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Mathematical modeling for COVID-19 transmission dynamics: A case study in Ethiopia

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A B S T R A C T

In this paper, we proposed a nonlinear deterministic mathematical model for the transmission dynamics of COVID-19. First, we analyzed the system properties such as boundedness of the solutions, existence of disease-free and endemic equilibria, local and global stability of equilibrium points. Besides, we computed the basic reproduction number $R_0$ and studied its normalized sensitivity for model parameters to identify the most influencing parameter. The local stability of the disease-free equilibrium point is also verified via the help of the Jacobian matrix and Routh Hurwitz criteria. Moreover, the global stability of the disease-free equilibrium point is proved by using the approach of Castillo-Chavez and Song. We also proved the existence of the forward bifurcation using the center manifold theory. Then the model is fitted with COVID-19 infected cases reported from March 13, 2020, to July 31, 2021, in Ethiopia. The values of model parameters are then estimated from the data reported using the least square method together with the fminsearch function in the MATLAB optimization toolbox. Finally, different simulation cases were performed using PYTHON software to compare with analytical results. The simulation results suggest that the spread of COVID-19 can be managed via minimizing the contact rate of infected and increasing the quarantine of exposed individuals.

Introduction

Coronavirus disease is a communicable disease caused by a family of novel coronavirus. The virus is referred to as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and the associated disease is COVID-19 [1]. This newly discovered family of novel coronavirus disease started in Wuhan, Hubei Province, China, in late December 2019 and spread rapidly around the world, causing major public health concerns [2–4]. In January 2020, the new virus was named the 2019 novel coronavirus, and later, it was renamed as Coronavirus Disease 2019 (COVID-19) by World Health Organization (WHO) in February 2020 [5,6]. The rate of infection of the disease was very high as a result, WHO has declared it as a pandemic on March 11, 2020 [7]. Most people infected with the COVID-19 virus experience mild to moderate respiratory illness and recover without requiring special treatment. However, people having medical problems (cardiovascular disease, diabetes, chronic respiratory disease, cancer) and older people are more likely to develop serious illnesses. Globally, the COVID-19 pandemic infected more than 197 million individuals up to the end of July 2021, and among these more than 4 million individuals have died.

The symptoms of COVID-19 are variable, ranging from mild symptoms to severe illness. The clinical symptoms of COVID-19 include fever, dry cough, fatigue, sore throat, diarrhea, loss of taste or smell, and headache. In severe cases, it may result in difficulty breathing or shortness of breath, chest pain or pressure, and loss of speech [8]. The estimated incubation period is 2–14 days after contact, but it might extend up to 27 days [9]. Moreover, asymptomatic individuals do not develop any symptoms and not aware of their infection, yet they are capable of transmitting the disease [10]. COVID-19 is by its nature very contagious and spread easily from person to person. Most commonly it transmits from person to person via respiratory droplets produced when an infected person coughing, or sneezing within a social distance less than 6 feet [11].

The first case report of COVID-19 in Ethiopia was on March 13, 2020, two days after the global pandemic was declared. Then on April 16, 2020, the Ethiopian Public Health Institute reported a total of 92 confirmed cases, three deaths and two recoveries [12,13]. Thereby, the Federal Government of Ethiopia declared lock down, imposed quarantine for all travelers, restricted public gatherings, and school closures to combat the pandemic. Besides, the government announced intensively through mass media, social media and cell-phone ring to keep physical distance, wear face masks, and to keep their personal hygiene. Moreover, the Minister of Health was also started daily briefings on the disease progress. In-between, the national politics spoiled and people neglect following COVID-19 protocol. As a consequence,
the disease spread throughout the country and continued claiming many lives. In addition to weak health care system, many Ethiopians live in poor and crowded conditions [14] in large cities. This would further facilitate the spread of the disease. For instance, up to July 31, 2021, the total confirmed and death cases reported due to COVID-19 in Ethiopia, respectively, are 280,365, and 4,385 [15]. Furthermore, peoples’ awareness on the COVID-19 is very weak and wearing face mask is also almost nil in the countryside.

Mathematical modeling plays a fundamental role in understanding, predicting, and controlling the transmission dynamics of infectious diseases. In this regard, its application has a long history, for example, in malaria [16–18], tuberculosis [19,20], and references cited therein. Since COVID-19 outbreak, many mathematicians studied the transmission dynamics of the disease considering different scenarios [21–26]. In particular, Yang and Wang [21] proposed and analyzed a mathematical model for COVID-19 incorporating multiple transmission pathways, including both human-to-human and environment-to-human transmission routes. Their model was further extended by Legesse and Shiferaw [22] by applying an optimal control theory to minimize the transmission risk of COVID-19. Ali et al. [23] proposed a mechanistic model to investigate the role of asymptomatic class, quarantine, and isolation in the transmission dynamics of COVID-19. They have performed a detailed theoretical analysis in terms of the basic reproduction number and predicted cumulative cases. Moreover, the study suggests that quarantine and isolation of individuals play a significant role in controlling the transmission of the disease.

Tripathi et al. [24] proposed a mathematical model to study the transmission dynamics of COVID-19 considering the role of social distancing, lockdown, quarantine, and isolation. The result of this study reveals that reducing the contacts through increasing the efficacy of lockdown and quarantine of asymptomatic individuals significantly minimized the epidemic of the disease. In [25], a mathematical model for COVID-19 is proposed by taking into consideration pharmaceutical and non-pharmaceutical intervention techniques to combat the pandemic of novel coronavirus. The authors estimated model parameter values using real data reported on COVID-19 from three provinces in Indonesia, namely, Jakarta, West Java, and East Java. Moreover, they implemented the theory of optimal control to determine the best intervention strategies with optimal cost. Their results showed that community awareness plays a crucial role in the success of COVID-19 eradication programs.

Bajia et al. [26] studied the impact of non-pharmaceutical interventions on the dynamics of COVID-19 using a mathematical model. They employed a mixing standard incidence rate based on the law of mass action and estimated parameter values using case data reported on COVID-19 from India. In [27], the authors proposed a mathematical model to analyze the dynamics and impact of non-pharmaceutical interventions on the COVID-19 in Pakistan. First, they developed a model without control variables and provided a good fit to the reported case data. Later, they reformulated the proposed model by incorporating two control functions in order to determine an appropriate innervation mechanism. In [28], a mathematical model for COVID-19 is proposed and analyzed by classifying the transmission routes as: symptomatic infectious, asymptomatic infectious, and hospitalized individuals. The model was parameterized based on the cumulative case report in Nigeria. Moreover, they extended the proposed model into an optimal control model to minimize the number of infectious humans in the population.

