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Future implications of COVID-19 through Mathematical modeling

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A B S T R A C T

COVID-19 is a pandemic respiratory illness. The disease spreads from human to human and is caused by a novel coronavirus SARS-CoV-2. In this study, we formulate a mathematical model of COVID-19 and discuss the disease free state and endemic equilibrium of the model. Based on the sensitivity indexes of the parameters, control strategies are designed. The strategies reduce the densities of the infected classes but do not satisfy the criteria/threshold condition of the global stability of disease free equilibrium. On the other hand, the endemic equilibrium of the disease is globally asymptotically stable. Therefore it is concluded that the disease cannot be eradicated with present resources and the human population needs to learn how to live with corona. For validation of the results, numerical simulations are obtained using fourth order Runge-Kutta method.

Introduction

At the end of 2020, the most dangerous communicable viral disease COVID-19 appeared in China. The disease’s main source was the virus SARS-CoV-2. Once the disease entered the human community, its alarming transmission rate was un-ignorable, therefore the experts of all areas and disciplines focused to stop the further spreading of the disease. Different strategies have been adopted but humanity is still at risk of the disease. Close human contact is the main root of the disease transmission and this ultimately leads to a disaster in business and education.

The SARS-CoV-2 released from the mouth or nose of the COVID-positive individuals may directly hit another susceptible human if it is within range of 5–6 ft. Otherwise, the virus contaminates the available surfaces. As such these surfaces play the role of the virus carrier [1]. There are currently 257,007,274 confirmed cases and 5,156,403 deaths from the coronavirus COVID-19 outbreak as of November 21, 2021, worldwide [2].

The disease fatality rate is not high because almost 75% of the COVID-positive individuals recover without treatment. Since these individuals do not develop any symptoms of the disease, therefore they are not treated [3]. In 20%–25% cases tiredness, severe headache, losing taste and smell, dry cough or high fever appears on the victim, from two to fourteen days after the attack of virus [4,5]. Most of the cases of COVID-19 are mild and recover in two weeks, however, in critical cases, the recovery may take 21 to 42 days [6]. The genetic features, history, and clinical features of the disease can be found in [7–12].

Round the world, all well-wishers of humanity are very much concerned about the future forecast of COVID-19. If the disease maintains its present status, humanity may face starvation and illiteracy. To forecast the future of the disease different mathematical models including [13–16] have been presented recently. The studies focussed on different dynamics of the disease. For a new update about modeling in fractional calculus, we refer to [17–24].

Motivating from the work presented in [25], where four control variables were used for the optimal control of COVID-19. We, in this study, formulate the mathematical model of the disease and discuss both the endemic equilibrium and disease free equilibrium of the model. Control strategies are designed based on sensitivity indexes of the transmission parameters. The results show that the conditions required for the globally asymptotically stable eradication of the disease do not hold. Furthermore, the endemic equilibrium of the disease is globally stable. Based on these results the study concludes that with the present resources the disease cannot be defeated, therefore humanity needs to learn how to live with corona.

Our paper is organized as: Section “Introduction” gives the brief introduction of COVID-19 and the contributions of different researchers. In Section “Model Formulation of COVID-19” different stages of the
disease and its mathematical modeling is discussed. Section “Analysis of the model” is concerned with analysis of the model and addresses invariant region, well-posedness and reproduction number. Section “Threshold condition” addresses threshold condition for global stability of disease free equilibrium. Section “Strategies for COVID-19 control” focusses sensitivity indexes of the parameters, control strategies and the results of the strategies obtained with help of numerical simulations. In Section “Endemic equilibrium”, we address global stability of endemic equilibrium of the model and the conclusion.

Model formulation of COVID-19

To formulate the mathematical model of COVID-19, we consider different stages of the disease and accordingly divide the human population and surfaces in different compartments as shown below:

- $W_i$ denotes surfaces/stuff contaminated with SARS-CoV-2.
- $S$ denotes the class of susceptible human population.
- $R$ denotes the Recovered class of human population.
- $E$ denote the surfaces or stuff that the human class can touch on daily basis.
- $I$ denotes the class of those individuals who have caught the virus but do not yet show any symptoms of the disease.
- $I_1$ denote the class of those individuals who have completed the incubation period and have got the symptoms of the disease.
- $I_2$ denote the class of those individuals who have completed the incubation period but do not show any symptoms of the disease.
- $I_3$ denote the class of those individuals whom have got breathing problem and are put ventilator, the vent Bol class.

