A mathematical model on COVID-19 studying the efficacy of testing to control the epidemic

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Abstract

This study models the efficacy of testing in controlling the global outbreak in the presence of two preventative strategies. A thorough investigation of the impacts of testing, that assists in the control of the epidemic, is done using the non-linear mathematical model of COVID-19 and optimal control theory. We demonstrate the existence of an optimal control set and examine the optimality, transversality, and necessary and sufficient requirements. The system's optimality is determined analytically and resolved numerically.

KEYWORDS
Efficacy, Optimal control, Sensitivity analysis, Stability analysis, Numerical simulation.

1 Introduction

The COVID-19 pandemic is one of the world's most serious public health crises. This pandemic occurred due to out break of SARS-CoV-2 virus [1]

in a medical institute in Hubei province, China in the month of December 2019. COVID-19 has traveled across several countries, and the sickness has spread to the majority of the world in a relatively short period of time. It spreads quickly when one person comes into touch with another, resulting in a human-to-human infection. The outbreak later infected more than 200 countries since March 2020, becoming a pandemic due to the high number of deaths. It has left an impact on the social, economical, political and cultural lives of people all over the world. Due to this with a confirmed death toll of 171, COVID-19 was decided to a Public Health Emergency of International Concern (PHEIC) on January 30, 2020. By the end of 2020, the number was 1813188. However, preliminary projections indicate that at least 3 million deaths will be directly related to the COVID-19 pandemic worldwide in 2020, which is 1.2 million more deaths than have been officially reported. According to WHO in the first week of June 2023, there have been 767,750,853 confirmed cases of COVID-19 including 6.941,095 deaths, reported to WHO (https:// covid19.who.int). AS of 5 June 2023, a total of 13,396,086,098 vaccine doses have been administered. From the COVID-19 total of more than 633 million cases and over 6.6 million deaths have been recorded worldwide.[2, 3, 4, 5, 6].

The use of vaccines is a very efficient way of preventing and alleviating viral infection [7]. The efficacy of the test could also be a great way to help control the pandemic. Many researchers have done many papers to study the efficacy of the vaccine. Because the effect of the vaccine can prevent the spread of disease inside the people. Vaccines offer direct protection by making people less susceptible to sickness or infection. Vaccines provide indirect safeguards by decreasing the number of people infected or contagious in a population. These vaccination effects can be examined in clinical trials by measuring the efficacy of the vaccine against disease, infection, and disease transmission, as well as in studies that investigate the vaccine's indirect impacts [8]. Human exposure vaccine research studies, in which participants in a randomized controlled trial are purposely exposing to the virus, have the potential to yield high-quality data on the effect of vaccines on the transmission of viruses [9]. A vaccine's purpose is typically infection prevention (i.e., sterilizing immunity). The true utility of an effective vaccination, on the other hand, is the avoidance of infectious disease caused by that infection. It is possible that it can be seen directly or indirectly in a vaccinated person. Wintachai and PrÃ thom (2021) [10] examine the stability of the SEIR model in relation to vaccination efficacy in the COVID-19 condition. By using Nano-materials we can optimize the efficacy of vaccines, it was discussed by Y.Liu. et al. (2014) [11].

With the use of mathematical concepts and hypotheses, overall flow of work,

technique, cartilage, and consequences can be simply examined. As a result, biologists are increasingly reliant on mathematics. Biological mathematical modeling has been done [12, 13, 14]. During simple mathematical modeling, the relationship combines biological mode, integer order differential equations that depict their dynamics, and a complex system that defines their dynamic structure. The optimal control theory is an effective way that gives several efficient control techniques to reduce or eliminate a disease in the population. This strategy has been used to remove a number of diseases from the population in recent years [15, 16, 17, 18, 19, 20, 21].

To the best of my knowledge, the efficacy of testing to control the epidemic by using optimal control theory has not been considered in any of the previous mathematical models. As discussed above, a lot of work has been done only on the efficacy of vaccines. So, we constructed a mathematical model of COVID-19 to study the efficacy of testing to control the epidemic. Our research paper is organised as follows: In section 2 we formulate mathematical model. In section 3 we studied equilibrium analysis. section 4 described the stability analysis of the model. The model formulation of the optimal control, invariance and positivity of solution, proof of existence and characterization of optimal control is given in section 5. In section 6 and 7 numerical simulations and conclusions are described respectively.

2 The model

We consider a SUDR mathematical model and using control theory on it. We use φ and ϑ as case detection parameters which represents the rate of testing and rate of asymptomatic people going for test that are detected as control parameter. At any time t, we consider a region with total population N. we divided whole population into four sub classes: Susceptible (S), Undetected infectious (U), Recovered (Removed) (R), Detected infectious (D) and Testing done (T). A diagram is shown in Figure1 to study the efficacy of testing.

