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Controlling of pandemic COVID-19 using optimal control theory

Shahriar Seddighi Chaharborj a,b, Sarkhosh Seddighi Chaharborj b,c, Jalal Hassanzadeh Asl a,b,,* and Pei See Phang c,1

a Department of Mathematics, Faculty of Science, Tabriz Branch, Islamic Azad University, Tabriz, Iran
b School of Mathematics and Statistics, Carleton University, Ottawa K1S 5B6, Canada
c Department of Mathematics, Faculty of Science, Universiti Putra Malaysia, 43400 UPM, Malaysia

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ABSTRACT

In 2019, a new infectious disease called pandemic COVID-19 began to spread from Wuhan, China. In spite of the efforts to stop the disease, being out of the control of the governments it spread rapidly all over the world. From then on, much research has been done in the world with the aim of controlling this contagious disease. A mathematical model for modeling the spread of COVID-19 and also controlling the spread of the disease has been presented in this paper. We find the disease-free equilibrium points as trivial equilibrium (TE), virus absenteeism equilibrium (VAE) and virus incidence equilibrium (VIE) for the proposed model; and at the trivial equilibrium point for the presented dynamic system we obtain the Jacobian matrix so as to be used in finding the largest eigenvalue. Radius spectral method has been used for finding the reproductive number. In the following, by adding a controller to the model and also using the theory of optimal control, we can improve the performance of the model. We must have a correct understanding of the system i.e. how it works, the various variables affecting the system, and the interaction of the variables on each other. To search for the optimal values, we need to use an appropriate optimization method. Given the limitations and needs of the problem, the aim of the optimization is to find the best solutions, to find conditions that result in the maximum of susceptibility, the minimum of infection, and optimal quarantination.

Introduction

The order Nidovirales includes the families Coronaviridae, Roniviridae, and Arteriviridae; a family of Coronaviridae is called coronavirus. Being atomic in size (65–125 nm in diameter), Coronavirus contains a single-stranded RNA as a nucleic material; size confining from 26 to 32kbs in length (Fig. 1) [1] COVID-19, In particular is a family of RNA Beta virus in Nidoviral order. The COVID-19 pandemic, also known as the coronavirus pandemic is a worldwide public health emergency which has been unrivaled in the recent time. Epidemics of infectious disease are generally caused by several factors that in case of being out of control might cause the ongoing death of hundreds or thousands of people all over the world. Coronavirus is a family of viruses that got its name from its appearance; that is, Coronavirus is derived from Latin corona, meaning crown. This virus was initially introduced in Wuhan, China; Huanan seafood wholesale market that was a live animal and seafood market was identified as a possible point of origin of COVID-19 [2,3]. It is the seventh Corona virus discovered that causes illness in humans. Some researchers believe that the virus transmitted from either bats or snakes to humans. Currently, scientists suspect bats as the source of COVID-19 [2]. Bats contain that highest proportion of mammalian viruses that are likely to infect people. When viruses transfer from one species to another species the epidemic occur. The cheeks that hostess the severe torrid respiratory syndrome corona virus 2 (SARS-CoV-2) is probably bat, containing 96% similar at the total-genome sequence level [2].

What epidemiological models have certainly given prominence to, is keeping proper social distances; that is this simple matter is considered as a key factor highly effective in decreasing the epidemic [4–7]; though yet there are many other points about Covid-19 pandemic which are not discovered yet [4]. According to the recent discoveries; although, fatality rate of COVID-19 is somewhat about 0.3–1% (CDC, 2020), the...
estimations by Baud et al., 2020 imply that COVID-19 fatality rate is about 20% in Wuhan [8]. At the beginning of the disease spread, the epidemic doubled in each 7.4 days [9] and the reproduction number was estimated 2.2 (95% CI, 1.4 to 3.9) [9]. COVID-19 has an incubation period of 5 or 6 days; thus, Anderson et al., 2020; CDC, 2020 suggested a significant pre-symptomatic infectiousness [4,10]. Many mathematical models have been developed for COVID-19 at epidemiological level with regard to SARS-CoV-2 transmission and de-confinement strategies [4–7,11,12]. There are rare models at the level of within-host for comprehending SARS-CoV-2 replication cycle, intercommunication with the immune system (natural defenses), and medicine effect [13–18]. Target cell limited model, among many other model structures that represent viral dynamics, is used for the delineation of diseases such as HIV [19–22], Hepatitis [23,24], Ebola [25,26], influenza [27–29]. A detailed reference for viral modelling can be found in Hernandez-Vargas, 2019 [30]. In this paper, a recent data obtained from patients infected with COVID-19 has shed light on the within-host viral dynamic. The reproduction cycle of SARS-CoV-2 lasts quite longer; over 10 days or even more [4,31]. With the aim of giving early stage epidemic predictions for Covid-19 pandemic, so far different mathematical models have been developed [32–44]; although, all of the models have been of deterministic type, they do not consider uncertainty and variations in the parameters; nevertheless, in the case of a growing epidemic it is somehow clear enough; particularly, it has been shown that uncertainty is certain in the disease transmission rate of Covid-19 and there have been large variation in its range [45]. Taking this into account, to address various epidemiological issues based on the simulation results, some stochastic models have been proposed for Covid-19 epidemic [46–50]. Humans societies are occasionally faced with breathtaking challenges like COVID-19. Thus, it would be necessary to do different thorough studies and analyses of COVID-19; so as to provide more knowledge for a better conception of the virus. In this study, a mathematical model has been presented for modeling and studying the spread of COVID-19 and more importantly controlling the spread of the. The disease-free equilibrium points as trivial equilibrium (TE), virus absenteeism equilibrium (VAE) and virus incidence equilibrium (VIE) has been studied for the proposed model. At the trivial equilibrium point, for the presented dynamic system we obtained the Jacobian matrix so as to use it in finding the largest eigenvalue. In order to improve the performance of the model, we added a controller to that

**Primary definitions about general epidemic model in a social network**

Generally, the population dynamic systems are shown as follows [51],

\[ \dot{X}(t) = f(t, X) \]  

where, the function \( f \) for the population dynamics (1) is defined as follows,

\[ f : N \rightarrow [0, \infty), \text{ for all } t \in [0, \infty). \]

**Spectral Radius:** Let matrix \( A \in \mathbb{R}_{m \times n} \) with eigenvalues \( \lambda_1, \lambda_2, \ldots, \lambda_n \). Then the spectral radius of matrix \( A \) denoted by \( \rho(A) \) and the spectral abscissa (spectral bound) of \( A \), denoted \( \alpha(A) \), are defined as,

\[ \rho(A) = \max_{i=1 \ldots n} |\lambda_i| \text{ and } \alpha(A) = \max_{i=1 \ldots n} \Re(\lambda_i) \]

**Lemma 1.** If \( X_0 \) is a disease-free equilibrium (DFE) of equation (2) and \( f(x) \) satisfies the assumptions(\( \sqrt{1} \)–\( \sqrt{5} \)), then the derivatives \( Df(X_0) \) and \( D\dot{f}(X_0) \) are partitioned as follows [52],

\[ Df(X_0) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}, \quad D\dot{f}(X_0) = \begin{bmatrix} V & 0 \\ A & B \end{bmatrix}, \]

where \( F \) and \( V \) are the \( m \times m \) matrices are defined as,

\[ F = \left[ \frac{\partial \dot{f}_i}{\partial X_j} (X_0) \right] \text{ and } V = \left[ \frac{\partial \dot{f}_j}{\partial X_i} (X_0) \right] \text{ with } 1 \leq i, j \leq m. \]

Then, \( F \) is non-negative, \( V \) is a non-singular M-matrix and all eigenvalues of \( B \) have positive real part.

