Dynamics of COVID-19 Outbreak and Optimal Control Strategies: A Model-Based Analysis

Naba Kumar Goswami^{1*}, B. Shanmukha²

¹Department of Mathematics, PET Research Foundation, Mysuru, India ²Department of Mathematics, PES College of Engineering, Mandya, India

Abstract: COVID-19 is an infectious disease caused by the SARS-CoV-2 virus, which spreads so fast in the inhabitants. The virus is transmitted through direct contact with respiratory droplets of an infected individuals through coughing and sneezing or indirect contact through contaminated objects or surface. In this article, a non-linear mathematical model is proposed and analyzed to manifest the impact of transmission dynamics of the COVID-19 pandemic based on Indian condition by considering asymptomatic and symptomatic infections. It is assumed that the transmission rates due to asymptomatic and symptomatic individuals are different. The basic reproduction number of the model is computed and studied the stability of different equilibria of the model in detail. The sensitivity analysis is presented to identify the key parameters that influence the basic reproduction number, which can be regulated to control the transmission dynamics of the disease. Also, this model is extended to the optimal control model and is analyzed by using the Pontryagin's Maximum Principal and solved numerically. It has been observed that the optimal control model gives better result as compacted to the model without optimal control model as it reduces the number of infectives significantly in a desired interval of time.

Keywords: COVID-19, basic reproduction number, stability analysis, sensitivity analysis, optimal control

1. INTRODUCTION

The ongoing COVID-19 outbreak has put mathematical models in the limelight. In 1960, the human coronavirus was first identified and In 21st century, a large number of people in this world have been affected by the three outbreaks such as SARS, MERS, and 2019-nCoV. In 2003, the Severe Acute Respiratory Syndrome(SARS) outbreaks, especially in the Chinese mainland, Hong Kong, Taiwan, and Canada of the World [1] and in 2012 and 2015, the Middle East Respiratory Syndrome(MERS) outbreak in Saudi Arabin [2] and South Korea [3] respectively. Coronaviruses belonging to the family of Coronaviridae and order of Nidovirales, enveloped, non-segmented, single-stranded positive-sense RNA viruses [4]. All coronavirus are zoonotic. They start in animals and can then, following mutation, recombination, and adaptation, be passed on to humans. In the human-to-human transmission of COVID-19 can occurs via respiratory droplets directly (through droplets from coughing or sneezing) or indirectly (touching surfaces or objects contaminated with virus and touching their mouth, nose, eyes) [5]. The incubation period for the new coronavirus from 2 to 14 days in human to human transmission [6]. The common symptoms of COVID-19 are fever, cough, difficulty of breathing, and fatigue. At present, there are no specific drugs for the disease to protect the people, only hygiene measures can reduce the

^{*}Corresponding author: nabakrgoswami@gmail.com

rate of transmission. According CDC COVID-19 vaccination is a safer way to help build protection measure. Covid vaccines can stop or reduce most of the people from getting sick, but not everyone. Despite taking all recommended doses of vaccines and waits a few weeks for immunity to build up, still there is a chance that people can get infected. So far there is no evidence, however, that human coronaviruses can be transmitted by animals.

In December 2019, a new outbreak of pneumonia of unknown cause has been identified in Wuhan city, the capital of China's Hubei province [9]. This outbreak has some other potential causes like influenza, avian influenza, adenovirus, and SARS, but there are no symptoms like MERS [7]. Later on 7th January, 2020, the causative pathogen was identified as a Novel Coronavirus (2019-nCoV) [8]. As the virus is very closely related to SARS and MERS, so the name 2019-nCoV can distinguish the virus from both [9]. On 30th January 2020, the World Health Organization (WHO) declared that the outbreak is a Public Health Emergency of International Concern(PHEIC) [10] as it is Worldwide spread. As the number infective is increasing rapidly and the epidemiological evidence of human-to-human (doorplates, direct contact, etc.) transmission suggests that 2019-nCoV is more contagious than both SARS and MERS [11-13]. On 11th February 2020, the World Health Organization announced a new name of coronavirus disease as COVID-19 [14]. On 11th March 2020, the World Health Organization officially declares the COVID-19 outbreak as a Pandemic as it spread worldwide. The most affected countries in this world are the USA, India, Brazil, Italy, Spain, France, UK, Germany, Iran, China, Belgium, Netherlands, etc. In this ongoing outbreak, in the USA more than 43,107,628 people are infected and more than 694,619 people have died. Presently more than 229,382,253 infected cases have been reported across 223 territories, where more than 206,052,168 infected cases are recovered and more than 4,707,336 infected cases have been died till 20^{th} Sept 2021.

In India, the first Novel coronavirus case was reported on 30th January 2020 [15] in the state of Kerala. Due to the crises of coronavirus pandemic, Prime Minister of India declared 21 + 19 = 40 days nationwide lockdown from 25^{th} of March to 3^{rd} of May 2020 as a preventive measure for the COVID-19 [16]. The Ministry of Health and Family Welfare of India has suggested various precautionary measures to prevent the spread of viruses such as washing hands frequently, physical/social distancing, wearing mask, avoiding touching face, nose, and eyes [17, 18]. Apart from lockdown the government of India performing many awareness programs about preventative measures through media and social networks (TV, radio, newspaper, Facebook, Twitter, etc.). The effect of lockdown and social distancing play an important role to reduce the coronavirus infection. In 2020, total number of infected cases are recorded in India are 10,266,674 and January to September 2021, number infected cases are 23,211,745. In 2021, also statewide or locality wise lockdown declared according to basis of active cases in that particular state or region. On 20th September, 2021, more than 33,478,419 infected COVID-19 cases have been reported in 32 states in India, while 32,715,105 cases are recovered and 445,165 many cases have been died. Most affected states in India are Maharashtra, Karnataka, Kerla, Delhi, Gujarat, Rajasthan, Madhya Pradesh, Uttar Pradesh, Tamil Nadu, Andhra Pradesh, Telangana, West Bengal, Jammu and Kashmir. Among the cities in India, Mumbai, Bangalore and Delhi are the badly effected by COVID-19 outbreak.

The primary aimed to study this model to study Indian conditions and will forecast future pandemic by using information available. In [1] authors presented a deterministic model and simplified from the SEIJR model, which is adapted to analyze the important parameters of the model of SARS epidemic; In [19] authors constructed a SEQIJR model of epidemic disease transmission which includes immunization and varying population size is studied; In [20] authors studied a mathematical about early transmission dynamics of the infection and evaluating the effectiveness of control measures; In [21] authours proposed and studied a

Copyright © 2021 ASSA.

model on the various impact of the intervention on the spread of COVID-19 in India; In [22] authors discussed a data-driven analysis in the early phase of the outbreak. They estimated the basic reproduction number of novel coronavirus (2019-nCoV) in China; In [23] authors develop a mathematical model for the spread of the coronavirus disease 2019 (COVID-19); In [24] authors developed a Bats-Hosts-Reservoir-People transmission network model for simulating the potential transmission from the infection source to the human infection. In [31] authors proposed a mathematical model and they focuses on the impacts of face mask, hospitalization of symptomatic individuals and quarantine of asymptomatic individuals on the transmission dynamics of COVID-19 pandemic in India.

This paper is organized as follows: In section 2 presents the model; In section 3 discussed existence of equilibria and basic reproduction number; In section 4 presents the stability analysis of the model; In section 5 deal with sensitivity analysis of basic reproduction number; In Section 6 illustrate the effects of parameters on disease outbreak; In Section 7 we extend the model to optimal control model and analysis it. Demonstrates the numerical simulation results of the optimal control model; Finally, Section 7 we conclude the paper.

