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Modeling the effect of contaminated objects for the transmission dynamics of COVID-19 pandemic with self protection behavior changes

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ABSTRACT

A mathematical model for the transmission dynamics of Coronavirus diseases (COVID-19) is proposed using a system of nonlinear ordinary differential equations by incorporating self protection behavior changes in the population. The disease free equilibrium point is computed, and both the local and global stability analysis was performed. The basic reproduction number ($R_0$) of the model is computed using the method of next generation matrix. The disease free equilibrium point is locally asymptotically and globally stable under certain conditions. Based on the available data, the unknown model parameters are estimated using a combination of least square and Bayesian estimation methods for different countries. The forward sensitivity index is applied to determine and identify the key model parameters for the spread of disease dynamics. The sensitive parameters for the spread of the virus vary from country to country. We found out that the reproduction number depends mostly on the infection rates, the threshold value of the force of infection for a population, the recovery rates, and the virus decay rate in the environment. It illustrates that control of the effective transmission rate (recommended human behavioral change towards self-protective measures) is essential to stop the spreading of the virus. Numerical simulations of the model were performed to supplement and verify the effectiveness of the analytical findings.

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1. Introduction

The outbreak of coronavirus was first informed to the World Health Organization (WHO) as pneumonia of unknown cause on December 31, 2019, in Wuhan City, Hubei Province, China. As of January 10, 2020, the virus causing the outbreak was further determined by gene sequencing to be the new novel coronavirus, the same category as the Middle Eastern Respiratory Syndrome virus (MERS-CoV) and the Severe Acute Respiratory Syndrome virus (SARS-CoV) [1]. Due to the rapid spread of the virus with consequences worldwide, on March 11, 2020, the World Health Organization declared a pandemic. The global report of COVID-19 by the WHO indicated that on August 16, 2020, above 21.2 million people were infected with the virus, and over 761,779 have died [2]. The outbreak of the disease is still rapidly increasing in South American, North American, Asian, and African Countries at an alarming rate.

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The novel coronavirus is a respiratory virus that spreads primarily through droplets of saliva or discharge from the nose generated when an infected person coughs or sneezes [3]. There is no known curing medicine nor vaccine to combat the COVID-19 pandemic. Standard recommendations by the WHO to prevent the spread of COVID-19 include frequent cleaning of hands using soap or alcohol-based sanitizer, covering the nose and mouth with a flexed elbow or disposable tissue when coughing and sneezing and avoiding close contact with anyone that has a fever and cough [4].

The COVID-19 virus may survive on surfaces for long periods with the right environmental conditions [4]. As a result, COVID-19 infection can spread from such contaminated surfaces to uninfected humans. Individuals will infect by contacting surfaces contaminated with the virus and touching their eyes, nose and mouth. We develop a compartmental mathematical model in combination with the effect of indirect transmission due to contaminated objects in the environment for the spread of COVID-19 dynamics. Understanding the transmission characteristics of the diseases in communities, regions, and countries leads to better approaches to decrease the transmission of these diseases [5]. The awareness of individuals for applying these preventive mechanisms vary from region to region and from country to country. In some places, protective measures are employed by volunteer individuals while in some other places, governments impose some kind of rules on the population to use strict physical distancing and wearing face masks in public places [6].

Mathematical models have become fundamental tools in understanding and analyzing the spread and control of infectious diseases, which clarifies variables and parameters to obtain conceptual results such as basic reproduction numbers [7]. Mathematical models and computer simulations are also useful for determining sensitivities to change in parameter values, and estimating key parameters from data that can contribute to identifying trends, make general forecasts, and estimate uncertainties [5]. Understanding the virus dynamics and host response is essential in formulating strategies for antiviral treatment, vaccination, and epidemiological control of COVID-19 [8]. The analysis from mathematical models may assist decision-makers to estimate the risk and the potential future growth of the disease in the population [6].

As epidemiological models play a fundamental role in the study of the dynamics of such COVID-19, various mathematical modelings have been used to investigate the transmission dynamics of the pandemic [9–17]. In [13], a generalized SEIR model was developed to analyze this pandemic. Based on the public data of the National Health Commission of China from Jan, 20th to Feb, 9th, 2020, they estimate epidemic parameters and make predictions on the possible ending time for five different regions.

Behavior change towards using preventive mechanisms by the population to protect themselves from an infectious disease is assumed to be dependent on the way that the disease is transmitted and its fatality [18]. Individuals who have awareness about the disease and decided to use preventive mechanisms have less susceptibility than those without awareness and demonstrating the usual risky behavior [6,19]. In this paper, we propose a SEIRDM mathematical model for the transmission dynamics of COVID-19 by introducing a behavior change function.

To get better predictions and to design and analyze various intervention strategies, one needs to estimate the model parameters from existing epidemiological data. It is most unusual to estimate parameter values from observed data in dynamical systems. But some authors estimate parameters based on the available data to determine the effects of various epidemiological factors on disease transmission and possible control strategy. Biegler and Grossmann [20] employed optimization techniques based on the seasonal data with the SIRS epidemic model to estimate the parameters of a generalized incidence rate function. The SIR model parameters were numerically estimated in [21] using the least squares method. In our case, we inferred or estimated the model parameters from observed data for different countries use a modified Bayesian approach combined with least square techniques. We also implement a forward sensitivity analysis of the model parameters to determine and identify the crucial factors for the spread of disease dynamics.

The paper is organized as follows: The mathematical model is formulated and described in Section 2. The qualitative analysis of the model by examining the equilibrium points and its stability analysis is studied in Section 3. Numerical simulations of the model by estimating the parameters are given in Section 4. The sensitivity analysis of the basic reproduction number for the model parameters is also discussed in this section. Conclusions and recommendations of the study are given in Section 5.

2. Model formulation

The model subdivides the human population into five disjoint compartments; Susceptible, Exposed, Infected, Recovered, Death; and one compartment, which is the contaminated material or surface. We consider the following basic assumptions to formulate the model.

1. The transmission dynamics of COVID-19 is similar to the SEIR model. We add the death compartment for which individuals die via the virus, in account with they may also die via the natural death.
2. We consider the contribution of the asymptomatic infectious individuals in the transmission dynamics of disease in the population.
3. The effect of indirect transmission through virus concentration in the environment due to shedding by infectious is considered.
4. We apply behavior change towards self-protective measures by the population to protect themselves from the virus.
We will then propose a mathematical model and analyze the effect of these factors to investigate in terms of their contribution to preventing the spread of disease.

The model state variables and parameters with their meanings are given in Tables 1 and 2 respectively. In the model, the force of infection $\lambda = \lambda(E, I, M)$, and the behavior change function $e = e(\lambda)$ are state dependent parameters, and they depend on the states $E, I$, and $M$. All other parameters (Greek letters) are instead constant numbers throughout our study, which do not depend on the states. We will estimate the values of all the parameters using available data in Section 4 by Matlab.