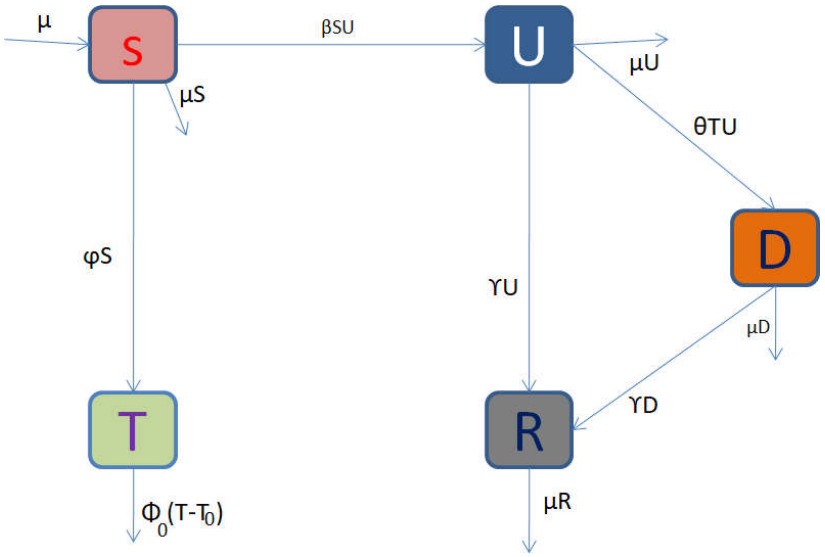

Figure 1: Flow diagram for a model system (2.1) to (2.5).

$$\frac{dS}{dt} = \mu - \beta SU - \mu S + (1-\psi)m, \tag{2.1}$$

$$\frac{dU}{dt} = \beta SU - \gamma U - \vartheta TU - \mu U + \psi m, \tag{2.2}$$

$$\frac{dD}{dt} = \vartheta TU - \gamma D - \mu D, \tag{2.3}$$

$$\frac{dR}{dt} = \gamma U + \gamma D - \mu R, \tag{2.4}$$

$$\frac{dT}{dt} = \varphi S - \varphi_0(T - T_0), \tag{2.5}$$

with the initial conditions $S(0) > 0$, $U(0) \geq 0$, $D(0) \geq 0$, $R(0) \geq 0$ and $T(0) \geq 0$.

Description of parameters is defined in Table(1).

Here note that,

$$S(t) + U(t) + D(t) + R(t) = N(t). \tag{2.6}$$

Adding equation (2.1) to (2.5), we have

Table 1: Table of Description

Variable and Parameter	Description
S	Susceptible
U	Undetected infectious
R	Recovered (Removed)
D	Detected infectious
T	Testing done
T_0	Boxline number of testing done
ψ	Infected migrants rate
m	Number of migrants
β	Transition rate of infection
μ	Inflow rate, Mortality rate (Natural)
ϑ	Rate of asymptomatic people going for test
γ	Recovery rate of asymptomatic people
φ	Rate of testing
φ_0	Failure rate of testing

$$\frac{dS}{dt} + \frac{dU}{dt} + \frac{dD}{dt} + \frac{dR}{dt} = \mu - \mu(S + U + D + R) + m$$

$$or, \quad \frac{d}{dt}(S + U + D + R) = \mu - \mu(S + U + D + R) + m.$$

Using equation (2.6) in above equation, we get

$$\frac{dT}{dt} \leq \mu - \mu N + m. \tag{2.7}$$

This shows that the total population $lim_{t\to\infty} supN\,(t) \leq 1$. In the context of model described above, the assumption holds that all variable and parameters associated with the model are non-negativity for every $t \geq 0$. This assumption is significant, reflecting the nature of the system being modeled. In the positively invariant set, we investigate the above model on

$$\Omega = \{(S, U, D, R, T) \in R^5_+ : 0 \leq S, U, D, R \leq 1, 0 \leq T \leq \frac{\varphi}{\varphi_0}\}. \tag{2.8}$$

This is the model's region of attraction.

Table 1 provides a description of the parameters used in the model system (Fig. 1).