**Note 1:** Following Diekmann et al. [53], we nominate \( FV^{-1} \) the next generation matrix for the model and the reproductive number \( R_0 \) can be define as the following form,

\[ R_0 = \rho(FV^{-1}). \]

Where \( \rho(A) \) represents the spectral radius of a matrix \( A \).

**Theorem 1.** Consider the disease transmission model given by (2) with \( f(x) \) satisfying conditions

![Fig. 1. Structure of respiratory syndrome causing human coronavirus.](image1)

![Fig. 2. Flow chart of the proposed model.](image2)
If $X_0$ is a disease-free equilibrium of the model, then $X_0$ would be locally asymptotically stable if $\Re_0 < 1$, but unstable if $\Re_0 > 1$.

**Proposed epidemic disease model**

finding the destructive parameters affecting the spread of COVID-19, obtaining strategies that stabilize coronavirus attacks, and also preventing the attacks in the social network, are very important. The proposed model for studying the COVID-19 is showed in the Fig. 2, it includes five classes, class $S$ indicates the people in the social networks who are potentially susceptible to infection (healthy), class $I$ represents the people already infected by coronavirus, class $Q$ shows the quarantined infected people, class $R$ represents people who recovered from COVID-19, and class $D$ represents dead people. As shown in the proposed flowchart each of the defined classes has outputs and inputs (see Fig. 2). The new people that have readiness and susceptibility to be infected by Covid-19 enter to class $S$ as constant migration $a_1S_0$. Coefficient $\eta$ indicates the mortality rates due to natural causes, parameters $\beta_1$ and $\beta_2$ indicate the mortality rates as a result reported for classes $I$ and $Q$, respectively. The rate of recovered people from among the infected ones without being quarantined is shown by parameter $\alpha_3$. The rate of quarantined people from among the infected ones, rate of recovered people from among the quarantined, and rate of susceptible people from among the recovered ones are shown by the constant parameters $\alpha_2$, $\alpha_1$, and $\alpha_0$, respectively.

The infection rate, $\alpha_2 = cpI/N$, depends on the number of partners per individual per unit time ($c > 0$), the transmission probability per partner ($p > 0$) and the proportion of infected individuals to infection active individuals $I/N$, with $N = S + I + Q + R$ as the total population size. In the following, the pandemic COVID-19 transmission dynamic system is derived as follows [54],

$$\begin{align*}
\frac{dS}{dt} &= a_1S_0 + a_2R - \eta S - \alpha_2S, \\
\frac{dI}{dt} &= a_2S - (\eta + \alpha_1 + \beta_1)I, \\
\frac{dQ}{dt} &= a_3I - (\eta + \alpha_2 + \beta_2)Q, \\
\frac{dR}{dt} &= a_4Q + a_1I - (\alpha_0 + \eta)R, \\
\frac{dD}{dt} &= \eta(S + I + Q + R) + \beta_1I + \beta_2Q.
\end{align*}$$

With initial conditions $S(0) = S_0, I(0) = I_0, Q(0) = Q_0, R(0) = R_0$ and $D(0) = D_0$, where, $a_2 = cpI/N$ and $N = S + I + Q + R$. The feasible region of the system (3) is as follows,

$$\Omega = \{ (S, I, Q, R) \mid S + I + Q + R \leq \frac{a_1S_0}{\eta}, S > 0, I > 0, Q > 0, R > 0 \}$$

**Equilibrium points**

For the system (3), the trivial equilibrium (TE), virus absenteeism equilibrium (VAE) and virus incidence equilibrium (VIE) respectively are obtained as follows,

**TE:** $(S, I, R, Q) = (0, 0, 0, 0)$, **VAE:** $(S^0, I^0, R^0, Q^0) = (\frac{a_1S_0}{\eta}, 0, 0, 0)$,

**VIE:** $(S^*, I^*, R^*, Q^*) = (\mathcal{S}, \mathcal{I}, \mathcal{R}, \mathcal{Q})$;

$$\mathcal{S} = \frac{a_1a_2S(\eta + \alpha_2)(\alpha_1 + \beta_1)(\beta_2)}{\eta^4 + (\alpha_1 + \alpha_2 + \alpha_3 + \beta_1)\eta^3 + (\mathcal{S}^* + \mathcal{R}^*)\eta + \mathcal{I}^*},$$

$$\mathcal{I}^* = \frac{a_1S^*\eta(\alpha_1 + \alpha_2)(\alpha_1 + \beta_1)(\beta_2)}{\eta^4 + (\alpha_1 + \alpha_2 + \alpha_3 + \beta_1)\eta^3 + (\mathcal{S}^* + \mathcal{R}^*)\eta + \mathcal{I}^*},$$

$$\mathcal{R}^* = \frac{a_1S^*\eta(\alpha_1 + \alpha_2)(\alpha_1 + \beta_1)(\beta_2)}{\eta^4 + (\alpha_1 + \alpha_2 + \alpha_3 + \beta_1)\eta^3 + (\mathcal{S}^* + \mathcal{R}^*)\eta + \mathcal{I}^*},$$

$$\mathcal{Q}^* = \frac{a_1S^*\eta(\alpha_1 + \alpha_2)(\alpha_1 + \beta_1)(\beta_2)}{\eta^4 + (\alpha_1 + \alpha_2 + \alpha_3 + \beta_1)\eta^3 + (\mathcal{S}^* + \mathcal{R}^*)\eta + \mathcal{I}^*}.$$