The total population at time $t$, denoted by $N(t)$, is given by

$$N(t) = S(t) + E(t) + I(t) + R(t) + D(t).$$

The flow diagram of the model is illustrated in Fig. 1.

Based on our assumptions and the flow diagram, it results in systems of the following non-linear differential equations:

$$\begin{align*}
\frac{dS}{dt} &= \Lambda - (\lambda + \alpha e + \mu)S, \\
\frac{dE}{dt} &= \alpha e S - (\theta + \mu + (1 - \eta)\lambda)E, \\
\frac{dI}{dt} &= \lambda S + (1 - \eta)\lambda E - (\mu + \delta + \gamma)I, \\
\frac{dR}{dt} &= \theta E + \gamma I - \mu R, \\
\frac{dD}{dt} &= \delta I - \mu D, \\
\frac{dM}{dt} &= \epsilon E + \xi I - \psi M.
\end{align*}$$

(1)

with non negative initial conditions $S(0) > 0$, $E(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$, $D(0) \geq 0$ and $M(0) \geq 0$.

The model in Eq. (1) looks decoupled, but as $\lambda$ depends on $M$, on $E$, and on $I$, the parameter (the force of infection) $\lambda$ couples the two subsystems because, through lambda, the state $M$ influences the epidemic evolution.

2.1. Model description

The susceptible population is increased by the recruitment of individuals at a rate $\Lambda$. All human individuals suffer from natural death at a constant rate of $\mu$. Susceptible individuals can acquire COVID-19 infection directly from human to human interaction and indirectly from the environment to human interaction. The incidence is after direct contact
Table 2

| Parameter | Biological description of the parameter |
|-----------|-----------------------------------------|
| \( \Lambda \) | Rate of recruitment to the susceptible individuals |
| \( \alpha \) | Rate of dissemination of information about the disease in the population |
| \( \beta_1 \) | Rate of disease transmission directly from humans |
| \( \beta_2 \) | Rate of disease transmission from the environment |
| \( \lambda \) | Force of infection (It is the probability of acquiring infection from an infected individual) |
| \( \lambda_0 \) | Threshold value of the force of infection for a population to start reacting swiftly |
| \( K \) | The pathogen concentration in the environment that yields 50% chance for a susceptible individual to catch the viral infection from the environment |
| \( \nu \) | Modification parameter |
| \( e \) | Behavior change function |
| \( \theta \) | Rate of recovery of the individuals from exposed class |
| \( \eta \) | The average effectiveness of existing self-preventive measures |
| \( \gamma \) | Rate of recovery of the individuals from virus in the infected class |
| \( \delta \) | Death rate due to the virus |
| \( \mu \) | Natural death rate of the individuals |
| \( \psi \) | Decay rate of the virus from the environment |
| \( \epsilon \) | Shedding rate of the virus from the exposed class to the environment |
| \( \xi \) | Shedding rate of the virus from the infected class to the environment |

with individuals from exposed and infected classes \((E, I)\) and contaminated environment \(M\) at the rates \(\beta_1\) and \(\beta_2\). The behavior change function \(e\) and the force of infection \(\lambda\) are given by

\[
\lambda = \frac{\beta_1 (E + \nu I)}{N} + \frac{\beta_2 M}{M + K} \quad \text{and} \quad e = \frac{\lambda^n}{\lambda_0^n + \lambda^n},
\]

where \(\beta_1\) is the rate of virus transmission directly from humans, \(\beta_2\) is the rate of virus transmission from the environment, \(K\) is the pathogen concentration in the environment that yields a 50% chance for a susceptible individual to catch the viral infection from the surface, \(\lambda_0\) is the value of the force of infection corresponding to the threshold infectivity in which individuals start reacting swiftly (the point at which the behavior change function changes its concavity), and \(n\) is a Hill coefficient that portrays the rate of reaction by the population. The modification parameter \(\nu \geq 1\) accounts for the relative infectiousness of individuals with COVID-19 symptoms, in comparison to those infected with the virus and with no symptoms. Individuals with virus symptoms are more infectious than those without symptoms because they have a higher viral load and there is a positive correlation between viral load and infectiousness [22].

At the beginning of an outbreak, individuals understand very little about the virus; there could be no reaction, and this can be related to the situation at the disease-free equilibrium such that \(e(\lambda) = e(0) = 0\). However, as the risk of the disease increases, individuals start to think about the type of measures they take to avoid all means of contracting the disease. These protection measures, if perfect, account for an increase in the values of \(e\) to unity. The order \(n\) of the function \(e(\lambda)\) is a Hill coefficient that portrays the rate of reaction by the population [18].

The rate of dissemination of information \(\alpha\) describes the awareness of individuals from the disease. Individuals acquire information through multiple ways, such as TV news, reports on a network, mouth-to-mouth communication, or even education. The information individuals gathered is an essential factor that impacts how individuals react to disease transmission and individuals make behavior changes to keep themselves from infection based on the information available to them during the pandemic [23].

Individuals leave the exposed class \(E\) by becoming symptomatic, at a rate \((1 - \eta)\lambda\) with the average effectiveness of existing self-preventive measures \(\eta \leq 1\), or recovered at rate \(\theta\). Individuals with the symptoms of COVID-19 disease dies due to the virus-induced death at a rate \(\delta\). The infected individuals also become recover at a rate of \(\gamma\). We assume that the recovered individuals \(R\) acquire partial immunity. The released COVID-19 from the exposed and infected individuals through coughing or sneezing landed on materials or surfaces around them and become infected at a rate \(\epsilon\) and \(\xi\), respectively [6,19]. The virus decays from the infected surfaces with the decay rate of \(\psi\).

3. Analysis of the model

In this section, we will see the qualitative analysis of the model Eq. (1) by examining the equilibrium points and its stability analysis.

3.1. Well-posedness

Let us begin understanding the dynamics of a model by investigating the behavior of its steady states. We first show that the model is well posed in a biologically feasible domain, and then proceed with stability analysis of the equilibrium points of the model.
Theorem 3.1.