In the case of Ethiopia, a limited number of mathematical models on COVID-19 outbreak have been studied with real data. In [29], Haileyesus and Getachew considered a SEIR compartmental model and extended the model into optimal control. In [30], Chernet and Gemechis proposed and analyzed an extension of the SEIR model to asymptomatic and hospitalized classes. Moreover, they presented a detail analysis and fitted the model to real data from Ethiopia. Their study concluded that the combined effects of public health education, personal protective, and treatment of hospitalized individuals are significantly reducing the expansion of the disease. However, the authors do not consider quarantined individuals and disease-induced death rate in asymptomatic compartment. Cases are not fully reported due to weak health infrastructure and limited well trained human power in the country. The asymptomatic individuals are identified as an important source of disease transmission and isolated infected individuals is a primary mechanism adopted to control the spread of the disease [5,11]. Thus, this study aims to study the transmission dynamics of COVID-19 via extending the SEIR model by including asymptomatic, quarantined, and hospitalized classes. The model parameters are fitted to the reported real data of COVID-19 from March 13, 2020 to July 31, 2021 in Ethiopia.

The remaining parts of the paper are arranged as follows: The proposed mathematical model is described in Section a. Detailed mathematical analysis including: existence and uniqueness, non-negativity and boundedness, equilibria, stability analysis, and bifurcation analysis of the model is presented in Section a. Parameter estimation and curve fitting are presented in Section a. The Sensitivity analysis of the control reproduction number to model parameters are discussed in Section a. We demonstrated numerical simulations in Section a. Finally, the discussion and conclusions are presented in Section a.

Model formulation

In this section, we propose an SEQAIJR type mathematical model to analyze the dynamics of COVID-19 pandemic. Here, the total population is divided into seven compartments: \( S \) denotes susceptible; \( E \) represents the compartment of exposed (individuals who may or may not have been infected). We assumed that some of the exposed individuals are quarantined to avoid contact with health individuals. This class is denoted by \( Q \). We assume that asymptomatic individuals contribute to the distribution of the disease and thus denoted by \( A \). A symptomatic individuals who are capable of spreading the disease but not hospitalized are also represented by \( I \). Moreover, seriously sick and hospitalized individuals are denoted by \( J \). These are isolated individuals with confirmed COVID-19 cases. Lastly, we denote the compartment of recovered individuals by \( R \). Thus, the total population at time \( t \) is \( N(t) = S(t) + E(t) + Q(t) + A(t) + I(t) + J(t) + R(t) \).

In our model, the parameter \( \Lambda \) denotes the recruitment rate of susceptible individuals. Besides, the population of susceptible are assumed to be increased at the rate \( \xi \) due to the influx from quarantined individuals after testing negative. Furthermore, we assumed that the susceptible individuals are acquired infection when they contact either with asymptomatic or symptomatic individuals. In this case, we assumed that the infection rate of asymptomatic individuals is lower than that of symptomatic individuals, with the reduction parameter \( \tau \in (0,1) \). This is because of as asymptomatic individuals do not show any symptoms which in turn do not spread pathogens by sneezing as often as symptomatic individuals. Thus, the force of infection is defined as \( \lambda = \frac{\beta(rA + I)}{N(t) - Q - J} \), excluding the quarantined and isolated from the total population [26]. Here, \( \beta \) denotes the number of new contagions per unit time due to contact with COVID-19 infectious, while \( r \beta \) stands for the modified transmission coefficient from the susceptible to infected class for asymptomatic class. We represented the natural death rate in each compartment by \( \mu \). The term \( \eta_q \) denotes a fraction of exposed population joining compartment \( Q \), while \( \eta_h \) represents the portion of individuals moving to compartment \( A \). Furthermore, we denote the proportion of exposed individuals showing symptoms by \( \eta_s \). The parameter \( \eta_h \) denotes the rate at which quarantined individuals show symptoms and moves to I class. The disease-induced death rates for populations in classes \( A, I \), and \( J \), respectively, are represented by the parameters \( \delta_1, \delta_2 \), and \( \delta_3 \). The infective individuals in classes \( A \) and \( I \) are assumed to be recovered at a constant rate \( r_1 \) and \( r_2 \), respectively, and treated individuals leave compartment \( J \) at a rate \( r_3 \).
joining compartment $R$. The infected individuals are isolated at a rate $a$.

The above model descriptions are illustrated in Fig. 1.

The resulting mathematical model is given by a system of nonlinear ordinary differential equations:

$$
\begin{align*}
\frac{dS}{dt} &= A - \lambda S - \mu S + \zeta Q, \\
\frac{dE}{dt} &= \lambda S - (\eta_1 + \eta_2 + \eta_3 + \mu)E, \\
\frac{dQ}{dt} &= \eta_1 E - (\xi + \eta_1 + \mu)Q, \\
\frac{dA}{dt} &= \eta_2 E - (r_1 + \delta_1 + \mu)A, \\
\frac{dI}{dt} &= \eta_3 E + q_1 Q - (a + r_2 + \delta_2 + \mu)I, \\
\frac{dJ}{dt} &= aI - (r_3 + \delta_3 + \mu)J, \\
\frac{dR}{dt} &= r_1 A + r_2 I + r_3 J - \mu R,
\end{align*}
$$

where $\lambda = \frac{\beta(rA + I)}{N - Q + j}$ with initial data $S(0) > 0$, $E(0) \geq 0$, $A(0) \geq 0$, $Q(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$ and $N(0) > 0$. All parameters in the model are assumed to be nonnegative. Compared to the model introduced in [30], our model considers the quarantine parameters in the model are assumed to be nonnegative. Compared to the model in [26], our model considers the asymptomatic compartment. In this case, the asymptomatic individuals move to a recovered class. The descriptions of the model parameters are presented in Table 1.

Model analysis

In this section, some basic properties of the model (1) including the existence and uniqueness of the solution, feasible region, the positivity of the solution, equilibria, and their stability are discussed.

**Theorem 1 (Existence and uniqueness of solutions). Solutions to model (1) are exists and unique for $t \in [0, \infty)$.**

**Proof.** The model (1) can be written as:

$$\dot{x} = f(t, x(t))$$

where $x(t) = (S(t), E(t), Q(t), A(t), I(t), J(t), R(t))$ and $f(t, x(t)) = (f_1(t, x(t)), \ldots, f_7(t, x(t)))$ denotes the right hand expressions.

The result is straightforward: applying the existence and uniqueness theorem in [31], one can easily show that there exists a unique solution for model (1) in the bounded region $\mathbb{R}^7_+$. This can be verified by checking that in the bounded region $\mathbb{R}^7_+$, each of the partial derivatives on the right side of the model (1) with respect to $S$, $E$, $Q$, $A$, $I$, $J$ or $R$ are bounded in $\mathbb{R}^7_+$. □

The next theorem implies that the solutions of the model (1) are nonnegative and bounded from above with nonnegative initial conditions.