**Reproductive number**

By determining the spectral radius of the next generation operator of system, we have derived an explicit formula for the reproductive number of infection (3). System (3) has a virus absenteeism equilibrium, given by $(S^0, I^0, R^0, Q^0) = (a_1S^0/\eta, 0, 0, 0)$, linearizing system (3) around the virus absenteeism equilibrium, we have the following Jacobian matrix,

$$\mathcal{J}(V) = \begin{bmatrix}
-\eta & -\eta & 0 & \alpha_1 \\
-cp & -cp & 0 & 0 \\
0 & \alpha_0 & -(\eta + \alpha_2) & 0 \\
0 & \alpha_4 & \eta + \alpha_2 & 0
\end{bmatrix}$$

We have derived an explicit formula for the reproductive number employing the spectral radius of the next generation operator. According to the Jacobian matrix (4), the transmission matrix $F$ and transition matrix $V$ [4, 8-12], along with virus absenteeism equilibrium (VAE) are as follows,

$$\mathcal{F} = \begin{bmatrix}
\alpha_2 & 0 & 0 \\
\alpha_3 & 0 & 0 \\
0 & \alpha_0 & 0 \\
0 & \alpha_4 & \eta + \alpha_2
\end{bmatrix}$$

where $F$ is a non-negative matrix and $V$ is a non-singular matrix, the reproductive number, $\Re_0$, would be equal to the spectral radius $\rho(FV^{-1})$ [51, 53, 55-58]. Matrix $V^{-1}$, inverses of matrix $V$ is given by,

$$\mathcal{F} = \frac{a_1a_2S(\eta + \alpha_2)(\alpha_1 + \beta_1)(\beta_2)}{\eta^4 + (\alpha_1 + \alpha_2 + \alpha_3 + \beta_1)\eta^3 + (\mathcal{S}^* + \mathcal{R}^*)\eta + \mathcal{I}^*}.$$
Thus, the reproductive number $R_0$ is derived as follows,

$$R_0 = \frac{c p}{\eta + \alpha_1 + \alpha_3 + \beta_1}$$

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**Theorem.** The virus absence equilibrium (VAE), $(S^0, F^0, R^0, Q^0) = \left( \frac{s^0}{q}, 0, 0, 0 \right)$ is locally asymptotically stable if $R_0 < 1$.

**Proof.** To find the eigenvalues of derived Jacobian matrix, we define the Characteristic Polynomial (CP) as follows,

$$P(\lambda) = \left| \begin{array}{cccc}
1 - \eta - \lambda & -c p & 0 & 0 \\
\eta + \alpha_1 & 1 - \eta - \lambda & 0 & 0 \\
\eta + \alpha_3 & \eta + \alpha_2 & 1 - \eta - \lambda & 0 \\
\eta + \alpha_3 & \eta + \alpha_2 & \eta + \alpha_3 + \beta_1 & 1 - \eta - \lambda \\
\end{array} \right|$$

$$= 0.$$

Therefore, the eigenvalues of the Jacobian matrix are as follows,

$$\lambda_1 = -\eta - \lambda, \lambda_2 = -\eta - \alpha_3, \lambda_3 = -\eta - \alpha_4, \lambda_4 = c p - \eta - \alpha_3 - \alpha_5 - \beta_1.$$

Three first eigenvalues are negative. By applying the second order Routh-Hurwitz Criterion at derived fourth eigenvalue we have,

$$\eta + \alpha_1 + \alpha_3 + \beta_1 > 0, \quad c p - \eta - \alpha_3 - \alpha_5 - \beta_1 < 0 \Rightarrow R_0 = \frac{c p}{\eta + \alpha_1 + \alpha_3 + \beta_1} > 1.$$

Thus,

$$\lambda_4 = c p - \eta - \alpha_3 - \alpha_5 - \beta_1 < 0 \Rightarrow R_0 < 1.$$

Hence, by Routh Hurwitz criteria, the given virus absence equilibrium, $(S^0, F^0, R^0, Q^0) = \left( \frac{s^0}{q}, 0, 0, 0 \right)$, is locally asymptotically stable.

**Theorem.** The virus incidence equilibrium (VIE), $(S, F, R, Q') = (S, F, C, R')$ is locally asymptotically stable (LAS) if $R_0 > 1$.

**Proof.** Characteristic Polynomial of Jacobian matrix at virus incidence equilibrium is given by,

$$P(\lambda) = \left| \begin{array}{cccc}
1 - \eta - \lambda & -c p & 0 & 0 \\
\eta + \alpha_1 & 1 - \eta - \lambda & 0 & 0 \\
\eta + \alpha_3 & \eta + \alpha_2 & 1 - \eta - \lambda & 0 \\
\eta + \alpha_3 & \eta + \alpha_2 & \eta + \alpha_3 + \beta_1 & 1 - \eta - \lambda \\
\end{array} \right|$$

$$= 0,$$

with $a_2 = c p / (\eta + \alpha_3 + \beta_1)$, three of eigenvalues are negative and last eigenvalue is as follows,

$$\lambda_4 = c p F / (\eta + \alpha_3 + \beta_1) - \eta - \alpha_3 - \alpha_5 - \beta_1.$$

If $c p F / (\eta + \alpha_3 + \beta_1) - \eta - \alpha_3 - \alpha_5 - \beta_1 > 0 \Rightarrow R_0 = \frac{c p}{\eta + \alpha_1 + \alpha_3 + \beta_1} > 1.$

So, the given virus absence equilibrium, $(S^*, F^*, R^*, Q^*) = (\eta + \alpha_3 + \beta_1)$, is locally asymptotically stable if $R_0 > 1$.

**Bifurcation analysis**

When there is a bifurcation at reproductive number equal to 1 ($R_0 = 1$), the disease invade to population wouldn’t be feasible if $R_0 < 1$, because with entering some infected individuals into the population, the system would then return to the disease-free equilibrium $I = 0$. For values of reproductive number, whatever greater than 1 and $R_0$ is shifted from stability to instability; in this case, the model embraces a unique endemic equilibrium, which is locally asymptotically stable [59,60]. To indicate the stability and existence of endemic equilibrium for the proposed model we find the contact rate of $c$, from reproductive equation as follows,

$$c = \frac{R_0}{p} (\eta + \alpha_1 + \alpha_3 + \beta_1).$$

By placing this equation in the $I'$ we will have,

$$I' (R_0) = \mathcal{M} \times \{ \eta + \alpha_1 + \alpha_3 + \alpha_5 + \beta_1 \} \eta^3 + \left( \beta_2 + \alpha_3 + \alpha_5 \right) \eta^2 - \alpha_2 \eta - \alpha_3 \eta - \alpha_5 \eta - \beta_1 \left( \beta_2 + \alpha_3 + \alpha_5 \right) \eta + \alpha_1 \eta^2 + \left( \beta_2 + \alpha_3 + \alpha_5 \right) \eta + \alpha_1 \eta^2 + \left( \beta_2 + \alpha_3 + \alpha_5 \right) \eta + \alpha_1 \eta - \alpha_5 \eta - \beta_1 \left( \beta_2 + \alpha_3 + \alpha_5 \right) \eta,$$

where $\mathcal{M}$ and $\mathcal{B}$ are defined as follows,

$\mathcal{M} = S^* \alpha_1 (\alpha_3 + \eta) (\beta_2 + \alpha_3 + \alpha_5 + \beta_1)$,

$\mathcal{B} = \eta + \alpha_3 + \alpha_5 + \beta_1$.