1. There exists a unique solution to the system of Eqs. (1) in the region 
   \[ D = (S, E, I, R, D, M) \in \mathbb{R}^6. \]
2. If \( S(0) > 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, D(0) \geq 0 \) and \( M(0) \geq 0 \), then \( S(t) > 0, E(t) \geq 0, I(t) \geq 0, R(t) \geq 0, D(t) \geq 0 \) and \( M(t) \geq 0 \) for all \( t \geq 0 \).
3. The solution trajectories of the model Eqs. (1) evolve in a positive invariant region
   \[ \Omega = \{(S, E, I, R, D) \in \mathbb{R}^5_+ : 0 \leq S + E + I + R + D \leq \frac{\Lambda}{\mu}, 0 \leq M \leq \frac{(\epsilon + \xi)\Lambda}{\mu \psi}\}. \]

Proof. The Well-Posedness of the Model is proved as follows:

1. All the functions on the right hand side of Eq. (1) are \( C^1 \) on \( \mathbb{R}^5 \). Thus, by the Picard–Lindelöf theorem [24], Eqs. (1) has a unique solution.
2. The positivity of model state variables are proved based on proposition A.1 in [25]. Let the model Eqs. (1) be written in the form \( x' = F(x, t) \), where \( x = (S, E, I, R, D, M) \), and \( F = \left( \frac{dS}{dt}, \frac{dE}{dt}, \frac{dI}{dt}, \frac{dR}{dt}, \frac{dD}{dt}, \frac{dM}{dt} \right) \). The functions \( F(x, t) \) on the right hand side of Eqs. (1) have the property of \( F(S, E, I, R, D, M, t) \geq 0 \) whenever \( x \in [0, \infty)^6 \), \( x_j = 0, \ t \geq 0 \). Here our \( x_j \)'s are \( x_1 = S, \ x_2 = E, \ x_3 = I, \ x_4 = R, \ x_5 = D \) and \( x_6 = M \). By Theorem 3.1.1, there exists a unique solution for the model Eqs. (1). Thus, it follows from the Proposition that \( x(t) \in [0, \infty)^6 \) for all \( t \geq t_0 \geq 0 \) whenever \( x(t_0) \geq 0 \).
3. The change of total population \( N(t) = S(t) + E(t) + I(t) + R(t) + D(t) \) at time \( t \) is governed by \( N'(t) = S'(t) + E'(t) + I'(t) + R'(t) + D'(t) \). That is:
   \[ \frac{dN}{dt} = \Lambda - \mu N. \]
   The solution for this linear first order ode is \( N(t) = N(0) e^{-\mu t} + \frac{\Lambda}{\mu} (1 - e^{-\mu t}) \). Thus, for the initial data \( 0 \leq N(0) \leq \frac{\Lambda}{\mu} \), we obtain
   \[ 0 \leq N(t) \leq \frac{\Lambda}{\mu}. \]
   Moreover, for the environmental variable \( M \), we have
   \[ \frac{dM}{dt} = \epsilon E + \xi I - \psi M \leq (\epsilon + \xi) \frac{\Lambda}{\mu} - \psi M. \]
   As \( E(t) \) and \( I(t) \) are less than \( \frac{\Lambda}{\mu} \). Using the same procedure or by applying the Gronwall inequality, for \( 0 \leq M(0) \leq \frac{(\epsilon + \xi)\Lambda}{\mu \psi} \), we obtain:
   \[ 0 \leq M(t) \leq \frac{(\epsilon + \xi)\Lambda}{\mu \psi}. \]
   If \( x_0 \) is a point in \( D \), then the solution of the initial value problem (1), exists for all times \( t \geq 0 \) by 3.1.1. By the result of Theorem 3.1.2, the solution lies in \( D \), for all \( t \geq 0 \). Hence the region \( \Omega \) is positive invariant.

Then we will analyze the model quantitative behaviors in the domain \( \Omega \). \( \Box \)

3.2. Local stability of disease-free equilibrium

The equilibrium solutions of the model are obtained by setting the right-hand side of Eq. (1) equal to zero:

\[
\begin{align*}
    \Lambda - (\lambda + \alpha e + \mu)S &= 0, \\
    \alpha e S - (\theta + \mu + (1 - \eta)\lambda)E &= 0, \\
    \lambda S + (1 - \eta)\lambda E - (\mu + \delta + \gamma)I &= 0, \\
    \theta E + \gamma I - \mu R &= 0, \\
    \gamma I - \mu D &= 0, \\
    \epsilon E + \xi I - \psi M &= 0.
\end{align*}
\]

(2)

The disease-free equilibrium point of our model is obtained by setting the disease state variables \( E = 0 \) and \( I = 0 \). If \( E = 0 \) and \( I = 0 \), then \( R = 0 \) and \( D = 0 \). It is then denoted and given by:

\[ \xi_0 = \left( \frac{\Lambda}{\mu}, 0, 0, 0, 0 \right). \]

Now let us calculate the basic reproduction number, which is denoted by \( R_0 \), and defined as the average number of secondary infections produced by a single infected individual in a completely susceptible population. Using the next
generation matrix method [26], we calculate the basic reproduction number as follows. From the model equation (1), using the notation \( X = (E, I, M) \), we have the vector functions:

\[
F(x) = \begin{bmatrix} \alpha e S \\ \lambda S \\ 0 \end{bmatrix} \quad \text{and} \quad V(x) = \begin{bmatrix} (\theta + \mu + (1-\eta)\lambda_1)E \\ (\mu + \delta + \gamma)I - (1-\eta)\lambda_1E \\ \psi M - (\epsilon E + \xi I) \end{bmatrix},
\]

representing the appearance of new infections, and the transfer of individuals in to and out of the infected compartments, respectively. The Jacobian matrices of \( F(x) \) and \( V(x) \) are, respectively

\[
F = DF(e_0) = \begin{bmatrix} \frac{\partial F_1}{\partial e_0} & \frac{\partial F_1}{\partial \theta} & \frac{\partial F_1}{\partial \mu} \\ \frac{\partial F_2}{\partial e_0} & \frac{\partial F_2}{\partial \theta} & \frac{\partial F_2}{\partial \mu} \\ \frac{\partial F_3}{\partial e_0} & \frac{\partial F_3}{\partial \theta} & \frac{\partial F_3}{\partial \mu} \end{bmatrix} \quad \text{and} \quad V = Dv(e_0) = \begin{bmatrix} p_1 & 0 & 0 \\ 0 & p_2 & 0 \\ -\epsilon & -\xi & \psi \end{bmatrix},
\]

where \( p_1 = \mu + \theta, p_2 = \mu + \delta + \gamma \) and the entries of \( F \) and \( V \) are obtained by \( \frac{\partial F_i(e_0)}{\partial e_0} \) and \( \frac{\partial V_i(e_0)}{\partial e_0} \) respectively. It is easy to calculate the inverse of \( V \) and given by

\[
V^{-1} = \begin{bmatrix} \frac{1}{p_1} & 0 & 0 \\ 0 & \frac{1}{p_2} & 0 \\ \frac{\epsilon}{p_1} & \frac{\xi}{p_2} & \frac{1}{\psi} \end{bmatrix}.
\]

The next-generation matrix \( FV^{-1} \) is

\[
FV^{-1} = \begin{bmatrix} \frac{\alpha_1}{\lambda_0 p_1} + \frac{\alpha_2 \lambda_0}{\lambda_0 p_1} & \frac{\alpha_1}{\lambda_0 p_2} + \frac{\alpha_2 \lambda_0}{\lambda_0 p_2} & \frac{\alpha_1}{\lambda_0 p_2} + \frac{\alpha_2 \lambda_0}{\lambda_0 p_2} \\ \frac{\beta_1}{p_1} + \frac{\beta_2 \lambda_0}{\lambda_0 p_1} & \frac{\beta_1}{p_2} + \frac{\beta_2 \lambda_0}{\lambda_0 p_2} & \frac{\beta_1}{p_2} + \frac{\beta_2 \lambda_0}{\lambda_0 p_2} \\ 0 & 0 & 0 \end{bmatrix},
\]

and its eigenvalues are \( \lambda_1 = 0, \lambda_2 = 0 \) and \( \lambda_3 = \frac{\Delta \beta_2 (\alpha \epsilon p_2 + \lambda_0 \xi p_1) + \beta_1 K \mu \psi (\alpha p_2 + \lambda_0 \nu p_1)}{\lambda_0 K \mu \nu p_1 p_2} \). The spectral radius (the largest eigenvalue) of the next generation matrix is the basic reproduction number of the model. Hence we have the following result.

