A Dynamic Optimal Control Model for SARS-CoV-2 in India

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Abstract

The pandemic coronavirus has explored all over the world with a devastating negative impact on humanity indeed. The epidemic virus has dynamic genetic diversity that has never been encountered earlier. The mortality rate of the disease is increasing rapidly as the approved drug is not available yet. Only isolation from the infected individual or community is the only choice to save our existence. As the human is the only carrier so in that case if the host carriers are isolated from each other then it might be possible to control the spread of positive rates of the infected population. In this research work, we have presented an optimal control model for COVID-19 by following two different steps. In the first step, we formulated a Susceptible-Exposed-Infected-Asymptomatic-Recovered (SEIAR) model for COVID-19, analyzed and reported the disease dynamics for time-independent disease control parameters. In the second step, we discussed an optimal control model with time-dependent control parameters. The theoretical findings are validated graphically with some realistic data for the pandemic COVID-19 concerning India's perspective.

Keywords: Epidemic; Asymptomatic; Susceptible; Isolation; Quarantine; Social distancing.

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1. Introduction

In the present scenario, it is very difficult to control the impact of the pandemic SARS-CoV-2, which has been named COVID-19. Though history itself has shown that the impact of epidemic disease is very dangerous which happened in the year 1918, Spanish Flu, Pandemic influenza.^[1] The history of human coronaviruses began in 1965 when Tyrrell and Bynoe found that they could passage a virus named B814.^[2] At about the same time, Hamre and Procknow invented 229E named coronavirus. The 229E agent and the previous OC-43 strain identified by McIntosh *et al.*^[3] had a similar morphology. In the late 1960s, Tyrrell was leading a group of virologists working with human strains and several animal viruses. These included infectious bronchitis virus, mouse hepatitis virus, and transmissible gastroenteritis virus of swine, all of which had been demonstrated to be morphologically the same as seen through electron microscopy. This new group of viruses was named coronavirus (corona denoting the crown-like appearance of the surface projections) and was later officially

accepted as a new genus of viruses.^[4]

The devastating impact of coronavirus emerged in 2002 – 2003 as Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV) from southern China and spread throughout the world with quantifiable speed.^[5,6,7] The origins of the virus are not fully understood, but according to the analysis of different virus genomes, it is believed that it may have originated in bats and was transmitted to Himalayan civets sometime in the distant past.^[8]

Then, in the year 2012 Middle East Respiratory Syndrome Coronavirus (MERS-CoV) appeared as another deadly epidemic disease to kill a huge number of the population.^[9] Again, it is believed that it may have originated in bats,^[10] and was transmitted to camels^[11] sometime in the distant past. The year 2019 is another deadly comeback of coronavirus with some ultimate novel properties as well. The host mutant is considered pangolin this time and confirmed as well.^[12,13,14] It has been first predicted in China by December 31, 2019. Wuhan is the place where it is first registered. This novel coronavirus is having an outstanding capacity mutation rate. It can adapt to the situation based on climatic conditions and so on.^[12] Rather, it has developed numerous types of its genotypes as well due to the super-fast mutation rate indeed. This category of coronavirus is genetically identical to SARS-CoV and that is why it has been named SARS-CoV-2^[15,16]. Due to the novel properties of SARS-CoV-2, it is very difficult to develop stable vaccines in the whole world to save mankind.

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In that case, all these corona-viruses are transferable between humans only if they come in close contact. It attacks the pulmonary system and kills people randomly with weaker lung compliance. Human deaths due to Acute Respiratory Distress Syndrome (ARDS).^[7,17] The only possible way to control this transmission is by maintaining social distancing for a certain period.^[18]