**Theorem 2 (Nonnegativity and boundedness of the solution). All solutions of model (1) with nonnegative initial value remain nonnegative for all $t \geq 0$. Moreover**

$$\limsup_{t \to \infty} N(t) \leq \frac{A}{\mu}$$

**Proof.** We show that all solutions of the model (1) are nonnegative as required in [32,33]. We use the proof by contradiction to show that the state variable $S$ of the model is positive for all $t \geq 0$. We suppose that a trajectory crosses the positive cone at time $t_1$ such that:

$$t_1 \quad S(t_1) = 0, \quad \frac{dS}{dt}(t_1) < 0, \quad E(t_1) > 0, \quad Q(t_1) > 0, \quad A(t_1) > 0, \quad I(t_1) > 0, \quad J(t_1) > 0, \quad R(t_1) > 0 \text{ for } t \in (0, t_1).$$

Using the first equation of the model (1), the first assumption leads to

$$\frac{dS}{dt}(t_1) = A + \zeta Q(t_1) > 0,$$

which contradicts the first assumption that $\frac{dS}{dt}(t_1) < 0$. Thus, $S(t)$ remains positive for all $t \geq 0$. Here, we choose $t_1$ in such away that our point to be on the positive axis of $S(t)$ so that $Q(t_1)$ is positive.

Based on the second equation of model (1),

$$\frac{dE}{dt} = \lambda S - (\eta_1 + \eta_2 + \eta_3 + \mu)E \geq -(\eta_1 + \eta_2 + \eta_3 + \mu)E,$$

because $S(t)$ is nonnegative for all $t \geq 0$. Solving this equation yields

$$E(t) \geq E(0) \exp\left(-\eta_1 - \eta_2 - \eta_3 - \mu\right) \geq 0.$$  

Correspondingly, from the third equation of model (1), we obtain

$$\frac{dQ}{dt} = \eta_1 E - (\xi + \eta_1 + \mu)Q \geq -(\xi + \eta_1 + \mu)Q.$$  

Solving this equation leads to

$$Q(t) \geq Q(0) \exp\left(-(\xi + \eta_1 + \mu)\right) \geq 0.$$
Similarly, using the last four equations of model (1), we have
\[
\frac{dA}{dt} = -\eta_1 E - (r_1 + \delta_1 + \mu)A \\
\frac{dI}{dt} = \eta_2 E + \eta_3 Q - (d_2 + \beta)I - (\alpha + r_3 + \delta_1 + \mu)I \\
\frac{dJ}{dt} = \alpha I - (r_3 + \delta_3 + \mu)J \\
\frac{dR}{dt} = -\delta_1 (I + J + R)
\]
and
\[
R(t) = r_1 A + r_2 I + r_3 J - \mu R \geq -\mu R,
\]
because \( S(t), E(t), \) and \( Q(t) \) are nonnegative for all \( t \geq 0 \). Solving the above equations gives
\[
A(t) \geq A(0)e^{-(r_1 + \delta_1 + \mu)t} \geq 0, \\
I(t) \geq I(0)e^{-(\alpha + r_3 + \delta_2 + \mu)t} \geq 0, \\
J(t) \geq J(0)e^{-(r_3 + \delta_3 + \mu)t} \geq 0,
\]
and
\[
R(t) \geq R(0)e^{-\mu t} \geq 0.
\]
respectively. Thus, any solution of model (1) is nonnegative with nonnegative initial data for all \( t \geq 0 \). Furthermore, adding the right-hand sides of model (1) together, we obtain
\[
\frac{dN}{dt} = \Lambda - \mu N - (\delta_1 A + \delta_2 I + \delta_3 J) \leq \Lambda - \mu N.
\]
It follows that
\[
N(t) \leq \frac{\Lambda}{\mu} (N(0) - \frac{\Lambda}{\mu}) e^{-\mu t}.
\]
Considering \( t \to \infty \), we have
\[
\lim_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu}
\]
Thus, the model (1) is bounded. This completes the proof of Theorem 2. \( \square \)

**Theorem 3.** The closed region
\[
\Omega = \{(S, E, Q, A, I, J, R) \in \mathbb{R}^7_+ : 0 < S + E + Q + A + I + J + R \leq \frac{\Lambda}{\mu}\}
\]
is positively invariant set for the model (1).

**Proof.** Positive invariance of \( \mathbb{R}^7_+ \) can be verified by examining the direction of the vector field \((f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T\) of the model (1) on each face [32]. Thus, to show that \( \mathbb{R}^7_+ \) is positively invariant, it suffices to show that the direction of the vector field points inward on the boundary of \( \mathbb{R}^7_+ \).

For example, for the model (1), on the face \( S = 0 \), we have
\[
\frac{dS}{dt}|_{S=0} = A + \zeta Q > 0.
\]
Therefore, the vector field on the \((E, Q, A, I, J, R)-\)face points to the interior of \( \mathbb{R}^7_+ \). No solution can escape the interior through the \((E, Q, A, I, J, R)-\)face. Similarly, one can show for the others. If \( N = S + E + Q + A + I + J + R = \frac{\Lambda}{\mu} \),
\[
\frac{dN}{dt}|_{N=\frac{\Lambda}{\mu}} = -(\delta_1 A + \delta_2 I + \delta_3 J) \leq 0.
\]
We thus have shown that all solutions starting in \( \mathbb{R}^7_+ \) remain in \( \mathbb{R}^7_+ \) for \( t > 0 \). That is, \( Q \) is positively invariant for the model (1). \( \square \)

**Disease free equilibrium**

We calculate the disease-free equilibrium of model (1) by equating the right-hand side equations to zero and then putting \( E = 0, Q = 0, A = 0, I = 0, \) and \( J = 0 \). Then we get;
\[
E^0 = (S^0, 0, 0, 0, 0, 0, 0), \quad (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0).
\]
The infected compartments of model (1) consist of \((E, Q, A, I, J)\) classes. Using the next generation method [34,35], the basic reproduction number \( R_0 \) can be calculated from the relation \( R_0 = \mu (FV^{-1}) \) (the spectral radius of the Eigenvalue of the Jacobian matrix evaluated at the COVID-19-free equilibrium point \( E^0 \)). The Jacobian matrix calculated at \( E^0 \) of the transmission terms, \( F \) and that of transition terms, \( V \) are, respectively
\[
F = \begin{pmatrix} 0 & 0 & \beta \tau & \beta \tau \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}
\]
and
\[
V = \begin{pmatrix} K_1 & 0 & 0 & 0 \\ -\eta_1 & K_2 & 0 & 0 \\ -\eta_2 & 0 & K_3 & 0 \\ 0 & 0 & 0 & -\alpha & K_4 \end{pmatrix}
\]
where \( K_1 = \eta_1, K_2 = \eta_2 + \eta_1 + \mu, K_3 = \zeta + \eta_1 + \mu, K_4 = \alpha + r_2 + \delta_2 + \mu \) and \( K_5 = r_3 + \delta_3 + \mu \). Thus, the next generation matrix is
\[
(FV^{-1}) = \begin{pmatrix} \beta \tau \eta_2 & \beta \eta_2 K_2 + \eta_1 \eta_1 & \beta \tau \eta_1 & \beta \tau \eta_1 & \beta \tau \eta_1 & \beta \tau \eta_1 \\ K_1 K_2 & \alpha \tau + \zeta & K_1 K_2 & K_1 K_2 & K_1 K_2 & K_1 K_2 \\ K_1 K_2 & K_1 K_2 & \alpha \tau + \zeta & K_1 K_2 & K_1 K_2 & K_1 K_2 \\ K_1 K_2 & K_1 K_2 & K_1 K_2 & \alpha \tau + \zeta & K_1 K_2 & K_1 K_2 \\ K_1 K_2 & K_1 K_2 & K_1 K_2 & K_1 K_2 & \alpha \tau + \zeta & K_1 K_2 \\ K_1 K_2 & K_1 K_2 & K_1 K_2 & K_1 K_2 & K_1 K_2 & \alpha \tau + \zeta \end{pmatrix}
\]
By solving the dominant eigenvalue of the next generation matrix \( FV^{-1} \), we get the basic reproduction number
\[
R_0 = \frac{\beta(\eta_1, K_1 + K_2 + \eta_1, q_1)}{K_1, K_2, K_3}
= \frac{\beta(\eta_1, \xi + q_1 + \mu)(a + r_1 + \delta_1 + \mu) + \beta(\eta_1, q_1 + \mu) + \eta_1, q_1)}{\eta_1 + \eta_1 + \mu}(\xi + q_1 + \mu)(r_1 + \delta_1 + \mu) + \eta_1, q_1 + \mu(a + r_2 + \delta_2 + \mu)).
\]