**Theorem 3.2.** The basic reproduction number of the model equation (1) is given by

\[
R_0 = \frac{\Delta \beta_2 (\alpha \epsilon p_2 + \lambda_0 \xi p_1) + \beta_1 K \mu \psi (\alpha p_2 + \lambda_0 \nu p_1)}{\lambda_0 K \mu \nu p_1 p_2}.
\]

**Remark 3.3.** In general, if \( R_0 > 1 \), then on average, the number of new infections resulting from one infected individual is greater than one. Thus, COVID-19 infections will persist in the populations. If \( R_0 < 1 \), then on average, the number of new infections generated by one infected individual is less than one. This implies that the infections will eventually disappear from the populations. This threshold can as well be used to depict parameters that are most important during the infection.

The local stability analysis of the equilibrium point is analyzed using linearization. For \( n = 1 \), the expression \( \alpha e \) is simplified as \( \alpha e = \frac{\beta_1}{\beta_2} \), where

\[
B_1 = \beta_1 (E + v I)(K + M) + \beta_2 MN. \quad \text{and} \quad B_2 = \lambda_0 N(K + M) + \beta_1 (E + v I)(K + M) + \beta_2 MN.
\]

The Jacobian matrix of the model equation (1) is

\[
J(S, E, I, R, D, M) = \begin{pmatrix} A_1 & A_2 & A_3 & 0 & 0 & A_4 \\ A_5 & A_6 & A_7 & 0 & 0 & A_8 \\ A_9 & A_{10} & A_{11} & 0 & 0 & A_{12} \\ 0 & 0 & \gamma & -\mu & 0 & 0 \\ 0 & 0 & \delta & 0 & -\mu & 0 \\ 0 & \epsilon & \xi & 0 & 0 & -\psi \end{pmatrix}
\]

where,

\[
A_1 = -\left[ \frac{\beta_1 (E + v I)(E + I + R + D)}{N^2} + \frac{\beta_2 M}{K + M} \right] + \frac{C_1}{B_2^2} + \mu,
\]

\[
A_2 = -\left[ \frac{\beta_1 S (S + I + R + D)}{N^2} + \frac{C_4}{B_2^2} \right],
\]

\[
A_3 = -\left[ \frac{\beta_1 v S (S + E + R + D)}{N^2} + \frac{C_7}{B_2^2} \right],
\]

\[
A_4 = \begin{pmatrix} 0 \\ \frac{\beta_1 v (S + I + R + D) + C_7}{B_2^2} \\ \frac{\beta_1 v S (S + E + R + D) + C_7}{B_2^2} \\ 0 \\ 0 \\ 0 \end{pmatrix}.
\]
By expanding the characteristic equation

The characteristic equation of $J$

Proof.
The disease-free equilibrium point $\varepsilon_0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 3.4. The disease-free equilibrium point $\varepsilon_0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. The locally asymptotical stability of $\varepsilon_0$ is analyzed using the sign of the eigenvalues of a Jacobian matrix at the disease-free equilibrium point $\varepsilon_0$. Substituting the equilibrium point $\varepsilon_0$ with $N_0 = \frac{3}{\mu}$ (at the disease-free equilibrium, the total population $N$ is equal to the total susceptible $S$ population) in the matrix equation (3), we have:

$$J(\varepsilon_0) =$$

$$= \begin{pmatrix}
-\mu & -\beta_1 \left(1 + \frac{\alpha}{\lambda_0}\right) & -\beta_1 \left(1 + \frac{\alpha}{\lambda_0}\right) & 0 & 0 & -\frac{\beta_2 \Lambda \left(1 + \frac{\alpha}{\lambda_0}\right)}{\mu \lambda_0} \\
0 & \frac{a_\beta p_1}{\alpha p_0} - p_1 & \frac{a_\beta p_1}{\alpha p_0} & 0 & 0 & \frac{\beta_2 \Lambda}{\mu K \lambda_0} \\
0 & \beta_1 & \beta_1 v - p_2 & 0 & 0 & \frac{\beta_2 \Lambda}{\mu K \lambda_0} \\
0 & 0 & \theta & -\mu & 0 & 0 \\
0 & 0 & 0 & \xi & 0 & 0 \\
0 & 0 & 0 & 0 & -\psi
\end{pmatrix}$$

By expanding the characteristic equation $|\lambda I - J(\varepsilon_0)| = 0$ with the first, fourth and fifth columns, we obtain three eigenvalues $\lambda_{1,2,3} = -\mu$. We calculate the remaining three eigenvalues from the reduced matrix

$$J_3 =$$

$$= \begin{pmatrix}
\frac{a_\beta p_1}{\alpha p_0} - p_1 & \frac{a_\beta p_1}{\alpha p_0} & \frac{a_\beta p_2}{\mu K \lambda_0} & \\
\beta_1 & \beta_1 v - p_2 & \frac{\beta_2 \Lambda}{\mu K \lambda_0} & \\
\epsilon & \xi & -\psi
\end{pmatrix}$$

The characteristic equation of $J_3$ is a third degree polynomial which is given by:

$$Q(\lambda) = \lambda^3 + D_1 \lambda^2 + D_2 \lambda + D_3,$$

where

$$D_1 = p_1 + \psi + p_2 - \beta_1(v + \frac{\alpha}{\lambda_0}) = p_1 + \psi + \frac{\beta_2 \Lambda (\alpha \epsilon p_2 + \lambda_0 \xi p_1)}{\mu K \lambda_0 \psi p_1} + p_2(1 - R_0),$$

$$D_2 = \psi(p_1 + p_2) + p_1 p_2 - \left(\beta_1 \left(\psi(v + p_1) + \frac{\alpha}{\lambda_0} (\psi + p_2)\right) + \frac{\beta_2 \Lambda}{\mu K} (\xi + \frac{\alpha \epsilon}{\lambda_0})\right),$$

and

$$D_3 = \psi p_1 p_2 - \left[p_1 \psi \beta_1 v + \frac{\beta_2 \Lambda \xi}{\mu K \lambda_0} + \frac{\alpha \beta_1 \psi p_2}{\lambda_0} + \frac{\alpha \beta_2 \Lambda \epsilon p_2}{\mu K \lambda_0}\right] = \psi p_1 p_2 (1 - R_0).$$
The sign of $D_1, D_2$ and $D_3$ is positive if $R_0 < 1$. It is also true that $D_1D_2 > D_3$. Using Routh–Hurwitz stability criterion the disease-free equilibrium point $\epsilon_0$ is stable if $R_0 < 1$ and unstable if $R_0 > 1$. □