Twenty percent of the susceptible individuals after getting an infection develop the symptom of the disease and the rest 80% remain asymptomatic and are accordingly placed in $(I_1)$ and $(I_2)$ respectively. Only five percent of these infectious individuals may face problems in inhalation and are kept in hospital for ventilation and are placed in Vent Bol class. About forty-nine percent of individuals in Vent Bol class die due to COVID-19. To reduce the speed of transmission of the infection, the infectious individuals are isolated. The herd immunity acquired by an individual after recovery from COVID-19, is not permanent. After the infectious individuals are isolated. The herd immunity acquired due to COVID-19. To reduce the speed of transmission of the infection, halation and are kept in hospital for ventilation and are placed in Vent

\[ S = I_0 - (\beta_1 I_1 + \gamma I_1)S + \beta_2 W_S + R - \mu S \]
\[ E = (\beta_1 I_1 + \gamma I_1)S + \beta_2 W_S - (\eta_1 + \mu)E \]
\[ I_1 = \eta_1 (1 - \delta) I - (\mu + I)I_1 \]
\[ I_2 = k(a_1 I_2 + \beta I_1) - (a_1 I_2 + (D_2 + \mu)I_2 \]
\[ I_3 = \delta \mu I - (k + \mu)I \]
\[ R = k(1 - \mu)I + a_1 I_1 + k(1 - a_2)I_2 - (\beta_1 + \mu)R \]
\[ W_i = A - (\theta_1 I_2 + \theta_2 I_2)W_i - e_s W_i \]
\[ W_i = (\theta_1 I_2 + \theta_2 I_2)W_i - (e + s)W_i \]

The flowchart of COVID-19 is shown in Fig. 1 below.

Table 1 contains the values of the parameters.

Analysis of the model

This section discusses three properties of the model; Invariant region, Disease-Free-Equilibrium (DFE) and the Basic Reproduction Number.

Invariant region

Let $N$ be the total population of humans and $Z$ be the total density of stuff in human use. Adding all the classes of human population together and the compartments in stuff together, we have $N$ and $Z$ as under:

\[ \dot{N} = I_0 - \mu N - D_2 I_2 \]

\[ \dot{Z} = A - e_z Z - \epsilon W_i \]

Solving Eqs. (2) and (3), we obtain the following results:

\[ N \leq N(0)e^{-\mu t} = \frac{I_0}{\mu} \quad \text{when } t \to \infty \]

\[ Z \leq Z(0)e^{-\epsilon x^t} = \frac{A}{\epsilon x} \quad \text{when } t \to \infty \]

Above two inequalities shows that both the trajectories representing human population and the density of stuff, are forward bounded. Thus the following result is proved:

Proposition 3.1 ([35]). The following region $\Phi$, $\Phi = (S, W_i, E, I_1, I_2, I_3, W_i, R) \in \mathbb{R}_8 \ni N \leq \frac{I_0}{\mu} \leq \frac{A}{\epsilon x}$ is +ve invariant domain. Furthermore the trajectories of human population and stuff density are bounded above. Hence the proposed model is well posed.

Disease reproduction number

Communicable diseases spread generally by the contact of infected and non-infected individuals. How many susceptible individuals, an infectious individual do infect, is called $R_0$ or the disease reproduction rate and is fire by next generation matrix ([27,28,36]).

Let $X = (E, I_1, I_2, W_i)^T$ and $X = (W_i, R, S)^T$

Following [37], we find $f(x)$ and $\psi(x)$ and their derivatives at the disease-free equilibrium point as:

\[ f(x) = \begin{pmatrix}
\beta_1 (I_1 + \gamma I_1) S + \beta_2 W_S \\
0 \\
0 \\
(\theta_1 I_1 + \theta_2 I_2)W_i
\end{pmatrix}
\]

and

\[ \psi(x) = \begin{pmatrix}
\eta_1 E + \mu E \\
(1 - \delta) \eta_1 E - (k + \mu) I_1 \\
\beta k I_1 + a_2 k I_2 - (a_1 + D_2 + \mu) I_2 \\
\delta \eta_1 E - (k + \mu) I_2 \\
(\epsilon + x) W_i
\end{pmatrix}
\]

\[ \psi(x) = \begin{pmatrix}
\eta_1 E + \mu E \\
(1 - \delta) \eta_1 E - (k + \mu) I_1 \\
\beta k I_1 + a_2 k I_2 - (a_1 + D_2 + \mu) I_2 \\
\delta \eta_1 E - (k + \mu) I_2 \\
(\epsilon + x) W_i
\end{pmatrix}
\]