Many research articles have been explored and discussed for understanding the current updates on COVID-19.^[19,12,20] In an overall analysis of the transmission rate due to COVID-19 has been presented using Susceptible-Exposed-Infected-Asymptomatic-Recovered (SEIAR) model using a differential equation. The origin of coronavirus from SARS is discussed in which the eminent authors have justified and considered bat as parent mutant and pangolin as host mutant of SARS-CoV-2. The evolution of SARS-CoV-2 is a novel cause and its pandemic impact on disasters has been presented in.^[12] The effectiveness of the quarantine of Wuhan city against the coronavirus disease 2019 (COVID-19) has been reported.^[21] Management strategies in an SEIR model of COVID-19 community spread have been discussed.^[22] Pandey *et al.*^[23] have shown SEIR and Regression Model based COVID-19 outbreak predictions in India. These articles are confined to some extent, firstly, it is based on the SEIR model that has been formulated by excluding the asymptomatic infected population. Secondly, most of the articles are presented by considering Wuhan due to the population density. On the other side, few articles have been found yet already published in India like.^[24, 25] As India is also considered to be a country with the second highest population just after China. It is also true that the density of the overall population in India is undoubtedly the maximum as compared to China. So, controlling this type of pandemic situation is more difficult as compared to less populated countries or regions.

In this paper, we have discussed and presented an optimal control model for COVID-19 in India by following two different steps. In the first step, we formulated a SEIAR model for COVID-19 and analyzed and reported the disease dynamics for time-independent disease control parameters. In the second step, an optimal control problem invader is discussed to avoid the third wave with a time-dependent control parameter. This paper has two main contributions:

1. SEIAR model formulation based on the differential equation to confirm the impact of lockdown in India due to huge population density;
2. Upgraded optimal control model with time-dependent control parameter for predicting best isolation time to reduce susceptibility rate for safer India;

The rest of the paper is organized as follows. Section 2 describes the mathematical modeling of SEIAR model, section 3 describes the reproduction number, section 4 explains the dynamical behavior of the system, section 5 describes the impact of quarantine and treatment on the endemic equilibrium, and section 6 explains the optimal control policies. Finally, the main conclusion and

contributions are highlighted in section 7.

2. Mathematical model

In this paper, we develop a dynamic system of epidemic control of Covid-19.

2.1 Notations of the concerned parameters

The entire work has been carried out by incorporating twenty-six parameters and the details of the entire parameters are given in Table 1.

Table 1. Notations of the concerned parameters followed throughout the model.

N	Total population
S	Susceptible population
E	Exposed class
I	Symptomatic infected population
A	Asymptomatic infected population
R	Removed (or recovered) population
u_1	Home isolation control
u_2	Quarantine control on the I and A infected population
u_3	Treatment control on the infected population
α_1	Transmission coefficient between S and infected(both I and A) population
α_2	The incubation period of E turned to A
α_3	The incubation period of E turned to I
β_1	Positive constant, the multiple of the transmissibility of A to that of I
$1/\beta_2$	Average viremic period of A
$1/\beta_3$	Average viremic period of I
γ_1	Disease-related death of A
γ_2	Disease-related death of I
τ_1	The treatment response rate of A
τ_2	A treatment response rate of I
d	The proportion of asymptomatic infection rate in people
$1/\delta$	The average lifespan of a person
α	The rate at which R becomes S again

$N = S + E + I + A + H + R$, this equation is very simple to understand. Let us explain this by taking an example. Suppose, N is the total population of a certain entity. S is the susceptible population of that concerned entity those who might get infected due to any close contact with an infected human. Now, the infected population is classified into two categories. One is considered with symptoms and named as symptomatic (I) and the other one is a silent carrier^[26] that is no such symptoms but is equally harmful in transmitting COVID-19. They are known as the asymptomatic (A) infected population. Both the I and A class passes through an intermediate class called exposed class (E) where an individual is infected but not yet infectious where the infected individuals remain in the incubation phase. Once an individual (both symptomatic and asymptomatic) is diagnosed to be infected are shifted to a hospital or quarantine center. These individuals are assumed to be hospitalized (H). The final percentage of the population

left might be clean or recovered (R). In India compared to other countries recovering rate is much better and death consequences are very less as compared to the overall population of the entire country. Because of this reason, we have not considered the death rate in our modeling formulation.

We suppressed time dependency in the notations for simplicity. Now, all these populations together are counted to get an exact figure equal to N .