3 Equilibrium analysis

The term ”equilibrium point” refers to a state of a dynamical system when there is no change over time. So, if a system begins from an equilibrium point, its state will always remain in equilibrium. Finding a disease free equilibrium point is our current objective. Then from equation (2.1) to (2.5), only pandemic equilibrium point $E^m(S^m, U^m, R^m, D^m, T^m)$ exists. The values of S^m, U^m, R^m, D^m and T^m are given as

$$S^m = \frac{\mu + (1-\psi)m}{\beta U^m + \mu}, \tag{3.1}$$

$$R^m = \frac{\gamma}{\mu}[U^m + \frac{\vartheta U^m}{\gamma + \mu}\{T_0 + \frac{\varphi}{\varphi_0}(\frac{\mu + (1-\psi)m}{\beta U^m + \mu})\}], \tag{3.2}$$

$$D^m = \frac{\vartheta U^m}{\gamma + \mu}[T_0 + \frac{\varphi}{\varphi_0}(\frac{\mu + (1-\psi)m}{\beta U^m + \mu})], \tag{3.3}$$

$$T^m = T_0 + \frac{\varphi}{\varphi_0}\{\frac{\mu + (1-\psi)m}{\beta U^m + \mu}\} \tag{3.4}$$

Then the root of the quadratic equation (3.5) provides U^m.

$$A_1(U^m)^2 + A_2U^m - A_3 = 0. \tag{3.5}$$

where the coefficients of equation (3.5) are

$$A_1 = \{\varphi_0(\gamma + \vartheta T_0) + \varphi_0\mu\}\beta,$$
$$A_2 = (\varphi\vartheta\mu + \varphi\vartheta m + \varphi_0\gamma\mu + \varphi_0\vartheta T_0\mu + \Phi_0\mu^2) - (\varphi\vartheta\psi m + \beta\varphi_0\mu + \beta\varphi_0 m)$$
$$A_3 = \psi m\varphi_0\mu.$$

This shows that the uniqueness of positive equilibrium point U^m exist if and only if

$$\sqrt{A^{22} + 4A_1A_2} > A_2$$

4 Stability analysis

The investigation of local stability at the equilibrium point of the system provides insight into the effects of perturbations. Our system can be linearized with regard to the random equilibrium points $E^m(S^m, U^m, R^m, D^m, T^m)$ in order to determine the epidemic equilibrium’s stability point and we are given the equivalent matrix called the Jacobian matrix by

$$J_E = \begin{bmatrix} b_{11} & b_{12} & 0 & 0 & 0 \\ b_{21} & b_{22} & 0 & 0 & 0 \\ 0 & b_{32} & b_{33} & 0 & 0 \\ 0 & b_{42} & b_{43} & b_{44} & 0 \\ b_{51} & 0 & 0 & 0 & b_{55} \end{bmatrix}$$

Where entries of the matrix J_E are given by

$$\begin{aligned} b_{11} &= -\beta U - \mu, & b_{12} &= -\beta S, & b_{15} &= \psi, \\ b_{21} &= \beta U, & b_{22} &= \beta S - \gamma - \vartheta T - \mu, & b_{42} &= \gamma, \\ b_{32} &= \vartheta T, & b_{33} &= -(\gamma + \mu), & b_{51} &= \varphi, \\ b_{43} &= \gamma, & b_{44} &= -\mu, & b_{55} &= -\varphi_0. \end{aligned}$$

Proposition. *For the fixed parameters of the model system* (2.1) *to* (2.5) *with initial condition the equilibrium point* $E^m(S^m, U^m, D^m, R^m, T^m)$ *is locally asymptotically stable if* $(\beta U + 2\mu + \gamma + \vartheta T) > \beta S$ *with* $(b_{11} - b_{22})^2 + 4b_{12}b_{21} < 0$.

Proof:- The Jacobian matrix of the model system (2.1) to (2.5) is given by J_E defined as above. The eigenvalues, λ, of the Jacobian matrix J_E is computed by the equation $det(\lambda I - J_E) = 0$; i.e., the eigenvalues are the solution of the characteristic polynomial

$$(b_{33} - \lambda)(b_{44} - \lambda)(b_{55} - \lambda)\{b_{11}b_{22} - b_{12}b_{21} - (b_{11} + b_{22}) + \lambda^2\} = 0. \qquad (4.1)$$

Now, by solving above equation (4.1) the eigenvalues of the Jacobian matrix J_E is given by

$$\begin{aligned} \lambda_1 &= \frac{1}{2}\{(b_{11} + b_{22}) - \sqrt{(b_{11} - b_{22})^2 + 4b_{12}b_{21}}\}, \\ \lambda_2 &= \frac{1}{2}\{(b_{11} + b_{22}) + \sqrt{(b_{11} - b_{22})^2 + 4b_{12}b_{21}}\} \\ \lambda_3 &= b_{33} = -(\gamma + \mu), \\ \lambda_4 &= b_{44} = -\mu, \\ \lambda_5 &= b_{55} = -\varphi_0. \end{aligned}$$

Where, $b_{11} = -\beta U - \mu$, $b_{12} = -\beta S$, $b_{21} = \beta U$ and $b_{22} = \beta S - \gamma - \vartheta T - \mu$. As, $b_{11} + b_{22} = -(\beta U + 2\mu + \gamma + \vartheta T) + \beta S$ Thus, if $(\beta U + 2\mu + \gamma + \vartheta T) > \beta S$ *with* $(b_{11} - b_{22})^2 + 4b_{12}b_{21} < 0$ then all eigenvalues have negative real part. Therefore, the equilibrium point of the model system (2.1) to (2.5) is locally asymptotic stable.