\[ FV^{-1} = \begin{pmatrix}
\frac{\Gamma_0(1-\delta) \eta_1}{\mu(\eta_1 + \mu)(k + \mu)} \\
\frac{\Gamma_0(\eta_1) \beta_1}{\mu(\eta_1 + \mu)(k + \mu)} \\
\frac{\Gamma_0(\eta_1) \beta_1}{\mu(\eta_1 + \mu)(k + \mu)} \\
\frac{\Gamma_0(\eta_1) \beta_1}{\mu(\eta_1 + \mu)(k + \mu)} \\
\frac{A(1-\delta) \eta_1}{e_x(\eta_1 + \mu)(k + \mu)} + \frac{\Lambda(\eta_1) \beta_1}{e_x(\eta_1 + \mu)(k + \mu)} + \frac{\eta_1 E}{\mu(\eta_1 + \mu)(k + \mu)}
\end{pmatrix}
\]

$R_0$ is the dominant eigenvalue of $FV^{-1}$ and is given in Box I.

$R_0$ denote the transmission of the disease both from human to human and contaminated surfaces to human. The transmission from human to human is denoted by $R_0^H$ and from contaminated surfaces to humans by $R_0^W$.

\[ R_0 = \frac{\Gamma_0}{\mu(\eta_1 + \mu)(k + \mu)} \left[ (1 - \delta) \eta_1 \beta_1 + \delta \eta_1 \gamma \beta_2 \right] \]
In this section, we derive threshold condition for global stability for DFE (disease free equilibrium) of the system (1), using theorem 3.1 of [38].

Let $P = (S, W, E, I_1, I_2, I_3, W_e, R)^T$ be the class of state variables of the proposed model, $P_i = (W_i, E, I_1, I_2, I_3, W_e, R)^T$ be the class of infected population and contaminated surfaces, and $P_i = (S, W_e, R)^T$ be the class of susceptible and recovered human population, and non-contaminated stuff/surfaces.

**Theorem 4.1.** Given the sub-system:

$$
\begin{align*}
\dot{S} &= \Gamma_h + \beta_1 R - (\beta_1 (I_1 + \gamma I_2) S + \beta_2 W_e S) - \mu S \\
\dot{R} &= k I_1 (1 - \beta) + \alpha_1 I_2 + (1 - \alpha_2 k) I_2 - (\mu + \beta_1) R \\
W_i &= A - (\theta_2 I_2) W_i - (\theta_1 I_1) W_i - \varepsilon_X W_i
\end{align*}
$$

System (7) is GAS at the domain $\Delta$, where

$$
\Delta = \{ P \in \Phi; P_i = 0, P_i \neq 0 \}.
$$

---

### Table 1

Table of the values of parameters.

| Notation | Parameter definition | Value | Source |
|----------|----------------------|-------|--------|
| $\Gamma_h$ | Humans recruitment rate | 0.0015875 day$^{-1}$ | [27] |
| $\mu$ | Humans natural mortality rate | 0.00004 day$^{-1}$ | [27, 28] |
| $\alpha_0$ | $\alpha_0$ is the incubation period of human | 0.1923 day$^{-1}$ | [16] |
| $\theta_1$ | The transition period at $I_1$ | 2-6 weeks | [29] |
| $\delta_2$ | The ratio of asymptomatic moving to vent bol | 2% | [31, 32] |
| $\beta_1$ | The transmission rate of infection from $I_1$ to $S$ | 0.65 day$^{-1}$ | [16] |
| $\beta_2$ | The transmission rate of infection from stuff | 0.165 day$^{-1}$ | [16] |
| $\eta_1$ | The shedding coefficient of $I_1$ on $W$ | 0.5 | [16] |
| $\eta_2$ | The shedding coefficient of $I_1$ on $W$ | 0.5 | [16] |
| $\gamma$ | The multiple of the transmissibility of $I_2$ to that of $I_i$ | 0.5 | [16] |
| $D_2$ | Disease induced death ratio of vent bol | 49% | [33] |
| $\delta$ | The ratio of symptomatic moving to vent bol | 5% | [34] |
| $\beta_3$ | The transmission rate of infection from stuff | 0.165 day$^{-1}$ | [16] |
| $\varepsilon_X$ | Stuff/food items expiry | 0.835 day$^{-1}$ | [25] |
| $\alpha$ | Per capita stuff supply to market | 0.635 day$^{-1}$ | [25] |