2.2 Model formulation

We have proposed a *SEIAHR* model by identifying humans as susceptible (S), exposed (E), symptomatically infected (I), asymptotically infected (A), hospitalized (H) and removed/resistant (R), for example, the propagation mechanism of H_1N_1 .^[27] The actual motivation for taking this model is to replace the *SEIR* model which is a bit weaker as compared to the proposed model in the case of India.^[23] The asymptomatic infected are classified among those who do not show any signs or symptoms but, are not detectable and are medically termed as a silent carrier^[26] for transmitting infections due to COVID-19. The Susceptible class has a constant recruitment rate $(1 - u_1)\delta N$ where at a rate of $u_1\delta N$ are home isolated and so they fall in the R class. We have also assumed that a susceptible person becomes infected when they come in contact with an asymptotically or symptomatically infected person. We have assumed that once a person is diagnosed to be infected (symptomatic or asymptomatic), an amount $u_2(I + \beta_1 A)$ are quarantined at hospitals or quarantine centers. So $(1 - u_2)(I + \beta_1 A)$ the amount comes in contact with the susceptible population and acquires infection. The susceptible then enter into their exposed E class in the dormant period. After this period, $\alpha_3(1 - d)E$ amount enter I class, where $\alpha_2 dE$ amount enters A class of infectious, who are infectious in the sense that they are capable of transmitting infections. The hospitalized individuals are treated with medication and supplementation *etc.* to develop immunity at a rate u_3 . When the transitional period is over, the person enters the recovered class R . We have assumed two types of recovery, namely natural recovery and recovery after treatment with proper medication.

It is observed that a symptomatic infected individual cannot get immunity without hospital support. Again, we assumed that an asymptomatic infected individual has a natural recovery rate β_2 , a hospitalized individual has a natural recovery rate β_3 and recovery rate under medication is τu_3 .

Further, we assumed that a symptomatic infected individual, asymptomatic infected individual, and hospitalized individual have disease-induced mortality at a rate γ_1 , γ_2 and γ_3 respectively. Since the recovery is not permanent, an amount of αR again becomes susceptible. All the classes have a natural death rate δ . We assumed here, a removed person may become susceptible or infected again due to multiple genotypes.^[15] In our model, newborns and migration are ignored. Therefore, the network model with infectious force follows during dormant and infected periods.

$$\begin{cases} \frac{dS}{dt} = \delta(1 - u_1)N - \alpha_1(1 - u_2)(I + \beta_1 A)\frac{S}{N} - \delta S + \alpha R \\ \frac{dE}{dt} = \alpha_1(1 - u_2)(I + \beta_1 A)\frac{S}{N} - ((1 - d)\alpha_2 + d\alpha_3 + \delta)E \\ \frac{dI}{dt} = (1 - d)\alpha_2 E - (u_2 + \gamma_1 + \delta)I \\ \frac{dA}{dt} = d\alpha_3 E - (\beta_1 u_2 + \beta_2 + \gamma_2 + \delta)A \\ \frac{dH}{dt} = u_2(I + \beta_1 A) - (\beta_3 + \tau u_3 + \gamma_3 + \delta)H \\ \frac{dR}{dt} = \delta u_1 \delta N + \beta_2 A + (\beta_3 + u_3 \tau)H - (\alpha + \delta)R \end{cases} \quad (1)$$

Initially, the mathematical introduction of the epidemic problem was completed with the conditions such as $S(0) > 0, E(0) > 0, I(0) > 0, A(0) > 0, H(0) > 0, R(0) > 0$. In the classical *SEIR* model,^[27] the infected population is taken as only symptomatic numbers, but in our model, we have introduced an asymptomatic infected population as well which is denoted by A as shown in Table 1. This function is very much necessary as SARS-CoV-2 is mutating at a faster rate and introduced many genotypes of it.^[15,28] Due to this some of the population are becoming silent carriers^[26] as well as those who are taken as asymptomatic in this model indeed. For this particular group of population is not that possible to evaluate modeling using the *SEIR* model.