Remark 4. The basic reproduction number, \( R_0 \), is defined as the expected number of secondary infections produced by an index case in a completely susceptible population by a typical infective individual [34]. This number is a measure of the potential for disease spread within a population. Mathematically, \( R_0 \) is a threshold for the stability of a disease-free equilibrium and that can be used as an indicator for disease control.

Local and global stability of DFE

In many epidemiological models, there is a disease-free equilibrium point (DFE) at which the population remains in the absence of disease. The following theorems discuss the local and global stability of DFE \( E^0 \).

**Theorem 5.** The disease-free equilibrium \( E^0 \) of model (1) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

**Proof.** We determine the local stability of DFE (\( E^0 \)) using the eigenvalues of the Jacobian matrix at \( E^0 \), which is given by
\[
J_{E^0} = 
\begin{pmatrix}
-\mu & 0 & \xi & -\beta r & -\beta \\
0 & -K_1 & 0 & \beta r & \beta \\
-\eta_1 & -K_2 & 0 & 0 & 0 \\
0 & -\eta_2 & 0 & -K_3 & 0 \\
0 & 0 & 0 & 0 & -K_4 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix}
\]
where \( K_1 = \eta_1 + \eta_2 + \eta_3 + \mu, \ K_2 = \xi + q_1 + \mu, \ K_3 = r_1 + \delta_1 + \mu, \ K_4 = a + r_2 + \delta_2 + \mu \), and \( K_5 = r_3 + \delta_3 + \mu \) as in section a. It is obvious that \( \lambda_1 = -\mu, \ \lambda_2 = -K_4 \) and \( \lambda_3 = -\mu \) are three negative eigenvalues of \( J_{E^0} \). We find the remaining eigenvalues of \( J_{E^0} \) from the following block matrix
\[
RJ_{E^0} = 
\begin{pmatrix}
-K_1 & 0 & \beta r & \beta \\
-\eta_1 & -K_2 & 0 & 0 \\
0 & -\eta_2 & 0 & -K_3 \\
0 & 0 & 0 & -K_4
\end{pmatrix}
\]
The characteristic polynomial of \( RJ_{E^0} \) is given by
\[
P(\lambda) = \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4,
\]
where
\[
a_1 = K_1 + K_2 + K_3 + K_4,
\]
a_2 = K_1(K_2 + K_3 + K_4) + K_3(K_4 + K_5) + K_5(K_1 + K_2) - \beta(\eta_3, q_1) + \eta_2(q_1),
\]
a_3 = K_1K_2(K_3 + K_4) + K_3K_4(K_1 + K_2) - \beta(\eta_3, q_1) + \eta_2(q_1),
\]
a_4 = K_1K_2K_3K_4(1 - R_0).

Applying the Routh–Hurwitz stability criterion [36] and after some little algebraic manipulations, it can be shown that the eigenvalues of the block matrix \( RJ_{E^0} \) have negative real parts i.e. \( \Re(\lambda_2), \Re(\lambda_3), \Re(\lambda_4), \Re(\lambda_5) \) < 0, if \( R_0 < 1 \). If \( R_0 > 1 \), then \( a_3 < 0 \), thus the matrix \( RJ_{E^0} \) has at least one eigenvalue with positive real part. Hence, disease-free equilibrium \( (E^0) \) of model (1) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

From the above theorem, we conclude that if \( R_0 < 1 \), then the DFE is locally asymptotically stable, and the disease cannot invade the population; but if \( R_0 > 1 \), then the DFE is unstable and invasion is always possible. In the next theorem, the global stability of disease-free equilibrium is investigated by using the technique implemented by Castillo-Chavez and Song [37].

**Theorem 6.** If \( R_0 < 1 \), the disease-free equilibrium \( E^0 \) of model (1) is globally asymptotically stable in its feasible region.

**Proof.** First, we rewrite the model (1) as follows:
\[
\frac{dX}{dt} = F(X, Y), \quad \frac{dY}{dt} = G(Y), \quad \text{with } G(X, 0) = 0,
\]
where \( X = (S, R) \in \mathbb{R}^2 \) represents the non-disease compartments, and \( Y = (E, Q, A, I, J) \in \mathbb{R}^5 \) represents the disease compartments. The following two conditions \( (H_1) \) and \( (H_2) \) are required for the global asymptotic stability of the DFE of model (1).

\( (H_1) \) For \( \frac{dX}{dt} = F(X, 0), X^* \) is globally asymptotically stable, where \( F(X^*, 0) = 0 \).

\( (H_2) \) \( G(Y) = BY - G(X, Y), G(X, Y) > 0 \) for \( (X, Y) \in \Omega \), where \( B = D_Y G(X^*, 0) \) is an M-matrix. The off-diagonal elements of \( B \) are nonnegative and is the region where the system makes biologically feasible in \( \Omega \).

For model (1), we have
\[
\frac{dX}{dt} = \begin{pmatrix} -\mu S \\ 0 \end{pmatrix}.
\]

Indeed, the system (2) is globally asymptotically stable around \( X^* = \left( \frac{A}{\mu}, 0 \right) \). This can be verified from the solution \( S(t) = \frac{A}{\mu} + (S(0) - \frac{A}{\mu}) e^{\mu t} \), such that \( \lim_{t \to \infty} S(t) = \frac{A}{\mu} \), which implies that the global convergence of (2) in \( \Omega \). Furthermore, from the model (1), we obtain
\[
B = 
\begin{pmatrix}
-K_1 & 0 & \beta r & \beta \\
-\eta_1 & -K_2 & 0 & 0 \\
0 & -\eta_2 & 0 & -K_3 \\
0 & 0 & 0 & -K_4
\end{pmatrix}
\]
and
\[
\hat{G}(X, Y) = 
\begin{pmatrix}
\beta(rA + I) \left( \frac{E + A + I + R}{S + E + A + I + R} \right) \\
0 \\
0 \\
0
\end{pmatrix}
\]

Clearly, \( \left( \frac{E + A + I + R}{S + E + A + I + R} \right) \geq 0 \) inside \( \Omega \) and therefore, \( \hat{G}(X, Y) \geq 0 \).