2. THE MODEL

In this section, a dynamic model for COVID-19 pandemic is presented and discussed based on India condition. The model divides the total population $N(t) = S + E + I_a + I_a$ $I_s + H + R$ into six different compartments according to the nature of the disease such as Susceptible individuals (S), Exposed individuals (E), Asymptomatic infective individuals (I_a) , Symptomatic infective individuals (I_s) , Hospitalized individuals (H) and Recovered individuals (R). It is assumed that the total population is varying and homogeneously mixed i.e., all people are equally likely to be infected by the infectious individuals if they come into contact. The individuals are employed in the province at a constant rate Λ and join the susceptible class. The Natural birth and deaths in the population are also considered in the model. It is assumed that susceptible individuals after being exposed to the COVID-19 infection can progress to asymptotic infective and symptomatic infective at the rates β . Assume that an asymptomatic individual joins the symptomatic populations class at the rate ρ . Further, both asymptomatic and symptomatic infectious individuals will progress to the hospitalized or quarantine compartment with clinical symptoms of COVID-19 at the rate δ_1 and δ_2 respectively. Also, some asymptotic and symptomatic individuals may recover without hospitalized or quarantine at the rates γ_1 and γ_2 respectively. Hospitalized individuals may recover and after recovery, it progresses to recovered class at the rate γ_3 . However, the rates of recovery may vary from one compartment to another. The natural mortality rate of each Individuals class is μ . Due to critical illness of COVID-19 disease some of the symptomatic and hospitalized individuals class have additional mortality rates μ_1 and μ_2 , respectively. The flow diagram of the model is given in Figure 1, and biological interpretations of parameters are shown in Table 1. Keeping the above facts/assumptions in mind, a mathematical model COVID-19 is proposed as follows:

$$\frac{dS}{dt} = \Lambda - \beta_a S I_a - \beta_s S I_s - \mu S$$

$$\frac{dE}{dt} = \beta_a S I_a + \beta_s S I_s - (\kappa + \mu) E$$

$$\frac{dI_a}{dt} = \xi \kappa E - (\rho + \gamma_1 + \mu + \delta_1) I_a$$

$$\frac{dI_s}{dt} = (1 - \xi) \kappa E + \rho I_a - (\gamma_2 + \mu_1 + \mu + \delta_2) I_s$$
(1)

Copyright © 2021 ASSA.

$$\frac{dH}{dt} = \delta_1 I_a + \delta_2 I_s - (\gamma_3 + \mu_2 + \mu)H$$
$$\frac{dR}{dt} = \gamma_1 I_a + \gamma_2 I_s + \gamma_3 H - \mu R$$

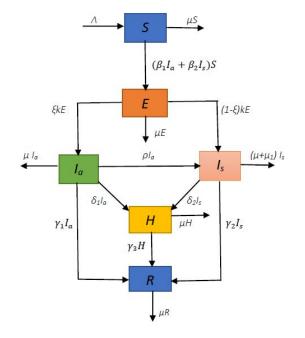


Fig. 1. Flow diagram of the model.

Table 1.	Biological	interpretations	of parameters

Parameter		Biological interpretations
Λ	:	Rate of recruitment in the susceptible class,
β_a	:	Rate of infection of susceptible with asymptomatic individuals
β_s	:	Rate of infection of susceptible with symptomatic individuals
κ	:	Rate of incubation
ρ	:	Rate of progression from asymptomatic to symptomatic
$\xi \\ \delta_1$:	Fraction of exposed individuals not showing symptoms
δ_1	:	Hospitalized/Quarantine rate of asymptomatic individuals
δ_2	:	Rate of hospitalized symptomatic individuals
γ_1	:	Recovery rate of asymptomatic individuals
γ_2	:	Recovery rate of symptomatic individuals
γ_3	:	Recovery rate of hospitalized individuals
μ	:	Natural mortality rate of human
μ_1	:	Disease related mortality rate for symptomatic individuals
μ_2	:	Disease related mortality rate for hospitalized individuals

Copyright © 2021 ASSA.

3. ANALYSIS OF THE MODEL SYSTEM

From the system (1), we have

$$\frac{dS}{dt}\Big|_{S=0} = \Lambda > 0, \frac{dE}{dt}\Big|_{E=0} = \beta_a SI_a + \beta_s SI_s \ge 0, \frac{dI_a}{dt}\Big|_{I_a=0} = \xi \kappa E \ge 0$$

$$\frac{dI_s}{dt}\Big|_{I_s=0} = (1-\xi)\kappa E + \rho I_a \ge 0, \frac{dH}{dt}\Big|_{H=0} = \delta_1 I_a + \delta_2 I_s \ge 0, \frac{dR}{dt}\Big|_{R=0} = \gamma_1 I_a + \gamma_2 I_s + \gamma_3 H \ge 0$$

Here, all the rates are non-negative on the bounding planes. So, if we start in the interior of the 6-dimensional closed hyperoctant \mathbb{R}^6_+ , we will always remain there, in view of the fact that the direction of the vector field is inward on all the bounding planes. Thus, non-negativity of all the solutions of the model system (1) is guaranteed.

Further, from the model system (1), we note that the total human population $N = S_1 + S_2 + I + H + Q + R$ satisfies,

$$\frac{dN}{dt} = \Lambda - \mu N - \mu_1 I_s - \mu_2 H$$

This gives

$$\limsup_{t \to \infty} N \le \frac{\Lambda}{\mu}$$

Therefore, all the solutions S(t), E(t), $I_a(t)$, $I_s(t)$, H(t), R(t) are bounded by $\frac{\Lambda}{\mu}$. Hence, the biologically feasible region for the system (1) is given by the following positively invariant set:

$$\Omega = \{ (S, E, I_a, I_s, H, R) \in \mathbb{R}^6_+ : 0 \le S + E + I_a + I_s + H + R \le \frac{\Lambda}{\mu} \}$$

3.1. Basic Reproduction Number

The disease-free equilibrium for the model (1) as $E_0 = (S^0, E^0, I_a^0, I_s^0, H^0, R^0) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0).$

We find the basic reproduction number R_0 by using the next generation matrix method [26]. The new infection terms of the matrix \mathcal{F} and the transition terms of the matrix \mathcal{V} of the system (1) are respectively, as follows:

$$\mathcal{F} = \left(\begin{array}{c} \beta_a S I_a + \beta_s S I_s \\ 0 \\ 0 \\ 0 \end{array} \right),$$

$$\mathcal{V} = \begin{pmatrix} (\kappa + \mu)E \\ -\xi\kappa E + (\rho + \gamma_1 + \delta_1 + \mu)I_a \\ -(1 - \xi)\kappa E - \rho I_a + (\gamma_2 + \delta_2 + \mu_1 + \mu)I_s \\ -\delta_1 I_a - \delta_2 I_s + (\gamma_3 + \mu_2 + \mu)H \end{pmatrix}$$

Now, we find the matrices F (of new infection terms) and V (of the transition terms) as

Copyright © 2021 ASSA.

It follows that

where,

where,

$$m_{11} = \frac{\kappa\xi\beta_{a}\Lambda}{\mu(\kappa+\mu)(\rho+\gamma_{1}+\delta_{1}+\mu)} + \frac{\beta_{s}\Lambda\kappa(\xi\rho+(1-\xi)(\rho+\gamma_{1}+\delta_{1}+\mu))}{\mu(\kappa+\mu)(\rho+\gamma_{1}+\delta_{1}+\mu)(\gamma_{2}+\delta_{2}+\mu_{1}+\mu)}$$

$$m_{12} = \frac{\beta_{a}\Lambda}{\mu(\rho+\gamma_{1}+\delta_{1}+\mu)} - \frac{\rho\beta_{s}\Lambda}{\mu(\rho+\gamma_{1}+\delta_{1}+\mu)(\gamma_{2}+\delta_{2}+\mu_{1}+\mu)},$$

$$m_{13} = \frac{\beta_{s}\Lambda}{\mu(\gamma_{2}+\delta_{2}+\mu_{1}+\mu)}$$

The basic reproduction number is same as the spectral radius of the next-generation matrix FV^{-1} . Thus, from above, we obtain the expression for R_0 as

$$R_{0} = \frac{\kappa\Lambda}{\mu(\kappa+\mu)(\rho+\gamma_{1}+\delta_{a}+\mu)} \left[\xi\beta_{a} + \frac{\beta_{s}\{\xi\rho+(1-\xi)(\rho+\gamma_{1}+\delta_{1}+\mu)\}}{\gamma_{2}+\delta_{2}+\mu_{1}+\mu}\right]$$

The quantity R_0 is known as basic reproduction number, the expected number of secondary cases produced in completely susceptible population, by a typical infective individual for the system (1).