3. Dynamical behavior of the system

The differential system (1) has two possible non-negative equilibria, *i. e.*

- (i) the disease-free equilibrium, $\varepsilon_1(S_1, 0, 0, 0, 0, R_1)$
- (ii) the endemic equilibrium, $\varepsilon^*(S^*, E^*, I^*, A^*, H^*, R^*)$

where $S_1 = \delta(1 - u_2)N + \alpha \frac{R_1}{\delta}$, $R_1 = \frac{\delta u_1 N}{\alpha + \delta}$ and

$$\begin{aligned} S^* &= \frac{C_3 C_5 C_6}{C_2(C_4 C_6 + \beta_1 d \alpha_3 C_5)}, \\ E^* &= \frac{(\alpha R^* + C_1 - \delta S^*)C_5 C_6}{C_2 C_4 C_6 S^* + \beta_1 C_2 C_5 d \alpha_3 S^*}, \\ I^* &= \frac{C_4}{C_5} E^*, A^* = \frac{d\alpha_3}{C_6} E^*, H^* = \frac{u_2}{C_7} (I^* + A^*), \\ R^* &= \frac{1}{C_{10}} (C_8 + \beta_2 A^* + C_9 H^*) \end{aligned}$$

where $C_1 = \delta(1 - u_1)N$, $C_2 = \frac{\alpha_1}{N}(1 - u_2)$, $C_3 = (1 - d)\alpha_2 + d\alpha_3 + \delta$, $C_4 = (1 - d)\alpha_2$, $C_5 = u_2 + \gamma_1 + \delta$, $C_6 = u_2 + \beta_2 + \gamma_2 + \delta$, $C_7 = \beta_3 + \tau u_3 + \gamma_3 + \delta$, $C_8 = \delta u_1 N$, $C_9 = \beta_3 + \tau u_3$, $C_{10} = \alpha + \delta$.

Reproduction number

Many epidemiological models have a disease-free equilibrium where there is a population in the absence of disease. These models usually have a threshold parameter, known as the basic reproduction number, $\mathcal{R}_0$.

In this section, we will find the reproduction number $\mathcal{R}_0$ and the stability of disease-free equilibrium. Following,^[29] we note it only E, A, I, H are involved in the calculation of $\mathcal{R}_0$. In a disease-free equilibrium, the rate of presence of new infection Φ and the transfer rate of individuals between the two categories Ψ is given by:

$$\Phi = \begin{pmatrix} \alpha_1 (1 - u_2) \frac{SI}{N} + \frac{\alpha_1}{\beta_1} (1 - u_2) S A \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (2)$$

$$\Psi = \begin{pmatrix} \{(1-d)\alpha_2 + d\alpha_3 + \delta\}E \\ -(1-d)\alpha_2 E + (u_2 + \gamma_1 + \delta)I \\ -d\alpha_3 E + (\beta_2 + u_2 + \gamma_2 + \delta)A \\ -u_2(I + A) + (\beta_3 + \tau u_3 + \gamma_3 + \delta)H \\ -(1-u_1)\delta N + \alpha_1(1-u_2)\frac{S}{N} + \alpha_1\beta_1(1-u_2)\frac{SA}{N} + \delta S - \alpha R \\ -u_1\delta N - \beta_2 A - (\beta_3 + \tau u_3)H - (\alpha + \delta)R \end{pmatrix} \quad (3)$$

According to the ideas of the next-generation matrix and the basic reproduction number proposed in,^[29] the reproduction number is $\rho(FV^{-1})$ for the following matrices F and V where ρ is the spectral radius of the matrix (FV^{-1}) .

$$F = \frac{\partial \Phi}{\partial x_i}(x_0) = \begin{pmatrix} 0 & C_2 S(0) & C_2 \beta_1 S(0) & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad (4)$$

$$V = \frac{\partial \Psi}{\partial x_i}(x_0) = \begin{pmatrix} C_2 & 0 & 0 & 0 \\ -C_4 & C_5 & 0 & 0 \\ -d\alpha_3 & 0 & C_6 & 0 \\ 0 & -u_2 & -u_2 & C_7 \end{pmatrix} \quad (5)$$

$$\frac{1}{|V|} \begin{pmatrix} C_5 C_6 C_7 & 0 & 0 & 0 \\ C_4 C_6 C_7 & -C_3 C_6 C_7 & 0 & 0 \\ C_5 C_7 d \alpha_3 & 0 & C_3 C_5 C_7 & 0 \\ u_2 C_4 C_6 - d \alpha_3 u_2 C_5 & u_2 C_3 C_6 & u_2 C_3 C_5 & C_3 C_5 C_6 \end{pmatrix} \quad (6)$$

where $|V| = C_3 C_5 C_6 C_7$.

