Optimal Control of the COVID-19 Transmission through Non-Pharmaceutical Intervention, Vaccination, and Quick Response Using SEIQRS Model

Muhammad Saefurrachman, Diny Zulkarnaen*, Mia S. Khumaeroh, Annisa Martina and Inne S. Putri

***Abstract*—This paper discusses the transmission of COVID-19 using the SEIQRS model consisting of five population compartments: Susceptible, Exposed, Infected, Quarantined, and Recovered, where population who have recovered from the disease can be reinfected. The model yields two equilibrium points: disease-free and endemic, which are then analyzed for stability using the Trace-Determinant method and the Routh-Hurwitz criterion. To mitigate the spread of COVID-19, optimization of control variables is implemented that covers non-pharmaceutical interventions, vaccination, and quick quarantine response. Utilizing the Pontryagin's minimum principle allows for the most efficient control, ensuring that the costs incurred for implementing these controls are minimized. Finally, several graphical simulations are presented to observe population dynamics for disease-free and endemic scenarios, as well as the sensitivity of parameters that affect the outbreak. Another graphical simulations are also given to illustrate the differences between scenarios with and without controls, showing that with controls in place, the number of infected individuals can be reduced to zero.**

***Index Terms*—COVID-19, SEIQRS model, Trace-Determinant, Routh-Hurwitz, Pontryagin's minimum principle.**

I. Introduction

IN early 2020, the World Health Organization (WHO) designated Corona Virus Disease 2019 or COVID-19 as a global health emergency. The first recorded case occurred in Wuhan, China [1]. WHO identified that this disease is caused by infection with the Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2). Transmission predominantly occurs among humans via respiratory droplets, direct contact, or objects contaminated with the virus.

COVID-19 represents a newly emerging disease. Therefore, understanding regarding its management and prevention remains limited. Researchers around the globe are actively developing vaccines aimed to establishing immunity against SARS-CoV-2, with the goal of reducing or even preventing its transmission. Consequently, the WHO advocates for strategies that interrupt the transmission chain, which include fundamental protective measures such as maintaining personal hygiene, early detection, isolation, and quarantine, all aimed at minimizing infection rates.

In addition to these basic protective measures, strengthening the immune system is critical to ensure the body can effectively resist viral attacks [2]. Even in cases where individuals contract the COVID-19 virus, they may experience only mild symptoms, allowing for self-management through isolation. Vaccination is another vital component [3], [4], enabling individuals to reduce their risk of infection and contribute to the suppression of the virus's spread.

Mathematical modeling has emerged as a significant tool for understanding the dynamics of COVID-19, particularly by indentifying the factors that influence its transmission and spread. These models are intended to serve as valuable references for policymakers in formulating strategies to control the outbreak.

Various mathematical models representing the transmission dynamics of COVID-19 have been documented in the literature [5], [6], including the SIR model (Susceptible-Infected-Recovered) and the SAIU model (Susceptible-Asymptomatic-Infectious-Unreported). Further studies focusing on populations with symptomatic and asymptomatic infections can be found in [7], [8]. Beyond the SIR and SAIU frameworks, the dynamics of COVID-19 can also be described by other models that incorporate quarantine measures, such as the SIQR model [9] and the SEIQR model, which includes exposed individuals [10], [11]. Additionally, the SEICR model considers populations with comorbidities [12].

Research has not only concentrated on the formulation of mathematical models and the dynamics affecting the Susceptible, Infected, and Recovered compartments but has also explored the impact of vaccination on disease transmission, as outlined in [13], [14], [15].

Furthermore, the control of COVID-19 spread can be achieved through non-pharmaceutical interventions, including mask-wearing, social distancing, and maintaining personal hygiene [16], [17], as well as specific measures like quarantine, hospitalization, and environmental controls [18]. Some studies have also examined the integration of vaccination, a pharmaceutical measure, with isolation strategies, a form of non-pharmaceutical intervention, to mitigate disease spread [19]. It should be noted that mathematical models of

Manuscript received June 27, 2024; revised November 1, 2024.

Muhammad Saefurrachman is an undergraduate student of Department of Mathematics, Universitas Islam Negeri Sunan Gunung Djati, Bandung, Indonesia (e-mail: m.saefurrachman29031999@gmail.com).

Diny Zulkarnaen is a lecturer of Department of Mathematics, Universitas Islam Negeri Sunan Gunung Djati, Bandung, Indonesia (corresponding author e-mail*: dzulkarnaen@uinsgd.ac.id).

