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MATHEMATICAL MODELING OF COVID-19 AND ITS TRANSMISSION IN CASE OF ETHIOPIA

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Abstract. In this paper, we attempt to describe the spread of COVID-19 based on an extensions of the well-known SEIR models. We bring in a new representation to appraise and manage the spread of infectious disease COVID-19 through SEIHR pandemic model, which is based on the supposition that the infected individuals are isolated and the necessary treatments are arranged so that they cannot taint the other residents in the community. Dynamics of the SEIHR model is presented by basic reproduction number $\mathcal{R}_0$ and the comprehensive stability analysis. The reproduction number was also determined for the epidemiological model and found to be consistent with the local stability condition for the disease-free equilibrium. We establish the positivity and boundedness of solutions, local and global stability analysis of equilibria to examine its epidemiological relevance. To validate the model and estimating the important model parameters and prediction about the disease, we consider the real cases of Ethiopia from 4th March to 20th August 2020.

1. Introduction

In December 2019, corona virus first reported in Wuhan, China, is an infectious disease caused by a newly discovered corona virus. The virus is now known as the severe acute respiratory syndrome corona virus 2 (SARS-CoV-2). The disease it causes is called corona virus disease 2019 (COVID-19) [1]. According to WHO Novel Corona virus disease situation report 21 January 2020, the first case outside of China was reported in Thailand on 13 January 2020, Japan on 15 January 2020 and Republic of Korea on 20 January 2020 [2]. Currently the COVID-19 outbreak has now spread over the world. According to WHO the symptoms of COVID-19 may appear two to 14 days after exposure. This time after exposure and before having symptoms is called the incubation period i.e., during this period the infected humans are not infectious. However, the patients infected by COVID-19 will have symptoms including fever, cough and difficulty breathing, and the severe patients may have renal failure. This list is not all inclusive [3]. Current evidence suggests that COVID-19

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spreads between people through direct, indirect (through contaminated objects or surfaces), or close contact with infected people via mouth and nose secretions. These include saliva, respiratory secretions or secretion droplets. These are released from the mouth or nose when an infected person coughs, sneezes, speaks or sings [1]. According to WHO, to prevent infection and to slow transmission of COVID-19, do the following: wash your hands regularly with soap and water, or clean them with alcohol-based hand rub, maintain at least 1 metre distance between you and people coughing or sneezing, avoid touching your face, cover your mouth and nose when coughing or sneezing, stay home if you feel unwell, refrain from smoking and other activities that weaken the lungs, practice physical distancing by avoiding unnecessary travel and staying away from large groups of people. According to WHO 2020 report, after the outbreak day to present September 18, 2020 globally, there were about 30,055,710 confirmed COVID-19 cases with 943,433 deaths. In Africa also the virus spread to 52 countries, but two countries (Lesotho and Comoros) are still registered as COVID-19 free. Through out Africa up to August 26, 2020 confirmed cases are 22,313, number of deaths are 1,124 and recoveries are 5,492 [4]. In Ethiopia the first case was reported on March 13, 2020, and up to September 18, 2020 the confirmed cases reached to 67,515, deaths are 1,072 and total recoveries are 27,638. In epidemiology, mathematical modeling has a great role in understanding the dynamics of infectious diseases in a population and widely used to predict the results of an epidemic successfully. Most commonly used epidemic models are SIS, SIR and SEIR models [4]. The SIR model is a very well established model and used widely for various epidemics [5]. Several Mathematical modeling have been proposed to study the dynamics of epidemic disease. Mathematical models that analyze the spread of COVID-19 have began to appear in published papers and online resources [6, 7, 8]. A number of compartmental models have already been proposed and analyzed for the COVID-19 outbreak in different countries [9]-[19]. For instance, [9] included compartments corresponding to suspected cases, which consists of the individuals that show similar symptoms but are not confirmed cases, and indirectly infected individuals. The modified SEIR model which included asymptomatic and treatment compartments for occurrences in Wuhan, China, the city where the outbreak started, and outside of Wuhan haven been used [10]. [12] included a separate compartment for quarantined individuals in the SIR model to account for the containment measures applied by the public for the virus. The stability of epidemic models has been studied in many papers [5]-[22]. Many authors paid attention to local stability of equilibria. Recently, the study of epidemic models mainly concerns global asymptotic stability. The most successful approaches to the problem are the direct Lyapunov method [23] and the geometric method [24]. In this paper, we consider a modefied SEIHR model depicting the transmission of corona virus with disease resistance in human. The model is given by a system of four differential equations depending on parameters. By using the method of next generation matrix [25], we found a threshold $\mathcal{R}_0$ called basic reproduction number. In general, when $\mathcal{R}_0 \leq 1$, the disease dies out and when $\mathcal{R}_0 > 1$, the disease persists in the