Therefore, the reproduction number,

$$\mathcal{R}_0 = C_2 \frac{C_4 C_6 S(0) + \beta_1 \alpha_3 C_5}{C_3 C_5 C_6} \quad (7)$$

where $S(0) = \frac{C_1 + \alpha R(0)}{\delta}$ and $R(0) = \frac{C_8}{C_{10}}$.

As a remark, we have the following theorems.

Theorem 1, If $\mathcal{R}_0 < 1$, the infection-free equilibrium is locally asymptotically stable, and if $\mathcal{R}_0 > 1$, the infection-free equilibrium is unstable, and an epidemic ensues.^[30]

Theorem 2, If $\mathcal{R}_0 < 1$, the infection-free equilibrium is globally asymptotically stable.^[31]

Theorem 3, The endemic equilibrium E^* is locally asymptotically stable if $\mathcal{R}_0 > 1$.^[32]

4. Impact of quarantine & treatment on the endemic equilibrium

To study the effect of quarantine and treatment of infected people, we have obtained partial derivatives of the local equilibrium coordinates in combination with the discontinuity parameters.

From the Eqs. (18) and (20) as given in the annexure, is

observed that the first-order partial derivative of E^*, I^*, A^* concerning the quarantine variable u_2 is negative. Also, those are negative with respect to the treatment variable as well shown in Eqs. (22) and (24) are given in the Annexure. That means the functions are monotonically decreasing function of u_2 & u_3 , it is possible to achieve $E^* \rightarrow 0, I^* \rightarrow 0, A^* \rightarrow 0$. That means infected individuals should be acquired as soon as possible with an appropriate quarantine and treated appropriately.

The result of the fixed time control analysis is shown in Fig. 1 by considering 100 days in this case. In Fig. 1, it is very clear that within 38 days susceptible population and exposed population have been reduced to almost zero. Whereas, infected curves of the symptomatic and asymptomatic population are presenting a monotonic decay graph. At the end of the 100th day, the infected, exposed, and the asymptomatic population is not existing anymore. The susceptible population is slightly increased to a certain value and thus recovery curve is almost a few gradients down as compared to the 90th day.

Fig. 1 Fixed time control analysis by taking 100 days of pandemic impact due to COVID-19.

In the case of Fig. 2, the same is almost happening as

susceptibility is increasing up to a certain constant figure at the end of the 1000th day. Whereas, the recovery rate is also decreasing up to a certain limit. All these are happening due to excessive interaction of the doctors and nurses with the infected patients in hospital quarantine. Also, the huge density of the population in India is not controlling the spread inside the home indeed due to a huge number of people staying together in slums area and joint families as well. The risk factor of getting infected is taking this curve higher at the end of the span. Thus, the entire exposed, infected, asymptomatic population is not considered as they are taken to the hospital. The doctors are still in touch with the patient and they are surviving within highly contaminated viral droplets suspended arena.^[33]

Fig. 2 Fixed time control analysis by taking 1000 days of pandemic impact due to COVID-19.

5. Optimal control policy

From theorems (1), (2), and (3) we observe that the COVID-19 disease has epidemic potential as the system has a stable endemic equilibrium. Also, the control measures have an impact on the spread of the epidemic. But, the application of fixed control takes a long time to control which costs a huge amount of loss of human lives. Also longer time of home isolation, quarantine in hospitals and locked-down situation brings huge loss to the economy. In this case, the application of optimal control policies is a more suitable option. The optimal control policies aim to optimize the use of the control measures, reduce the severity (reducing the number of Exposed, Symptomatic and Asymptomatic cases), and reduce the side effects or overdoses of control measures in a short period. The optimal control model is given by:

$$J = \min_{u_1, u_2, u_3} \int_0^T (a_1 u_1^2 + a_2 u_2^2 + a_3 u_3^2 + a_4 E + a_5 I + a_6 A) dt \quad (8)$$

subject to the system of ODE (1). The objective here is to find the triplet (u_1^*, u_2^*, u_3^*) such that,

$$J(u_1^*, u_2^*, u_3^*) = \min_{u_1, u_2, u_3 \in \Theta} J(u_1, u_2, u_3) \quad (9)$$

where, $\Theta = \{u: \text{measurable and } 0 \leq u_i(t) \leq 1\}$ for $\{t \in [0, T], i = 1, 2, 3\}$ is the set of controls.