4.1 Basic reproduction number

The basic reproduction number is defined as a threshold digit that delivers a value of secondary infections caused by an infected individual class in total infection duration if every member of the community is susceptible. The next-generation matrix is a method that allows us to determine the basic reproduction number. The model is divided into two subparts, R_1 and R_2, and the system (2.1) to (2.5) with $m = 0$ has the following form:-

$$X = R_1 - R_2. \tag{4.2}$$

Where $R_1 = \begin{bmatrix} \beta SU - \vartheta TU \\ \vartheta TU \\ 0 \\ 0 \\ -\beta SU + \mu \end{bmatrix}$,

$$R_2 = \begin{bmatrix} (\gamma + \mu)U \\ (\gamma + \mu)D \\ \mu R - \gamma(U + D) \\ \varphi_0(T - T_0) - \varphi S \\ \mu S \end{bmatrix}$$

and $X = \begin{bmatrix} \frac{dU}{dt} & \frac{dD}{dt} & \frac{dR}{dt} & \frac{dT}{dt} & \frac{dS}{dt} \end{bmatrix}$.

In the case of disease free equilibrium point the infected compartment are U, D, R and T.

Now, $\tilde{R}_1 = \left[\frac{\delta(R1)_i}{\delta x_j}\right]$ & $\tilde{R}_2 = \left[\frac{\delta(R2)_i}{\delta x_j}\right]$ for $1 \le i, j \le 4$.

Thus, $\tilde{R}_1 = \begin{bmatrix} \beta S - \vartheta T & 0 & 0 & -\vartheta U \\ \vartheta T & 0 & 0 & \vartheta U \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$,

$\tilde{R}_2 = \begin{bmatrix} \gamma + \mu & 0 & 0 & 0 \\ 0 & \gamma + \mu & 0 & 0 \\ -\gamma & -\gamma & \mu & 0 \\ 0 & 0 & 0 & \varphi_0 \end{bmatrix}$.

Here $\tilde{R}_1 \ge 0$ and $\tilde{R}_2$ is a matrix whose determinant is not equal to zero $\tilde{R}_2^{-1} \ge 0$ and $\tilde{R}_1 \tilde{R}_2^{-1}$ as non negative matrix; $\tilde{R}_1 \tilde{R}_2^{-1}$ is the next generation matrix.

$$\text{now } \tilde{R}_2^{-1} = \begin{bmatrix} \frac{1}{\gamma+\mu} & 0 & 0 & 0 \\ 0 & \frac{1}{\gamma+\mu} & 0 & 0 \\ \frac{\gamma}{\gamma\mu+\mu^2} & \frac{\gamma}{\gamma\mu+\mu^2} & \frac{1}{\mu} & 0 \\ 0 & 0 & 0 & \frac{1}{\varphi_0} \end{bmatrix}.$$

In this case

$$\tilde{R}_1\tilde{R}_2^{-1} = \begin{bmatrix} \frac{\beta S - \vartheta T}{\gamma+\mu} & 0 & 0 & -\frac{\vartheta U}{\varphi_0} \\ \frac{\vartheta T}{\gamma+\mu} & 0 & 0 & \frac{\vartheta U}{\varphi_0} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Then the spectral eigen value about the equilibrium $E(1, 0, 0, 0, T_0)$ point of the matrix is

$$\lambda = \frac{\beta - \vartheta T_0}{(\gamma + \mu)}. \tag{4.3}$$

As a result, the basic reproduction number is provided by

$$R_0 = \frac{\beta - \vartheta T_0}{(\gamma + \mu)}. \tag{4.4}$$

Thus, by controlling the value of these parameters we can control spread of the disease.