Thus, the two conditions \( (H_1) \) and \( (H_2) \) are satisfied. Therefore, the DFE \( E^0 \) of model (1) is globally asymptotically stable when \( R_0 < 1 \).

**Existence of an endemic equilibrium**

In this subsection, we investigate the existence of the endemic equilibrium \( E^*_1 \) of model (1).

**Theorem 7.** If \( R_0 > 1 \), there exists a unique endemic equilibrium \( E^*_1 = (S^*, E^*, Q^*, A^*, I^*, J^*, R^*) \) of system (1) and there is no endemic equilibrium when \( R_0 < 1 \).
Proof. For the existence of an endemic equilibrium, we need to solve the following system of equations:

\[ \begin{align*}
A - \beta S^* (r A^* + I^*) &- \mu S^* + \zeta Q^* = 0, \\
\beta S^* (r A^* + I^*) &- (\eta_1 + \eta_2 + \eta_3 + \mu) E^* = 0, \\
\eta_1 E^* - (\zeta + \eta_4) Q^* & = 0, \\
\eta_2 E^* - (r_1 + \delta_1 + \mu) A^* = 0, \\
\eta_3 E^* + \eta_4 Q^* - (a + r_2 + \delta_2 + \mu) I^* & = 0, \\
\mu A^* - (r_1 + \delta_1 + \mu) I^* & = 0, \\
r_1 A^* + r_2 I^* + r_3 J^* - \mu R^* & = 0.
\end{align*} \]

We use the same definition as in section a, \( K_1 = \eta_1 + \eta_2 + \eta_3 + \mu, \)
\( K_2 = \zeta + \eta_4 + \mu, \) \( K_3 = r_1 + \delta_1 + \mu, \)
\( K_4 = a + r_2 + \delta_2 + \mu \), and \( K_5 = r_1 + \delta_1 + \mu. \)
Solving the above system of equations (3) yields Box 1

Since \( K_1 K_2 - \zeta \eta_1 = \eta_1 (\zeta + \mu) + (\eta_2 + \eta_3 + \mu (\zeta + \eta_4) + \mu) > 0, \) we can easily observe that there exists a unique endemic equilibrium of the model (1) when \( R_0 > 1 \) and no endemic equilibrium when \( R_0 < 1. \)

\textbf{Bifurcation analysis}

In this subsection, we establish the conditions on the parameters using Theorem 4.1 from [37] and center manifold theory [38]. In this theorem, there are two coefficients that represent dynamics on the center manifold. If we say these coefficients that 'decide' the bifurcation a and b, we have \( a < 0 \) and \( b > 0 \) for the occurrence of forward bifurcation.

By solving \( R_0 = 1, \) we obtain

\[ \beta = \beta^* = \frac{(\eta_1 + \eta_2 + \eta_3 + \mu) (\zeta + \eta_4) (r_1 + \delta_1 + \mu) (a + r_2 +\delta_2 + \mu) (r_1 + \delta_1 + \mu)}{r_2 (\zeta + \eta_4) (r_1 + \delta_1 + \mu) (a + r_2 +\delta_2 + \mu) (r_1 + \delta_1 + \mu)} \cdot \]

The Jacobian matrix \( J_{\beta_0, \rho}, \) for the model (1) evaluated at \( E^0 \) and
\( \beta = \beta^* \) has a simple zero eigenvalue and other eigenvalues have negative sign. Hence \( E^0 \) is a non-hyperbolic equilibrium, when \( \beta = \beta^*. \)
Therefore, we can use the center manifold theory [37,38] to establish the local stability of the endemic equilibrium \( E^0. \) Calculating a right eigenvector \( W = (w_1, w_2, w_3, w_4, w_5, w_6)^T \) and a left eigenvector \( V = (v_1, v_2, v_3, v_4, v_5, v_6)^T \) associated to the zero eigenvalues, we get

\[ \begin{align*}
w_1 &= \frac{r_1 \eta_1 K_1 K_2 K_3 (\zeta - \eta_1)}{\mu K_1 K_3 K_2} + \frac{K_3 (r_2 + \eta_3) (\eta_1 K_2 + \zeta)}{\mu K_1 K_3 K_2}, \\
w_2 &= \frac{r_2 \eta_2 K_1 K_2 K_3 (r_1 + \delta_1 + \mu) (\eta_1 K_2 + \zeta)}{\mu K_1 K_3 K_2}, \\
w_3 &= \frac{r_1 \eta_3 K_1 K_2 K_3 (r_1 + \delta_1 + \mu) (\eta_1 K_2 + \zeta)}{\mu K_1 K_3 K_2}, \\
w_4 &= \frac{r_2 \eta_4 K_1 K_2 K_3 (r_1 + \delta_1 + \mu) (\eta_1 K_2 + \zeta)}{\mu K_1 K_3 K_2}, \\
w_5 &= \frac{w_6}{\mu K_1 K_2 K_3 (\eta_1 K_2 + \zeta)}, \\
w_6 &= \frac{w_6}{\mu K_1 K_2 K_3 (\eta_1 K_2 + \zeta)} \cdot \end{align*} \]

and

\[ \begin{align*}
v_1 &= \frac{v_2}{\tau K_2 K_3} + \frac{v_3}{\tau K_2 K_3}, \\
v_2 &= \frac{\tau K_2 K_4}{\tau K_2 K_3}, \\
v_3 &= \frac{\tau K_2 K_4}{\tau K_2 K_3}, \\
v_4 &= \frac{\tau K_2 K_4}{\tau K_2 K_3}, \\
v_5 &= 1, \\
v_6 &= 0. \end{align*} \]

Now from Theorem 4.1 of [37], we need to calculate the bifurcation constants \( a \) and \( b \):

\[ \begin{align*}
a &= \sum_{k=1}^2 v_k w_k \frac{\partial^2 f_k}{\partial x_1 \partial x_2} (E^0, \beta^*) = -\frac{\beta \tau \mu}{A}, \\
b &= \sum_{k=1}^2 v_k w_k \frac{\partial^2 f_k}{\partial x_1 \partial \beta} (E^0, \beta^*) = -\frac{\beta \tau \mu}{A}. \end{align*} \]