Here, the time-dependent control variables $u_1(t)$ is the home isolation for susceptible classes, $u_2(t)$ and $u_3(t)$ are respectively the quarantine control (hospitalization) and treatment (medication) control on both symptomatic and asymptomatic infected classes. Also, a_1, a_2, a_3 are the positive constants associated with the squares of the control to balance the size of the terms? a_4, a_5, a_6 are the constant weights corresponding to $E(t), I(t)$ and $A(t)$, respectively. The squares of the controls are taken to consider the side effect and overdoses of the controls. Now the optimal control problem in Eq. (8) is solved, subject to the condition given in Eq. (1).

The Lagrangian of the problem is given by:

$$L = a_1 u_1^2 + a_2 u_2^2 + a_3 u_3^2 + a_4 E + a_5 I + a_6 A.$$

To minimize L , the Hamiltonian of the objective function is:

$$H(u_1, u_2, u_3, S, E, I, A, R, t) = a_1 u_1^2 + a_2 u_2^2 + a_3 u_3^2 + a_4 E + a_5 I + a_6 A + \lambda_1(t) S'(t) + \lambda_2(t) E'(t) + \lambda_3(t) I'(t) + \lambda_4(t) A'(t) + \lambda_5(t) R'(t) \quad (10)$$

Where, $\lambda_i(t)$, $i = 1, 2, \dots, 5$ are adjoint (co-state) variables to be determined by the following system of differential equations

$$\dot{\lambda}_1(t) = -\frac{\partial H}{\partial S(t)} = B_1 (I + \beta_1 A) (\lambda_1(t) - \lambda_2(t)) \quad (11)$$

$$\begin{aligned} \dot{\lambda}_2(t) &= -\frac{\partial H}{\partial E(t)} \\ &= -a_4 + B_3 \lambda_2(t) - B_2 \lambda_3(t) - d \alpha_3 \lambda_4(t) \end{aligned} \quad (12)$$

$$\begin{aligned} \dot{\lambda}_3(t) &= -\frac{\partial H}{\partial I(t)} \\ &= -a_5 + B_1 S (\lambda_1(t) - \lambda_2(t)) + B_4 \lambda_3(t) - B_7 \lambda_5(t) \end{aligned} \quad (13)$$

$$\begin{aligned} \dot{\lambda}_4(t) &= -\frac{\partial H}{\partial A(t)} \\ &= -a_6 + \beta_1 B_1 S (\lambda_1(t) - \lambda_2(t)) + B_5 \lambda_4(t) - B_6 \lambda_5(t) \end{aligned} \quad (14)$$

$$\dot{\lambda}_5(t) = -\frac{\partial H}{\partial R(t)} = (\alpha + \delta) \lambda_5(t) - \alpha \lambda_1(t) \quad (15)$$

satisfying the conditions,

$$\lambda_i(T) = 0, \quad i = 1, 2, \dots, 5 \quad (16)$$

We have considered that $\bar{S}, \bar{E}, \bar{I}, \bar{A}, \bar{R}$ are the optimum values of A, E, I, A, R . Also, let $\bar{\lambda}_1, \bar{\lambda}_2, \bar{\lambda}_3, \bar{\lambda}_4, \bar{\lambda}_5$ be the solution of the system is given by Eq. (11) to Eq. (15).

Following Luke,^[34] and Pontryagin *et al.*,^[35] we have proved the following theorem:

Theorem 4. There exist optimal controls u_1^*, u_2^*, u_3^* for $t \in [0, T]$ such that,

$$J(u_1^*, u_2^*, u_3^*, E, I, A) = \min_{u_1, u_2, u_3} J(u_1, u_2, u_3, E, I, A)$$

subject to the system (1).