5 Developing an optimal control model

By selecting appropriate control parameters, we may do this using optimal control theory. In the model system, we have selected two control parameters, namely (i) Rate of testing (*i.e.,* φ) (ii) Rate of asymptomatic people going for test (*i.e.,* ϑ). The parameters φ and ϑ are represented by Lebesgue measurable functions $v_1(t)$ and $v_2(t)$, respectively on a finite interval $[0, t_f]$. Now, the challenge is to reduce the total cost functional J as stated by

$$J(v_1, v_2) = \int_0^{t_f} (AU(t) + \frac{B}{2}v_1^2 + \frac{C}{2}v_2^2)dt \tag{5.1}$$

Subject to

$$\frac{dS}{dt} = \mu - \beta SU - \mu S + (1 - \psi)m, \tag{5.2}$$

$$\frac{dU}{dt} = \beta SU - \gamma U - v_2(t)TU - \mu U + \psi m, \tag{5.3}$$

$$\frac{dD}{dt} = v_2(t)TU - \gamma D - \mu D, \tag{5.4}$$

$$\frac{dR}{dt} = \gamma U + \gamma D - \mu R, \tag{5.5}$$

$$\frac{dT}{dt} = v_1 S - \varphi_0(T - T_0), \tag{5.6}$$

with the initial conditions $S(0) > 0$, $U(0) \geq 0$, $D(0) \geq 0$, $R(0) \geq 0$ and $T(0) \geq 0$.

Here note that,

$$S(t) + U(t) + D(t) + R(t) = N(t). \tag{5.7}$$

The positive constant A, B and C within the cost function serve as weight constants, influencing the integral components of the functional cost, J. Our objective is to find an optimal control pair $(v_1^*, v_2^*) \in \Theta$ that minimizes the functional J, aiming to

$$J(v_1^*, v_2^*) = min_{(v_1, v_2) \in \Theta} J(v_1, v_2) \tag{5.8}$$

in which the control set is defined as

$$\Theta = \{(v_1, v_2) | \quad v_i \quad \text{is measurable and} \quad 0 \leq v_i(t) \leq 1 \quad for \quad t \in [0, t_f], i = 1, 2\}$$

is the needed set for the controls.

To keep things simple, we can write $v_1(t) = v_1$ and $v_2(t) = v_2$

5.1 Consistency and positivity of solutions

In this section, we demonstrate the persistence and positive of the existing solutions for the considered system (5.2) to (5.6).

Theorem 5.1.1 *The system of equation (5.2) to (5.6) makes biological sense in the region*

$$\Omega = \{(S, U, D, R, T) \in R_+^5 : 0 \leq S, U, D, R \leq 1, 0 \leq T \leq \frac{\varphi}{\varphi_0}\}.$$

It is attractive and positively invariant with regard to the system of equation (5.2) to (5.6).

Proof By combining the first four equations of the model system (5.2) to (5.6) mentioned before and taking $0 \leq v_i(t) \leq 1$ *for* $i = 1, 2$
Thus,

$$\frac{dT}{dt} \leq (\mu + m) - \mu T.$$

The remainder of the proof is straightforward by using integration and limits.

Theorem 5.1.2 *Our model system's (5.2) to (5.6) solutions are all positive.*
Proof The theorem's proof 5.1.2 is rather simple.

5.2 Existence of optimal control

The theorem establishes the existence of an optimal control state, as demonstrated in theorem 5.2.1 (by Fleming et al. 1975), in the following manner:

Theorem 5.2.1 *Consider the control challenge associated with model system (5.2) to (5.6) there exist* $\tilde{v} = (v_1^*, v_2^*) \in \Theta$ *in such a way that*

$$J(v_1^*, v_2^*) = min_{(v_1, v_2) \in \Theta} J(v_1, v_2)$$

Proof The following prerequisites must be met for the optimum control to exist:

(i) The associated state variables and set of controls indicated by the symbol Ω are not empty. The solutions of model system (5.2) to (5.6) exists found in (Lukes 1982) Theorem 9.2.1.
As a result, the set Ω is not empty.

(ii) The set Θ is convex and closed. From definition, it is clear that Θ is closed. Given $(\vartheta_1, \vartheta_2) \in \Theta$ and $t \in [0, 1]$ such that $\vartheta_1 = (v_{11}, v_{21})$ and $\vartheta_2 = (v_{12}, v_{22})$, the line $t\vartheta_1+(1-t)\vartheta_2 = (tv_{11}+(1-t)v_{12}), tv_{21}+(1-t)v_{22})$ belongs to Θ since each of its components is in between zero and one. Thus Θ is convex.

(iii) The right hand side of the state model system (5.2) to (5.6) is bounded by a linear function in the state and control variable. System (5.2) to

(5.6) is linear with respect to v_1 and v_2. Beside its solution are absolutely continuous(Lukes 1982), which implies that they are bounded and statement (iii) is satisfied.

(iv) The content of the objective function is concave over Θ. The specific expression for the integral component K of the objective function is provided by by

$$K(U, v_1, v_2) = AU(t) + \frac{B}{2}v_1^2 + \frac{C}{2}v_2^2$$

.

It can be verify for any $(\vartheta_1, \vartheta_2) \in \Theta$ and $t \in [0, 1]$,

$$K(t\vartheta_1 + (1-t)\vartheta_2) \geq tK(\vartheta_1) + (1-t)K(\vartheta_2)$$

This shows that K is concave.