3.2. Existence of Endemic Equilibrium Point

The endemic equilibrium of the system (1) satisfies the following algebraic equations such as

$$\frac{dS}{dt} = 0, \ \frac{dE}{dt} = 0, \ \frac{dI_a}{dt} = 0, \ \frac{dI_s}{dt} = 0, \ \frac{dI_s}{dt} = 0, \ \frac{dH}{dt} = 0, \ \frac{dR}{dt} = 0$$

The system (1) realize a unique positive solution $E_1 = (S^*, E^*, I_a^*, I_s^*, H^*, R^*)$

$$S^* = \frac{\kappa + \mu}{(\beta_a d_1 + \beta_s d_2)}, \quad E^* = \frac{\Lambda(\beta_a d_1 + \beta_s d_2) - \mu(\kappa + \mu)}{(\beta_a d_1 + \beta_s d_2)(\kappa + \mu)},$$
$$I_a^* = \frac{\xi \kappa E^*}{\rho + \gamma_1 + \delta_1 + \mu} = d_1 E^*, \quad I_s^* = \frac{(1 - \xi)\kappa E^* + \rho d_1 E^*}{\gamma_2 + \delta_2 + \mu_1 + \mu} = d_2 E^*,$$
$$H^* = \frac{(\delta_1 d_1 + \delta_2 d_2)E^*}{\gamma_3 + \mu_2 + \mu} = d_3 E^*, \quad R^* = \frac{(\gamma_1 d_1 + \gamma_2 d_2 + \gamma_3 d_3)E^*}{\mu}$$

where,

$$d_1 = \frac{\kappa\xi}{\rho + \gamma_1 + \delta_1 + \mu}, \ d_2 = \frac{(1 - \xi)\kappa + \rho d_1}{\gamma_2 + \delta_2 + \mu_1 + \mu}, \ d_3 = \frac{(\delta_1 d_1 + \delta_2 d_2)}{\gamma_3 + \mu_2 + \mu}$$

Copyright © 2021 ASSA.

4. STABILITY ANALYSIS OF THE MODEL

Theorem 4.1:

For model system (1), the disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

4.1. Global stability of disease-free equilibrium

To prove the global stability of disease-free equilibrium, we are using the theorem by Castillochavez et al. [25]

Theorem 4.2:

If the given mathematical model can be written in the form:

$$\frac{dX}{dt} = F(X, Y), and \quad \frac{dY}{dt} = G(X, Y), \quad G(X, 0) = 0$$
 (2)

where X = S, $Y = (E, I_a, I_s, H)^T$, denoting the number of uninfected and denoting the number of Covid-19 infected people respectively. Then the disease-free equilibrium is represented here by

$$E_0 = (X_0, 0) = (\frac{\Lambda}{\mu}, 0)$$

For the global asymptotically stable, the condition (H_1) and (H_2) given below must be satisfied.

$$H_{1}: for \frac{dX}{dt} = F(X_{0}, 0),$$

$$H_{2}: G(X, Y) = AY - \widehat{G}(X, Y), \ \widehat{G}(X, Y) \ge 0,$$

Here $A = D_Y G(X_0, 0)$ is M-matrix (In M-matrix, all the off diagonal element of matrix are non-negative). If the given system of differential equation in mathematical model satisfies the given condition in (2) then the point $E_0 = (X_0, 0)$ is a global asymptotically stable equilibrium of given mathematical model provided $R_0 < 1$. And for the given mathematical model, the result is shown in the next theorem, as given below.

Theorem 4.3:

The point $E_0 = (X_0, 0)$ of the system (1) is global asymptotically stable (G.A.S.), provided $R_0 < 1$. and the condition given in (2) are satisfied.

Proof

By using theorem (2.1) to our model system (1), we get

$$F(X_0, 0) = \Lambda - \mu S, \ G(X, Y) = AY - \widehat{G}(X, Y)$$

where,

$$A = F - V = \begin{pmatrix} -(\kappa + \mu) & \beta_a S & \beta_s S & 0\\ \xi \kappa & -(\rho + \gamma_1 + \delta_1 + \mu) & 0 & 0\\ (1 - \xi)\kappa & \rho & -(\gamma_2 + \delta_2 + \mu_1 + \mu) & 0\\ 0 & \delta_1 & \delta_2 & -(\gamma_3 + \mu_2 + \mu) \end{pmatrix}$$

Copyright © 2021 ASSA.

then

$$G(X,Y) = AY - \hat{G}(X,Y) = AY - \begin{pmatrix} \hat{G}_1(X,Y) \\ \hat{G}_2(X,Y) \\ \hat{G}_3(X,Y) \\ \hat{G}_4(X,Y) \end{pmatrix} = \begin{pmatrix} (S^0 - S)(\beta_a I_a + \beta_s I_s) \\ 0 \\ 0 \end{pmatrix}$$

where

$$AY = \begin{pmatrix} -(\kappa + \mu)E + S^{0}(\beta_{a}I_{a} + \beta_{s}I_{s}) \\ \xi\kappa E - (\rho + \gamma_{1} + \delta_{1} + \mu)I_{a} \\ (1 - \xi)\kappa E + \rho I_{a} + (\gamma_{2} + \delta_{2} + \mu_{1} + \mu)I_{s} \\ \delta_{1}I_{a} + \delta_{2}I_{s} + (\gamma_{3} + \mu_{2} + \mu)H \end{pmatrix}$$

and

$$\widehat{G}(X,Y) = \begin{pmatrix} (\kappa+\mu)E + S(\beta_a I_a + \beta_s I_s) \\ \xi \kappa E - (\rho + \gamma_1 + \delta_1 + \mu)I_a \\ (1-\xi)\kappa E + \rho I_a + (\gamma_2 + \delta_2 + \mu_1 + \mu)I_s \\ \delta_1 I_a + \delta_2 I_s + (\gamma_3 + \mu_2 + \mu)H \end{pmatrix}$$

Here, we can easily see $S^0 \ge S$, hence $G(X, Y) \ge 0$ for all (X, Y). Also by the defination of M matrix we can say that the matrix A is M matrix. Hence, disease-free equilibrium (E_0) is global asymptotically stable.

4.2. Global stability of endemic equilibrium

Theorem 4.4:

The endemic equilibrium $E_1 = (S^*, E^*, I_a^*, I_s^*, H^*, R^*)$ of the given mathematical model is globally asymptotically stable.

Proof

For the global stability result, we will use the method discussed in Korobeinikov and Wake [27], Li and Muldowney [28, 29]. Here we consider the following Lyapunov function:

$$L = C_1 \left(S - S^* - S^* ln \frac{S}{S^*} \right) + C_2 \left(E - E^* - E^* ln \frac{E}{E^*} \right) + C_3 \left(I_a - I_a^* - I_a^* ln \frac{I_a}{I_a^*} \right) + C_4 \left(I_s - I_s^* - I_s^* ln \frac{I_s}{I_s^*} \right)$$

Then the time derivative of L is given by

$$\frac{dL}{dt} = C_1 \left(1 - \frac{S^*}{S} \right) \frac{dS}{dt} + C_2 \left(1 - \frac{E^*}{E} \right) \frac{dE}{dt} + C_3 \left(1 - \frac{I_1^*}{I_1} \right) \frac{dI_1}{dt} + C_4 \left(1 - \frac{I_2^*}{I_2} \right) \frac{dI_2}{dt}$$

Now from the mathematical model we put the expressions for $\frac{dS}{dt}$, $\frac{dE}{dt}$, $\frac{dI_a}{dt}$, $\frac{dI_s}{dt}$, in the above equation, which gives

$$\frac{dL}{dt} = C_1 \left(1 - \frac{S^*}{S} \right) \left\{ \Lambda - \beta_a I_a S - \beta_s I_s S - \mu S \right\}$$

+
$$C_2 \left(1 - \frac{E^*}{E} \right) \left\{ \beta_a I_a S + \beta_s I_s S - (\kappa + \mu) E \right\}$$

+
$$C_3 \left(1 - \frac{I_a^*}{I_a} \right) \left\{ \xi \kappa E - \rho I_a - (\gamma_1 + \mu) I_a - \delta_1 I_a \right\}$$

Copyright © 2021 ASSA.