Proof: As all the state variables and control variables are non-negative, all the control variables u_1, u_2, u_3 are convex. Also, the control space Θ is closed and convex. So the optimal control (u_1^*, u_2^*, u_3^*) exists and is bounded which minimizes Eq. (1) for $t \in [0, T]$ with the help of the system given by Eq. (1). Hence the theorem.

With the help of Pontryagin's maximum principle^[35] and the above theorem (4), the following theorem is proved.

Theorem 5. The optimal control triplet (u_1^*, u_2^*, u_3^*) which minimizes J over Θ is given by:

$$\begin{aligned} u_1^* &= \max\{0, \min(u_1, 1)\} \\ u_2^* &= \max\{0, \min(u_2, 1)\} \\ u_3^* &= \max\{0, \min(u_3, 1)\} \end{aligned}$$

where,

$$\begin{aligned} \bar{u}_1 &= \frac{\delta N}{2a_1}(\bar{\lambda}_1 - \bar{\lambda}_5) \\ \bar{u}_2 &= \frac{\alpha \bar{S}}{2a_2 N}(\bar{I} + \beta_1 \bar{A})(\bar{\lambda}_2 - \bar{\lambda}_1) \\ \bar{u}_3 &= \frac{1}{2a_3}(\tau_2 \bar{I}(\bar{\lambda}_3 - \bar{\lambda}_5)) + \tau_1 A(\bar{\lambda}_4 - \bar{\lambda}_5) \end{aligned}$$

Proof: With the help of the optimality condition, that is $\frac{\partial H}{\partial u_i} = 0$, $i = 1, 2, 3$.

$$\begin{aligned} \bar{u}_1 &= \frac{\delta N}{2a_1}(\bar{\lambda}_1 - \bar{\lambda}_5) \\ \bar{u}_2 &= \frac{\alpha \bar{S}}{2a_2 N}(\bar{I} + \beta_1 \bar{A})(\bar{\lambda}_2 - \bar{\lambda}_1) \\ \bar{u}_3 &= \frac{1}{2a_3}(\tau_2 \bar{I}(\bar{\lambda}_3 - \bar{\lambda}_5)) + \tau_1 A(\bar{\lambda}_4 - \bar{\lambda}_5) \end{aligned}$$

Using Theorem (5), all the 3 controls are bounded with 0 and 1 as lower and upper bounds respectively. That is $u_i^* = 0$, if $\bar{u}_i \leq 0$ and $u_i^* = 1$, if $\bar{u}_i \geq 1$, where $i = 1, 2, 3$. Therefore, for this 3-tuple of controls (u_1^*, u_2^*, u_3^*) , we have the optimum value of the functional J^* given by Eq. (9). Hence the theorem.

5.1 Numerical analysis

In this section, we are going to discuss the results obtained after performing optimal control theory-based model analysis for COVID-19 in India by using the data given in Table 2. In Table 2, all the parameters are taken using [36]. Few of them are accumulated from other sources [37,38,39,40,41]. These values are considered from both the first and second waves.

From Figs. 3 and 4, it is clearly showing that isolation is not the appropriate solution for the susceptible population. Whereas, only quarantine along with treatment somehow is the best optimal control to be followed further. From Figs. 5 and 6, quarantine and treatment both should have to be followed to achieve an optimal control model against this COVID-19 for the exposed population. In the case of the symptomatic infected population, as per Figs. 7 and 8 as well as the asymptomatic infected population too as per Fig. 9 and 10, hospital quarantine and treatment is the one and only recommended process to be followed to obtain an optimal

control model. From Figs. 11 and 12, it is clear that up to a certain point home isolation or treatment is possible to follow to have a good number of the recovered patient. To some extent for achieving better perception, it is safe to follow both hospital quarantine and treatment as well. This might be faster indeed as compared to the previous processes indeed. Since COVID-19 is mutating faster and evolution provoked multiple genotypes though it is not safe indeed, and can't assure one recovered from COVID-19 that he or she might not get infected again. [15] The expected formulation of 38 days of locked down under section 5 is validated in Fig. 13.