(v) There exist constants $q_1, q_2 > 0$ and $r > 1$ such that the integrand K of the objective functional satisfied

$$K(U, v_1, v_2) \leq q_1 + q_2(|v_1|^2 + |v_2|^2)^{r/2}.$$

Since S is bounded [22], there exist a constant $q_1 > 0$ in such a way that $AU \leq q_1$ and also assuming $q_2 = maxv_i$ where $i = 1, 2$ we get

$$K(U, v_1, v_2) \leq q_1 + q_2(|v_1|^2 + |v_2|^2)^{r/2}$$

with $r = 2$.

Hence, condition (v) is met, affirming that the optimal system possesses a unique solution for t_f that's adequately small [22].

5.3 Characterization of optimal control

The Pontryagin's Maximum Principle (Pontryagin et al. 1962) provides the fundamental requirements that an optimum solution must meet.This principle turns equations (5.1)-(5.6) into a problem that characterizes the following Hamiltonian H in terms of control variable $H(S, U, D, R, T, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5)$ by minimizing it point wise. $H(S, U, D, R, T, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5) = AU(t) + \frac{B}{2}v_1^2 + \frac{C}{2}v_2^2 + \lambda_1\{\mu - \beta SU - \mu S + (1-\psi)m\} + \lambda_2\{\beta SU - \gamma U - v_2(t)TU - \mu U + \psi m\} + \lambda_3\{v_2(t)TU - \gamma D - \mu D\} + \lambda_4\{\gamma U + \gamma D - \mu R\} + \lambda_5\{v_1 S - \varphi_0(T - T_0)\}$

where λ_i, $i = 1, 2, 3, 4$ are co-state functions, also known as adjoint functions which can be defined through solving the system of differential equations:

$$\lambda_1^r = -\frac{\partial H}{\partial S} = -\{\lambda_1(-\beta U - \mu) + \lambda_2 \beta U + \lambda_5 \nu_1\},$$
$$\lambda_2^r = -\frac{\partial H}{\partial U} = -\{A + \lambda_1(-\beta U) + \lambda_2(\beta S - \gamma - \nu_2(t)T - \mu) + \lambda_3 \nu_2(t)T + \lambda_4 \gamma\},$$
$$\lambda_3^r = -\frac{\partial H}{\partial D} = -\{\lambda_3(-\gamma - \mu) + \lambda_4 \gamma\},$$
$$\lambda_4^r = -\frac{\partial H}{\partial R} = -\{\lambda_4(-\mu)\},$$
$$\lambda_5^r = -\frac{\partial H}{\partial T} = -\{-\lambda_2 \nu_2(t)U + \lambda_3 \nu_2(t)U - \lambda_5 \varphi_0\},$$

meeting the condition $\lambda_i(t_f) = 0$ for $i = 1, 2, 3, 4$ satisfies the transversality requirement. These condition are due to independent of state at the final time of the objective functional.

The Hamiltonian is minimized concerning v_1 and v_2 at the optimal value v_1^* and v_2^*, indicating the derivative of the H concerning v_1 and v_2 at v_1^* and v_2^*, must be zero. Since

$$H = \frac{B}{2}v_1^2 + \frac{C}{2}v_2^2 + (\lambda_3 - \lambda_2)\nu_2(t)TU + \lambda_5 \nu_1 S \tag{5.9}$$

Now, differentiating equation (5.9) partially with respect to v_1 and v_2 respectively, we get

$$\frac{\partial H}{\partial v_1} = v_1 B + \lambda_5 S \tag{5.10}$$

and

$$\frac{\partial H}{\partial v_2} = v_2 C + (\lambda_3 - \lambda_2)TU \tag{5.11}$$

The optimal control without restriction v_1^* and v_2^* satisfied $\frac{\partial H}{\partial v_1} = 0$ and $\frac{\partial H}{\partial v_2} = 0$ at $v_1 = v_1^*$ and $v_2 = v_2^*$ respectively due to Pontryagin's Maximum Principle (PMP).

Thus, we have $v_1^* = -\frac{\lambda_5 S}{B}$ and $v_2^* = \frac{(\lambda_2 - \lambda_3)TU}{C}$

Now, making use of the control's bound, the ideal control is defined as

$$v_1^* = max\{0, min(1, -\frac{\lambda_5 S}{B})\} \tag{5.12}$$

and

$$v_2^* = max\{0, min(1, \frac{(\lambda_2 - \lambda_3)TU}{C})\} \tag{5.13}$$

As a result, we have the following theorem.

Theorem 7.1 Equation (5.12) and (5.13) describes the optimal control v_1^* and v_2^* that optimizing the model system (6)to maximizes the objective function (5.1).