Mia S. Khumaeroh is a lecturer of Department of Mathematics, Universitas Islam Negeri Sunan Gunung Djati, Bandung, Indonesia (e-mail: miasitihumairoh@uinsgd.ac.id).

Annisa Martina is a lecturer of Department of Mathematics, Universitas Islam Negeri Sunan Gunung Djati, Bandung, Indonesia (e-mail: annisamartina@uinsgd.ac.id).

Inne S. Putri is a lecturer of Department of Mathematics, Universitas Islam Negeri Sunan Gunung Djati, Bandung, Indonesia (e-mail: innesyafrian@uinsgd.ac.id).

COVID-19 are not merely theoretical constructs; they can be practically applied to address real-world challenges faced by various countries, such as India [6] and Nigeria [20].

In this paper, we employ the SEIQRS model, which accounts for the possibility of reinfection among individuals who have recovered from COVID-19. We investigate the feasibility of the model solutions by analyzing their positivity and boundedness. Qualitative analyses are conducted on the equilibrium points and their stability to determine the conditions necessary for achieving both disease-free and endemic states. To avoid endemic scenarios, we implement an optimization control strategy focusing on three variables: non-pharmaceutical interventions, quarantine measures, and rapid responses by health authorities to isolate infected individuals. The Pontryagin's Minimum Principle is utilized to ensure that control costs are minimized while effectively preventing disease transmission. Finally, graphical simulations are presented to visualize population dynamics and the influence of control variables on the mitigation of disease spread.

II. SEIQRS Model

In this study, the dynamics of the COVID-19 outbreak are modeled using the SEIQRS framework, which comprises five compartments: Susceptible individuals $\bar{S}$, who are at risk of contracting COVID-19; Exposed individuals $\bar{E}$, who have been exposed but are not yet infectious; Infected individuals $\bar{I}$, who are currently infectious; Quarantined individuals $\bar{Q}$, who are isolated to prevent further spread; and Recovered individuals $\bar{R}$, who have regained health following infection.

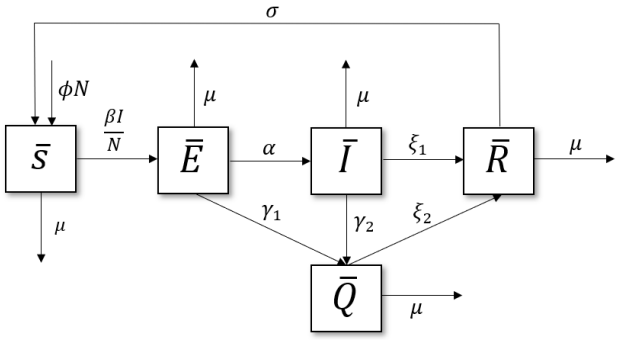

Fig. 1: Illustration of COVID-19 transmission dynamics based on the SEIQRS model.

Figure 1 depicts the COVID-19 transmission process through these five population compartments. The susceptible population grows due to births at a rate ϕ and also through recovered individuals who can be reinfected at a rate σ. Conversely, the susceptible group decreases due to natural mortality at a rate μ and by transitioning to the exposed compartment at a rate β due to contact with infectious individuals. Once the incubation period concludes, exposed individuals transition to the infectious state at a rate α. It is also imperative to implement quick responses, which may include quarantining exposed individuals at a rate γ_1 and infectious individuals at a rate γ_2. These infected populations can recover at rates ξ_1 and ξ_2, respectively. As previously mentioned, recovered individuals may be reinfected, reverting to the susceptible at a rate σ. It is assumed that all compartments experience mortality at the same rate μ.

Based on the description and the diagram in Figure 1, the SEIQRS model for COVID-19 transmission can be expressed as a system of differential equations as

$$\begin{aligned}
\frac{d\bar{S}}{dt} &= \phi N - \frac{\beta \bar{S}\bar{I}}{N} - \mu\bar{S} + \sigma\bar{R},\\
\frac{d\bar{E}}{dt} &= \frac{\beta \bar{S}\bar{I}}{N} - (\gamma_1 + \alpha + \mu)\bar{E},\\
\frac{d\bar{I}}{dt} &= \alpha\bar{E} - (\gamma_2 + \xi_1 + \mu)\bar{I},\\
\frac{d\bar{Q}}{dt} &= \gamma_1\bar{E} + \gamma_2\bar{I} - (\xi_2 + \mu)\bar{Q},\\
\frac{d\bar{R}}{dt} &= \xi_1\bar{I} + \xi_2\bar{Q} - (\mu + \sigma)\bar{R}.
\end{aligned} \tag{1}$$