Fig. 3 Susceptible population, all control, no control, and single controls.

5.2 Sensitivity analysis

Here, we have plotted some figures for the Exposed class (E), Asymptomatic infected class (A), and Symptomatic infected class (I) with respect to time t for α_2 and α_3 .

The incubation period is the time when it comes to exposure to a pathogen, chemical, or radiation, and when the signs and symptoms are first revealed. Here, α_2 is the incubation period of the exposed class to the infected class. From Fig. 14, it is seen that the exposed class people decreased with the increment of the incubation period. The infectious classes are increased with the α_2 (shown in Figs. 14, 15, and 16). The same thing happened for the incubation rate α_3 for E, A, and I (shown in Figs. 17, 18, and 19). So incubation period is the more sensitive parameter for becoming the infectious class of the exposed class people. That means that as the incubation period decreases, the number of an infected persons will decrease.

Table 2. Values of the parameters for controlling the pandemic situation.

Parameters	Values	Parameters	Values	Parameters	Values	Parameters	Values
α_1	0.5	α_2	0.07	α_3	0.03	β_1	0.3
β_2	0.05	β_3	0.06	α	0.001	d	0.3
δ	0.00004	N	100000	γ_1	0.025	γ_2	0.035
τ_1	0.07	τ_2	0.05	S_0	60000	E_0	15000
I_0	10000	A_0	10000	R_0	5000	A_1	0.001
A_2	0.002	A_3	0.1	A_4	1.0	A_5	1.0
A_6	1.0

Fig. 4 Susceptible population, all control, no control, and pairs of controls.

Fig. 7 Infected population, all control, no control, and single controls.

Fig. 5 Exposed population, all control, no control, and single controls.

Fig. 8 Infected population, all control, no control, and pairs of controls.

Fig. 6 Exposed population, all control, no control, and pairs of controls.

Fig. 9 Asymptomatic infected population, all control, no control, and single controls.

Fig. 10 Asymptomatic infected population, all control, no control, and pairs of controls.

Fig. 13 Time Vs Controls.

Fig. 11 Recovered or removed population, all control, no control, and single controls.

Fig. 14 Sensitivity analysis of α_2 on $E(t)$.

Fig. 12 Recovered or removed population, all control, no control, and pairs of controls.

Fig. 15 Sensitivity analysis of α_2 on $I(t)$.

Fig. 16 Sensitivity analysis of α_2 on $A(t)$.

Fig. 19 Sensitivity analysis of α_3 on $A(t)$.

Fig. 17 Sensitivity analysis of α_3 on $E(t)$.

Fig. 18 Sensitivity analysis of α_3 on $I(t)$.

6. Conclusions

In this paper, we have discussed the impact of the pandemic COVID-19 in India by developing the dynamic optimal control model using *SEIAR* model. Since India is a densely populated country therefore isolation by introducing a lockdown for around consecutive 40 days might be able to control the pandemic impact anyhow. However, it has also been observed that only isolation is not a good solution indeed. This might help in controlling up to a certain limit. This might lead to some adverse impacts in the future but for 100% perception, it is better to follow up hospital quarantine along with proper treatment. Identification of the infected population is very much necessary by following proper testing facilities. This will reduce the exposed population faster. As India is densely populated thus home isolation might lead to an increase in symptomatic or asymptomatic infected population which might lead to an uncontrollable situation as time goes on^[28,36]. So, it is quite evident from the study that adequate testing and hospital quarantine along with proper treatment affirmatively after following a restricted lockdown phase for around at least a minimum of 40 days is highly recommended. From Figs.1 and 2, it is also clear that COVID-19 is going to be a sustainable outbreak for around more than a decade not only in India but also throughout the world. Future research direction is to develop another mathematical model by including the death parameter and vaccination as well. The next objective is to find another optimal control solution using fuzzy theory by considering vaccination as the most important parameter. Along with this, it is also decided to forecast the impact of COVID-19 on the world economy as the second future scope of our entire research. Machine learning is considered to be implemented for finding the approximate model parameters for enriching the sustainability of the endemic stage.

Conflict of interest

There is no conflict of interest.

Supporting information

Not Applicable.

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