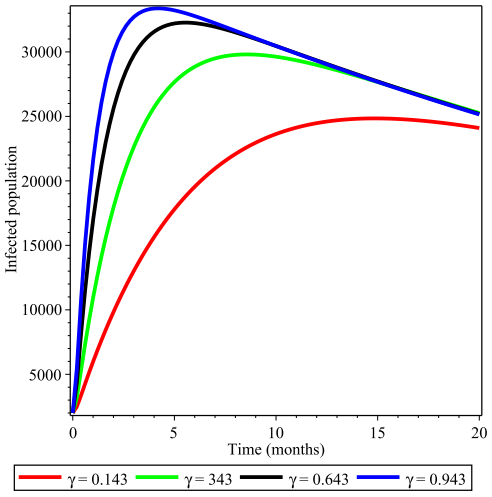


Figure 4: Impact of γ on Infected population

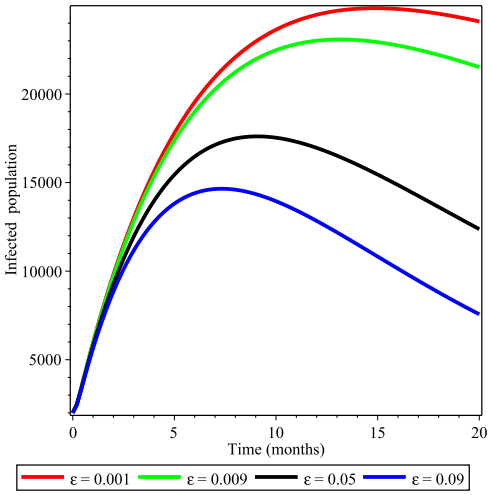


Figure 5: Impact of ε on Infected population

Figure 6, presents the dynamics of the mode with and without quarantine/hospitalize and treatment. From the figure, one can see that, using quarantine/hospitalize and treatment, it is possible to increase the number of recovered individuals. Therefore, here stakeholders should work on using those combating ways to fight the pandemic. A comparison figure is shown to see the effects on th number of total recovered individuals, as seen in the Figure 7. It is evident from figure that from the individual management techniques hospitalize/quarantine infective individuals is better than taking treatment at home. However, instead of using them separately, it is best to use the integration of both techniques to produce a big number of recovered population from the pandemic.

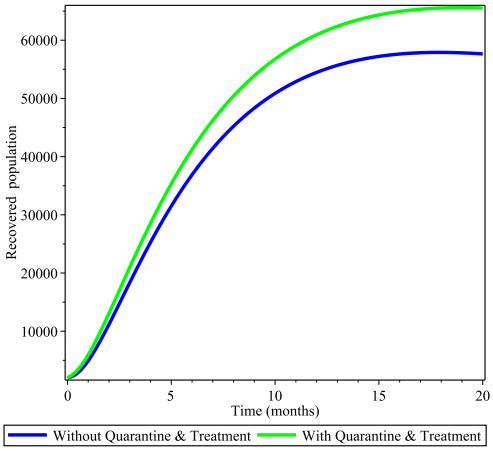


Figure 6: Comparison between with and without quarantine & Treatment

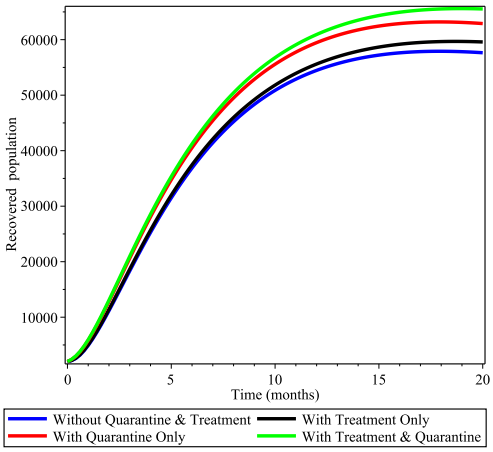


Figure 7: Comparison between quarantine only, Treatment only, with and without quarantine & Treatment

5. Conclusions

In this paper an SEAIR deterministic model with quarantine and treatment for the transmission dynamics of the pandemic COVID-19 was formulated. The mathematical results for the model were shown. The basic reproduction number $\mathcal{R}_0$ was computed and the stability of equilibria points was investigated. Using Castillo-Chavez theorem, the disease free equilibrium point globally asymptotically stable whenever the $\mathcal{R}_0 < 1$ was proven. We consider some parameters and their effect on the model graphically, which can be regarded as the controls for disease eradication. Also we show the effect of using quarantine and treatment in geting better number of recovered individuals. Therefore, as it is

shown in the figure, stakeholders should apply both quarantine and treatment simultaneously in cambating the pandemic.

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