It is assumed that the total population $N(t)$ across all compartments remains constant over time, implying that $dN/dt = 0$. Thus, from (1), we can derive

$$\begin{aligned}
\frac{dN}{dt} = \frac{d(\bar{S} + \bar{E} + \bar{I} + \bar{Q} + \bar{R})}{dt} &= 0\\
\phi N - \mu(\bar{S} + \bar{E} + \bar{I} + \bar{Q} + \bar{R}) &= 0\\
(\phi - \mu)N &= 0.
\end{aligned}$$

Consequently, for the total population to remain constant, it is necessary that ϕ equals μ, indicating that the birth and death rate must match. Under this assumption, we define the proportions for each compartment as follows

$$S = \frac{\bar{S}}{N}, E = \frac{\bar{E}}{N}, I = \frac{\bar{I}}{N}, Q = \frac{\bar{Q}}{N}, R = \frac{\bar{R}}{N},$$

which allows us to reformulate the ordinary differential equations in (1) as

$$\begin{aligned}
\frac{dS}{dt} &= \phi - \beta SI - \mu S + \sigma R,\\
\frac{dE}{dt} &= \beta SI - k_1 E,\\
\frac{dI}{dt} &= \alpha E - k_2 I,\\
\frac{dQ}{dt} &= \gamma_1 E + \gamma_2 I - k_3 Q,\\
\frac{dR}{dt} &= \xi_1 I + \xi_2 Q - k_4 R.
\end{aligned} \tag{2}$$

where

$$\begin{aligned}
k_1 &= \alpha + \gamma_1 + \mu, & k_2 &= \gamma_2 + \xi_1 + \mu,\\
k_3 &= \xi_2 + \mu, & k_4 &= \mu + \sigma.
\end{aligned}$$

A. Positivity and Boundedness

This section investigates the solutions of the SEIQRS model to ascertain whether the values remain positive and are bounded above. The positivity of all population compartments suggests that the model articulated in (2) is biologically viable.

Theorem 1. Let $S(0), E(0), I(0), Q(0)$, and $R(0)$ represent non-negative initial conditions. The solutions of the model given in (1) are positive for all $t > 0$.

Proof. To demonstrate the positivity of the model's solutions, we consider the rates of change for each compartment within the system (2). We obtain

$$\frac{dS}{dt}\Big|_{S=0} = \phi + \sigma R \geq 0; \quad \frac{dE}{dt}\Big|_{E=0} = \beta SI \geq 0;$$
$$\frac{dI}{dt}\Big|_{I=0} = \alpha E \geq 0; \quad \frac{dQ}{dt}\Big|_{Q=0} = \gamma_1 E + \gamma_2 I \geq 0;$$
$$\frac{dR}{dt}\Big|_{R=0} = \xi_1 I + \xi_2 Q \geq 0.$$

Since the rates of change for all compartments are non-negative, we conclude that the solutions $S(t)$, $E(t)$, $I(t)$, $Q(t)$, and $R(t)$ remain positive for all $t > 0$.

However, ensuring that all compartment variables are positive does not necessarily confirm the biological feasibility of the SEIQRS model. It is equally essential to demonstrate that the population sizes are finite or bounded.

Theorem 2. Let $S(0), E(0), I(0), Q(0)$, and $R(0)$ be non-negative initial conditions. The solutions of the SEIQRS model in (1) are bounded within the region

$$D = \left\{(S, E, I, Q, R) \in \mathbb{R} : 0 \leq S, E, I, Q, R, \leq \frac{\phi}{\mu}\right\}.$$

Proof. To establish boundedness, we consider the total population across all compartments $N = S+E+I+Q+R$ and sum the differential equations in the system (1), yielding

$$\frac{dN}{dt} = \phi - \mu N.$$

This implies that

$$\lim_{t\to\infty} \sup(N(t)) \leq \frac{\phi}{\mu}.$$

Without loss of generality, we conclude that each population compartment is constrained to a size no greater than $\frac{\phi}{\mu}$, establishing an upper bound. Thus, the domain of the solutions can be expressed as

$$D = \left\{(S, E, I, Q, R) \in \mathbb{R}^5_+ : 0 \leq S, E, I, Q, R, \leq \frac{\phi}{\mu}\right\}.$$