+
$$C_4 \left(1 - \frac{I_s^*}{I_s} \right) \left\{ (1 - \xi) \kappa E + \rho I_a - (\gamma_2 + \mu_1 + \mu) I_s - \delta_2 I_s \right\}$$
 (3)

The mathematical model system satisfies the following relation at the equilibrium point.

$$\Lambda = \beta_a I_a^* S^* + \beta_s I_s^* S^* + \mu S^*, \quad \kappa + \mu = \frac{\beta_a I_a^* S^* + \beta_s I_s^* S^*}{E^*},$$
$$\rho + \gamma_1 + \delta_1 + \mu = \frac{\xi \kappa E^*}{I_a^*}, \quad \gamma_2 + \delta_2 + \mu_1 + \mu = \frac{(1 - \xi)\kappa E^* + \rho I_a^*}{I_s^*}$$

Putting all the above expressions in (3) we get,

$$\begin{aligned} \frac{dL}{dt} &= C_1 \left(1 - \frac{S^*}{S} \right) \left[\beta_a I_a^* S^* + \beta_s I_s^* S^* + \mu S^* - \beta_a I_a S - \beta_s I_s S - \mu S \right] \\ &+ C_2 \left(1 - \frac{E^*}{E} \right) \left[\beta_a I_a S + \beta_s I_s S - \left(\frac{\beta_a I_a^* S^* + \beta_s I_s^* S^*}{E^*} \right) E \right] \\ &+ C_3 \left(1 - \frac{I_a^*}{I_a} \right) \left[\xi \kappa E - \left(\frac{\xi \kappa E^*}{I_a^*} \right) I_a \right] \\ &+ C_4 \left(1 - \frac{I_s^*}{I_s} \right) \left[(1 - \xi) \kappa E + \rho I_a - \left(\frac{(1 - \xi) \kappa E^* + \rho I_a^*}{I_s^*} \right) I_s \right] \end{aligned}$$

Then,

$$\begin{aligned} \frac{dL}{dt} &= -C_1 \left(\frac{(S^* - S)^2}{S} \right) \mu + C_1 \left[\left(1 - \frac{S^*}{S} \right) \left\{ \beta_a I_a^* S^* + \beta_s I_s^* S^* - \beta_1 I_a S - \beta_2 I_s S \right\} \right] \\ &+ C_2 \left(1 - \frac{E^*}{E} \right) \left[\beta_a I_a S + \beta_s I_s S - \left(\frac{\beta_a I_a^* S^* + \beta_s I_s^* S^*}{E^*} \right) E \right] \\ &+ C_3 \left(1 - \frac{I_a^*}{I_a} \right) \left[\xi \kappa E - \left(\frac{\xi \kappa E^*}{I_a^*} \right) I_a \right] \\ &+ C_4 \left(1 - \frac{I_s^*}{I_s} \right) \left[(1 - \xi) \kappa E + \rho I_a - \left(\frac{(1 - \xi) \kappa E^* + \rho I_a^*}{I_s^*} \right) I_s \right] \\ \frac{dL}{dt} &= -C_1 \left(\frac{(S^* - S)^2}{S} \right) \mu + g(x_1, x_2, x_3, x_4) \end{aligned}$$

where,

$$\frac{S}{S^*} = x_1, \quad \frac{E}{E^*} = x_2, \quad \frac{I_a}{I_a^*} = x_3, \quad \frac{I_s}{I_s^*} = x_4,$$

$$\beta_a S^* I_a^* = p, \quad \beta_s S^* I_s^* = q, \quad \xi \kappa E^* = r, \quad (1 - \xi) \kappa E^* = s$$

Now,

$$g(x_1, x_2, x_3, x_4) = C_1(p + q - px_1x_3 - qx_1x_4) - C_1p\frac{1}{x_1} - C_1q\frac{1}{x_1} + C_1px_3 + C_1qx_4 + C_2(px_1x_2 + qx_1x_4 - ax_2 - qx_2) - C_2p\left(\frac{x_1x_3}{x_2}\right) - C_2q\left(\frac{x_1x_4}{x_2}\right)$$

Copyright © 2021 ASSA.

$$+ C_{2}(p+q) + C_{3}(rx_{2} - rx_{3} - r\frac{x_{2}}{x_{3}} + r) + C_{4}(sx_{2} - sx_{4} - s\frac{x_{2}}{x_{4}} + s)$$

$$= x_{2}(-C_{2}p + C_{3}r + C_{4}s) + x_{3}(C_{1}p - C_{3}r) + x_{4}(C_{1}q - C_{4}s)$$

$$+ x_{1}x_{3}(-C_{1}p + C_{2}p) + x_{1}x_{4}(C_{1}q + C_{2}q) + C_{1}(2p+q)$$

$$+ C_{2}(p+q) + C_{3}r - C_{3}r\left(\frac{x_{2}}{x_{3}}\right) + C_{4}s - C_{4}s\left(\frac{x_{2}}{x_{4}}\right) - C_{1}(p+q)\left(\frac{1}{x_{1}}\right)$$

$$- C_{2}(px_{1}x_{3} + qx_{1}x_{4})\left(\frac{1}{x_{2}}\right)$$

To get the values of C_1, C_2, C_3, C_4 we take the coefficients of $x_1x_3, x_1x_4, x_2, x_3, x_4$ equal to zero and solve the algebraic equations in C_1, C_2, C_3, C_4 . This gives

$$C_1 = C_2; \ C_3 = \frac{C_1 p}{r}; \ C_4 = \frac{C_1 q}{s}$$

Choosing $C_1 = C_2 = 1$ and n = 1, we get

$$g(x_1, x_2, x_3, x_4) = p\left(3 - \frac{1}{x_1} - \frac{x_1 x_3}{x_2} - \frac{x_2}{x_3}\right) + q\left(3 - \frac{1}{x_1} - \frac{x_1 x_4}{x_2} - \frac{x_2}{x_4}\right)$$

Since the arithmetic mean is greater than or equal to geometric mean, we have

$$\frac{1}{x_1} + \frac{x_1 x_3}{x_2} + \frac{x_2}{x_3} \ge 3$$

and

$$\frac{1}{x_1} + \frac{x_1 x_4}{x_2} + \frac{x_2}{x_4} \ge 3$$

Hence,

$$\frac{dL}{dt} = -\left(\frac{(S^* - S)^2}{S}\right)\mu + p\left(3 - \frac{1}{x_1} - \frac{x_1x_3}{x_2} - \frac{x_2}{x_3}\right) + q\left(3 - \frac{1}{x_1} - \frac{x_1x_4}{x_2} - \frac{x_2}{x_4}\right)$$

Thus it is easy to observe that $\frac{dL}{dt} \le 0$ and the equality $\frac{dL}{dt} = 0$ hold on; y for $x_1 = x_2 = x_3 = x_4 = 1$ for which $S = S^*$, $E = E^*$, $I_a = I_a^*$, $I_s = I_s^*$. From the LaSalle's invariance principle [30], the equilibrium E_1 of the given system is

globally asymptotically stable for $R_0 > 1$.

5. SENSITIVITY ANALYSIS OF REPRODUCTION NUMBER

In this section, we also perform sensitivity analysis for the parameters involved in reproduction number R_0 , which reflects that increase or decrease in these parameter causes increase or decrease in R_0 . The sensitivity of R_0 to different parameters is shown in Figure 3. It is used to discover the parameters that have a high impact on R_0 and should be targeted by intervention strategies. Sensitivity indices allows to measure the relative change in a variable when parameter changes. For that we use the forward sensitivity index of a variable, with

Copyright © 2021 ASSA.

respect to a given parameter, which is defined as the ratio of the relative change in the variable to the relative change in the parameter. If such variable is differentiable with respect to the parameter, then the sensitivity index is defined using partial derivatives. The normalized forward sensitivity index of R_0 , which is differentiable with respect to a given parameter α , is defined by

$$Y_{\alpha}^{R_0} = \frac{\alpha}{R_0} \frac{\partial R_0}{\partial \alpha}$$

The above formula can be used to compute the analytical expression for the sensitivity of R_0 to each parameter that it includes. Accordingly, the sensitivity indexes of the model (1) are illustrate in Figure 2. Consequently, the value of R_0 increases with increase in the values of all positive indices parameters Λ , β_a , β_s , κ , and ξ with R_0 . Also, the parameters γ_1 , γ_2 , δ_1 , δ_2 , ρ , μ , and μ_1 have negative index with R_0 . It is clearly observed that the effect of the parameter Λ is the maximum and hence it is the most sensitive parameter of R_0 . It means that small change (increase or decrease) in the parameters (Λ) will significant change in the value of R_0 by 100%. It is obvious that phenomenon of a lower value of R_0 will boost to prevent the disease prevalence. Thus, to control the disease from the population, we have to control the increase of parameters having positive indices with R_0 , whereas parameters with negative indices should be maintained.