population. If we suppose that the endemic equilibrium also exists for $\mathcal{R}_0 < 1$, although it is not true, then the bifurcation occurring in the model can be explained as a trans critical bifurcation. Several various methods are used to determine the stability of equilibria. We concentrate our study on the globally stability of equilibria. This is obtained by Lyapunov functional approach and geometric approach

2. Model formulation

In this section, we develop mathematical model for the transmission of COVID-19 which spreads in a populations. Based on the development and epidemiological characteristics of COVID-19 infection, we extend the classical SEIR model to describe the transmission of COVID-19 in Ethiopia. In this model the entire population is considered into four disjoint compartments. Susceptible individuals (S) are those who are not infected by the disease but there is a possibility to be infectious. Exposed individuals (E) are individuals who are in the incubation period after being infected by the disease, and have no visible clinical signs. This individuals could infect other people with a higher probability than people in the infectious compartments. After the incubation period, the person passes to the Infectious compartment; Infected Individuals (I) are individuals who developed the symptom of the disease. As far as the diagnosed individual are concerned, we assume that infected individuals will be immediately sent to designated hospitals for isolation and treatment, these group of the population will be converted to hospitalized H. Finally, Recovered individuals (R) are those individuals who recovered (through treatment or natural recovery) from the disease.

The recruitment rate denoted by π is either through immigration or birth. The susceptible individuals got corona virus disease by contact rate of β either from exposed individuals with probability of $1-\tau$ or from infected individuals with probability of τ and move to the exposed compartment. The exposed individuals become infectious and join the infected compartment with the proportion α. σ is the average rate at which symptomatic individuals become hospitalized and ϵ is the recovery rate without being hospitalized. γ is the recovery rate of the isolated infected by treatment from the disease or die due to the corona virus disease at a rate of δ. The recovered individuals become again susceptible to the disease with a rate of η.

Based on the above assumptions and the actual isolation strategy, the spread of COVID-19 in the populations is depicted in Figure 1.

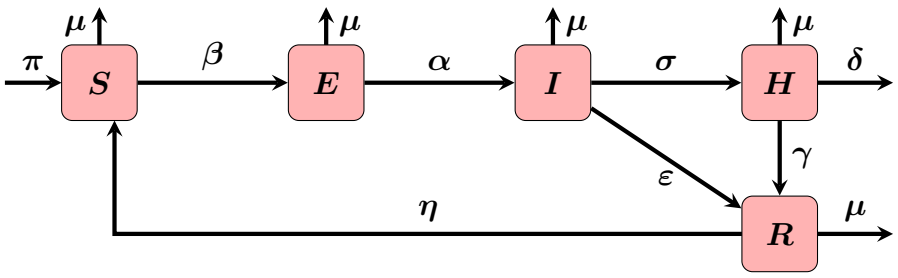


FIGURE 1. Flow diagram of the compartmental model of COVID-19

Using the above depiction, we formulate the corresponding dynamical model as follows.

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \pi - \beta[(\tau I + (1-\tau)E]S + \eta R - \mu S \\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta[(\tau I + (1-\tau)E]S - (\alpha+\mu)E \\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \alpha E - (\sigma+\varepsilon+\mu)I \\
\frac{\mathrm{d}H}{\mathrm{d}t} &= \sigma I - (\gamma+\delta+\mu)H \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \varepsilon I + \gamma H - (\eta+\mu)R
\end{aligned}
\qquad (2.1)
$$

With the initial condition

$$S(0) = S_0 \geq 0,\ E(0) = E_0 \geq 0,\ I(0) = I_0 \geq 0,\ H(0) = H_0 \geq 0,\ R(0) = R_0 \geq 0,$$

There are a total of 12 model parameters in system (2.1).