B. Basic Reproduction Number

The basic reproduction number, denoted as R_0, represents the average number of secondary infections produced by an infected individual in a completely susceptible population. If R_0 exceeds one, the virus is capable of continuing its spread among the susceptible individuals; conversely, if R_0 is less than one, the infection will eventually die out. A common approach to calculating R_0 is the Next Generation Matrix (NGM) method. In this method, the compartments relevant to the infected population, specifically the exposed (E), infected (I), and quarantined (Q) compartments, are utilized.

To initiate this process, the Jacobian matrix is derived from the three compartments referenced in equation (2), yielding

$$J = \begin{bmatrix} -k_1 & \frac{\phi\beta}{\mu} & 0 \\ \alpha & -k_2 & 0 \\ \gamma_1 & \gamma_2 & -k_3 \end{bmatrix}.$$

In the disease-free equilibrium, where all populations except the susceptible compartment are zero, the value of S is determined as $S = \frac{\phi}{\mu}$. Hence, the Jacobian matrix is constructed based on this disease-free equilibrium condition. The matrix is then decomposed into the difference between the transmission matrix F and the transition matrix V, expressed as

$$J = F - V,$$

where

$$F = \begin{bmatrix} 0 & \frac{\phi\beta}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} k_1 & 0 & 0 \\ -\alpha & k_2 & 0 \\ -\gamma_1 & -\gamma_2 & k_3 \end{bmatrix}.$$

Subsequently, the inverse of the transition matrix V is computed as

$$V^{-1} = \begin{bmatrix} \frac{1}{k_1} & 0 & 0 \\ \frac{\alpha}{k_1 k_2} & \frac{1}{k_2} & 0 \\ \frac{\gamma_1 k_2 + \alpha\gamma_2}{k_1 k_2 k_3} & \frac{\gamma_2}{k_2 k_3} & \frac{1}{k_3} \end{bmatrix}.$$

This allows us to compute

$$FV^{-1} = \begin{bmatrix} \frac{\phi\beta\alpha}{\mu k_1 k_2} & \frac{\phi\beta}{\mu k_2} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

From this matrix, we can derive the characteristic equation

$$\lambda^3 - \left(\frac{\phi\beta\alpha}{\mu k_1 k_2}\right)\lambda^2 = 0, \tag{3}$$

which leads to the eigenvalues

$$\lambda_1 = \frac{\phi\beta\alpha}{\mu k_1 k_2}, \quad \lambda_2 = 0, \quad \lambda_3 = 0.$$

According to the NGM method, the basic reproduction number (R_0) is defined as $R_0 = \rho(FV^{-1})$, where ρ denotes the largest positive eigenvalue of the matrix FV^{-1} [21]. Therefore, the basic reproduction number is equivalent to the eigenvalue λ_1, which can be expressed as

$$R_0 = \frac{\phi\beta\alpha}{\mu k_1 k_2}. \tag{4}$$

C. Equilibrium Point

In the SEIQRS model described in equation (2), we identify two equilibrium points: the disease-free equilibrium and the endemic equilibrium. These are represented as

$$\epsilon_0 = \left(\frac{\phi}{\mu}, 0, 0, 0, 0\right) \tag{5}$$

and

$$\epsilon^* = (S^*, E^*, I^*, Q^*, R^*), \tag{6}$$

where

$$S^* = \frac{k_1 k_2}{\beta\alpha},$$
$$E^* = \frac{k_2 k_3 k_4 (R_0 - 1)}{\beta\alpha\left(k_1 k_2 k_3 k_4 - \alpha\xi_1\sigma k_3 - \gamma_1\xi_2\sigma k_2 - \alpha\gamma_2\xi_2\sigma\right)},$$
$$I^* = \frac{k_3 k_4 (R_0 - 1)}{\beta\left(k_1 k_2 k_3 k_4 - \alpha\xi_1\sigma k_3 - \gamma_1\xi_2\sigma k_2 - \alpha\gamma_2\xi_2\sigma\right)},$$
$$Q^* = \frac{k_4 (R_0 - 1)(\gamma_1 k_2 + \alpha\gamma_2)}{\beta\alpha\left(k_1 k_2 k_3 k_4 - \alpha\xi_1\sigma k_3 - \gamma_1\xi_2\sigma k_2 - \alpha\gamma_2\xi_2\sigma\right)},$$
$$R^* = \frac{(R_0 - 1)(\alpha\xi_1 k_3 + \xi_2\gamma_1 k_2 + \alpha\xi_2\gamma_2)}{\beta\alpha\left(k_1 k_2 k_3 k_4 - \alpha\xi_1\sigma k_3 - \gamma_1\xi_2\sigma k_2 - \alpha\gamma_2\xi_2\sigma\right)}.$$