We achieve the uniqueness of the optimum control for small t_f due to the a priori boundedness of the state and adjoint functions and the resulting Lipschitz structure of the ordinary differential equations.

6 Numerical Simulation

We demonstrate numerical simulation of the dynamical systems (2.1) to (2.5) in this section to support the findings of theory. We have selected the following as the default values for this purpose:

$\beta = 0.9$, $\mu = 0.2$, $\vartheta = 0.05$, $T_0 = 4$, $\gamma = 0.25$, $m = 2500$, $\varphi = 0.6$, $\psi = 0.5$, $\varphi_0 = 0.1$.

It is possible to verify that the stability conditions and the presence of an

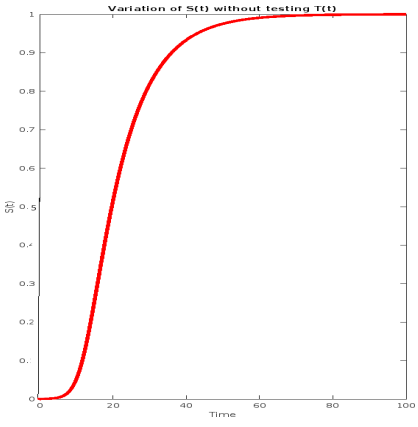

Figure 2: Variation of susceptible population without testing.

endemic equilibrium E_2 are fulfilled. With regard to E_2, the point of equilibrium values have been determined as:

$S^* = 0.4975313$, $U^* = 2791.785$, $D^* = 2166.793$, $R^* = 2231.36$, $T^* = 6.985188$.

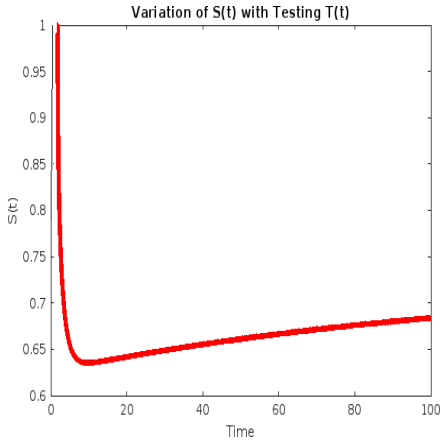

Figure 3: Variation of susceptible population with testing.

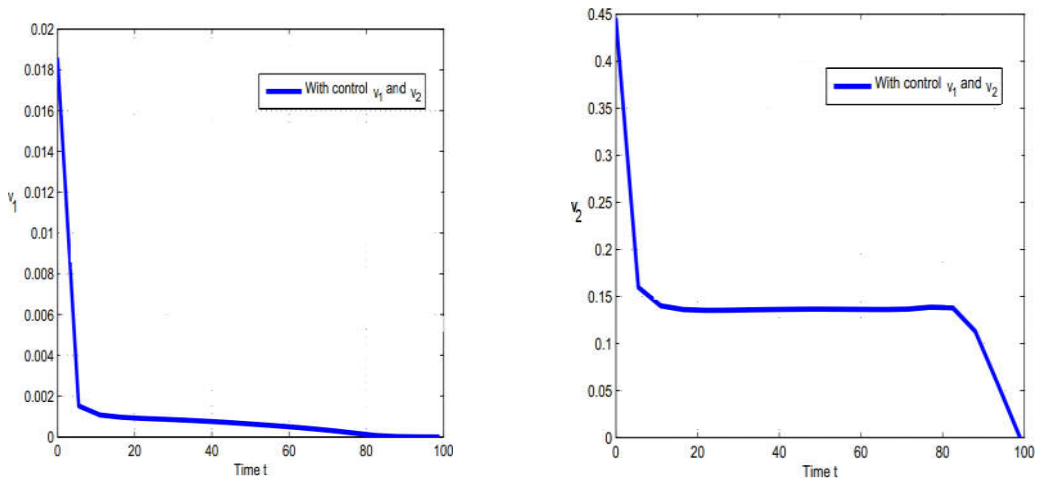

Figure 4: Optimal control profile v_1 and v_2 over time, considering $A = 3$, $B = 6$, and $C = 3$.