6. EFFECTS OF PARAMETERS ON DISEASE OUTBREAK

For the Numerical simulation of the model, we consider all the parameters are in per day basis. First we consider the following set of parameters which corresponds to disease-free equilibrium.

$$\Lambda = 2; \beta_a = 0.00085; \beta_s = 0.00029; \gamma_1 = 1/14; \gamma_2 = 0.00245; \gamma_3 = 0.002; \kappa = 1.19;$$

$$\mu_1 = 0.1; \mu_2 = 0.015; \mu = 0.00425; \rho = 0.001; \xi = 0.0015; \delta_1 = 0.16; \delta_2 = 0.01;$$

For the above set of parameters we get $R_0 = 0.0932 < 1$ and the disease-free equilibrium point $E_0(462.52, 0, 0, 0, 0, 0)$ is stable. This fact is demonstrated in Figure 4 (a). Later, we did few changes for endemic equilibrium as follows:

$$\Lambda = 40; \ \beta_a = 0.00085; \ \beta_s = 0.00029; \ \gamma_1 = 1/14; \ \gamma_2 = 0.000245; \ \gamma_3 = 0.002;$$

$$\kappa = 0.89; \ \mu_1 = 1.05; \ \mu_2 = 0.015; \ \mu = 0.0125; \ \rho = 0.69; \ \xi = 0.95; \ \delta 1 = 0.012; \ \delta 2 = 0.019;$$

From the above set of parameter we get $R_0 = 2.0075 > 1$, and the endemic equilibrium $E_1(801.56, 26.82, 25.04, 24, 43, 38.75, 208.6,)$ is stable. The stability of the equilibrium point E_1 is shown in Figure 4(b). The effect of different values of recovery rate from asymptomatic (γ_1) , symptomatic (γ_2) and hospitalized (γ_3) , which corresponds to infective human is demonstrated in Figure 4(c), 4(d), and 4(e) respectively. It is clear that the parameter of recovery rate γ_1 , γ_2 , and γ_3 increases, simultaneously the infected population decreases. The effect of S, H, and R also shown in the figure 4(f) with respect to endemic equilibrium point.

Copyright © 2021 ASSA.

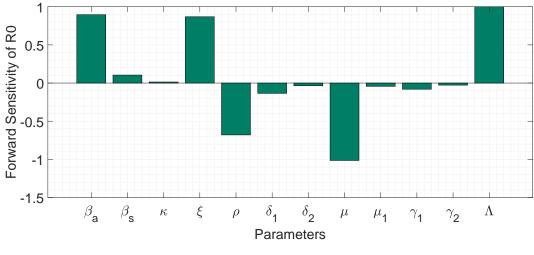


Fig. 2. Forward sensitivity of R_0

7. THE OPTIMAL CONTROL MODEL

In this section, the model (1) is extended to formulate optimal control problem by incorporating two time-dependent optimal control parameters, namely $u_1(t)$, and $u_2(t)$. If u_1 , and u_2 equal zero, no effort is placed in these controls at time t, and if they equal one, maximum effort is applied. Thus, optimal control variables are given, as follows:

The control variable $u_1(t)$ represents the reduction in the transmission between humanto-human via using surgical face masks, social distancing, self-isolation, sensitization and awareness of transmission of the disease.

The control variable $u_2(t)$ represents the increase in the testing facility and treatment, which can lead to fast detection of Covid infected cases and recovery and add additional time-dependent parameter $\eta u_2(t)$ in the rate of direction δ_2 . Keeping in view of the above assumptions, the optimal control model is formulated as follows:

$$\frac{dS}{dt} = \Lambda - (1 - u_1)\beta_a I_a S - (1 - u_1)\beta_s I_s S - \mu S
\frac{dE}{dt} = (1 - u_1)\beta_a I_a S + (1 - u_1)\beta_s I_s S - (\kappa + \mu)E
\frac{dI_a}{dt} = \xi \kappa E - (\rho + \gamma_1 + \mu + \delta_1)I_a
\frac{dI_s}{dt} = (1 - \xi)\kappa E + \rho I_a - (\gamma_2 + \mu_1 + \mu)I_s - (\delta_2 + \eta u_2)I_s$$
(4)
$$\frac{dH}{dt} = \delta_1 I_a + (\delta_2 + \eta u_2)I_s - (\gamma_3 + \mu_2 + \mu)H
\frac{dR}{dt} = \gamma_1 I_a + \gamma_2 I_s + \gamma_3 H - \mu R$$

Copyright © 2021 ASSA.

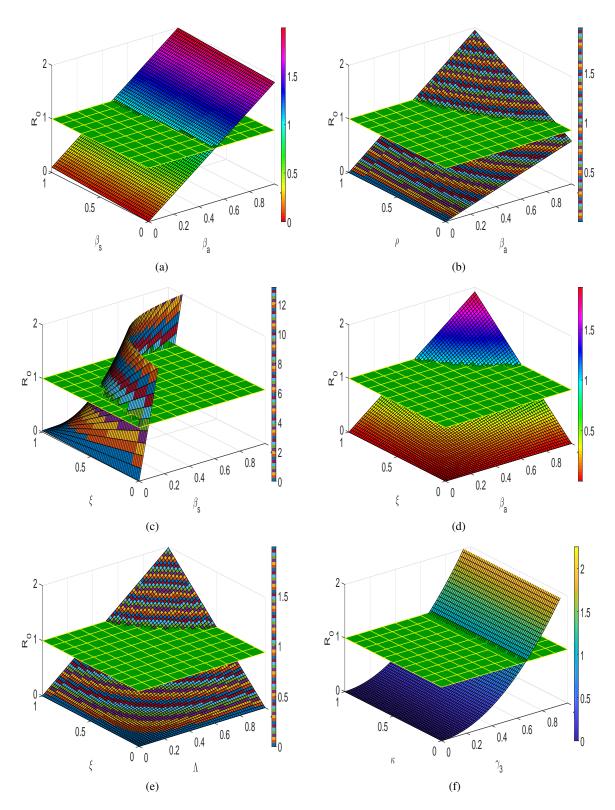


Fig. 3. (a) Influence of β_a and β_s on R_0 , (b) Influence of β_a and ρ on R_0 , (c) Influence of β_s and ξ on R_0 (d) Influence of β_a and ξ on R_0 , (e) Influence of Λ and ξ on R_0 , (f) Influence of γ_3 and κ on R_0

Copyright © 2021 ASSA.

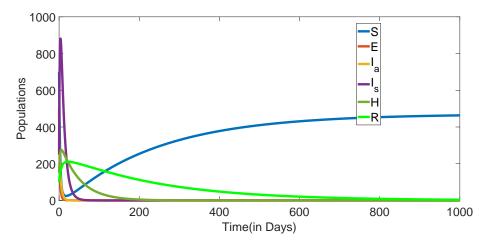


Fig. 4. Variation of S, E, I_a , I_s , H, R performing the stability of disease-free equilibrium point with $R_0 = 0.0932 < 1$.

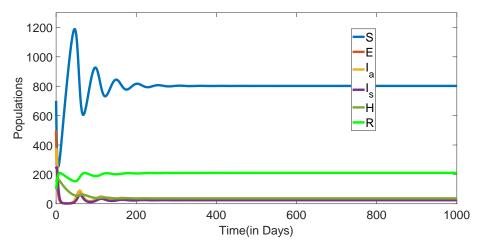


Fig. 5. Variation of S, E, I_a , I_s , H, R performing the stability of endemic equilibrium point with $R_0 = 2.0075 > 1$.