For the endemic equilibrium point to exist, the following conditions must hold:

$$R_0 > 1,$$
$$k_1k_2k_3k_4 > \alpha\xi_1\sigma k_3 + \gamma_1\xi_2\sigma k_2 + \alpha\gamma_2\xi_2\sigma.$$

III. Stability Analysis

To determine the stability of the equilibrium points, we first derive the Jacobian matrix from equation (2), that is

$$J_0 = \begin{bmatrix} -\beta I - \mu & 0 & -\beta S & 0 & \sigma \\ \beta I & -k_1 & \beta S & 0 & 0 \\ 0 & \alpha & -k_2 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -k_3 & 0 \\ 0 & 0 & \xi_1 & \xi_2 & -k_4 \end{bmatrix} \tag{7}$$

Here, the variables S and I can be replaced with their corresponding equilibrium values to analyze stability. Next, we investigate the signs of the eigenvalues of the Jacobian matrix, which are obtained by solving the characteristic equation $\det(J_0 - \lambda I) = 0$. The equilibrium point is stable when all eigenvalues are negative.

A. Disease-Free Equilibrium

To evaluate the stability of the Disease-Free Equilibrium (DFE), we substitute the equilibrium values from (5) into the Jacobian matrix (7), resulting

$$J_1 = \begin{bmatrix} -\mu & 0 & -\frac{\beta\phi}{\mu} & 0 & \sigma \\ 0 & -k_1 & \frac{\beta\phi}{\mu} & 0 & 0 \\ 0 & \alpha & -k_2 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -k_3 & 0 \\ 0 & 0 & \xi_1 & \xi_2 & -k_4 \end{bmatrix}.$$

Calculating $\det(J_1 - \lambda I) = 0$ gives the eigenvalues $\lambda_1 = -\mu$, $\lambda_2 = -k_4$, and $\lambda_3 = -k_3$, along with the remaining characteristic equation:

$$\lambda^2 - T\lambda + D = 0,$$

where T (trace) and D (determinant) are defined as

$$T = -(k_1 + k_2),$$
$$D = k_1k_2 - \frac{\beta\phi\alpha}{\mu}. \tag{8}$$

Substituting the basic reproduction number R_0 from (4) into D, we obtain:

$$D = k_1k_2(1 - R_0).$$

The conditions for the characteristic equation to yield negative eigenvalues are $T < 0$ and $D > 0$ (see [22] for details). It follows that T is always negative, while D is positive when:

$$k_1k_2 > \frac{\beta\phi\alpha}{\mu} \quad \text{or equivalently,} \quad 1 > R_0.$$

Thus, we conclude the stable condition for the DFE through the following theorem.

Theorem 3. The disease-free equilibrium ϵ_0 is locally asymptotically stable if the basic reproduction number $R_0 < 1$.

B. Endemic Equilibrium

To analyze the stability of the Endemic Equilibrium (END), we similarly substitute the equilibrium values from (6) into the Jacobian matrix (7), yielding

$$J_2 = \begin{bmatrix} \frac{k_3k_4(R_0-1)}{\beta W_1} - \mu & 0 & -\frac{k_1k_2}{\alpha} & 0 & \sigma \\ -\frac{k_3k_4(R_0-1)}{\beta W_1} & -k_1 & \frac{k_1k_2}{\alpha} & 0 & 0 \\ 0 & \alpha & -k_2 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -k_3 & 0 \\ 0 & 0 & \xi_1 & \xi_2 & -k_4 \end{bmatrix}.$$