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**Box I.**

$$
R_0 = \frac{\Gamma_h (1 - \delta) \eta_1 \beta_1 + \Gamma_h \delta \eta_1 \beta_1}{\Gamma_h (1 - \delta) \eta_1 \beta_1 + \Gamma_h \delta \eta_1 \beta_1} \left[ (1 - \delta) \eta_1 \beta_1 + \delta \eta_1 \beta_2 \right]
$$

So,

$$
R_0^W = \frac{A \beta_2 \Gamma_h}{\varepsilon_X \mu (\varepsilon + \varepsilon_X) (\theta_1 + \mu) (k + \mu)} \left[ (1 - \delta) \eta_1 \beta_1 + \delta \eta_1 \beta_2 \right]
$$

$$
R_3 = \frac{R_0^W + \sqrt{(R_0^W)^2 + 4 R_0^W}}{2}
$$
Proof. If $P_j = 0$, the system;

\[
\begin{align*}
\dot{S} &= \Gamma_h + \beta_1 R - (\beta_1 (I_1 + I_2) S + \beta_2 W S) - \mu S \\
\dot{R} &= (1 - \beta_1) k I_1 + a_1 I_1 + (1 - a_2) k I_2 - (\mu + \beta_1) R \\
\dot{W}_i &= \mu - (\delta I_1 + \theta_1) W_i - \epsilon_X W_i
\end{align*}
\]

reduces to the form:

\[
\begin{align*}
\dot{S} &= \Gamma_h + \beta_1 R - \mu S \\
\dot{R} &= -(\mu + \beta_1) R \\
\dot{W}_i &= A - e_x W_i
\end{align*}
\]

OR

\[
P_j = M_j(P) + \mathcal{K}_s
\]

Where

\[
M_j = \begin{pmatrix}
-\mu & \beta_1 & e_x \\
0 & -(\mu + \beta_1) & 0 \\
0 & 0 & -e_x
\end{pmatrix}, \quad \mathcal{K}_s = \begin{pmatrix}
\Gamma_h \\
0 \\
A
\end{pmatrix}
\]

Here all the entries $M_{i,(n)}$ are $-ve$ for $n\neq 0$. Therefore The Disease Free equilibrium $(\bar{S}, \bar{R}, \bar{W}_i, \ldots) = (0, 0, 0, 0)$ is stable globally. □

The sub-system:

\[
\begin{align*}
\dot{E} &= (\gamma I_2 + \Gamma_1) S + \beta_2 W S - (\mu E + \eta_1) E \\
\dot{I}_1 &= \mu (\gamma I_2 + 1) - (\mu + \beta_1) I_1 \\
\dot{I}_2 &= \mu (\gamma I_2 + 1) - (\mu + \beta_2) I_2 \\
\dot{W}_i &= (\theta_1 I_1 + \theta_1 I_2) W_i - (e_x) W_i
\end{align*}
\]

is simply written as:

\[
P_j = G_J(P) P_j.
\]

Theorem 4.2. Given the sub-system (10). The matrix $G_J$, as defined in (11), is Metzler and irreducible, for all $P \in \Phi$.

Proof. Consider the system (11):

\[
P_j = G_J(P) P_j
\]

With

\[
G_J(P) = \begin{pmatrix}
-(\eta_1 + \mu) & \beta_1 S & 0 & \gamma \beta_1 S & \mu S \\
(1 - \delta) \eta_1 & -(\mu + \beta_1) & 0 & 0 & 0 \\
0 & \beta k & -(a_1 + D_2 + \mu) & a_2 k & 0 \\
0 & 0 & -\delta_1 & 0 & 0 \\
0 & 0 & 0 & -\delta_2 & -(\epsilon + e_x)
\end{pmatrix}
\]

For $m=n$ the entries $G_{m,n}$ are $-ve$ and non-negative for $f$ or $m \neq n$. Hence $G_J(P)$ is Metzler and irreducible for all $P \in \Phi$. □

Theorem 4.3. There always exist $G_J^\prime$, for matrix $G_J$ as defined in Eq. (11), such that:

\[
G_J(P) \leq G_J^\prime(P) \text{ for } P \in \Phi.
\]

Also

\[
G_J^\prime \in \mathcal{N} = \{G_J(P), P \in \Phi\} \quad \text{and} \quad \mathcal{N} = \mathcal{N}_{max}^g.
\]

\[
\phi(G_J^\prime) \leq 0.
\]

\[
\phi \text{ being dominant eigenvalue of } G_J^\prime. \quad \text{Where } G_J^\prime \text{ is the upper bound matrix of } G_J.
\]