Parameters	Description
π	Recruitment rate of individuals
β	Contact rate of individuals
τ	Transmission rate of infection
α	Proportion of exposed individuals leaving the compartment
ε	recovery rate without being hospitalized
σ	Proposition of infected individuals become hospitalized
γ	Recovery rate of hospitalized patients
η	Proportion of recovered individuals to be susceptible
μ	Natural death rate
δ	Death rate due to coronavirus

TABLE 1. Description of the model parameters

3. Analysis of the model

In this section, we study the quantitative and qualitative analysis of Eqn. (2.1).

3.1. **Well-posedness.** An important feature of an epidemiological relevant model is the positivity and boundedness of the solutions. Therefore, it is important to prove that all the variables are nonnegative for all time $t > 0$ which implies that any solution that has positive initial values will remain positive for all time $t \geq 0$. We begin by determining the biologically feasible set for the model (2.1).

Theorem 3.1. *The closed region* $\Omega = \{(S, E, I, H, R) \in \mathbb{R}^5_+ \ : \ N(t) \leq \frac{\pi}{\mu}\}$ *is positively invariant set for the system (2.1).*

Proof. For the model the total population is

$$N = S(t) + E(t) + I(t) + H(t) + R(t)$$

Then, differentiating N with respect to time t, we obtain:

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \frac{\mathrm{d}S}{\mathrm{d}t} + \frac{\mathrm{d}E}{\mathrm{d}t} + \frac{\mathrm{d}I}{\mathrm{d}t} + \frac{\mathrm{d}H}{\mathrm{d}t} + \frac{\mathrm{d}R}{\mathrm{d}t} = \pi - \delta H - \mu N$$

If there is no death due to the disease, we get

$$\frac{\mathrm{d}N}{\mathrm{d}t} \leq \pi - \mu N \tag{3.1}$$

Integrating the inequality 3.1, using initial condition, we obtain

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}\left[1 - e^{-\mu t}\right]$$

Evaluating it as $t \to \infty$, we get

$$N(t) \leq \frac{\pi}{\mu}$$

Therefore

$$\Omega = \{(S, E, I, H, R) \in \mathbb{R}^5_+ \ : \ N(t) \leq \frac{\pi}{\mu}\}$$

Which is the feasible solution set for the model (2.1) and all the solution set is bounded in it. □

3.2. **Asymptomatic Stability of Disease Free Equilibrium.** When there is no disease in the population, I.e E = I = 0, the disease free equilibrium occur and to determine the equilibrium solutions, we set the right side of Eq. (2.1) equal to zero and obtain

$$\begin{aligned}
\pi - \beta[(\tau I + (1-\tau)E]S + \eta R - \mu S &= 0 \\
\beta[(\tau I + (1-\tau)E]S - (\alpha + \mu)E &= 0 \\
\alpha E - (\sigma + \varepsilon + \mu)I &= 0 \\
\sigma I - (\gamma + \delta + \mu)H &= 0 \\
\varepsilon I + \gamma H - (\eta + \mu)R &= 0
\end{aligned} \tag{3.2}$$

Therefore the disease free equilibrium point is obtained to be

$$\varepsilon_0 = \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right) \tag{3.3}$$

Basic reproduction number. The basic reproduction number is an important threshold condition in the analysis of an infectious disease. It determines whether the disease will die out or persist in the population as time increases. We calculate the basic reproduction number $\mathcal{R}_0$ of the system by applying the next generation matrix method used in [**?**]-[11]. The first step to get $\mathcal{R}_0$ is rewritten the model equations starting with newly infective classes:

$$\begin{aligned} \frac{\mathrm{d}E}{\mathrm{d}t} &= \beta[(\tau I + (1-\tau)E]S - (\alpha+\mu)E \\ \frac{\mathrm{d}I}{\mathrm{d}t} &= \alpha E - (\sigma+\varepsilon+\mu)I \\ \frac{\mathrm{d}H}{\mathrm{d}t} &= \sigma I - (\gamma+\delta+\mu)H \end{aligned} \quad , \tag{3.4}$$

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

$$\mathcal{F} = \begin{pmatrix} \frac{\beta(1-\tau)\pi}{\mu} & \frac{\beta\tau\pi}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} \alpha+\mu & 0 & 0 \\ -\alpha & \sigma+\varepsilon+\mu & 0 \\ 0 & -\sigma & \gamma+\delta+\mu \end{pmatrix},$$