The characteristic equation $\det(J_2 - \lambda I) = 0$ leads to two eigenvalues $\lambda_1 = -k_3$ and $\lambda_2 = -k_4$, along with a third-order characteristic equation

$$a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0, \tag{9}$$

where the coefficients are given by

$$\begin{aligned} a_3 &= 1, \\ a_2 &= \frac{(\mu + k_1 + k_2)(W_2 + k_1k_2k_3k_4(k_1 + k_2))}{W_1}, \\ a_1 &= \frac{\mu(k_1 + k_2)W_2}{W_1}, \\ a_0 &= \frac{k_1k_2k_3k_4(R_0 - 1)}{W_2}. \end{aligned} \tag{10}$$

The terms W_1 and W_2 from (10) are defined as follows

$$W_1 = k_1k_2k_3k_4 - \alpha\xi_1\sigma k_3 - \gamma_1\xi_2\sigma k_2 - \alpha\gamma_2\sigma,$$
$$W_2 = \phi\alpha\beta k_3k_4 - \alpha\xi_1\sigma k_3 - \alpha\gamma_2\xi_2\sigma - \gamma_1\xi_2\sigma k_2.$$

According to the Routh-Hurwitz criterion [23], the necessary conditions for the characteristic equation to have negative roots are that all coefficients in (10) must be positive, and $a_2a_1 - a_3a_0 > 0$. Thus, the conditions for a stable endemic equilibrium are:

$$\begin{aligned} \phi\alpha\beta k_3k_4 &> \alpha\xi_1\sigma k_3 + \alpha\gamma_2\xi_2\sigma + \gamma_1\xi_2\sigma k_2, \\ k_1k_2k_3k_4 &> \alpha\xi_1\sigma k_3 + \gamma_1\xi_2\sigma k_2 + \alpha\gamma_2\xi_2\sigma, \\ R_0 &> 1, \\ a_2a_1 &> a_3a_0. \end{aligned} \tag{11}$$

Thus, we formulate another theorem regarding the stability and existence of the endemic equilibrium as follows.

Theorem 4. The endemic equilibrium ϵ^* exists and is stable when it satisfies the following conditions:

(i) $\phi\alpha\beta k_3k_4 > \alpha\xi_1\sigma k_3 + \alpha\gamma_2\xi_2\sigma + \gamma_1\xi_2\sigma k_2$,
(ii) $k_1k_2k_3k_4 > \alpha\xi_1\sigma k_3 + \gamma_1\xi_2\sigma k_2 + \alpha\gamma_2\xi_2\sigma$,
(iii) $R_0 > 1$,
(iv) $a_2a_1 > a_3a_0$.

IV. Optimal Control Problem

In this section, we introduce control variables as strategies to suppress the spread of the virus while minimizing costs. The SEIQRS model in equation (2) is modified by incorporating three control variables. The first control variable, denoted by $u(t)$, represents the implementation of non-pharmaceutical interventions, which include measures such as mask usage, social distancing, and proper sanitation. This variable is applied to both susceptible and infectious

individuals. The second variable is the vaccination rate $v(t)$, administered to the susceptible population to confer immunity against COVID-19. Lastly, the quick response variable $w(t)$ is applied to exposed individuals through isolation measures, which may be self-imposed or enforced by healthcare authorities [10]. Therefore, the model in (2) can be modified as

$$\begin{aligned}
\frac{dS}{dt} &= \phi - \beta(1-u(t))SI - v(t)S - \mu S + \sigma R,\\
\frac{dE}{dt} &= \beta(1-u(t))SI - (\alpha+\gamma_1+\mu)E - w(t)E,\\
\frac{dI}{dt} &= \alpha E - (\gamma_2+\xi_1+\mu)I,\\
\frac{dQ}{dt} &= \gamma_1 E + \gamma_2 I - (\xi_2+\mu)Q + w(t)E,\\
\frac{dR}{dt} &= \xi_1 I + \xi_2 Q + v(t)S - (\mu+\sigma)R.
\end{aligned} \tag{12}$$

The variables in equation (12) are referred to as the state variables.

Our objective here is to minimize the costs incurred from non-pharmaceutical interventions, vaccination, and quick responses to suppress the spread of COVID-19. By adapting the objective functional from [12], we propose

$$J = \int_0^{t_f} \left[\epsilon_e E + \epsilon_i I + \epsilon_q Q + \frac{1}{2}\left(\epsilon_u u^2 + \epsilon_v v^2 + \epsilon_w w^2\right)\right] dt. \tag{13}$$

As evident from (13), the cost includes contributions from exposed individuals E, infected individuals I, and quarantined individuals Q, with weighting factors ϵ_e, ϵ_i, and ϵ_q assigned to each category. The non-linear terms reflect the relative costs associated with more restrictive strategies.