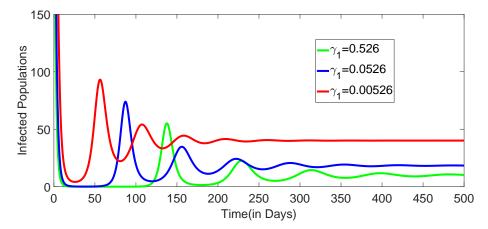


Fig. 6. Variation of I_a with time performing different values of γ_1

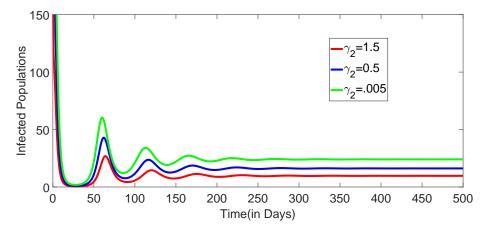


Fig. 7. Variation of I_s with time performing different values of γ_2

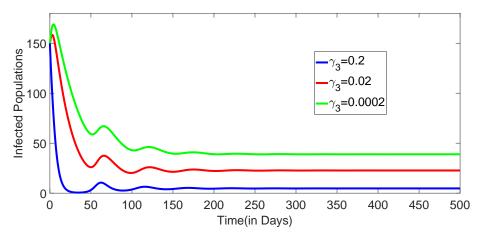


Fig. 8. Variation of H with time performing different values of γ_3

7.1. The Optimal Control Problem

In this section, we study the behavior of the proposed model by using optimal control theory. The objective functional for fixed time T_f if given by

$$J(u_1, u_2) = \int_0^{T_f} \left[A_1 I_a + A_2 I_s + \frac{1}{2} (B_1 u_1^2 + B_2 u_2^2) \right] dt$$
(5)

subject to the model system (4). The parameter $A_1 \ge 0$, $A_2 \ge 0$, $B_1 \ge 0$, $B_2 \ge 0$ are the weight and balancing constants, which measure the respective cost involvement over the interval $[0, T_f]$. In order to find an optimal control, u_1^* , and u_2^* such that

$$J(u_1^*, u_2^*) = \min_{(u_1, u_2) \in \Omega} J(u_1, u_2),$$
(6)

where Ω is the control set and is defined as

$$\Omega = \{(u_1, u_2) : 0 \le u_1, u_2 \le 1, t \in [0, T_f]\}$$

Here, all the controls are bounded and measurable.

7.1.1. Existence and characterization of optimal controls Here, we shall first establish the existence of such control functions that minimizes the cost functional J. The Lagrangian L

Copyright © 2021 ASSA.

of this problem is defined as:

$$L(I_a, I_s, u_1, u_2) = A_1 I_a + A_2 I_s + \frac{1}{2} B_1 u_1^2 + \frac{1}{2} B_2 u_2^2$$

Now, we shall use Pontryagin's maximum principle [32, 33] for necessary conditions for optimal controls system (4). For that by choosing $X = (S, E, I_a, I_s, H, R)$, $\Omega = (u_1, u_2)$ and $\lambda = (\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6)$, the associated Hamiltonian \mathcal{H} can be written as

$$\mathcal{H}(X,\Omega,\lambda) = L(I_a, I_s, u_1, u_2) + \lambda_1 \frac{dS}{dt} + \lambda_2 \frac{dE}{dt} + \lambda_3 \frac{dI_a}{dt} + \lambda_4 \frac{dI_s}{dt} + \lambda_5 \frac{dH}{dt} + \lambda_6 \frac{dR}{dt} (7)$$

Since u_1^* , and u_2^* are solutions to the control problem (4), there exists the adjoint variables $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ satisfying the following conditions.

$$\frac{dx}{dt} = \frac{\partial \mathcal{H}(t, x, u_1^*, u_2^*, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6)}{\partial \lambda} \\
0 = \frac{\partial \mathcal{H}(t, x, u_1^*, u_2^*, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6)}{\partial u} \\
\frac{d\lambda}{dt} = -\frac{\partial \mathcal{H}(t, x, u_1^*, u_2^*, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6)}{\partial x}$$
(8)

Theorem 7.1:

For the objective functional (5) and the control set (8) subject to control system (4) there exists an optimal control $u^* = (u_1^*, u_2^*) \in \Omega$ such that

$$J(u_1^*, u_2^*) = \min_{\Omega} J(u_1, u_2).$$

Proof

To establish this result, we follow the Theorem 4.1 mentioned in [40] for the existence of optimal controls. As, we have discussed above that all the state variables (population) are bounded for each bounded controls coming from the control set Ω . Furthermore, Lipschitz condition with respect to state variables is satisfied by the right hand part of the model system (4). The control variable set Ω is also convex and closed by the definition and the model system (4) is linear in control variables.

Thus, all the conditions for the existence of controls are fulfilled (for more details one can follow [28, 29]). Hence the result. \Box

Theorem 7.2:

For optimal controls measures u_1^*, u_2^* and the state solutions $S^*, E^*, I_a^*, I_s^*, H^*, R^*$ of the state system (4), there exists adjoint variables $\lambda = (\lambda_i)^{T_f} \in \mathbb{R}^6, i = 1, 2, 3, 4, 5, 6$ such that

$$\frac{d\lambda_1}{dt} = (1 - u_1)\beta_a I_a(\lambda_1 - \lambda_2) + (1 - u_1)\beta_s I_s(\lambda_1 - \lambda_2) + \mu\lambda_1$$

$$\frac{d\lambda_2}{dt} = (\kappa + \mu)\lambda_2 + \xi\kappa(\lambda_4 - \lambda_3) - \kappa\lambda_4$$

$$\frac{d\lambda_3}{dt} = -A_1 + (1 - u_1)\beta_a S(\lambda_1 - \lambda_2) + \rho(\lambda_3 - \lambda_4) + \gamma_1(\lambda_3 - \lambda_6) + (\mu + \delta_1)\lambda_3$$

$$\frac{d\lambda_4}{dt} = -A_2 + (1 - u_1)\beta_s S(\lambda_1 - \lambda_2) + \gamma_2(\lambda_4 - \lambda_6) + (\mu_1 + \mu)\lambda_4 + (\delta_2 + \eta u_2)(\lambda_4 - \lambda_5)$$

$$\frac{d\lambda_5}{dt} = \gamma_3(\lambda_5 - \lambda_6) + (\mu_2 + \mu)\lambda_5$$
(9)
$$\frac{d\lambda_6}{dt} = \mu\lambda_6$$

Copyright © 2021 ASSA.

with transversality conditions

$$\lambda_1(T_f) = \lambda_2(T_f) = \lambda_3(T_f) = \lambda_4(T_f) = \lambda_5(T_f) = \lambda_6(T_f) = 0$$
(10)

Proof

Let u_1^*, u_2^* be the optimal control functions and $S^*, E^*, I_a^*, I_s^*, H^*, R^*$ are the corresponding state variables. Then, Pontryagin's Maximum Principle ensures the existence of the following adjoint variable $\lambda_i (i = 1, 2, 3, 4, 5, 6) \in \mathbb{R}^6$, which satisfies the following canonical equations:

$$\frac{d\lambda_1}{dt} = -\frac{\partial \mathcal{H}}{\partial S}, \quad \frac{d\lambda_2}{dt} = -\frac{\partial \mathcal{H}}{\partial E}, \quad \frac{d\lambda_3}{dt} = -\frac{\partial \mathcal{H}}{\partial I_a}, \quad \frac{d\lambda_4}{dt} = -\frac{\partial \mathcal{H}}{\partial I_s}, \quad \frac{d\lambda_5}{dt} = -\frac{\partial \mathcal{H}}{\partial H}, \quad \frac{d\lambda_6}{dt} = -\frac{\partial \mathcal{H}}{\partial R}$$

with transversality conditions (10) and the Hamiltonian (7). The adjoint system (9) can be obtained. $\hfill \Box$

In the following result, we shall state the analytical forms of the optimal controls.