Proof. Since

\[
S \leq S_0 \text{ and } W_i \leq W_i^0.
\]

Then obviously the matrix $G_J^\prime(P)$, given below, is the upper bound of the matrix $G_J(P)$,

\[
G_J^\prime(P) = \begin{pmatrix}
-(\eta_1 + \mu) & \beta_1 S & 0 & \gamma \beta_1 S & \mu S \\
1 - \delta \eta_1 & -(\mu + \beta_1) & 0 & 0 & 0 \\
0 & \beta k & -(a_1 + D_2 + \mu) & a_2 k & 0 \\
0 & 0 & -\delta_1 & 0 & 0 \\
0 & 0 & 0 & -\delta_2 & -(\epsilon + e_x)
\end{pmatrix}
\]

Simplifying the above matrix and putting $S_0 = \frac{s}{e_x}$ and $W_i^0 = \frac{W_i}{e_x}$, we have:

\[
G_J^\prime(P) = \begin{pmatrix}
-p_1 & \frac{\Gamma_1}{\mu} & 0 & \gamma \frac{\Gamma_1}{\mu} & \frac{\Gamma_2}{\mu} \\
0 & \beta k & -(a_1 + D_2 + \mu) & a_2 k & 0 \\
0 & 0 & -\delta_1 & 0 & 0 \\
0 & 0 & 0 & -\delta_2 & -(\epsilon + e_x)
\end{pmatrix}
\]

The above upper bound matrix $G_J^\prime(P)$ is obtained if $S = N$. And the model attains the status of $N = N$ at DFE point. Hence $G_J^\prime(P)$ is the upper bound matrix obtained at disease free equilibrium.

The Jacobian of the system (10) is

\[
J_I = \begin{pmatrix}
-(\eta_1 + \mu) & \beta_1 S & 0 & \gamma \beta_1 S & \mu S \\
(1 - \delta \eta_1) & -(\mu + \beta_1) & 0 & 0 & 0 \\
0 & \beta k & -(a_1 + D_2 + \mu) & a_2 k & 0 \\
0 & 0 & -\delta_1 & 0 & 0 \\
0 & 0 & 0 & -\delta_2 & -(\epsilon + e_x)
\end{pmatrix}
\]

Matrix $J_I$ at disease free equilibrium is given by:

\[
\begin{pmatrix}
-p_1 & \frac{\Gamma_1}{\mu} & 0 & \gamma \frac{\Gamma_1}{\mu} & \frac{\Gamma_2}{\mu} \\
0 & \beta k & -(a_1 + D_2 + \mu) & a_2 k & 0 \\
0 & 0 & -\delta_1 & 0 & 0 \\
0 & 0 & 0 & -\delta_2 & -(\epsilon + e_x)
\end{pmatrix}
\]

Clearly $G_J^\prime(P)$ is equal to the block of the Jacobian $J_I$ at the DFE. Which proves Eqs. (13) and (12). □

To prove Eq. (14), we state the following theorem:

Theorem 4.4. For the matrix

\[
G_J^\prime(P) = \begin{pmatrix}
-p_1 & \frac{\Gamma_1}{\mu} & 0 & \gamma \frac{\Gamma_1}{\mu} & \frac{\Gamma_2}{\mu} \\
0 & \beta k & -(a_1 + D_2 + \mu) & a_2 k & 0 \\
0 & 0 & -\delta_1 & 0 & 0 \\
0 & 0 & 0 & -\delta_2 & -(\epsilon + e_x)
\end{pmatrix}
\]

\[
\phi(G_J^\prime(P)) \leq 0, \; \text{if} \; \tau < 1.
\]

Where $\tau$ is given by:

\[
\tau = \frac{\delta \eta_1 \beta \Gamma_2 \Gamma_1 A}{e_x} + \frac{(1 - \delta) \eta_1 \beta \Gamma_2 \Gamma_1 A}{e_x} + \frac{(1 - \delta) \eta_1 \beta \Gamma_2 \Gamma_1 A}{e_x}
\]

And

\[
\begin{align*}
&n_1 = \beta_1 \Gamma_1, \\
&n_2 = \gamma \beta_1 \Gamma_1, \\
&n_3 = \beta_2 \Gamma_1, \\
&p_1 = \eta_1 + \mu, \\
&p_2 = k + \mu, \\
&p_3 = \mu + e_x, \\
&d_1 = (1 - \delta) \eta_1, \\
&d_2 = \delta \eta_1.
\end{align*}
\]