Next, we need to calculate the values of the control variables to minimize the cost J. Thus, we define the optimal control as

$$J(u^*, v^*, w^*) = \min\{J(u, v, w) : u, v, w \in \Omega\},$$

where Ω is defined on the interval $[0, t_f]$, and the optimal controls $(u^*(t), v^*(t), w^*(t))$ are constrained between 0 and 1. A value of 0 indicates no application of non-pharmaceutical interventions, vaccination, or quick response, while a value of 1 indicates maximum implementation of these controls.

A. Hamiltonian Function

To determine the optimal solution to the problem stated in (12), we utilize the Hamiltonian function based on Pontryagin's minimum principle. The Hamiltonian function, which combines the objective functional (13) with the state equations from (12), is defined as

$$\begin{aligned}
H = {} & \epsilon_e E + \epsilon_i I + \epsilon_q Q + \frac{1}{2}\left(\epsilon_u u^2 + \epsilon_v v^2 + \epsilon_w w^2\right)\\
& + \lambda_1\left(\phi - \beta(1-u(t))SI - v(t)S - \mu S + \sigma R\right)\\
& + \lambda_2\left(\beta(1-u(t))SI - (\alpha+\gamma_1+\mu)E - w(t)E\right)\\
& + \lambda_3\left(\alpha E - (\gamma_2+\xi_1+\mu)I\right)\\
& + \lambda_4\left(\gamma_1 E + \gamma_2 I - (\xi_2+\mu)Q + w(t)E\right)\\
& + \lambda_5\left(\xi_1 I + \xi_2 Q + v(t)S - (\mu+\sigma)R\right).
\end{aligned} \tag{14}$$

Here, $\lambda_1, \lambda_2, \lambda_3, \lambda_4$, and λ_5 are referred to as the adjoint or co-state variables.

The values of these unknown co-state variables can be determined by establishing their differential equations from the negative derivative of the Hamiltonian function with respect to S, E, I, Q, and R, yielding

$$\begin{aligned}
\frac{d\lambda_1}{dt} =& (\lambda_1-\lambda_2)\left[\beta(1-u(t))I - \lambda_1(v(t)-\mu) - \lambda_5 v(t)\right],\\
\frac{d\lambda_2}{dt} =& -\epsilon_e + \lambda_2\left(\alpha+\gamma_1+\mu-w(t)\right) - \lambda_3\alpha\\
& - \lambda_4\left(\gamma_1 + w(t)\right),\\
\frac{d\lambda_3}{dt} =& -\epsilon_i + (\lambda_1-\lambda_2)\beta(1-u(t))S + \lambda_3(\gamma_2+\xi_1+\mu)\\
& - \lambda_4\gamma_2 - \lambda_5\xi_1,\\
\frac{d\lambda_4}{dt} =& -\epsilon_q - \lambda_4(\xi_2+\mu) - \lambda_5\xi_2,\\
\frac{d\lambda_5}{dt} =& -\lambda_1\sigma + \lambda_5(\mu+\sigma),
\end{aligned} \tag{15}$$

with the transversality condition $\lambda_i(t_f) = 0$ for $i = 1, 2, \ldots, 5$.

B. Stationary Condition

To achieve optimality, the Hamiltonian function must be optimized with respect to the control variables. This leads to the conditions

$$\frac{\partial H}{\partial u} = 0, \quad \frac{\partial H}{\partial v} = 0, \quad \frac{\partial H}{\partial w} = 0.$$

Solving these equations gives us the control variable values that minimize cost

$$\begin{aligned}
u^*(t) &= \frac{(\lambda_2-\lambda_1)\beta SI}{\epsilon_u},\\
v^*(t) &= \frac{(\lambda_1-\lambda_5)S}{\epsilon_v},\\
w^*(t) &= \frac{(\lambda_2-\lambda_4)E}{\epsilon_w}.
\end{aligned}$$

Since these values are constrained between 0 and 1, the optimal solutions can be expressed as:

$$\begin{aligned}
u^*(t) &= \min\left\{1, \max\left[0, \frac{(\lambda_2-\lambda_1)\beta SI}{\epsilon_u}\right]\right\},\\
v^*(t) &= \min\left\{1, \max\left[0, \frac{(\lambda_1-\lambda_5)S}{\epsilon_v}\right]\right\},\\
w^*(t) &= \min\left\{1, \max\left[0, \frac{(\lambda_2-\lambda_4)E}{\epsilon_w}\right]\right\}.
\end{aligned} \tag{16}$$

V. Numerical Simulations and Discussion

This section presents graphical simulations to illustrate stability in both disease-free and endemic scenarios. Additionally, we compare population dynamics under conditions with and without the implementation of control strategies. Furthermore, we examine the parameters that are sensitive to the spread of the outbreak, as well as the variable controls.