With the production number of $R_0 > 1$, the classical compartmental epidemic model includes just one endemic equilibrium. Moreover, the stability of the disease-free equilibrium is achieved by $R_0 < 1$, while it is unstable when $R_0 > 1$.

Accordingly, there exist a forward bifurcation from the disease-free equilibrium towards an endemic equilibrium. However, backward bifurcation has attracted attentions towards disease control in recent years [57,61-68]. Here, the reproduction number is no longer capable of explaining factors leading to disease elimination. In this case, backward bifurcation is needed along with disease control threshold determination.

In these classical compartmental epidemic models, it is assumed that disease treatment rate is determined relative to the basic reproduction number of an infection. Thus, each community should have an appropriate capacity for disease treatment because of the limited number of resources. It is inferred that this hypothesis is confirmed with enough treatment resources and lower reproduction number; otherwise, it is incorrect. According to [57,65-68], a constant treatment rate is achieved, which is applicable when the infection reproduction number is high. The modified treatment rate is presented in [67] as follows,

$$f(I) = \begin{cases} \frac{\gamma I}{I_k}, & 0 \leq I \leq I_k, \\ \frac{I_k}{I_0}, & I > I_k, \end{cases}$$

where $\gamma$ and $I_0$ are positive constants. This again asserts that the abovementioned hypothesis is true in case of lack of treatment capacity. Otherwise, it requires the maximum treatment capacity of the community. This leads to an improvement of both proportional and constant treatments as proposed in [57,67,68]. Accordingly, equation (3) can be rewritten as follows:
By linearizing equation (6) around the virus absenteeism equilibrium, the following Jacobian matrix is achieved,

\[
J_{\text{VAE}} = \begin{bmatrix}
-\eta & -c_p \\
-\eta & 0 \\
0 & c_p \\
0 & 0
\end{bmatrix},
\]

the Characteristic Polynomial (CP) of matrix (7) is as follows,

\[
\lambda = \begin{cases}
\eta + a_1 + \beta_1 & I > I_0 \\
\eta + a_1 + \beta_1 + \gamma & 0 \leq I \leq I_0 \\
0 & \lambda > I_0
\end{cases}
\]

\[
= \begin{cases}
1 + \frac{\gamma}{c_p} & I > I_0 \\
\frac{\gamma}{c_p} & 0 \leq I \leq I_0
\end{cases}
\]

According to \( N(t) = S(t) \times I(t) \times Q(t) + R(t) \) and (5), \( N(t) \) satisfies the following equation,

\[
\frac{dN}{dt} = a_1 S^0 - \eta N - \beta_1 I - \beta_1 Q - a_1 R,
\]

\[
\frac{dI}{dt} = a_1 (N - I - Q - R) - \gamma I - f(I),
\]

\[
\frac{dQ}{dt} = a_1 I - \gamma Q + a_1 I - \gamma Q + f(I),
\]

\[
\frac{dR}{dt} = \gamma Q + a_1 I - \gamma Q + f(I),
\]

where \( I_1, I_2, \) and \( I_3 \) as defined before,

\[
I_1 = \eta + a_1 + \beta_1, I_2 = \eta + a_1 + \beta_1, I_3 = \eta + a_1.
\]

If \( I_0 \geq 0 \), then \( I > 0 \), system (8) admits a unique positive solution \( E^* = (I_1^*, I_2^*, I_3^*) \) supposed that \( f(I) = \gamma I \) as follows,

\[
N^* = \frac{1}{\eta} \left( a_1 S^0 - \beta_1 - \frac{a_1 a_6}{\gamma} - \frac{a_1 \beta_1}{\gamma} - \frac{a_1 a_6}{\gamma} - \frac{\gamma a_6}{\gamma} - \frac{a_1 a_6}{\gamma} \right) I^*,
\]

\[
Q^* = \frac{a_1 I^*}{\gamma},
\]

\[
R^* = \frac{1}{\gamma} \left( a_1 + \gamma + \frac{a_1 a_6}{\gamma} \right) I^*,
\]

\[
I^* = \frac{a_1 I^* (cp - \gamma - \gamma I)^S}{\eta (cp - \gamma - \gamma I)}.
\]

where \( \Lambda_1 \) and \( \Lambda_2 \) are defined as follows,

\[
\Lambda_1 = \frac{1}{\eta} \left( \beta_1 + \frac{a_1 a_6}{\gamma} + \frac{a_1 \beta_1}{\gamma} + \frac{\gamma a_6}{\gamma} + \frac{a_1 a_6}{\gamma} \right),
\]

\[
\Lambda_2 = \frac{1}{\gamma} \left( a_1 + \gamma + \frac{a_1 a_6}{\gamma} \right).
\]

Also, supposed that \( f(I) = I_0 I \), we will have,

\[
N^* = \frac{1}{\eta} \left( a_1 S^0 - \frac{a_1 a_6}{\gamma} \right) - \frac{1}{\eta} \left( \beta_1 + \frac{a_1 \beta_1}{\gamma} + \frac{\gamma a_6}{\gamma} + \frac{a_1 a_6}{\gamma} \right) I^*,
\]

\[
Q^* = \frac{a_1 I^*}{\gamma},
\]

\[
R^* = \frac{1}{\gamma} I_0 + \left( \frac{a_1 a_6}{\gamma} + \frac{a_1 a_6}{\gamma} \right) I^*.
\]

A \times I^* + B \times I^* + C = 0,

with \( A, B, C, \Lambda_3, \Lambda_4, \Lambda_5 \) and \( \Lambda_6 \) as follows,

\[
A = cp (I_2 \Lambda_4 - I_2 - a_1 - I_1 \gamma \Lambda_1 - I_1 \gamma \Lambda_3),
\]

\[
B = cp (I_2 \Lambda_4 - I_2 - a_1 - I_1 \gamma \Lambda_1 - I_1 \gamma \Lambda_3),
\]

\[
C = -\gamma I_2 \Lambda_1 I_0,
\]

\[
\Lambda_3 = \frac{1}{\eta} \left( a_1 S^0 - \frac{a_1 a_6}{\gamma} \right), \Lambda_4 = \Lambda_1 - \frac{1}{\eta} \left( \frac{\gamma a_6}{\gamma} \right), \Lambda_5 = \frac{I_0}{\gamma}, \Lambda_6 = \frac{\gamma}{\gamma},
\]