3.3. Global stability of disease-free equilibrium

Let us rewrite our model system (1) as

\[
\begin{aligned}
\frac{dX}{dt} &= F(X, Z) \\
\frac{dZ}{dt} &= G(X, Z), \quad G(X, 0) = 0.
\end{aligned}
\]

where $X = (S, R, D)$ and $Z = (E, I, M)$, with the components of $X \in \mathbb{R}^2_+$ denoting the number of uninfected individuals and $Z \in \mathbb{R}^4_+$ denoting the number of infected ones [27]. The disease-free equilibrium is denoted now as

\[U_0 = (X_0, 0), \quad \text{where,} \quad X_0 = \left( \frac{A}{\mu}, 0, 0 \right).\]

The conditions $(H_1)$ and $(H_2)$ below must be met to guarantee global asymptotically stability:

$(H_1)$ For $\frac{dX}{dt} = F(X, 0)$, $U_0$ is globally asymptotically stable;

$(H_2)$ $G(X, Z) = AZ - \hat{G}(X, Z), \quad \hat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$, where $A = D_2G(U_0, 0)$ is a Metzler matrix (the off diagonal elements of $A$ are non-negative) and $\Omega$ is the region where the model makes biological sense.

**Theorem 3.5.** The disease-free equilibrium point $U_0 = (X_0, 0)$ is a globally asymptotically stable equilibrium of (1) if $R_0 < 1$ and the assumptions $(H_1)$ and $(H_2)$ are satisfied.

**Proof.** We have

\[
\frac{dX}{dt} = F(X, Z) = \begin{bmatrix}
A - (\lambda + \alpha e + \mu)S \\
\theta E + \gamma I - \mu R \\
\delta I - \mu D
\end{bmatrix}
\]

\[
F(X, 0) = \begin{bmatrix}
A - \mu S \\
0 \\
0
\end{bmatrix}
\]

Therefore,

\[
A = D_2G(U_0, 0) = \begin{bmatrix}
\frac{\alpha \beta_1}{\lambda_0} - p_1 & \frac{\alpha \beta_1 v}{\lambda_0} & \frac{\alpha \beta_1 \lambda}{\mu K \lambda_0} \\
\beta_1 & \beta_1 v - p_2 & \frac{\beta_2 \lambda}{\mu} \\
\epsilon & \xi & -\psi
\end{bmatrix}
\]

which is a Metzler Matrix.

Here $\hat{G}(X, Z) = AZ - G(X, Z)$, and so,

\[
\hat{G}(X, Z) = \begin{bmatrix}
\hat{G}_1(X, Z) \\
\hat{G}_2(X, Z) \\
\hat{G}_3(X, Z)
\end{bmatrix} = \begin{bmatrix}
\frac{\alpha}{\lambda_0} \left( \beta_1(E + vI) + \frac{\beta_2 \lambda M}{\mu} \right) + \alpha eS + (1 - \eta)\lambda E \\
\beta_1(E + vI) + \frac{\beta_2 \lambda}{\mu}M - \lambda(S + (1 - \eta)E) \\
0
\end{bmatrix}.
\]

Since $N \leq \frac{\lambda}{\mu}$ and $(1 - \eta) \leq 1$,

\[
\hat{G}_2 = \beta_1(E + vI) + \frac{\beta_2 \lambda}{K \mu}M - \lambda(S + (1 - \eta)E) \geq \beta_1(E + vI) + N\frac{\beta_2}{K}M - \lambda(S + E + I + R + D).
\]

Taking $N$ as a common factor, implying

\[
\hat{G}_2 \geq N \left( \frac{\beta_1(E + vI)}{N} + \frac{\beta_2}{K}M - \lambda \right) \geq 0.
\]

It follows that $\hat{G}_1(X, Z) \geq 0, \quad \hat{G}_2(X, Z) \geq 0$ and $\hat{G}_3(X, Z) = 0$. Thus, $\hat{G} \geq 0$. Conditions $(H_1)$ and $(H_2)$ are satisfied, and we conclude that $U_0$ is globally asymptotically stable for $R_0 < 1$. □
4. Parameter estimation and numerical simulations

In this section, we discuss and estimate the parameter choices and the numerical solutions of the model Eqs. (1). We outline the initial conditions and fit the existing WHO data with the model for that choice of parameters.

4.1. Parameter estimation

The systems of model Eqs. (1) can be expressed as a dynamical system of the form:

\[
\frac{dX}{dt} = F(t, X, \Phi), \quad X(0) = X_0,
\]

where \( t \) is the independent variable (time), \( X \) is the state vector of the system, \( \frac{dX}{dt} = [\frac{dx_1}{dt}, \ldots, \frac{dx_{N'}}{dt}]^T \), \( X = [x_1, \ldots, x_{N'}] \), \( F = [f_1, \ldots, f_{N'}] \) and \( N' \) is the number of compartments in the population. \( \Phi = [\phi_1, \ldots, \phi_p] \) are \( p \) unknown parameters of the system and \( X_0 \) are the initial values [28].

In order to estimate the unknown parameters \( \Phi \), the state variable \( X(t) \) is observed at \( L \) time instants \( t_1, \ldots, t_r \), so that we have

\[ Y(t_i) = X(t_i) + E_i, \quad i = 1, \ldots, T, \]

where \( Y(t_i) \) is the observed values of the state variables at time instant \( t_i \) and \( \{E_i\}_{i=1}^T \) are the difference between the observed value \( y_i \) and the corresponding fitted value \( x_i \), i.e., \( E_i = y_i - x_i \). The objective is to determine appropriate parameter values so that the sum of squared errors between the outputs of the estimated model \( \{X(t)\} \) and the measured data \( \{Y(t)\} \) should be minimized.

We wish to find the vector of least-square estimators, \( \Phi \), that minimizes

\[ \sum_{i=1}^{l} E^2_i = \sum_{i=1}^{l} (y_i - x_i)^2. \]  \[ (5) \]

To find the values of parameters \( \Phi \) that minimizes Eq. (5), various methods have been used for handling this problem. The first technique is to differentiate \( \sum_{i=1}^{l} E^2_i \) with respect to each \( \Phi \) and set the results equal to zero to obtain a system of equations that can be solved simultaneously for the \( \Phi \)'s [29].