Then

$$\mathcal{F}\mathcal{V}^{-1} = \begin{pmatrix} \frac{\beta(1-\tau)\pi(\sigma+\varepsilon+\mu)+\beta\tau\pi\alpha}{\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)} & \frac{\beta\tau\pi}{\mu(\sigma+\varepsilon+\mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

Therefore, the basic reproduction number is given us

$$\mathcal{R}_0 = \frac{\beta(1-\tau)\pi(\sigma+\varepsilon+\mu)+\beta\tau\pi\alpha}{\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)} \tag{3.5}$$

which is is a threshold parameter that represents the average number of infection caused by one infectious individual when introduced in the susceptible population [19].

Local Stability of DFE. Local stability is attained when the eigenvalues of the Jacobian, λ, are negative or have negative real parts. In other words, the solutions for λ such that $|J_{\varepsilon_0} - \lambda I| = 0$ should be negative or have negative real parts if the solution is complex [27].

Theorem 3.2. *The DFE ε_0 of the model (2.1) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. The Jacobian matrix at ε_0 is as:

$$J_{\varepsilon_0} = \begin{pmatrix} -\mu & -\frac{\beta(1-\tau)\pi}{\mu} & -\frac{\beta\tau\pi}{\mu} & 0 & \eta \\ 0 & \frac{\beta(1-\tau)\pi}{\mu} - \alpha - \mu & \frac{\beta\tau\pi}{\mu} & 0 & 0 \\ 0 & \alpha & -\sigma - \varepsilon - \mu & 0 & 0 \\ 0 & 0 & \sigma & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \varepsilon & \gamma & -\eta - \mu \end{pmatrix}$$

and the characteristic polynomial is $|J_{\varepsilon_0} - \lambda I| = 0$. Solving this polynomial, we get:

$$(-\mu - \lambda)(-\gamma - \delta - \mu - \lambda)(-\eta - \mu - \lambda)\left(\lambda^2 + A\lambda + B\right) = 0 \tag{3.6}$$

where

$$\begin{aligned} A &= -\frac{\beta(1-\tau)\pi}{\mu} + 2\mu + \alpha + \sigma + \varepsilon \\ B &= -\frac{\beta(1-\tau)\pi}{\mu}(\sigma + \varepsilon + \mu) - \frac{\beta\tau\pi\alpha}{\mu} + (\alpha + \mu)(\sigma + \varepsilon + \mu) \end{aligned}$$

Thus, the eigen values of the first expression of eqn. (3.6) are given by

$$\begin{aligned} \lambda 1 &= -\mu < 0 \\ \lambda 2 &= -(\eta + \mu) < 0 \\ \lambda 3 &= -(\gamma + \delta + \mu) < 0 \end{aligned}$$

The last expression of eqn. (3.6), that is

$$\lambda^2 + A\lambda + B = 0 \tag{3.7}$$

We applied Rourth-Hurwitz criteria and by the principle eqn. (3.6) has strictly negative real root iff $A > 0, B > 0$ and $AB > 0$. Clearly, we see that $A > 0$ because it is the sum of positive parameters.

$$\begin{aligned} A &= \frac{\sigma + \varepsilon + \mu}{\alpha + \mu} + \frac{\beta\tau\pi\alpha}{\mu(\alpha + \mu)(\sigma + \varepsilon + \mu)} + 1 - \mathcal{R}_0 > 0 \\ B &= (\alpha + \mu)(\sigma + \varepsilon + \mu)(1 - \mathcal{R}_0) > 0 \end{aligned}$$

Therefore, ε_0 is locally asymptotically stable. □

Global Stability of DFE.