Therefore, the quadratic equation \( A \times I^* + B \times I^* + C = 0 \) can be derived as,

\[
I^* = \frac{-B \pm \sqrt{B^2 - 4AC}}{2A}.
\]

To take \( \Delta \geq 0 \) as the case,

\[
\Delta = B^2 - 4AC = cp (I_2 \Lambda_4 - I_2 - a_1 - I_1 \gamma \Lambda_1 - I_1 \gamma \Lambda_3)^2
\]

\[
+ 4 (\gamma I_2 \Lambda_1 I_0)(cp (I_2 \Lambda_4 - I_2 - a_1 - I_1 \gamma \Lambda_1 - I_1 \gamma \Lambda_3),
\]

**Theorem.** In order to have \( \Delta \geq 0 \), we must have,

\[
\min \left\{ \frac{a_1 I_2 \Lambda_4 - I_1 \Lambda_4 - a_1 I_1 \gamma}{\gamma I_2 I_0} \right\} \leq I_0
\]

\[
\leq \max \left\{ \frac{a_1 I_2 \Lambda_4 - I_1 \Lambda_4 - a_1 I_1 \gamma}{\gamma I_2 I_0} \right\}
\]

**Proof.** To make a condition \( \Delta \geq 0 \) we must have,

\[
\left\{ \begin{array}{l}
\gamma I_2 \Lambda_1 I_0 \geq 0 \\
(\gamma I_2 \Lambda_1 I_0 - a_1 - I_1 \gamma \Lambda_1 - I_1 \gamma \Lambda_3) \leq 0
\end{array} \right.
\]

and because of \( cp \geq 0 \), from \( cp (I_2 \Lambda_4 - I_2 - a_1 - I_1 \gamma \Lambda_1 - I_1 \gamma \Lambda_3) \)

\[
\left\{ \begin{array}{l}
\gamma I_2 \Lambda_1 I_0 \geq 0 \\
(\gamma I_2 \Lambda_1 I_0 - a_1 - I_1 \gamma \Lambda_1 - I_1 \gamma \Lambda_3) \leq 0
\end{array} \right.
\]

Therefore, it can be easily concluded that:
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> only if, Bilgehan in 2021 for the outbreak of COVID-19 [72].

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Optimization of the proposed epidemic model

Optimization of a system means minimizing or maximizing a function which measures the performance of a system. This would ultimately improve the performance of the system. Generally, we can name three important steps for system optimization as follows,

- The first step is to understand the system and the various variables that effect on that.
- The second step is to select functions as system performance measures. This criterion depends on system variables and has a great positive effect on system efficiency.
- The third step is to select the value of the system variables, and this selection is done in a way that the system is eventually optimized.

We must have a correct understanding of the system, how it works, the various variables affecting the system and the interaction of the variables on each other. We need to use an appropriate optimization method so as to search for optimal values. The aim of optimization is to find the best solutions offering the limitations and the needs of the problem. There may might different solutions for a problem; to compare the solutions and selecting the optimal solution, a function called the objective function has been defined. Choosing the right objective function is one of the most important optimization steps.

Sometimes in optimization several goals are being considered simultaneously; such optimization problems, which involve multiple objective functions, are called multi-objective problems. The simplest way to deal with such problems is to form a new objective function in the form of a linear combination of the main objective functions; that way the effectiveness of each function is determined by the weight assigned to that. Each optimization problem has a number of independent variables, called target variables, represented by the n-dimensional vector $x$ [69].

Optimal control model was used by Keshri and Mishra in 2014 to control the attack of worms in the wireless sensor network [69]. In their paper, they used a proper optimal control countermeasure to minimize the attack of worms, susceptible number of sensor nodes, and also the infected ones. Optimal control, with including extra compartment namely exposed class to the basic SIR epidemic model, was applied to a nonlinear fractional order epidemic model for HIV transmission by Nai et al. in 2020 [70]. An optimal control strategy for sequencing social distancing and testing events has been used by Tsy et al. in 2021 in a way that the number of infections is minimized, [71]. The analysis of our extensive computational efforts reveals that both social distancing and quarantining, in case of being implemented early, would have a great efficiency; worth mentioning that the in-time quarantining of the confirmed cases of infection, would have a higher efficiency. Using a mathematical model with fractional order derivative in the Caputo sense, a fractional optimal control problem was formulated by Baba and Bilgehan in 2021 for the outbreak of COVID-19 [72].

Some of the most recent articles about pandemic Covid-19 disease, which provide models for predicting and controlling the disease are presented in the Refs. [73–76]. In these articles, the optimal control theory is used for optimizing the models; so, we apply the optimal control theory to maximize the number of people returning to the “normal life” and minimizing the number of active infected individuals with minimal economical costs while warranting a low level of hospitalizations [77]. Optimal control analysis showed that, a combination of optimal preventive strategies such as public health education, personal protective measures and treatment of hospitalized cases are significantly effective in decreasing the number of COVID-19 cases in different compartments of the model [78]. The optimal control theory is established using Pontryagins Maximum Principle [79]; this theory, being applied based on SEIRDs model, suggests taking COVID-19 preventive measures such as physical distancing and mask wearing.

Fig. 3 shows a flow chart of the proposed model with controller. In this paper, we applied the optimal control theory to SIQR model with protection covering $P$. The objective function includes the three classes of susceptible, infected and quarantined people. The goal is to find conditions that result in the maximum of susceptible people, the minimum of infected people, and the optimal quarantined people.

The purpose of optimization is to determine the target variables so that the objective function is minimized or maximized,

$$J = \int_0^T \left[ w_1 S(t) + w_2 I(t) + w_3 Q(t) + \frac{1}{2} \psi^2(t)^2 \right] dt; \ t \in [0, T].$$

With the following conditions,

$$\begin{align*}
\dot{S} &= a_S S - a_S R - \eta S - a_S S - \psi S, \\
\dot{I} &= a_I S - (\eta + \alpha + \beta) I, \\
\dot{Q} &= a_Q I - (\eta + \beta) Q, \\
\dot{R} &= a_R Q + a_I I - (\alpha + \eta) R + \psi S, \\
D &= \eta (S + I + Q + R) + \beta I + \beta Q.
\end{align*}$$

(9)

where, $\psi$ is the safekeeping coating rate of the susceptible people and it is a crucial factor in specifying the measure of protection covering $P$; then, it is assumed that $A$ is directly proportionate to the incidence of individuals COVID-19 infection. Thus, $\psi = uP$; where $u$ is a positive coefficient (utility function or controller) and $P$ is information variable (protection covering) [57,58], then the parameter $P$ prepares the information about both the current and past condition of COVID-19 infection as defined in the following formula,

$$P = \int_0^t \Psi(\beta, I(t), S(t), \tau, \tau) dt; \ \Psi(\beta, I, S, \tau, \tau) = \beta \times I \times S \times \frac{1}{2} \times e^{\left(\frac{-\tau}{\psi}\right)}.$$ 