To estimate the model parameters, we use a two-step approach

a. The first approach requires solving the ordinary differential equation (1). As using analytic methods are difficult, we use the numerical techniques to solve ode’s like Runge–Kutta methods.

b. The second approach is finding the optimization algorithm to update the parameters based on Eq. (5)

The process of updating the parameters continues until no significant improvement (convergence) in the objective function is observed. We use a type of Bayesian technique to estimate the unknown parameters and to solve the optimization algorithm.

In this parameter estimation procedure, we modified and combined the Bayesian estimation techniques with methods of least square techniques. The acceptance and rejection procedure in the Metropolis–Hastings (MH) algorithm is replaced by comparing the minimum of the sum squared errors between the proposed parameter and the previously assigned parameter. \( \Phi = [\phi_1, \ldots, \phi_p] \) is a vector of parameters, where, \( p \) is the number of parameters to be estimated. In our case, the number of parameters is \( p = 15 \). We take one parameter at a time and consider the other parameters held constant in the objective function, i.e

\[ \sum_{i=1}^{l} E^2_i(\phi_j \mid \phi_1, \ldots, \phi_{j-1}, \phi_{j+1}, \ldots, \phi_p) = \sum_{i=1}^{l} (y_i - x_i)^2 \]  \[ (6) \]

This is a kind of Gibbs Sampling technique for parameter estimation [30]. By combining these parameter estimation techniques we estimate all vector of parameters until convergence.

First, we have to initialize the parameters in their parameter space and propose the next parameter value by sampling from the proposal density. The proposal density we assign \( \phi_j^{prop} = \phi_i + U[\phi_1 - c_1, \phi_1 + c_1] \), where \( c_1 \) is tuning value which is a small number and help us to move the parameter \( \phi_i \) up and down through the parameter estimation process. A rough outline of the algorithm is given in Algorithm 1.

Convergence analysis of the parameter estimation is assessed by line plots of separate parameters.
Algorithm 1 MH algorithm with Least square

Input: $B$ (number of iteration), $\phi^0$ (initial value for parameters), $Y$ (Observed data)

Step 1. For $b = 0, 1, \ldots, B$.

Step 2. Solve ode's for Equation (1) at $\phi^l_i$ and compute the sum of squared errors for Equation (6).

Step 3. Select new parameter $\phi^{\text{prop}}_i \sim q(\phi^{\text{prop}}_i | \phi^l_i)$.

Step 4. Solve Ode's at $\phi^{\text{prop}}_i$ and Compute Equation (6).

Step 5. If the sum of squared errors in Step 4 is less than in Step 2
   
   $\phi^{l+1}_i = \phi^{\text{prop}}_i$.

   else,
   
   $\phi^{l+1}_i = \phi^l_i$.

Step 6. $l = l + 1$.

Table 3

| Parameter | Estimated value |
|-----------|-----------------|
|           | China | Italy | Ethiopia | Brazil | South Africa |
| $\alpha$  | 0.9865 | 0.8642 | 0.6808   | 0.6594 | 0.2459       |
| $\mu$     | 0.0078 | 0.0122 | 0.0477   | 0.0022 | 0.0233       |
| $\eta$    | 0.9998 | 0.9999 | 0.0685   | 0.0962 | 0.0725       |
| $\delta$  | 0.0039 | 0.0105 | 0.0023   | 0.0443 | 0.0018       |
| $\gamma$  | 0.0326 | 0.0200 | 0.0650   | 0.0461 | 0.0060       |
| $\epsilon$| 0.0075 | 0.0755 | 0.0210   | 0.0155 | 0.2355       |
| $\xi$     | 0.0323 | 0.0151 | 0.0064   | 0.0081 | 0.0002       |
| $\psi$    | 0.7862 | 0.7224 | 0.9787   | 0.8808 | 0.5788       |
| $\beta_1$ | 0.0081 | 0.0328 | 0.1458   | 0.0116 | 0.0407       |
| $\beta_2$ | 0.0089 | 0.0023 | 0.0032   | 0.0003 | 0.0020       |
| $\lambda_0$| 0.1062 | 0.071756 | 0.4599 | 0.0541 | 0.0437       |
| $\Lambda$ | 437.9314 | 674.8087 | 590.5512 | 842.5115 | 874.1602     |
| $\theta$  | 105.9383 | 1195.8673 | 19595.5597 | 712.7638 | 1128.3940    |
| $\nu$     | 1.0023 | 1.0015 | 1.0250   | 4.3520 | 1.8138       |

4.2. Numerical results and discussion

The parameters must be estimated and assigned a value to make the model operable. In this paper, we use total active cases, totally recovered and total death data extracted from WHO situation reports 1 – 192, and worldometer [31], with the daily data from January 21, 2020 to August 16, 2020. We use this data in parameter estimation for countries such as China, Italy, Brazil, South Africa, and Ethiopia. We denote the observed data $Y = [I, R, D]$ with having different length of time $T$ for those countries. The goal is to find the value of the parameters which minimize the squared errors between the model predictions and the observed data. We also use different initial states of the dynamics for each of the countries. We take initial values for Infected ($I_0$), Recovered ($R_0$) and Death ($D_0$) cases reasonably the same as the observed data $Y_0$. We assume that about 80% of the disease is asymptomatic that helps us to outline the initial value for Exposed ($E_0$). By an initial guess of the parameters $\phi^0$, we use $B = 10,000$ number of iterations for estimation in the MH algorithm.

Based on the available data and the prediction of the proposed model, we compute the error terms for the three state compartments. We then update the parameter based on the minimization of sum squared differences between measurements and the model predictions.

The convergence analysis of some estimated parameters is given in Fig. 2 and our parameter estimation algorithm seems to converge at 2000 iterations. We take the values at the final iteration as the estimated parameter value for the proposed model. The estimated parameters for the countries China, Italy, Ethiopia, South Africa, and Brazil are given in Table 3.

From the estimated parameter values in Table 3, the natural death rate $\mu$ is relatively higher in Ethiopia and South Africa, while the induced death rate due to COVID-19 $\delta$ is relatively high in Italy. The contact rates of the virus from the environment is relatively high in China, and the virus decay rate from the infected objects is relatively small in Brazil. From the estimated parameters, one can find the number of days required for the incubation and recovery periods.

Fig. 3 shows the fitted model with the observed data for South Africa. The estimated parameters for the infected compartment is well fitted and approximated compared with the observed data. It is also shown that the recovered and death compartment has relatively small under predictions.

The Figures in 4 shows the fitted model with the observed data for Brazil. We observe that the estimated parameters for the infected and recovered compartments are well fitted and approximated compared with the observed data. The death compartment has relatively small over predictions.
Fig. 2. Convergence analysis of a sample of parameters for the COVID-19 induced death rate $\delta$, recovery rate $\gamma$ and the contact rates $\beta_1$, $\beta_2$.

Fig. 3. Numerical results of the fitted and observed values of Infected, Recovered and Death cases for South Africa.

Fig. 4. Numerical results of the fitted and observed values of Infected, Recovered and Death cases for Brazil.