Theorem 3.3. *The DFE ε_0 of the model (2.1) is globally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. To prove the global stability of the equilibrium point we construct the Lyapunov function as

$$L = c_1 E + c_2 I \tag{3.8}$$

Differentiating equation (3.13) with respect to t gives

$$\frac{dL}{dt} = c_1\frac{dE}{dt} + c_2\frac{dI}{dt} \tag{3.9}$$

Substituting $\frac{dE}{dt}$ and $\frac{dI}{dt}$ from the model (2.1), we obtain:

$$\begin{aligned}\frac{dL}{dt} &= c_1\left(\beta[(\tau I + (1-\tau)E]S - (\alpha+\mu)E\right) + c_2(\alpha E - (\sigma+\varepsilon+\mu)I)\\ &= c_1\beta(1-\tau)ES - c_1(\alpha+\mu)E + c_2\alpha E + c_1\beta\tau I - c_2(\sigma+\varepsilon+\mu)I \qquad (3.10)\end{aligned}$$

Take $c_2 = -\frac{\beta(1-\tau)S-(\alpha+\mu)}{\alpha}c_1$, then we get:

$$\frac{dL}{dt} = c_1\beta\tau IS - \left(-\frac{\beta(1-\tau)S-(\alpha+\mu)}{\alpha}c_1\right)(\sigma+\varepsilon+\mu)I \tag{3.11}$$

$$= \left[\frac{\beta(1-\tau)(\sigma+\varepsilon+\mu)S + \beta\tau\alpha S - (\alpha+\mu)(\sigma+\varepsilon+\mu)}{\alpha}\right]c_1 I \tag{3.12}$$

Taking $c_2 = 1$, and substituting $\mathcal{R}_0$ we get

$$\begin{aligned}\frac{dL}{dt} &\leq \left[\frac{\beta(1-\tau)\pi(\sigma+\varepsilon+\mu) + \beta\tau\alpha\pi - (\alpha+\mu)(\sigma+\varepsilon+\mu)}{\mu\alpha}\right]I\\ &= \left(\frac{(\alpha+\mu)(\sigma+\varepsilon+\mu)}{\alpha}\right)\left(\frac{\beta(1-\tau)\pi(\sigma+\varepsilon+\mu)+\beta\tau\alpha\pi}{\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)} - 1\right)I\\ &= \left(\frac{(\alpha+\mu)(\sigma+\varepsilon+\mu)}{\alpha}\right)(\mathcal{R}_0 - 1)\,I\end{aligned}$$

for $S \leq S_0 \leq \frac{\pi}{\mu}$ and $\frac{dL}{dt} \leq 0$ for $\mathcal{R}_0 \leq 1$ and trajectory of the model (2.1) on which $\frac{dL}{dt} = 0$ if and only if $I = 0$. This implies that the only $\frac{dL}{dt} \leq 0$ is ε_0. Therefore by Lasalle's invariance principle, ε_0 is globally asymptotically stable in Ω. □

The Endemic Equilibrium point. The endemic equilibrium point shows that the disease will persist in the system in the steady state. Here solve the equations (3.2) to obtain S, E, I, H and R. But for easy identification S, E, I, H, R are represented by $(S^*, E^*, I^*, H^*, R^*)$ at the steady state of the endemic respectively.

$$\begin{aligned}S^* &= \frac{\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)}{\beta[\tau\alpha + (1-\tau)(\sigma+\varepsilon+\mu)]}\\ E^* &= \frac{\mu(\alpha+\mu)(\eta+\mu)(\sigma+\varepsilon+\mu)^2(\gamma+\delta+\mu)}{\beta\kappa}(\mathcal{R}_0-1)\\ I^* &= \frac{\alpha\mu(\alpha+\mu)(\eta+\mu)(\sigma+\varepsilon+\mu)(\gamma+\delta+\mu)}{\beta\kappa}(\mathcal{R}_0-1) \qquad (3.13)\\ H^* &= \frac{\alpha\mu\sigma(\alpha+\mu)(\eta+\mu)(\sigma+\varepsilon+\mu)}{\beta\kappa}(\mathcal{R}_0-1)\\ R^* &= \frac{\alpha\mu(\alpha+\mu)(\eta+\mu)(\sigma+\varepsilon+\mu)[\varepsilon(\gamma+\delta+\mu)+\gamma\sigma]}{\beta\kappa}(\mathcal{R}_0-1)\end{aligned}$$

where

$$\kappa = [\tau\alpha + (1-\tau)(\sigma+\varepsilon+\mu)][(\alpha+\mu)(\eta+\mu)(\sigma+\varepsilon+\mu)(\gamma+\delta+\mu) - \eta\alpha((\gamma+\delta+\mu)+\gamma\sigma)]$$