Then, using an iterative approach and the Runga-Kutta fourth order procedure from Lenhart et al.(2007), we deal with the optimality system through numerical calculations while making some preliminary assumptions about the control variables regarding the same set of values for the parameters as above. Making use of the Runga-Kutta fourth order iterative approach, the system of state variables is first solved forward in time, and then the system of adjoint variables is solved backward in time. The cost for such controls is modified after every iteration. As long as the intended convergence criteria is not met, we will have continue the evolution. We start by setting the ini-

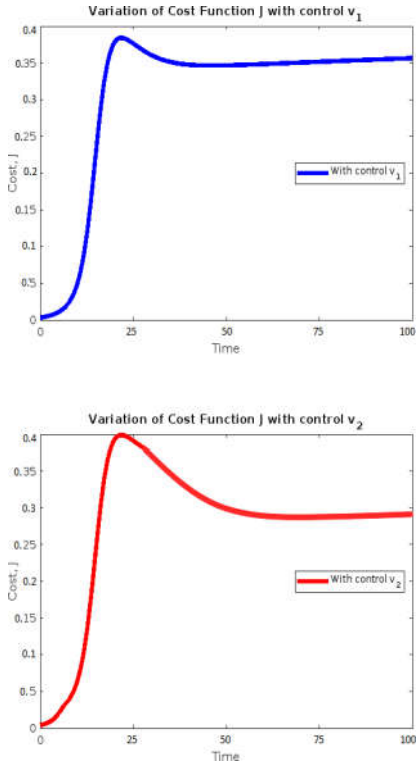

Figure 5: Variation of cost function with respect to time with control v_1 and v_2 individually.

tial conditions as follows: S(0) = 0.8, U(0) = 0.1, D(0) = 0.05, R(0) = 0.1, T(0)= 6.5. The maximum values for the parameters of control are restricted to u_{1max} = 1 and u_{2max} = 1. To ensure the enforcement of control strategies, the duration of the period is reduced to 100 days.

In the analysis, we observe the influence of testing on susceptible individu-

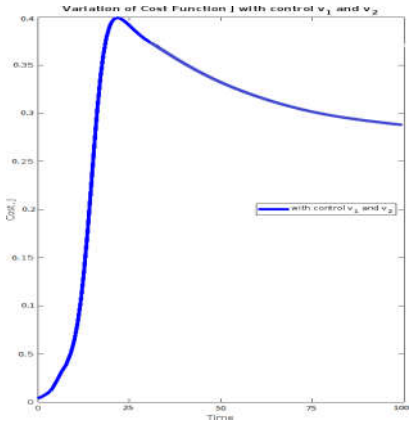

Figure 6: Variation of cost function with respect to time with control v_1 and v_2.

als, as depicted in Figure 2, 3. It was noted that the number of susceptible individuals increases when there is no COVID testing conducted, whereas in its presence, the count of susceptible individuals decreases.

We chose to set the weight factors associated with susceptibility and the effectiveness of testing to be equal, specifically A = 3, B = 6, and C = 3. The impact of these weight constants on the pattern of optimal controls is depicted in Figure 4. These figures distinctly demonstrate the crucial role played by the optimal control profile in guiding the optimal control strategy. Furthermore, we've analyzed the breakdown of cost formation to determine more effective wellness control strategies in Figure 5, 6. From these figures, it's evident that the cost is notably high in the absence of any control measures. Additionally, it's observed that with a single control measure, the cost differs only slightly compared to the scenario where no control measures are used. However, it's notable that when both control measures are employed, the optimal cost significantly decreases compared to the cases where either a single or no control measure is used. Hence, it suggests that employing both control strategies is far more effective than utilizing only a single control measure on the susceptible population, consequently reducing their numbers.

7 Conclusions

In this study, we have constructed a mathematical model to show the efficacy of testing by using two control parameters: the rate of testing and the rate at which asymptomatic people going for test to control the epidemic. Our findings reveal the presence of two equilibrium points: one representing the disease-free equilibrium $E_1(1, 0, 0, 0, T_0)$, and the other representing the endemic equilibrium point $E_2(S^m, U^m, D^m, R^m, T^m)$ for $R_0 > 1$. The disease-free equilibrium point is locally asymptotically stable when $R_0 < 1$. Additionally, our analysis indicates that a pandemic equilibrium point is both locally and globally stable under specific conditions. Numerical simulations have been conducted to verify these analytical findings. It has been observed that in the absence and presence of rate of testing, the population of susceptible increases and decreases, respectively.

Our findings strongly indicate that testing plays a significant role in reducing the susceptible population and subsequently curbing the spread of diseases. This comprehensive investigation highlights the effectiveness of testing strategies coupled with appropriate treatments in eradicating susceptibility to diseases within a population. Implementing testing protocols offers the potential for disease monitoring and prevention, reducing complications, and enhancing overall quality of life. However, given the considerable costs associated with testing programs, we have sought an optimal solution using optimal control theory. This theory enables us to identify the optimal rates for implementing and disseminating testing actions. We've established the presence of a controlled system modality and employed Pontryagin?s Maximum Principle to derive optimal control characteristics, which are corroborated by numerical examples. Therefore, the optimal solution provides a cost-effective approach to controlling epidemics by employing efficacy testing strategies on the susceptible population, consequently aiding in disease control.

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