The Fig. 5 shows the fitted model with the observed values for Ethiopian data. We see that the estimated parameters for the infected and death compartments are well fitted and approximated compared with the observed data. When we estimate parameters with the existing Ethiopian COVID-19 data, the recovered compartment have relatively small under predictions.

4.3. Sensitivity of the basic reproduction number

Sensitivity analysis is used to determine how sensitive a model is to changes in the value of the parameters and to changes in the structure of the model [32,33]. In this paper, we focus on parameter sensitivity. Sensitivity analysis is a useful tool in model building as well as in model evaluation by showing how the model behavior responds to changes in parameter values [34].
The numerical results of the fitted and observed values of Infected, Recovered and Death cases for Ethiopia.

The visual representation of the elasticity indices of $R_0$ with respect to the estimated parameters of countries China, Ethiopia and Brazil cases.

**Definition 4.1.** The Sensitivity and elasticity indices of the basic reproduction ratio, $R_0$, with respect to model parameter $p$ are respectively given by $S_p = \frac{\partial R_0}{\partial p}$ and $e_p = \frac{\partial R_0}{\partial p} \frac{p}{R_0}$. That is, the elasticity indices is given by $e_p = S_p \frac{p}{R_0}$ [34,35].

The graph in Fig. 6 shows that in all countries the infection rates $\beta_1$, $\beta_2$, the recruitment rate $\Lambda$, the modification parameter $\nu$ and shedding rate of the virus from the infected class to the environment $\xi$ has the highest elasticity indices with COVID-19 being positively correlated to $R_0$. An increase in these parameters will increase the spread of the COVID-19 pandemic. The recovery rates $\theta, \gamma$, the natural and virus-induced death rates $\mu, \delta$, the threshold value of the force of infection $\lambda_0$, the pathogen concentration in the environment $K$ and the virus decay rate in the environment $\psi$ have a negative correlation of $R_0$ implying the virus decreases with an increase of these parameters.

In Fig. 7(a), we observe that for the basic reproduction number $R_0 < 1$, all solutions curve goes to the disease-free equilibrium point. These indicate that the disease-free equilibrium point is locally and globally asymptotically stable for the values of $R_0 < 1$. In Fig. 7(b) with $R_0 < 1$ and different initial conditions for the susceptible population, all trajectory goes to the equilibrium point $\Lambda^*$, which indicates its stability.

In Fig. 8, we observe that for the basic reproduction number $R_0 > 1$, all solutions curves goes away from the disease-free equilibrium point. These indicate that the disease-free equilibrium point is unstable for the values of $R_0 > 1$, and the solutions will go to the endemic equilibrium point.

### 4.4. Predictions

One of the most crucial applications of the dynamical system is to predict the future spread of the disease in the population. As the number of infected individuals increases, the new predictions will help the government to prepare medical facilities and health care workers for the patients. Here we predicted the number of active infections, recoveries and deaths for the countries Ethiopia, Brazil and South Africa for the next 45 days. In Fig. 9, the fitted areas are similar to Figs. 3–5 while the prediction areas show the possible values of cases (active, recovered and death) predicted until September 30, 2020.

According to our prediction, the total number of active infected populations of Ethiopia (Fig. 9(a)) on September 30, 2020, will be around 55,000, the number of recovered individuals will surpass 60,000, and the total deaths will reach above 1000. The number of infected populations will also stabilize and the number of recovered individuals will overshadow those of the infected ones thereafter on September 25, 2020.
Fig. 7. The trajectories of state variables for $R_0 = 0.2552$, which is less than one.

Fig. 8. Trajectories of state variables for $R_0 = 8.3636$, which is greater than one.

Fig. 9. Predictions of active cases, recovered cases and death Ethiopia, Brazil and South Africa on September 30, 2020.

When we see the predictions of Brazil (Fig. 9(b)), the number of active infections and deaths is stable, and the number of recovered classes increases. The number of deaths will reach 100,000 by the last of September 2020. In Fig. 9(c), the
total number of active infections will decrease (after reaching the peak of the curve around 98,000 active infections) on August 15, 2020; those of recovered and death class will also stabilize on that day for South Africa.

5. Conclusion

In this paper, we developed a Susceptible–Exposed–Infected–Recovered-Death Mathematical model with the addition of environmental infection with the virus for the transmission dynamics of the COVID-19 pandemic. The well-posedness of the model is examined by proving the existence, positivity, and boundedness of the solutions.

The disease-free equilibrium point is computed. We performed a local and global stability analysis of the disease free equilibrium point, by finding the basic reproduction number of the model. The result shows that the disease-free equilibrium point is locally as well as globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

The parameters are estimated using the combination of least square and Bayesian estimation techniques. We develop Matlab codes for the combination of the MH algorithm with the least square. The estimation results of the model parameters vary from country to country as the case of the spread of the virus varies accordingly. The sensitivity analysis on the countries show the infection rates $\beta_1$ (human to human) and $\beta_2$ (from the infected surfaces or environment) have high positive impacts on the spread of COVID-19. The threshold value of the force of infection for a population $\lambda_0$, the recovery rates $\theta$, $\gamma$ and the virus decay rate in the environment $\psi$ hurt the spread of the virus. We have also observed that high numbers of people with knowledge about the virus, that are practicing the prescribed self-protective measures can slow down the outbreak.