Theorem 3.4. *The endemic equilibrium point ε_0^* of model (2.1) is locally asymptotically stable in Ω if $\mathcal{R}_0 > 1$.*

Proof. Recall the Jacobian of the system (2.1) at any equilibrium point (S, E, I, H, R) and we have

$$J = \begin{pmatrix} -\beta[\tau I + (1-\tau)E] - \mu & -\beta(1-\tau)S & -\beta\tau S & 0 & \eta \\ \beta[\tau I + (1-\tau)E] & \beta(1-\tau)S - \alpha - \mu & \beta\tau S & 0 & 0 \\ 0 & \alpha & -\sigma - \varepsilon - \mu & 0 & 0 \\ 0 & 0 & \sigma & -\gamma - \delta - \mu & 0 \\ 0 & 0 & \varepsilon & \gamma & -\eta - \mu \end{pmatrix}$$

At the endemic equilibrium point ε_0^*, calculating the Jacobian matrix J and then solving $\det(J - \lambda I)$, the characteristic equation is

$$(-\gamma - \delta - \mu - \lambda)(-\eta - \mu - \lambda)(\lambda^3 + a\lambda^2 + b\lambda + c) \tag{3.14}$$

where

$$\begin{aligned} a &= \beta(1-\tau)S^* + \beta(\tau I + (1-\tau)E) - 3\mu - \alpha - \sigma - \varepsilon \\ b &= [-\beta(1-\tau)S^* + 2\mu + \alpha + \sigma + \varepsilon]\,[\beta(\tau I + (1-\tau)E) - \mu] \\ &\quad - [\beta(1-\tau)S^*\beta(\tau I + (1-\tau)E) + (\alpha+\mu)(\delta+\varepsilon+\mu)] \\ c &= \beta(\tau I + (1-\tau)E)\,[(\delta+\varepsilon+\mu)(\beta(1-\tau)S^* + (\alpha+\mu)) - \beta\tau\alpha s^*] \\ &\quad -\mu(\alpha+\mu)(\delta+\varepsilon+\mu) \end{aligned}$$

Since roots of the characteristic equations corresponding to $J(\varepsilon_0^*)$ are

$$\begin{aligned} \lambda_1 &= -(\gamma+\delta+\mu) < 0 \\ \lambda_2 &= -(\eta+\mu) < 0 \end{aligned}$$

and other three satisfies the following cubic equation

$$\lambda^3 + a\lambda^2 + b\lambda + c = 0$$

applied Routh-Hurwitz criteria.

□

4. Sensitivity Analysis

We carried out sensitivity analysis, on the basic parameters, to identify their effect to the transmition of the disease. To go through sensitivity analysis, we used the normalized

sensitivity index definition as defined in [29]. The Normalized forward sensitivity index of a variable, $\mathcal{R}_0$, that depends differentiably on a parameter, u, is defined as:

$$\Lambda_u^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial u} \times \frac{u}{\mathcal{R}_0}$$

for u represents all the basic parameters. Here we have

$$\mathcal{R}_0 = \frac{\beta(1-\tau)\pi(\sigma+\varepsilon+\mu)+\beta\tau\pi\alpha}{\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)}$$

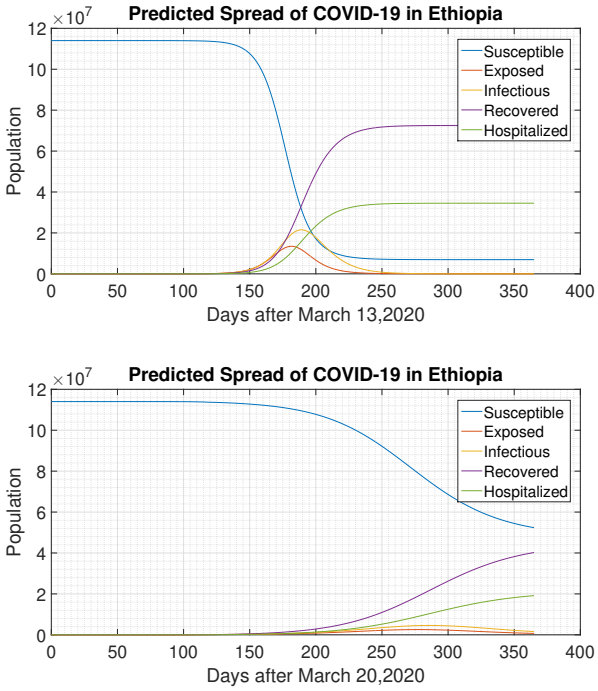
For the sensitivity index of $\mathcal{R}_0$ to the parameters:

$$\begin{aligned}
\Lambda_\beta^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \beta} \times \frac{\beta}{\mathcal{R}_0} = 1 > 0 \\
\Lambda_\pi^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \pi} \times \frac{\pi}{\mathcal{R}_0} = 1 > 0 \\
\Lambda_\tau^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \tau} \times \frac{\tau}{\mathcal{R}_0} = 1 - \frac{\beta\pi(\sigma+\varepsilon+\mu)}{[\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)]\mathcal{R}_0} > 0 \\
\Lambda_\alpha^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \alpha} \times \frac{\alpha}{\mathcal{R}_0} = -\frac{\beta\pi\mu(\sigma+\varepsilon+\mu)[\tau-(1-\tau)(\sigma+\varepsilon+\mu)]}{[\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)]^2} < 0 \\
\Lambda_\varepsilon^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \varepsilon} \times \frac{\varepsilon}{\mathcal{R}_0} = -\frac{\beta\tau\pi\alpha\mu(\alpha+\mu)}{[\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)]^2} < 0 \\
\Lambda_\sigma^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \sigma} \times \frac{\sigma}{\mathcal{R}_0} = -\frac{\beta\tau\pi\alpha\mu(\alpha+\mu)}{[\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)]^2} < 0 \\
\Lambda_\mu^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \mu} \times \frac{\mu}{\mathcal{R}_0} = -\mathcal{R}_0\frac{(\alpha+2\mu)(\sigma+\varepsilon+\mu)}{\mu(\alpha+\mu)(\sigma+\varepsilon+\mu)} < 0
\end{aligned}$$

The sensitivity indices of the basic reproductive number with respect to main parameters are π, β and τ. Which show that they have great impact on expanding the disease in the community if their values are increasing. Also those parameters in which their sensitivity indices are negative $\alpha, \varepsilon, \sigma$ and μ have an effect of minimizing the burden of the disease in the community as their values increase. Therefore, research advice for steack holders to work on decreasing the positive indeces and increasing negative indices parameters

5. Numerical Simulations

In this section, we examine the COVID 19 model and studied the effects of combined strategies on controlling the transmission of the disease. We start to solve the state equations with a guess for the controls over the simulated time using the fourth order Runge-Kutta scheme. Because of the transversality conditions, the adjoint equations were solved by a backward fourth order Runge-Kutta scheme using the current iterations' solutions of the state equation. This process is repeated and iterations are stopped if the values of the



unknowns at the previous iteration are very close to the ones at the present iteration. For numerical simulation purpose, we have used empiricaldata of COVID-19 cases in Ethiopia.

Moreover up to the April 23, 2020 total recoveries were 21 and registered deaths were 3. But, after march 20, 2020, COVID-19 in Ethiopia.

6. Conclusion

In this current paper we have formulated and studied an epidemic model of COVID-19 virus which is transferred from human to human. Therefore, prediction about infected individual is very much important for health concern arrangement of the citizens. It is also important to control spread rate of the COVID-19 virus with restricted supply. In this study, we have formulated a SEIHRS epidemic model for pandemic COVID-19. Considering all major parameters of the progression of the disease, several analytic results established. Theoretically it is proven that the dynamics depends on the basic reproduction number to examine the stability of the system. All the properties necessary for epidemiological relevance have also been proved. We have estimated the parametric values for Ethiopian using the existing real discrete data. The acceptable agreement between data analysis and numerical solutions are established. Finally the inclusion is that to estimate the situation of global pandemic COVID-19, mathematical modelling is an efficient method if the parameters can be estimated properly. We propose a novel dynamic system with time delay to predict the trend of outbreak for the 2019-nCoV. In this model, the instantaneous increment of

cumulative diagnosed people depends on the history of cumulative infected people, by which the latent period can be taken into consideration. The numerical simulation and parameter identification were carried out to verify the effectiveness and accuracy of the novel dynamic system. In addition, this model can well approximate the true data in this event, which may further predict the tendency of event.

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