Here, $\tau$ is the distributed delay, $\beta$ is a positive constant denoting the mean of the gathered information on the COVID-19 infection [59]. Thus, the model (9) can be reformulated as the following model of the
pandemic COVID-19 diffusion,
\[ \dot{S} = a_S S^0 + a_S R - \eta S - \beta SI - uPS, \]
\[ \dot{I} = \beta S - (\eta + a_I + a_S + \beta_I) I, \]
\[ \dot{Q} = a_I I - (\eta + a_I + \beta_I) Q, \]
\[ \dot{R} = a_Q I + a_I - (a_S + \eta) R + uPS, \]
\[ D = \eta (S + I + Q + R) + \beta I + \beta_Q Q. \]
\[ \tilde{P} = \frac{1}{2} (\beta S - \tilde{P}), \]
(10)

The System (10) might be reduced to the equivalent system,
\[ \dot{S} = a_S S^0 + a_S R - \eta S - \beta SI - uPS, \]
\[ \dot{I} = \beta S - (\eta + a_I + a_S + \beta_I) I, \]
\[ \dot{Q} = a_I I - (\eta + a_I + \beta_I) Q, \]
\[ \dot{P} = \frac{1}{2} (\beta S - \tilde{P}), \]

Thus, characteristic polynomial for Jacobian matrix \( J(\text{VAE}) \) is given by,
\[ P(\lambda) = |J(\text{VAE}) - \lambda I| = -\frac{1}{2} \{ -uP - \eta - \lambda (cp - \eta - \alpha_3 - \beta_1 - \beta_2) (\eta + \alpha_I + \beta_I) \} = 0. \]

Solving of this equation would result in the eigenvalues listed as the follows,
\[ \lambda_1 = -uP - \eta, \lambda_2 = cp - \eta - \alpha_3 - \beta_1 - \beta_2, \lambda_3 = -\eta - a_I, \lambda_4 = -1/\tau. \]

Theorem. If we take \( N_0 \) as the reproductive number at the disease-free equilibrium, then if \( N_0 < 1 \), the disease-free equilibrium will be locally asymptotically stable, and if \( N_0 > 1 \) the disease-free equilibrium would be unstable.

Proof. According the eigenvalue \( \lambda_2 = cp - \eta - \alpha_3 - \beta_1 - \beta_2 \), we have,
- If \( \lambda_2 = cp - \eta - \alpha_3 - \beta_1 - \beta_2 < 0 \), then \( N_0 = \frac{cp}{a_S + a_S + \beta_I} < 1. \)
- If \( \lambda_2 = cp - \eta - \alpha_3 - \beta_1 - \beta_2 < 0 \), then \( N_0 = \frac{cp}{a_S + a_S + \beta_I} < 1. \)

For the proposed optimal control problem, we gain the Langrangian and Hamiltonian functions \([59,77]\) to obtain an optimal solution,
Langrangian: \( \mathcal{L}(S, I, Q, P) = w_S S(t) + w_I I(t) + w_Q Q(t) + w_P P(t) + \frac{1}{2} ru(t)^2. \)
Hamiltonian: \[ \tilde{H} = \mathcal{L}(S, I, Q, P) + \lambda_1 (\frac{\partial \mathcal{L}}{\partial S} + \frac{\partial \lambda_1}{\partial S} + \lambda_2 (\frac{\partial \mathcal{L}}{\partial I} + \frac{\partial \lambda_2}{\partial I} + \lambda_3 (\frac{\partial \mathcal{L}}{\partial Q} + \frac{\partial \lambda_3}{\partial Q} + \lambda_4 (\frac{\partial \mathcal{L}}{\partial P} + \frac{\partial \lambda_4}{\partial P} + \lambda_5 (\frac{\partial \mathcal{L}}{\partial \tilde{P}}. \]

Therefore, the Hamiltonian function can be rewritten as,
\[ \tilde{H} = w_S S + w_I I + w_Q Q + w_P P + \frac{1}{2} ru(t)^2 + \lambda_1 (a_S S^0 + a_S R - \eta S - \beta SI - uPS) + \lambda_2 (\beta SI - (\eta + \alpha_I + \beta_I) I + \lambda_1 (\alpha_S I - (\eta + a_S + \beta_I) Q) + \lambda_2 (a_I I + \alpha_S I - (a_S + \eta) R + uPS) + \lambda_3 (\eta (S + I + Q + R) + \beta I + \beta_Q Q) + \lambda_4 \left( \frac{1}{2} (\beta S - \tilde{P}) \right), \]

where \( \lambda_1, \lambda_2, \lambda_3 \) and \( \lambda_4 \) stand as the adjoint functions \([78]\), the adjoint equation for \( \lambda_1, \lambda_2, \lambda_3 \) and \( \lambda_4 \) are given by,
\[ \frac{\partial \lambda_1}{\partial t} = \frac{d}{ds} \frac{\partial \tilde{H}}{\partial S} = -\left( w_1 - \eta - \lambda_1 \beta I - \lambda_1 uP + \lambda_2 \beta I + \lambda_3 uP + \lambda_4 \eta + \lambda_5 \frac{1}{2} \beta \right), \]
\[ \frac{\partial \lambda_2}{\partial t} = \frac{d}{ds} \frac{\partial \tilde{H}}{\partial I} = -\left( w_2 - \lambda_1 \beta S + \lambda_2 \beta S - \lambda_2 (\eta + \alpha_3 ) + \lambda_1 \alpha_3 + \lambda_3 \eta + \lambda_5 \beta_1 + \lambda_6 \frac{1}{2} \beta S \right), \]
\[ \frac{\partial \lambda_3}{\partial t} = \frac{d}{ds} \frac{\partial \tilde{H}}{\partial Q} = -\left( w_3 - \lambda_1 \alpha_3 - \lambda_2 \alpha_3 - \lambda_3 \eta + \lambda_5 \eta \right), \]
\[ \frac{\partial \lambda_4}{\partial t} = \frac{d}{ds} \frac{\partial \tilde{H}}{\partial P} = 0, \]
\[ \frac{\partial \lambda_5}{\partial t} = \frac{d}{ds} \frac{\partial \tilde{H}}{\partial \tilde{P}} = -\left( w_4 - \lambda_1 \alpha_3 + \lambda_2 \alpha_3 + \lambda_3 \eta + \lambda_4 \alpha_3 + \lambda_5 \beta_2 \right). \]