Theorem 7.3:

The optimal controls (u_1^*, u_2^*) which minimizes J over the region Ω are given by

$$u_1^* = \min\{1, \max(0, \tilde{u_1})\}$$
$$u_2^* = \min\{1, \max(0, \tilde{u_2})\}$$

where

$$\tilde{u_1} = \frac{(\beta_1 I_a + \beta_2 I_s) S(\lambda_2 - \lambda_1)}{B_1},$$
$$\tilde{u_2} = \frac{\eta I_s(\lambda_4 - \lambda_5)}{B_2},$$

Proof

Using optimally condition, we have

$$\frac{\partial \mathcal{H}}{\partial u_1} = 0, \quad \frac{\partial \mathcal{H}}{\partial u_2} = 0$$

We have

$$\frac{\partial \mathcal{H}}{\partial u_1} = B_1 u_1 + (\beta_a I_a + \beta_s I_s) S(\lambda_1 - \lambda_2) = 0$$

This gives

$$u_1 = \frac{\left((\beta_a I_a + \beta_s I_s) S \right) (\lambda_2 - \lambda_1)}{B_1} := \widetilde{u_1}$$

Similarly,

$$\frac{\partial \mathcal{H}}{\partial u_2} = B_2 u_2 + \eta I_s (\lambda_5 - \lambda_4) = 0$$

This implies

$$u_2 = \frac{\eta I_s(\lambda_4 - \lambda_5)}{B_2} := \widetilde{u_2}$$

Moreover, lower and upper bounds of these control are 0 and 1 respectively. Thus, if $\tilde{u_1} > 1$, $\tilde{u_2} > 1$, then

$$u_1 = u_2 = 1.$$

Copyright © 2021 ASSA.

Also, $\tilde{u_1} < 0$, $\tilde{u_2} < 0$ then

 $u_1 = u_2 = 0.$

Otherwise, we have

 $u_1 = \tilde{u_1}, and \ u_2 = \tilde{u_2}$

Hence, for these controls u_1^* , u_2^* we get optimum value of the function J.

8. SIMULATION OF OPTIMAL CONTROL PROBLEM

In this section, we simulate our optimal control model using MATLAB. The parameter values are keeping same the parameters corresponding to stability of endemic equilibrium point E_1 of the model (1). The weight constants for the optimal control problem are taken as $A_1 = 1, A_2 = 1, B_1 = 45, B_2 = 65$. We solve the optimality system by iterative method with the help of forward and backward difference approximations [32, 34–36]. We consider the time interval as [0,180]. First we solve the state equations by the forward difference approximation method to solve the adjoint equations. We consider different types of strategies to see the impact of optimal control in the total number of human infectives.

8.1. Strategy A: Employing hygiene promotion, social distancing and self-isolation (u_1) , only.

Here, only control measure $u_1(t)$ is used to optimize the objective function J, while control intervention $u_2(t) = 0$, were not employed. The influence of $u_1(t)$ is demonstrated in Figure 5(a), to minimize the objective function, the optimal control $u_1(t)$ is maintained at the maximum level. A single preventive measure can influence the spread of the Covid-19 in the population. Maintaining social distancing and self-isolation leads to control significant number reduction in asymptomatic and symptomatic cases in the population. From the figures, it is clear that the optimal control $u_1(t)$ is a little more effective compared to other types of controls but we need to maintain it to one for a longer period which is not easy to achieve. This control strategy is for using surgical face masks, social distancing, self-isolation, awareness of the transmission of disease, and sensitization.

8.2. Strategy B: Increase testing facility and treatment of the symptomatic individuals (u_2) only.

Here, only control measure $u_2(t)$ is used to optimize the objective function J, while control intervention $u_1(t) = 0$, were not employed. In Figure 5(b), we present the plots of population and the effects of the increase in testing facility and treatment are demonstrated to minimizing the cost and reducing the number of coronavirus infections in the population.

8.3. Strategy C: Employing both the control interventions (u_1, u_2) .

Here both the control interventions $(u_1(t), u_2(t))$ are used to optimize the objective function J. From Figure 5(c), it is easy to say that by combining both optimal controls $u_1(t)$ and $u_2(t)$, the total number of infectious individuals decreases significantly. The simulation result indicates the effectiveness of optimal control strategies in reducing the number of infectives. It is observed that combined controls are more useful in reducing the number of infected cases significantly. Finally, it observed that from Figure 5(d), the optimal control model gives a better result as compacted to the model without the optimal control model as it reduces the number of infectives significantly in a desired interval of time.

 \square

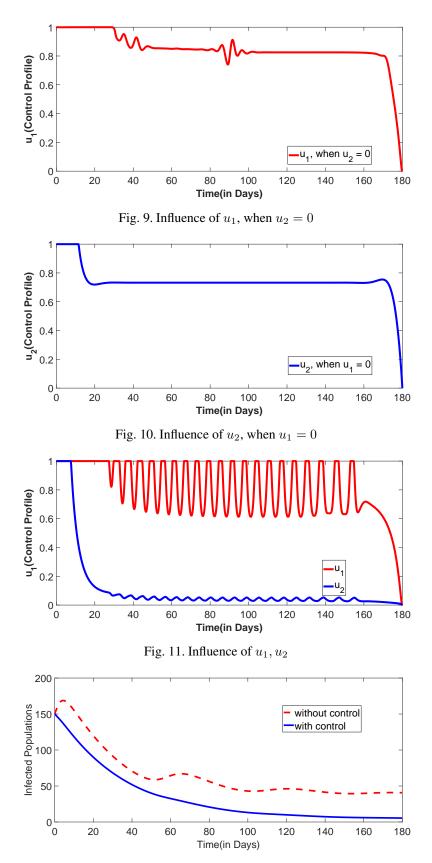


Fig. 12. Variation of infected human populations against time with and without optimal control

Copyright © 2021 ASSA.

9. CONCLUSION

Dynamics of Covid-19 pandemic and its optimal control strategies are discussed in this present study through a mathematical model. The host population of the model is subdividing into six different compartment according to nature of the disease such as susceptible, exposed, asymptomatic infective, symptomatic infective, hospitalized and recovery. This non-linear mathematical model is proposed based on Indian condition by considering asymptomatic and symptomatic infections populations. It is assumed that the transmission rates due to asymptomatic and symptomatic individuals are different. The epidemiological threshold (basic reproduction number) is computed by using next generation matrix method and discussed different equilibria of the model in details. The model is globally asymptotically stable for the global stability of disease-free equilibrium when basic reproduction number is less than unity using the Castillo-chavez theorem. Also the theoretical analysis is carried out the global stability of endemic equilibrium is globally asymptotically stable using Lyapunov method when basic reproduction number is greater than one. Sensitivity analysis of the model performed to identify the key parameter that influence the basic reproduction number, which will regulate to control transmission dynamics of the disease. It emphasized that Λ and μ are the most sensitive parameter, followed by asymptomatic transmission coefficient β_1 and ξ . Furthermore, the mathematical model is extended to optimal control problem by incorporating two time-dependent optimal control parameters to reducing the burden due to Covid-19 using Pontryagin's Maximum Principal. Introduced control parameter u_1 in the model as surgical face masks, social distancing, self-isolation, sensitization and awareness of transmission of the disease in asymptomatic and symptomatic invective individuals, which effectively reduce transmission rate. Social distancing should always implement at a higher percentage than self-isolation. The optimal control drives a significant reduction in the asymptomatic and symptomatic infected populations. The control parameter u_2 included reducing the cost infrastructure like testing facility, which can lead to fast detection of Covid infected cases. The optimal control suggests that unless the cost is very high, the social distancing should implement at the maximum level throughout the time examined. The optimal control model provides a more reliable result as compacted to the model without the optimal control model. This control strategy reduces the number of infectives significantly in a desired interval of time.

ACKNOWLEDGEMENTS

The authors would like to thank the editor and anonymous referees for their valuable comments and suggestions which led to an improvement of our original manuscript.