Proof. Let $M$, $N$, $O$ and $P$ be the decomposition of the matrix $G_J^\prime$, so that;

\[
P = \begin{pmatrix}
-p_1 & 0 & 0 \\
0 & -p_2 & 0 \\
0 & \beta k & -p_3
\end{pmatrix}
\]

\[
M = \begin{pmatrix}
-p_1 & n_1 & 0 \\
0 & -p_2 & 0 \\
0 & \beta k & -p_3
\end{pmatrix}
\]

\[
O = \begin{pmatrix}
-p_1 & n_1 & 0 \\
0 & -p_2 & 0 \\
0 & \beta k & -p_3
\end{pmatrix}
\]

\[
N = \begin{pmatrix}
-p_1 & n_1 & 0 \\
0 & -p_2 & 0 \\
0 & \beta k & -p_3
\end{pmatrix}
\]
We need to show that $G_1^p$ is stable. For this we show that $P - OM^{-1} N$ and $M$ are stable.

Since for $i = j$ all the entries $M_{i,j}$ are negative. Therefore all eigenvalues of $M$ are $-i\omega$ and the entries $M_{i,j} \geq 0$ for $i \neq j$. Hence $M$ is Metzler stable.

To show that $G_1^p$ is stable, we need to prove that $D = P - OM^{-1} N$ is stable. Applying Routh–Hurwitz criteria [39], we obtain the following inequality:

$$D \text{ is stable only if } \frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$$

$$D \text{ is stable only if } \frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$$

$$D = \text{stable only if } \frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$$

$$\Rightarrow D \text{ is stable only if } \frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$$

$$M \text{ and } D \text{ are stable and thus we have proved that}$$

$$G_1^p \text{ is stable only if } \frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$$

$$\text{That is (14) is satisfied if } \frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$$

In the above discussion we have proved all the five assumptions of global stability of disease free equilibrium, so we claim the following theorem:

**Theorem 4.5.** Disease free equilibrium of COVID-19 is globally stable subject to the condition $\frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$, where

$$f = \frac{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2}{\delta_1 \beta_1 \gamma_1 \delta_2 \alpha_2} + \frac{(1-\delta_1) \beta_1 \gamma_1 \delta_1}{(1-\delta_1) \beta_1 \gamma_1 \delta_1} < 1 < 0 \quad (15)$$

**Strategies for COVID-19 control**

In Theorem 4.5, it is concluded that the disease free state obtained with the help of different interventions, is globally stable subject to the condition $f < 1$. We, therefore, first need to decide the interventions and control strategies. In this section we discuss

- The sensitivity/role of different parameters in the transmission of COVID-19, and decide intervention.
- Formulation of control strategies based on decisive parameters.
- The results of control strategies with help of numerical simulations.

**Sensitive parameters**

The change observed in the dependent variable $R$ by changing the value of independent parameter $\beta$ is called sensitivity of $R$ for parameter $\beta$, denoted by $Y_\beta^R$ and is given by:

$$Y_\beta^R = \frac{\partial R}{\partial \beta} \beta \frac{\partial R}{\partial \beta}. \quad (2)$$

The result obtained from above equation is called the sensitivity index of $\beta$. For further detail on sensitivity the reader is refer to [25,27,40].

Using the above formula we have obtained the following Table 2 of sensitivities, $P$ denotes parameter, $V$ the value of parameter and $I$ the index of parameter:

| $P$ | $V$ | $I$ |
|-----|-----|-----|
| $\delta$ | 0.75 | -0.0037 |
| $\beta_1$ | 0.65 | 0.0014 |
| $\beta_2$ | 0.165 | 0.0015875 |
| $k$ | 0.041429 | -0.4966 |
| $\mu$ | 0.00004 | -0.5015 |
| $\eta_1$ | 0.1923 | 0.000333 |
| $r$ | 0.5 | 0.00053591 |

The magnitude of the sensitivity index of a parameter represents the role of the parameter in the transmission of COVID. Highest the magnitude of sensitivity index of parameter, greatest the role of the parameter in disease transmission. However, some parameters have high sensitivity indexes yet we do not consider these parameters for intervention or decisive parameters. Because these parameters cannot be addressed so simply, for example, human birth rate or mortality rate.