We recommend for individuals to develop their behavioral changes about the pandemic by following WHO recommendations such as using face masks, practicing social distancing, washing their hands with soap and an alcohol-based sanitizer, and disinfecting the surface. These mechanisms will decline and stop the spread of the virus from the contaminated environment to uninfected individuals and from infected individuals to the surrounding. It is also essential to create awareness and disseminate information for societies to keep themselves from the virus, which can reduce the pandemic threshold of the infections.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Li H, Liu S-M, Yu X-H, Tang S-L, Tang C-K. Coronavirus disease 2019 (COVID-19): current status and future perspective. Int J Antimicrob Agents 2020;105951.
[2] Organization WH, et al. Coronavirus disease 2019 (COVID-19): situation report, 153, 2020.
[3] World Health Organization, WHO Director- general’s opening remarks at the media briefing on COVID-19—11 March 2020. 2020, Available at: https://www.who.int/dg/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-covid-19---11-march-2020.
[4] World Health Organization, et al. Key messages and actions for COVID-19 prevention and control in schools. 2020.
[5] Hethcote HW. The mathematics of infectious diseases. SIAM Rev 2000;42(4):599–653.
[6] Kassa SM, Ngajarah JB, Terefe YA. Analysis of the mitigation strategies for COVID-19: from mathematical modelling perspective. Chaos Solitons Fractals 2020;109968.
[7] Ouakhoumi Noutchie SC, Kito Kwuimy CA, Tewa JJ, Nyabadza F, Bildik N. Computational and theoretical analysis of human diseases associated with infectious pathogens, 2015.
[8] Chen Y, Li L. SARS-CoV-2: virus dynamics and host response. Lancet Infect Dis 2020;20(5):515–6.
[9] Cao J, Jiang X, Zhao B, et al. Mathematical modeling and epidemic prediction of COVID-19 and its significance to epidemic prevention and control measures. J Biomed Res Innov 2020;1(1):1–19.
[10] Jewell NP, Lewnard JA, Jewell BL. Predictive mathematical models of the COVID-19 pandemic: underlying principles and value of projections. JAMA 2020;323(19):1893–4.
[11] d’Onofrio A, Manfredi P, Salinelli E. Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases. Theor Popul Biol 2007;71(3):301–17.
[12] Sameni R. Mathematical modeling of epidemic diseases; a case study of the COVID-19 coronavirus. 2020, arXiv preprint arXiv:2003.11371.
[13] Peng L, Yang W, Zhang D, Zhuge C, Hong L. Epidemic analysis of COVID-19 in China by dynamical modeling. 2020, arXiv preprint arXiv: 2002.06563.
[14] Giordano G, Blanchini F, Bruno R, Colaneri P, Di Filippo A, Di Matteo A, et al. Modelling the COVID–19 epidemic and implementation of population-wide interventions in Italy. Nature Med 2020;1–6.
[15] Tuite AR, Fisman DN, Greer AL. Mathematical modelling of COVID-19 transmission and mitigation strategies in the population of ontario, Canada. CMAJ 2020;192(19):E497–505.
[16] Lemecha Obsu L, Feyissa Balcha S. Optimal control strategies for the transmission risk of COVID-19. J Biol Dyn 2020;14(1):590–607.
[17] Mamo DK. Model the transmission dynamics of COVID-19 propagation with public health intervention. Res Appl Math August 2020, 100123;7.
[18] Kassa SM, Ouhinou A. Epidemiological models with prevalence dependent endogenous self-protection measure. Math Biosci 2011;229(1):41–9.
[19] BERGE T, Chapwanya M, Lubuma JM-S, Terefe YA. A mathematical model for ebola epidemic with self-protection measures. Math Biosci 2015;271(3):301–17.
[20] Jewell NP, Lewnard JA, Jewell BL. Predictive mathematical models of the COVID-19 pandemic: underlying principles and value of projections. JAMA 2020;323(19):1893–4.
[21] Cao J, Jiang X, Zhao B, et al. Mathematical modeling and epidemic prediction of COVID-19 and its significance to epidemic prevention and control measures. J Biomed Res Innov 2020;1(1):1–19.
[22] Jewell NP, Lewnard JA, Jewell BL. Predictive mathematical models of the COVID-19 pandemic: underlying principles and value of projections. JAMA 2020;323(19):1893–4.
[23] d’Onofrio A, Manfredi P, Salinelli E. Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases. Theor Popul Biol 2007;71(3):301–17.
[24] Sameni R. Mathematical modeling of epidemic diseases; a case study of the COVID-19 coronavirus. 2020, arXiv preprint arXiv:2003.11371.
[25] Peng L, Yang W, Zhang D, Zhuge C, Hong L. Epidemic analysis of COVID-19 in China by dynamical modeling. 2020, arXiv preprint arXiv: 2002.06563.
[26] Giordano G, Blanchini F, Bruno R, Colaneri P, Di Filippo A, Di Matteo A, et al. Modelling the COVID–19 epidemic and implementation of population-wide interventions in Italy. Nature Med 2020;1–6.
[27] Tuite AR, Fisman DN, Greer AL. Mathematical modelling of COVID-19 transmission and mitigation strategies in the population of ontario, Canada. CMAJ 2020;192(19):E497–505.
[28] Lemecha Obsu L, Feyissa Balcha S. Optimal control strategies for the transmission risk of COVID-19. J Biol Dyn 2020;14(1):590–607.
[29] Mamo DK. Model the transmission dynamics of COVID-19 propagation with public health intervention. Res Appl Math August 2020, 100123;7.
[30] Kassa SM, Ouhinou A. Epidemiological models with prevalence dependent endogenous self-protection measure. Math Biosci 2011;229(1):41–9.
[31] BERGE T, Chapwanya M, Lubuma JM-S, Terefe YA. A mathematical model for ebola epidemic with self-protection measures. J Biol Systems 2018;26(1):107–31.
[32] Biegler LT, Grossmann IE. Retrospective on optimization. Comput Chem Eng 2004;28(8):1169–92.
[33] Capaldi A, Behrend S, Berman B, Smith J, Wright J, Lloyd AL. Parameter estimation and uncertainty quantification for an epidemic model. Math Biosci Eng 2012;53.
[34] Silva CJ, Torres DF. A TB-HIV/AIDS coinfected model and optimal control treatment. 2015, arXiv preprint arXiv:1501.03322.
[23] Zhao S, Kuang Y, Ben-Arieh D, et al. Information dissemination and human behaviors in epidemics. In: IIE annual conference. Proceedings. Institute of Industrial and Systems Engineers (IISE); 2015, p. 1907.

[24] Schroers BJ. Ordinary differential equations: a practical guide. Cambridge University Press; 2011.

[25] Thieme HR. Mathematics in population biology. Princeton University Press; 2018.

[26] Van den Driessche P, Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Math Biosci 2002;180(1–2):29–48.

[27] Castillo-Chavez C, Feng Z, Huang W. On the computation of ro and its role on. In: Mathematical approaches for emerging and reemerging infectious diseases: an introduction, vol. 1. 2002, p. 229.

[28] Mehrkanoon S, Falck T, Suykens J. Parameter estimation for time varying dynamical systems using least squares support vector machines. In: Proc. of the 16th IFAC symposium on system identification (SYSID 2012). 16, (PART 1):2012, p. 1300–5.

[29] Montgomery DC, Peck EA, Vining GC. Introduction to linear regression analysis, vol. 821. John Wiley & Sons; 2012.

[30] Gamerman D, Lopes HF. Markov chain Monte Carlo: stochastic simulation for Bayesian inference. CRC Press; 2006.

[31] Worldometers. 2020, https://www.worldometers.info/coronavirus/#countries. (Accessed January to July 2020).

[32] Breierova L. An introduction to sensitivity analysis. MIT system dynamics in education project. 1996, http://sysdyn.clexchange.org/sdep/ Roadmaps/RM8/D–4526–2.pdf.

[33] Saltelli A, Ratto M, Andres T, Campolongo F, Cariboni J, Gatelli D, et al. Global sensitivity analysis: the primer. John Wiley & Sons; 2008.

[34] Martcheva M. An introduction to mathematical epidemiology, vol. 61. Springer; 2015.

[35] Malinzi J, Ouifki R, Eladdadi A, Torres DF, White K. Enhancement of chemotherapy using oncolytic virotherapy: mathematical and optimal control analysis. 2018, arXiv preprint arXiv:1807.04329.