Denote \( x_1 = S, x_2 = E, x_3 = Q, x_4 = A, x_5 = I, x_6 = J, x_7 = R. \) Since \( v_1 = 0, v_6 = 0 \) and \( v_5 = 0, \) we do not need the derivatives of \( f_1, f_6 \) and \( f_7. \) From the second-order partial derivatives of \( f_2, f_3, f_4 \) and \( f_5, \) the only ones that are nonzero are the following:

\[ \begin{align*}
\frac{\partial^2 f_2}{\partial x_1 \partial x_2} (E^0, \beta^*) &= \frac{\partial^2 f_2}{\partial x_1 \partial x_2} (E^0, \beta^*) = -\frac{\beta \tau \mu}{A}, \\
\frac{\partial^2 f_2}{\partial x_1 \partial x_2} (E^0, \beta^*) &= \frac{\partial^2 f_2}{\partial x_1 \partial x_2} (E^0, \beta^*) = -\frac{\tau \mu}{A}, \\
\frac{\partial^2 f_2}{\partial x_1 \partial \beta} (E^0, \beta^*) &= \frac{\partial^2 f_2}{\partial x_1 \partial \beta} (E^0, \beta^*) = -\frac{\tau \mu}{A}. \end{align*} \]

Furthermore,

\[ \frac{\partial^2 f_2}{\partial x_1 \partial \beta} (E^0, \beta^*) = \frac{\partial^2 f_2}{\partial x_1 \partial \beta} (E^0, \beta^*) = 1. \]

Hence, we obtain

\[ a = v_2 \left[ 2 \left( \frac{\partial^2 f_2}{\partial A \partial \beta} + \frac{\partial^2 f_2}{\partial I \partial \beta} + \frac{\partial^2 f_2}{\partial A \partial I} + \frac{\partial^2 f_2}{\partial I \partial A} \right) + \frac{\partial^2 f_2}{\partial A^2} + \frac{\partial^2 f_2}{\partial I^2} \right] = -v_2 \frac{\beta \tau \mu}{A} \left[ 2 (w_1 w_2 + w_2 w_3 + w_3 w_4 + w_4 w_5 + w_5 w_6 \right] + 2 \left( w_1 w_2 + w_2 w_3 \right) + w_1 w_2 + w_2 w_3 + w_3 w_4 + w_4 w_5 + w_5 w_6 \}

\[ = -2v_2 \frac{\beta \tau \mu}{A} \left[ w_1 w_2 + w_2 w_3 + w_3 w_4 + w_4 w_5 + w_5 w_6 \right] + w_1 w_2 + w_2 w_3 + w_3 w_4 + w_4 w_5 + w_5 w_6 < 0, \]

\[ b = v_2 \left( w_1 \frac{\partial^2 f_2}{\partial A \partial \beta} + w_2 \frac{\partial^2 f_2}{\partial I \partial \beta} \right) \]

\[ = v_2 (w_1 \tau + w_4) > 0. \]
Clearly, \(a < 0\) and \(b > 0\), at \(\beta = \beta^*\). Therefore, by applying Theorem 4.1 stated in Castillo-Chavez and Song [37], the model (1) undergoes a forward/transcritical bifurcation at \(R_0 = 1\) (See the Fig. 11). Hence, we get the following result.

**Lemma 8** (Local stability of endemic equilibrium). The unique endemic equilibrium \(E^*_1\) of model (1) is locally asymptotically stable (LAS) if \(R_0 > 1\).

The forward bifurcation tells us that as the basic reproduction number crosses a critical value, \(R_0 = 1\) the system transit from one equilibrium point, which is typically stable, to two non-negative equilibrium points, with one stable and one unstable. This implies that COVID-19 disease cannot invade the population for \(R_0 < 1\). On the contrary, the disease persist in the community for \(R_0 > 1\), exceeds unity.

**Global stability analysis of endemic equilibrium point**

Here, we apply the high-dimensional Bendixson criterion which is developed by Li and Muldowney [39] to prove the global stability of the endemic equilibrium point \(E^*_1\). The following lemma will be useful in the sequel before proving the global stability of the endemic equilibrium point \(E^*_1\).

**Lemma 9.** The model (1) has no periodic orbits.

**Proof.** We establish the Dulac’s criterion to prove the theorem. Let \(X = (S, E, Q, A, I, J, R)\). Define the Dulac’s function

\[
B = \frac{1}{SQ}
\]

Then we obtain

\[
\frac{dBX}{dt} = \frac{\partial}{\partial S} \left( \frac{B}{dS} \right) + \frac{\partial}{\partial E} \left( \frac{B}{dE} \right) + \frac{\partial}{\partial Q} \left( \frac{B}{dQ} \right) + \frac{\partial}{\partial A} \left( \frac{B}{dA} \right) + \frac{\partial}{\partial J} \left( \frac{B}{dJ} \right) + \frac{\partial}{\partial R} \left( \frac{B}{dR} \right)
\]

\[
= \frac{\partial}{\partial S} \left( \frac{A}{SQ} \right) + \frac{\partial}{\partial E} \left( \frac{\beta(a+1)}{SQ} \right) + \frac{\partial}{\partial Q} \left( \frac{\beta(a+1)}{SQ} \right) + \frac{\partial}{\partial A} \left( \frac{\beta(a+1)}{SQ} \right) + \frac{\partial}{\partial J} \left( \frac{\eta I E}{SQ} \right) + \frac{\partial}{\partial R} \left( \frac{\eta I E}{SQ} \right)
\]

\[
= \frac{-\left( \frac{A}{SQ^2} + \frac{\gamma}{S^2} + \frac{\eta I}{SQ^2} + \frac{K_1 + K_2 + K_3 + \mu}{SQ} \right)}{<0}
\]

Hence, Dulac’s criterion implies that there does not exist any periodic solution in \(\Omega\) for the model (1). □

From an epidemiological point of view, the non-existence of periodic orbit implies that there are fluctuations in the number of infectives which makes it difficult to allocate resources for disease control.

**Theorem 10.** The endemic equilibrium \(E^*_1\) of model (1) is globally asymptotically stable whenever \(R_0 > 1\).

**Proof.** The result can be established by using the approach given [39,40] and Lemma 9. □

**Table 2**

| Months          | Total confirmed cases | Months          | Total confirmed cases |
|-----------------|-----------------------|-----------------|-----------------------|
| March 31, 2020  | 26                    | Dec 31, 2020    | 124264                |
| April 30, 2020  | 131                   | Jan 31, 2021    | 137565                |
| May 31, 2020    | 1172                  | Feb 28, 2021    | 159072                |
| June 30, 2020   | 5846                  | March 31, 2021  | 206589                |
| July 31, 2020   | 17530                 | Apr 30, 2021    | 257442                |
| August 31, 2020 | 52131                 | May 31, 2021    | 271541                |
| Sep 30, 2020    | 75368                 | June 30, 2021   | 276174                |
| Oct 31, 2020    | 96169                 | July 31, 2021   | 280365                |
| Nov 30, 2020    | 110074                |                 |                       |

**Parameter estimation**

In this section, we estimate the parameters in model (1) based on real data of COVID-19 infected cases in Ethiopia. In this study, we consider the COVID-19 monthly confirmed cases from March 13, 2020, until July 31, 2021 in Ethiopia (see Table 2). The data were collected from Ethiopian Public Health Institute (EPHI) [13] and available online at [41,42]. The COVID-19 data for Ethiopia are fitted using the nonlinear least-squares curve fitting method with the help of “fminsearch” function from the MATLAB Optimization Toolbox. Some of the model parameters are estimated from the literature. The average life expectancy of Ethiopians for the year 2021 is 67.8 [30] and hence, the natural death rate of individuals is calculated by taking the reciprocal of the average life expectancy (in months) which is \(\mu = 1/(67.8 \times 12)\). The total population of Ethiopia for the year 2021 is estimated about \(N(0) = 114,963,588\) people [42]. The recruitment rate of susceptible individuals \(A\) is obtained from \(A/\mu = N(0)\), and it is assumed to be 141,302. The incubation period \((1/\eta)\) for COVID-19, which is the time from exposure to symptom development, is on average five to seven days. The quarantine period \((1/\zeta)\) for COVID-19 is 2 weeks.