**Decisive parameters**

The sensitivity index of non of the parameters shown in Table 2 is zero. So all these parameters have got a role in the transmission of COVID-19. The value of the parameter is directly proportional to the rate of disease transmission, $R_0$, if the sign of its sensitivity index is $+$ve and inversely proportional if the sign is $-$ve. For example, as shown in the table, $k$, the transition period of an individual at the stage $I_1$, has got sensitivity index of $-0.99$. Since the index is negative, hence an increase in the value of the parameter will cause a decrease in the transmission rate $R_0$. We select parameters $k, \beta_1, \beta_2, \theta_1, \theta_2, \eta_1, \epsilon, \delta_1, r, e_1$ fit for intervention or the decisive parameters.

We intervene in the above defined decisive parameters according to the available resources and formulate control strategies. Accordingly, the following 3 strategies are designed, as shown in Table 3. Due to the high transmission rate of COVID-19, the increase in densities of infected classes is too rapid to be accommodated by the hospitals. The proposed three strategies would help in flattening the curve of infection of COVID-19, resulting in a reduction of the burden of the patients in hospitals.

**Results of control strategies**

In the following numerical simulations we have used Runge–Kutta method. The effect of control strategies on control of COVID-19 is shown in Figs. 2, 3, 4, 5, 6 and 7. To facilitate the comparison of the strategies, we have displayed the results of all the three control strategies in the same figure, for each targeted particular class.

For global stability of the disease free state the following two conditions must hold:

- 1: The densities of the infected classes must reduce to zero.
- 2: The value of $f$ must be less than 1, for the selected values of decisive parameters.

All the above figures show that the densities of the targeted infected classes have reduced to zero. Thus satisfying condition 1 of global stability. But the second condition of global stability, $f < 1$, is not satisfied as shown in Table 3. This shows that the disease-free state so obtained is not globally stable and hence the disease cannot be permanently eliminated with help of present tools of elimination. Since the disease is highly communicable it therefore appears again in the disease free zones. As such the disease attains the endemic mood.

In the following section, we show that the endemic equilibrium is globally asymptotically stable.
Table 3
Control strategies.

| Strategy  | \( k \)   | \( \beta_1 \) | \( \beta_2 \) | \( \delta_1 \) | \( \delta_2 \) | \( \eta_1 \) | \( \delta \) | \( e \) | \( c_x \) | \( r \) | \( R_0 \) | \( f \) |
|-----------|------------|---------------|---------------|---------------|---------------|-------------|------|------|--------|------|-------------|------|
| Strategy 1| 0.041429   | 0.65          | 0.165         | 0.5           | 0.5           | 0.1923      | 0.75 | 0.1  | 0.835  | 0.5  | 3.5210      | 452.9218 |
| Strategy 2| 0.61429    | 0.35          | 0.135         | 0.3           | 0.3           | 0.1723      | 0.55 | 0.3  | 0.89   | 0.03 | 0.5809      | 12.1139 |
| Strategy 3| 0.81429    | 0.15          | 0.105         | 0.1           | 0.1           | 0.1523      | 0.25 | 0.5  | 0.98   | 0.05 | 0.2323      | 5.7967 |

Fig. 2. The graph represents the comparison of the strategies regarding exposed human population.

Fig. 3. The graph represents the comparison of the strategies regarding infectious human population.

Fig. 4. The graph represents the comparison of the strategies regarding the density of vent bol human population.
Fig. 5. The graph represents the comparison of the strategies regarding asymptomatic infectious human population.

Fig. 6. The graph represents the comparison of the strategies regarding recovered human population.

Fig. 7. The graph represents the comparison of the strategies regarding the density of the stuff stained/shedded with corona virus.

Endemic equilibrium

Let \((S^*, E^*, I_1^*, I_2^*, I_v^*, R^*, W_s^*, W_i^*)\) be the endemic equilibrium of model (1). Then

\[
S^* = \frac{(\eta_1 + \mu)(k + \mu)I_1}{\beta_r((1-\delta)\eta_1 + \delta\eta_2)I_1 + ((1-\delta)\eta_1\beta_sW_s)}
\]

\[
E^* = \frac{(k + \mu)I_1}{\eta_1(1-\delta)}
\]

\[
I_1^* = \frac{\delta I_1}{(1-\delta)}
\]

\[
I_2^* = \frac{k(1-\delta)\beta + ka_2\delta}{(1-\delta)(a_2 + D_1 + \mu)}I_1
\]

\[
I_v^* = \frac{k(1-\delta)\beta + ka_2\delta}{(1-\delta)(a_2 + D_1 + \mu)}I_1
\]
We choose the function $\nu = (e + e_X)^{-1}$.

To check the global stability of endemic equilibrium we use the concept used in [41].