With the transversality conditions \( \lambda_i(T) = 0(i = 1 \ldots 6) \). Using the optimal conditions, we can derive the optimal utility function \( u'(t) \) as follows,
\[ \frac{\partial \tilde{H}}{\partial u} = ru - \lambda_1 PS + \lambda_4 PS = 0, \]
\[ u'(t) = \max \left\{ \min \left\{ \frac{\lambda_1 P'(t) S'(t) - \lambda_2 (t) P'(t) S'(t)}{r} \right\}, 0 \right\} \]

The optimal points \( S', I', Q', R', \lambda' \) and \( P' \) can be derived by solving the following system,
where $\phi_1 = \eta + \alpha_3 + \beta_1$, $\phi_2 = \eta + \alpha_4 + \beta_2$ and $\phi_3 = \alpha_6 + \eta$, the optimal controller is given by,

$$ u^* = \max \left\{ \frac{1}{r} \left( \tilde{\lambda}_{1,n} - \lambda_{4,n} \right) P^n S^n_1, 0 \right \} $$. 

In the following, optimal values for $S', I', Q', R', D'$ and $P'$ can be obtained by solving the modified following matrix equation,

\[
\begin{bmatrix}
S'_{n+1} \\
I'_{n+1} \\
Q'_{n+1} \\
R'_{n+1} \\
D'_{n+1} \\
P'_{n+1}
\end{bmatrix} = \begin{bmatrix}
S_n \\
I_n \\
Q_n \\
R_n \\
D_n \\
P_n
\end{bmatrix} + h \begin{bmatrix}
\alpha S_n & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix} \begin{bmatrix}
\eta & -\beta S_n & 0 & \alpha_5 & 0 & S_n \\
0 & \alpha_5 & 0 & 0 & 0 & 0 \\
0 & \alpha_5 & -(\eta + \alpha_4 + \beta_2) & 0 & 0 & 0 \\
\eta & \beta_1 + \eta & \beta_2 + \eta & \eta & 0 & \eta \\
0 & \beta_n \tau S_n & 0 & 0 & 0 & -\beta_n / \tau
\end{bmatrix}
\end{equation}

\[
\begin{align*}
S'_{n+1} &= S_n + h \alpha S_n \\
I'_{n+1} &= I_n + h \alpha_5 \\
Q'_{n+1} &= Q_n + h \alpha_4 \\
R'_{n+1} &= R_n + h \beta_2 \\
D'_{n+1} &= D_n + h \beta_n \tau S_n \\
P'_{n+1} &= P_n + h \beta_n / \tau
\end{align*}
\]

With $\beta_n = cp/N_n$ and $h = (t_n - t_{n-1})/M$ (M stands as the number of interval $[t_0, t_n]$ iterations). With the optimal Hamiltonian function defined as follows,

$$ H^* = w_1 S_n + w_2 I_n + w_3 Q_n + w_4 P_n + \frac{1}{2} \left( \max \left\{ \frac{1}{r} \left( \tilde{\lambda}_{1,n} - \lambda_{4,n} \right) P^n S^n_1, 0 \right \} \right)^2 $$. 

Programing algorithm

For programing algorithm, we used the limitation definition of first order derivative as follows,

$$ \mathcal{S}'(t) = \lim_{\Delta t \to 0} \frac{\mathcal{S}(t + \Delta t) - \mathcal{S}(t)}{\Delta t}. $$

By using definition (11), algorithm of programing is given by,

\[
\begin{align*}
S'_{n+1} &= S_n + h \alpha S_n \\
I'_{n+1} &= I_n + h \alpha_5 \\
Q'_{n+1} &= Q_n + h \alpha_4 \\
R'_{n+1} &= R_n + h \beta_2 \\
D'_{n+1} &= D_n + h \beta_n \tau S_n \\
P'_{n+1} &= P_n + h \beta_n / \tau
\end{align*}
\]

With $\beta_n = cp/N_n$ and $h = (t_n - t_{n-1})/M$ (M stands as the number of interval $[t_0, t_n]$ iterations).
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between disease-free and endemic equilibria at $\alpha$.

The disease-free becomes unstable. This bifurcation diagram confirms our analytic results; it shows

of stability between the disease free and endemic equilibria. If $\alpha > 1$, the disease-free state is always the final reachable stable condition. When $\Re_0 > 1$, the disease establishes itself in a community at endemic levels. This bifurcation diagram confirms our analytic results; it shows that when $\Re_0 > 1$, a stable endemic equilibrium exists; and when the same condition holds, the disease free becomes unstable.

Fig. 5 shows $\Re_0$ versus $\Re_0$ when $\gamma = 1$ and $c \times p = 1$ and 2. As can be seen, $\Re_0$ is less than 1 for $c \times p = 1$ and for all $0 < \Re_0 < 4$ values. When $c \times p = 2$, for all $0 < \Re_0 < 2$ values, $\Re_0 < 1$ and for $2 < \Re_0 < 4$, $\Re_0$ is slowly increasing from 1 to 1.35. Fig. 6 indicates $\Re_0$ versus $\gamma$ when $\Re_0 = 1$ and $c \times p = 1, 2, 3, 4, 5$. For all values $c \times p = 1, 2, 3, 4, 5$, $\Re_0$ is less than 1, and when $\gamma$ increases from 0 to 1, $\Re_0$ decreases rapidly from 1 to 0.5. Backward bifurcation diagram when constant treatment rate is included in the model (3) is shown in Fig. 7. As can be seen, when $\Re_0 < \Re_0^*$, the disease-free equilibrium is globally asymptotically stable and when $\Re_0^* < \Re_0 < 1$, there are two endemic equilibria, the upper is stable and the lower is unstable. To plot the constant model, the following parameters have been used as, $\alpha_1 = 0.001; \alpha_3 = 0.3; \alpha_4 = 0.03; \alpha_5 = 0.25; \alpha_6 = 0.02; \beta_1 = 0.22; \beta_2 = 0.03; \eta = 0.05; c = 3; p = 0.35; S^0 = 2000; S_0 = 120; I_0 = 5; Q_0 = 0; R_0 = 0; D_0 = 0$.

Fig. 8 shows the respectively susceptible people and infected people when $\Re_0 < 1$ and $\Re_0 > 1$. As seen in this figure, in the case that $\Re_0 < 1$, for different values of $c = 0.1, 2.3$ all the graphs are convex and a critical point at $I = 0$ is occurred. For the mode that $\Re_0 > 1$, all diagrams are convex for different $c = 4, 5, 6, 7$ values and a critical point at $I =$

Numerical simulations and discussion

In this section, materials, methods and the numerical study of the presented models with controller have been discussed.