To estimate the rest of parameters, the initial conditions for the state variables are used. According to EPHI report, in March 2020, there have been 26 COVID-19 confirmed cases in Ethiopia, so that \(I(0) = 26\). As in other parts of the world, also in Ethiopia there is a limited COVID-19 test and there is the possibility for the presence of asymptomatic individuals (infected individuals with no symptoms) [30]. This results the actual number of cases is more likely higher than the reported cases. Thus, we estimate the initial conditions for the exposed, asymptomatic, quarantined, isolated, and recovered individuals to account for the possible actual number of cases. Now, it is easy to determine the initial susceptible population as \(S(0) = N(0) - (E(0) + Q(0) + A(0) + I(0) + J(0)) + R(0))\). Therefore, we can assume the initial conditions for the state variables used in model fitting to the real data as follows: \(E(0) = 5000\), \(Q(0) = 6000\), \(A(0) = 600\), \(J(0) = 24\) and \(R(0) = 500\).

The best fit to the monthly reported active cases via our model is displayed in Fig. 2. The values of the calculated and estimated parameters are summarized in Table 3 and it is to be noted that the values of the parameters used in this work are obtained from estimation, model fitting and from the literature and the unit of parameters (rate constants) is per month. Using the parameter values in Table 3, the basic reproductive value is \(R_0 = 1.0029\), which is greater than COVID-19 threshold value 1.

**Sensitivity analysis**

In this section, the sensitivity of model parameters with respect to the basic reproduction number \(R_0\) is performed and analyzed to limit COVID-19 cases for Ethiopia. To implement the sensitivity analysis, we calculate the sensitivity indices of the reproductive number, \(R_0\), to the parameters in the model. To investigate the most influential control parameters, we compute the normalized forward sensitivity indices, which tell us how crucial each parameter is to disease transmission and...
prevalence. Mathematically, using the approach in Chitnis et al. [43], we can define the normalized forward sensitivity index of a variable, $M$, which depends differentiably on a parameter, $p$, as

$$Y_p^M = \frac{\partial M}{\partial p} \times \frac{p}{M}.$$ 

From an explicit formula for $R_0$,

$$R_0 = \frac{\beta (r_1 + \delta_2 + \rho_1/\eta_1)(\zeta + q_1 + \mu) + (r_1 + \delta_1 + \mu)(\eta_1(\zeta + q_1 + \mu) + \eta_1 q_1)}{\eta_1 + \eta_2 + \rho_1(\zeta + q_1 + \mu) + (r_1 + \delta_1 + \mu)(\eta_1(\zeta + q_1 + \mu) + \eta_1 q_1)},$$

we derive an analytical expression for the normalized forward sensitivity indices of $R_0$ to different parameters involved in $R_0$. For example, $Y_\beta^R = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1$.

is the normalized forward sensitivity indices of $R_0$ with respect to the parameter $\beta$. Similarly, we can find for the remaining parameters. The sensitivity indices evaluated at the baseline parameter values are given in Table 3 were written in Table 4.

From Table 4, parameters with positive sensitivity indices implies that an increase in that parameter’s values will have a major effect on the frequency of the disease spread. For example, from $Y_\beta^R = 1$, we can see that increasing (or decreasing) the contact rate $\beta$ by 10%, increases (or decrease) the $R_0$ by 10%. On the other hand, parameters with negative sensitivity indices implies that an increase in the importance of these parameters would help to decrease the violence of the disease.

For example, the increasing (or decreasing) rate of exposed individuals become quarantined $\eta_1$ by 10%, decreases (or increase) the $R_0$ by 10%.

In addition, the contour plots of the basic reproduction number $R_0$ irrespective of different parameters of the model (1) are illustrated in Figs. 3–7 to investigate the effect of the control parameters on $R_0$. In Fig. 3, the contours shows that increasing the individuals joining the susceptible group from quarantine after the negative test of COVID-19 reduces the amount of basic reproduction number and, therefore, COVID cases, while the increase of coefficient of transmission increases the basic reproduction number $R_0$. Furthermore, in Fig. 4, we have seen that increasing the quarantine of exposed individuals reduces the amount of basic reproduction number $R_0$, but only this is not enough to control the COVID-19 outbreak. From our sensitivity analysis, we found that $R_0$ is most sensitive to the parameters $\beta$, $\tau$, $q_1$, $\eta_2$, $\eta_3$, and $\alpha$. This shows us the effectiveness of quarantine of exposed and isolation individuals via healthcare treatment in controlling the pandemic.

The contour in Fig. 5 show that when the rate of transfer of individuals becoming infected from quarantined is reduced together with transmission coefficient, then the amount of basic reproduction number $R_0$ is also reduced. From Fig. 6, we can see that increasing the quarantine of exposed individuals and the decrease of quarantined individuals showing symptoms reduces the basic reproduction number and, therefore, COVID-19 cases. From Fig. 7, we can see that decreasing the coefficient of transmission and modification factor for asymptomatic coefficient reduces the basic reproduction number and, consequently, the COVID-19 burden would be reduced.
Fig. 4. Contour plots of $R_0$ versus transmission rate ($\beta$) and exposed individuals become quarantined at rate $\eta_1$. All parameter values are given in Table 3 except the varied parameters.

Fig. 5. Contour plots of $R_0$ versus transmission rate ($\beta$) and quarantined individuals become showing symptoms at rate $q_1$. All parameter values are given in Table 3 except the varied parameters.

Fig. 6. Contour plots of $R_0$ versus quarantined individuals showing symptoms ($q_1$) and exposed individuals become quarantined at rate $\eta_1$. All parameter values are given in Table 3 except the varied parameters.

Fig. 7. Contour plots of $R_0$ versus transmission rate ($\beta$) and modification factor for asymptomatic exposed individuals at rate $r$. All parameter values are given in Table 3 except the varied parameters.

Fig. 8. Variation of $R_0$ with respect to coefficient of transmission $\beta$.

Further, Fig. 8 reveals the variation of $R_0$ with respect to transmission rate $\beta$. It can easily be observed that $R_0$ increases with an increase in transmission rate $\beta$ and after a certain value of $\beta$, $R_0$ becomes greater than 1. It implies that up to a certain value of $\beta$, disease-free equilibrium is stable and beyond that value of $\beta$, disease-free equilibrium becomes unstable.