\textbf{Theorem 6.1.} If $R_0 > 1$, the endemic equilibrium $(S^*, E^*, I_1^*, I_2^*, R^*, W^*)$ is globally asymptotically stable.

\textbf{Proof.} Consider the following sub-system of (1)
\[
\begin{aligned}
S &= I_1^* + \beta_W\rho (I_1^* + \gamma I_2^*) + \beta_TW S - \mu S \\
E &= (\beta_W(I_1^* + \gamma I_2^*) + \beta_TW S) - \eta E - \mu E \\
I_1 &= (1 - \delta)\eta E - (k + \mu)I_1
\end{aligned}
\]
(16)

The Jacobian matrix of system (16) is
\[
J = \begin{pmatrix}
-\beta_W I_1^* & -\beta_W I_1^* & 0 \\
\beta_W I_1^* & \beta_W I_1^* & -\eta I_1^* \\
0 & \beta_W I_1^* & -(k + \mu) \\
\end{pmatrix}
\]
and its second additive compound matrix is
\[
J^{[2]} = \begin{pmatrix}
-m & \beta_W S & \beta_W S \\
0 & \beta_W I_1^* & -(2k + \eta) \\
\end{pmatrix}
\]

Where
\[
m = \beta_W(I_1^* + \gamma I_2^*) + \beta_TW + 2\mu + \eta_i \\
n = \beta_W(I_1^* + \gamma I_2^*) + \beta_TW + 2\mu + k
\]

We choose the function $\nu(x)$ as:
\[
\nu(x) = P(S, E, I_1) = \frac{d\nu}{d\chi} \begin{pmatrix} 1 & E \end{pmatrix},
\]
then
\[
P \nu^{-1} = \begin{pmatrix}
E & -E I_1^* - E I_1' \\
I_1' & I_1
\end{pmatrix} = \begin{pmatrix} E & -E I_1^* - E I_1' \\
I_1' & I_1
\end{pmatrix} = \begin{pmatrix} E & -E I_1^* - E I_1' \\
I_1' & I_1
\end{pmatrix}
\]

Let
\[
D = P \nu^{-1} + P J^{[2]} \nu^{-1} = \begin{pmatrix} D_{11} & D_{12} \\
D_{12} & D_{22}
\end{pmatrix}
\]

\[
D_{11} = -(\beta_W(I_1^* + \gamma I_2^*) + \beta_TW + 2\mu + \eta_i) \\
D_{12} = (\frac{I_1}{E_1} - \beta_W S) \\
D_{21} = \begin{pmatrix} E & (1 - \delta)\eta_1 \\
0 & \beta_W I_1^* + \gamma I_2^* + \beta_TW
\end{pmatrix}
\]
\[
D_{22} = \begin{pmatrix} E & -E I_1^* - (2\mu + k) \\
I_1' & I_1
\end{pmatrix}
\]

Let $W = \{u, v, \nu\}$ be a vector in $\mathbb{R}^3$. The $\|W\|$ norm of $V$ defined by:
\[
\|W\| = \max \{|u|, |v|, |\nu|\}
\]

Then following [42], we choose
\[
\mu_1(D) \leq \rho(W_1, W_2) \text{ with respect to the norm defined above.}
\]

Then following [42], we choose
\[
\mu_1(D) = \rho(W_1, W_2) \text{ with respect to the norm defined above.}
\]

For future work it is recommended to address the effect of vaccination on the global stability of disease free state.

\textbf{Conclusion}

In this work, we discussed different dynamics of the COVID-19 pandemic. The sensitivity test of the reproduction number shows that non of the parameters and particularly easily addressable parameters, have got dominant role in disease transmission. Therefore the control of this disease is comparably tough but not disappointing, however, permanent eradication of the disease with present resources does not seem effective. What would be the long last effect of vaccination is still awaited.

Our result shows that a disease-free state can be achieved but the state is not globally stable. This means that reinfection and new outbreaks of the disease in the community may happen time and again. The endemic equilibrium of the disease is globally stable.

As a conclusion, it is recommended that the public shall be given awareness about 'how to live with corona using non-pharmaceutical approach'.

\textbf{CRedT authorship contribution statement}

\textbf{Muhammad Zamir:} Writing – original draft, Methodology, Conceptualization.

\textbf{Fawad Nadeem:} Writing – original draft, Methodology.

\textbf{Manar A. Alqudah:} Fromal analysis, Conceptualization, Funding acquisition, Writing – review & editing.

\textbf{Thabet Abdeljawad:} Writing – review & editing, Supervision, Validation, Formal analysis.
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