Materials and methods

In the current paper, Maple software was used to calculate, analyze and find analytical parts. MATLAB software was used for programming and numerical simulation. The algorithm mentioned in Section (4), was developed in Matlab software. For programming and numerical simulations, parameters were fixed at \([53, 54, 57, 79]\),

\[
\alpha_1 = 0.001; \alpha_3 = 0.3; \alpha_4 = 0.03; \alpha_5 = 0.25; \alpha_6 = 0.02; \beta_1 = 0.22; \beta_2 = 0.03; \eta = 0.05; c = 3; p = 0.35; T = 5; S^0 = 2000; S_0 = 120; I_0 = 5; Q_0 = 0; R_0 = 0; D_0 = 0;
\]

Simulation and discussion

Fig. 4 shows the bifurcation diagram so, that was obtained through numerical simulations using the relation $I'(\Re_0)$; it indicates an exchange of stability between the disease free and endemic equilibria. If $\Re_0 < 1$ the disease-free state is always the final reachable stable condition. When $\Re_0 > 1$, the disease establishes itself in a community at endemic levels. This bifurcation diagram confirms our analytic results; it shows that when $\Re_0 > 1$, a stable endemic equilibrium exists; and when the same condition holds, the disease free becomes unstable.

Fig. 5 shows $\Re_0$ versus $\Re_0$ when $\gamma = 1$ and $c \times p = 1$ and 2. As can be seen, $\Re_0$ is less than 1 for $c \times p = 1$ and for all $0 < \Re_0 < 4$ values. When $c \times p = 2$, for all $0 < \Re_0 < 2$ values, $\Re_0 < 1$ and for $2 < \Re_0 < 4$, $\Re_0$ is slowly increasing from 1 to 1.35. Fig. 6 indicates $\Re_0$ versus $\gamma$ when $\Re_0 = 1$ and $c \times p = 1, 2, 3, 4, 5$. For all values $c \times p = 1, 2, 3, 4, 5$, $\Re_0$ is less than 1, and when $\gamma$ increases from 0 to 1, $\Re_0$ decreases rapidly from 1 to 0.5. Backward bifurcation diagram when constant treatment rate is included in the model (3) is shown in Fig. 7. As can be seen, when $\Re_0 < \Re_0^*$, the disease-free equilibrium is globally asymptotically stable and when $\Re_0^* < \Re_0 < 1$, there are two endemic equilibria, the upper is stable and the lower is unstable. To plot the constant model, the following parameters have been used as, $\alpha_1 = 0.001; \alpha_3 = 0.3; \alpha_4 = 0.03; \alpha_5 = 0.25; \alpha_6 = 0.02; \beta_1 = 0.22; \beta_2 = 0.03; \eta = 0.05; c = 3; p = 0.35; S^0 = 2000; S_0 = 120; I_0 = 5; Q_0 = 0; R_0 = 0; D_0 = 0$.

Fig. 8 shows the respectively susceptible people and infected people when $\Re_0 < 1$ and $\Re_0 > 1$. As seen in this figure, in the case that $\Re_0 < 1$, for different values of $c = 0.1, 2.3$ all the graphs are convex and a critical point at $I = 0$ is occurred. For the mode that $\Re_0 > 1$, all diagrams are convex for different $c = 4, 5, 6, 7$ values and a critical point at $I =$

Fig. 4. Bifurcation diagram for the model which shows an exchange of stability between disease-free and endemic equilibria at $\Re_0 = 1$.

Fig. 5. $\Re_0$ versus $\Re_0$ when $\gamma = 1$ and $c \times p = 1$ and 2.

Fig. 6. $\Re_0^*$ versus $\gamma$ when $\Re_0 = 1$ and $c \times p = 1, 2, 3, 4, 5$. 
$I_0 = 0$ is occurred. In both cases, all the curves converge to the $S = S'$ point. Fig. 9 shows the optimal controller function, $u'(t)$, for $R_0 = 0.91 < 1$ and $R_0 = 1.28 > 1$. As seen in these figures, for the mode in which $R_0 > 1$, the number of changes of the optimal controller function is more than the number of changes in the mode in which $R_0 < 1$. Most changes for the state of $R_0 > 1$ occurred in the range $I = [0, I_0] = [0, 5]$.

Fig. 10 presents the susceptible people, $S(t)$, with and without controller when $R_0 < 1$ and $R_0 > 1$. As shown in this figure, by using the controller function, the number of susceptible people is increased significantly. As can be seen from this figure, for mode in which, the controller affects the increase of healthy people at a slower rate, but eventually, comparing with the mode $R_0 < 1$, the number of healthy people in the mode $R_0 > 1$ is more. In the Fig. 11 the infected people, $I(t)$, is shown with and without controller when $R_0 < 1$ and $R_0 > 1$ respectively. In the proposed model, the use of controller has caused the number of infected people to drop to zero straight away. During all time, the number of infected people with the controller is less than the number of infected people without the controller. For mode $R_0 > 1$, the diagram of infected people initially resists the controller function, but eventually begins to decline faster than mode $R_0 < 1$.

**Fig. 7.** Backward bifurcation diagram when constant treatment rate is included in model (3). When $R_0 < R'_0$, the disease-free equilibrium is globally asymptotically stable. However, when $R'_0 < R_0 < 1$, there are two endemic equilibria. The upper is stable and the lower is unstable.

**Fig. 8.** Susceptible vs infected people when $R_0 < 1$ and $R_0 > 1$.

**Fig. 9.** Optimal controller function, $u'(t)$, when $R_0 < 1$ and $R_0 > 1$.
Conclusion

Mathematical modelling of the pandemic diseases plays an important role in the prediction and control of the epidemic disease. A mathematical model has been presented for pandemic COVID-19 to study the behavior of this epidemic disease. The disease-free equilibrium points as the trivial equilibrium (TE), virus absenteeism equilibrium (VAE) and virus incidence equilibrium (VIE) are studied. A protection covering class and an utility function were added to the proposed model; due to the optimization by the theory of optimal control. For the optimal control problem, the Lagrangian and Hamiltonian functions are gained for obtaining an optimal solution to improve the performance of the model. The results show that the use of controllers might play an important and effective role in controlling infectious diseases such as Covid-19. Numerical simulations were also carried out to show the significance of the control programs. It is clear that when the control measures are applied optimally the number of infected people reduces; however, the number of susceptible people increases.

CRediT authorship contribution statement

Shahriar Seddighi Chaharborj: Conceptualization, Data curation, Formal analysis, Investigation, Resources, Validation, Writing - original draft, Writing - review & editing. Sarkhosh Seddighi Chaharborj: Conceptualization, Formal analysis, Methodology, Project administration, Software, Supervision, Visualization. Jalal Hassanzadeh Asl: Supervision, Writing - review & editing. Pei See Phang: Funding acquisition, Validation.

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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