Numerical simulation

In this section, the numerical simulation is performed to support the analytical results. To validate the local stability of DFE of model (1), we choose $\beta = 0.65$, and the other parametric values are similar to the baseline values given in Table 3. For $E_0^1$, the reproduction number $R_0 = 0.7387$ and DFE, $E_0^1 = (114963588, 0, 0, 0, 0, 0)$. The local stability of DFE for the model (1) is portrayed in Fig. 9. In this figure, the infective population gradually tend to disease-free equilibrium.

For EE we have, $E_0^1 = (1.149630984 \times 10^6, 2638, 5604, 382, 2789, 10303, 150773)$ and the reproduction number $R_0 = 1.0029$. The local stability of EE for the model (1) is portrayed in Fig. 10. In this figure,
all the distinct classes coexist in the population and approach endemic equilibrium.

The occurrence of the transcritical bifurcation at $R_0 = 1$ for the model (1) is illustrated in Fig. 11. Precisely, when $R_0 < 1$, the model (1) has no endemic equilibrium and the disease-free equilibrium is stable. When $R_0 > 1$, a stable endemic equilibrium appears and the disease-free equilibrium becomes unstable, i.e., the exchange of stability of the equilibrium (transcritical bifurcation) arises.

The global stability (Theorem 6) of the COVID-19-free equilibrium is illustrated in Fig. 12. From this figure, we observe that for different initial sizes of the infectious individuals in the population, all solution trajectories converge to the COVID-19-free equilibrium. In Fig. 13, it is observed that reducing the coefficient of transmission reduces the burden of infected individuals in hospital. Thus, this graphical suggests that the Ethiopian government takes progressive control measures to reduce the contact rate.

In Fig. 14, the effects of the modification factor on asymptomatic coefficient at different values are displayed. The projection shows that the cumulative number of individuals becoming infected is high when $r$ becomes high and becomes decreasing with low values of $r$, which minimize also the values of basic reproduction number less than one. As shown in Fig. 15, when the contact rate increases, the number of infected individuals become high and decreasing the value of $\beta$ decreases the number of infected population. This shows that the infected people with symptoms are significantly contributing the disease burden in the Ethiopian community.

From Fig. 16 we observe that increasing the rate of infected individuals exposed to healthcare treatment will decrease the transmission rate from the infected people to susceptible individuals. This implies that isolation of the infected human overall can reduce the risk of future COVID-19 spread in Ethiopia. In Fig. 17, we can see that when the rate of exposed individuals becoming quarantined is increased, then the number of individuals getting COVID-19 disease will decrease. Therefore, by increasing the quarantine of exposed individuals, we can minimize the spread of COVID-19 pandemic.

Discussion and conclusions

In this paper, we formulated and analyzed a nonlinear deterministic mathematical model to investigate the transmission dynamics of COVID-19. We first obtained the feasible region where the model is epidemiologically and mathematically well-posed. Then, the basic reproduction number, $R_0$ is computed using the next-generation matrix method. We then analyzed both local and global stability of the disease-free equilibrium point based on $R_0$. The analytical result shows that the COVID-19-free equilibrium point is locally as well as globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. The existence and stability of the endemic equilibrium point was determined by using the method stated in Castillo-Chavez and Song. Besides, the coexistence equilibrium point is locally asymptotically stable if $R_0 > 1$. From the epidemiological point of view, the disease can be managed if $R_0$ is less than unity. Otherwise, the disease can persist in the community. We also proved that the model exhibits forward bifurcation with the help of center manifold theory. Thus, the possible implication of this is that to reduce the disease burden in the community, policymakers have to work on bringing the reproduction number below one.

In ‘Parameter estimation’, the model parameters are fitted using COVID-19 infected data reported from March 13, 2020 to July 31, 2021 in Ethiopia. In ‘Sensitivity analysis’, the normalized sensitivity indices of $R_0$ show that the most sensitivity parameters are $\beta$ and $r$ with positive sign, which shows that decreasing the contact rate with infected and asymptomatic individuals reduces $R_0$ and so does the disease load.
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Fig. 12. Convergence of solution trajectories for (a) exposed, (b) asymptomatic, (c) infected and (d) hospitalized individuals at different initial values in line with Theorem 6 by using parameter values given in Table 3 except for \( \beta = 0.65 \), so that \( R_0 = 0.7387 < 1 \).

Again, the parameters \( \eta_1 \) and \( \sigma \) are the most sensitive parameters with negative sign. This indicates that the increase of quarantine of exposed and treating infected individuals decreases the contact rate which in turn reduces \( R_0 \) and so does the disease load.

We performed the numerical simulation in ‘Numerical Simulation’. The simulation result shows that the diseases always persist in the community whenever the reproduction number exceeds unity. This supports the fact that decrease in the transmission rate \( \beta \) reduces the value of \( R_0 \). Moreover, the decrease in the parameter \( \tau \) (the modified contact rate at which the asymptomatic individuals interact with susceptible) also shows a positive impact on \( R_0 \). In addition, an increase in the parameter \( \eta_1 \) which is the rate that exposed individuals become quarantined, reduces the value of \( R_0 \). Thus, the simulation result concludes that the spread of COVID-19 can be managed via minimizing the contact rate of infected and increasing the quarantine of exposed individuals. In the future, it is reasonable extending the present mathematical model via vaccination into an optimal control problem to investigate optimal intervention strategies.

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.

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Projections with varying effect of quarantine rate of exposed individuals at values of $\eta_1 = 0.81692$, $R_0 = 1.0029 > 1$, $0.82692$, $R_0 = 0.9994 < 1$, $0.83692$, $R_0 = 0.9987 < 1$, $0.84692$, $R_0 = 0.9981 < 1$.

Projections with varying effect of infected individuals become exposed to healthcare treatment at values of $\alpha = (0.98414; R_0 = 0.7804 < 1), 0.96314; (R_0 = 0.7959 < 1), 0.94214; (R_0 = 0.8122 < 1), 0.92114; (R_0 = 0.8292 < 1)$.

Fig. 15. Projections with varying effect of coefficient of transmission at values of $\beta = 0.7070; R_0 = 0.7955 < 1), 0.65; R_0 = 0.7386 < 1), 0.55; R_0 = 0.6250 < 1), 0.45; R_0 = 0.5114 < 1)$. 

Fig. 16. Projections with varying effect of infected individuals become exposed to quarantine at values of $\alpha = 0.98414; R_0 = 0.7804 < 1), 0.96314; (R_0 = 0.7959 < 1), 0.94214; (R_0 = 0.8122 < 1), 0.92114; (R_0 = 0.8292 < 1)$. 

Fig. 17. Projections with varying effect of quarantine rate of exposed individuals at values of $\eta_1 = 0.81692$, $R_0 = 1.0029 > 1$, $0.82692$, $R_0 = 0.9994 < 1$, $0.83692$, $R_0 = 0.9987 < 1$, $0.84692$, $R_0 = 0.9981 < 1$.

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