REFERENCES

- 1. Guanghong, D., Chang, L., Jianqiu, G., Ling, W., CHENG, K., and ZHANG, D. (2004) SARS epidemical forecast research in mathematical model, Chinese Science Bulletin, 49 (21), 2332–2338.
- 2. Killerby, M., H. Biggs, H., Midgleys, C., Gerber, S., Watson, J. (2020) Middle Esat Respiratory Syndrome coronavirus transmission, Emerg.Infect.Dis. 26 (4),191-198.
- 3. Willman, M., Kobasa, D., Kindrachuk, J. (2019) A comparative analysis of factors influencing two outbreaks of middle eastern respiratory syndrome (MERS) in Saudi Arabia and South Korea, Viruses 11(12):1119.Doi :10.3390/v11121119.PMID 31817037.
- 4. Kolifarhood, G., Aghaali, M., Saadati, H. M., Taherpour, N., Rahimi, S., Izadi, N., and Nazari, S.S.H. (2020) Epidemiological and Clinical Aspects of COVID-19: A Narative review, Archives of Academic Emergency Medicine, 8(1).
- 5. Chan, J.F.W., Yuan, S., Kok, K.H., Chu,H., Yang, J. (2019) Afamilial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person

Copyright © 2021 ASSA.

transmission: a study of a family cluster. Lancet. http://dx.doi.org/10.1016/S0140-6736(20) 30154-9.

- Bai, Y., Yao, L., Wei, T., Tian, F., Jin, D. Y., Chen, L. (2020) Presumed Asymptomatic Carrier Transmission of COVID-19. Jama. 2020 Feb 21. PubMed PMID: 32083643. Pubmed Central, PMCID: PMC7042844. Epub 2020/02/23. eng.
- 7. World Health Organization. Novel coronavirus China. Geneva,Switzerland: World Health Organization.https://www.who.int/csr/https://www.who.int/csr/don/12-january-2020-novel-coronavirus-china/en/
- 8. Hui, D. S., Azhar, E. I., Madani, T. A., Ntoumi, F., Kock, R., Dar, O. (2020) The continuing 2019 nCoV epidemic threat of novel coronaviruses to global health the latest 2019 novel coronavirus outbreak in Wuhan, China. Int. J. Infect. Dis.
- 9. N. Zhu, D. Zhang, W. Wang, X. W. Li, B. Yang, J. D. Song , et al. A novel coronavirus from patients with pneumonia in China, 2019, N Engl JMed.http: //dx.doi.org/10.1056/NEJMoa2001017. [2020-01-24].
- 10. World Health Organization. Statement on the second meeting of the International Health Regulations (2005)Emergency Committee regarding the outbreak Switzerland: coronavirus (2019-nCoV). Geneva, of novel World Health Organization.https://www.who.int/newsroom/detail/30-01-2020-statement-on-thesecond-meeting-of-theinternational-health-regulations-(2005)-emergency-committeeregar ding-the-outbreak-of-novel-coronavirus-(2019-ncov). [2020-01-30].
- 11. Wang, C., Hornby, P. W., Hayden, F. W., Gao, G. F. (2020) A novel coronavirus outbreak of global health concern. Lancet. http://dx.doi.org/10. 1016/S0140-6736(20)30185-9..
- 12. Munster, V.J., Koopmans, M., Doremalen, N. V., Riel, D. V., Wit, E. d. (2020) A novel coronavirus emerging in China key questions for impact assessment. N Engl J Med. http://dx.doi.org/10.1056/NEJMp2000929.
- 13. Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y. (2020) Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. http://dx.doi.org/10.1016/S0140-6736(20)30183-5.
- 14. WHO, (2020) Naming the coronavirus disease (COVID-19) and the virus that causes it. Available online:,https://www.who.int/emergencies/diseases/novel-coronavirus-2019/technical-guidance/[Retrieved: 25/03/2020].
- 15. WHO, (2020) Coronavirus disease 2019 (COVID-19), situation report -10. Available online:, https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports/[Retrieved: 25/03/2020].
- 16. Pulla, P. (2020) Covid-19: India imposes lockdown for 21 days and cases rise, BMJ 368, Doi: 10.1136/bmj.m1251.
- 17. Ministry of Health and Family Welfare (MOHFW), (2020) Coronavirus disease 2019 (COVID-19). Available online:, https://www.mohfw.gov.in/, [Retrieved: 25/03/2020].
- 18. Khanna, R.C., Honavar, S.G., (2020) All eyes on coronavirus what do we need to know as ophthalmologists, Indian Journal of Ophthalmology, 68(4) 549–553.
- 19. Siriprapaiwana, .S , Moorea, E. J., Koonpraserta, S. (2018) Generalized reproduction numbers, sensitivity analysis and critical immunity levels of an SEQIJR disease model with immunization and varying total population size, Mathematics and Computers in Simulation,146,70-89.
- Kucharski, A.J., Russell, T. W., Diamond, C., Liu, Y., Edmunds, J., Funk, S., Eggo, R. M. (2020) Early dynamics of transmission and control COVID-19: A mathematical modelling study, Lancet Infect. Dis. Doi:10.1016/S1473-3099(20)30144-4.
- 21. Senapati, A., Rana1b, S., Dasb, T., Chattopadhyaya, J. (2021)Impact of intervention on the spread of COVID-19 in India: A model based study, J. Theor Biol., doi:10.1016/j.jtbi.2021.110711, PMID:33862090.
- 22. Zhao, S., Lin, Q., Ran, J. (2020) Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCoV) in China, from 2019 to 2020: A data-driven analysis in the early phase of the outbreak, Int. J. Infect. Dis. 92, 214–217.

Copyright © 2021 ASSA.

- 23. Ivorra, B., Ferrández, M. R., Vela-Pérez, M.,and Ramos.A. M. (2020) Mathematical modeling of the spread of the coronavirus disease 2019 (COVID-19) considering its particular characteristics. The case of China. Communications in Nonlinear Science and Numerical Simulation. Accepted . Preprint version in ResearchGate , DOI link: http://www.doi.org/10.13140/RG.2.2.21543.29604.
- Chen, T., Rui, J., Wang, Q., Zhao1, Z., Cui, J., and Yin, L. (2020) A mathematical model for simulating the phase-based transmissibility of a novel coronavirus, Infectious Diseases of Poverty 9:24, https://doi.org/10.1186/s40249-020-00640-3
 Castillo-Chavez, C., Feng, Z., and Huang, W. (2002) On the computation of R₀ and its
- 25. Castillo-Chavez, C., Feng, Z., and Huang, W. (2002) On the computation of R_0 and its role on global stability, in: Mathematical Approaches for For Emerging and Reemerging Infectious Diseases. Springer-Verlag, 229-250.
- 26. Driessche, P.V. and Watmough, J. (2002) Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission Mathematical Biosciences, 180, 29-48.
- 27. Korobeinikov, A., Wake, G. C. (2002) Lyapunov function and global stability for SIR, SIRS, and SIS epidemiological models. Appl. Math. Lett. 15:955–960.
- 28. Li MY, Muldowney JS (1996) A geometric approach to global stability problems. SIAM J Math Anal 27(4):1070–1083.
- 29. Li, M. Y., Muldowney, J. S. (1995) Global stability for the SEIR model in epidemiology. Math Biosci 125:155–164.
- 30. LaSalle, J.P. (1976) The stability of dynamical systems. Regional conference series in applied mathematics. SIAM, Philadelphia.
- 31. Srivastav, A.K., Tiwari, P.K., Srivastava, P.K., Ghosh, M., Kang, Y. (2020) A mathematical model for the impacts of face mask, hospitalization and quarantine on the dynamics of COVID-19 in India: deterministic vs. stochastic, Mathematical Biosciences and Engineering, 18 (1), 182–213.
- 32. Lenhart, S., Workman, J. T. (2007) Optimal control applied to biological models. CRC Press, Boca Raton.
- 33. Pontryagin, L.S., Boltyanskii, V. G., Gamkrelidze, R.V., Mishchenko, E. F. (1962) The mathematical theory of optimal processes, Inter science Publishers, Geneva.
- 34. Goswami, N. K. and Shanmukha, B. (2020) 'Modeling and Analysis of Symptomatic and Asymptomatic Infections of Zika Virus Disease with Non-Monotonic Incidence Rate', Applied Mathematics and Information Sciences, 14(4), 655-671.
- 35. Goswami , N. K. (2021)'Modeling analysis of Zika virus with saturated incidence using optimal control theory', International journal of dynamical systems and differential equations, 11 (3/4), 287–301.
- 36. Olaniyi, S., Obabiyi, O.S., Okosun, K.O., Adewale, A.T., Adewale, S.O. (2020)Mathematical Modeling and Optimal cost-effecting control of Covid-19 transmission dynamics. Eur Phys J Plus.135(11).

Copyright © 2021 ASSA.