TABLE I: Parameter values for (a) disease-free and (b) endemic conditions.

(a)

Parameter	Value
ϕ	0.0113
β	0.1
μ	0.0113
σ	0.0005
α	0.4
γ_1	0.2
γ_2	0.3980
ξ_1	0.182
ξ_2	0.3

(b)

Parameter	Value
ϕ	0.0113
β	0.1
μ	0.0113
σ	0.0005
α	0.4
γ_1	0.0084
γ_2	0.0040
ξ_1	0.0199
ξ_2	0.0416

A. *Equilibria Stability*

We commence with a simulation for the disease-free equilibrium (DFE) utilizing the parameter values outlined in Table Ia, where all initial population values are set to 0.2. To achieve stability, it is essential to satisfy the condition $R_0 < 1$, as stated in Theorem 1. Using the parameters from Table Ia, we calculate (4) to obtain $R_0 = 0.1107$, which confirms a stable DFE. Consequently, the susceptible population increases and approaches its limit, as illustrated in Figure 2. The graph indicates that the susceptible population converges

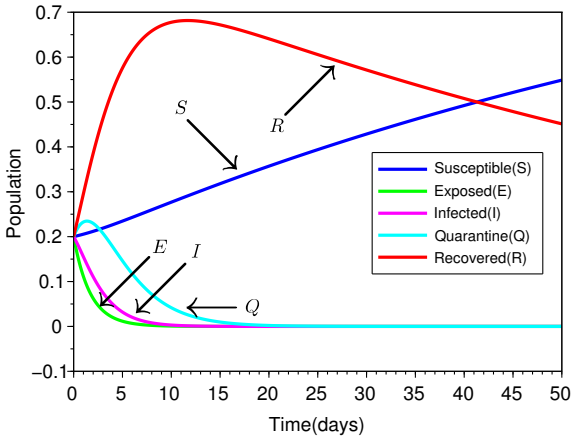

Fig. 2: Population dynamics for the SEIQRS model under disease-free conditions.

to 1, while other compartments ultimately diminish to zero, aligning with the equilibrium values outlined in (5).

Subsequently, we evaluate the endemic condition (END) using distinct parameter values provided in Table Ib, ensuring that $R_0 > 1$ for stability, in accordance with Theorem 2. Calculating R_0 from (4) yields $R_0 = 2.8717$, signifying that the disease is propagating. The infected compartments (exposed, infected, and quarantined) stabilize at positive values, as confirmed by Figure 3. It can be observed from this figure that the number of infected individuals experiences an initial sharp increase due to the spread of the virus. This is followed by a gradual decline as the infected individuals begin to recover and leading to a stable state.

B. *Population Dynamics with Control Variables*

The SEIQRS model incorporating control variables is simulated using the fourth-order Runge-Kutta method. The

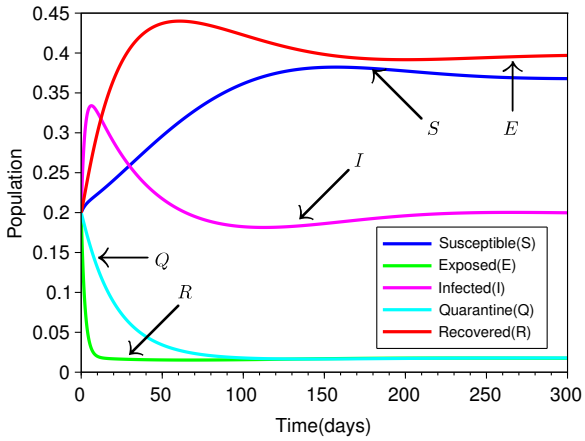

Fig. 3: Population dynamics for the SEIQRS model under endemic conditions.

state equations presented in (12) are solved using a forward step approach, while the co-state equations are resolved with a backward step method.

TABLE II: Parameter values for the optimization problem.

Parameter	ϵ_e	ϵ_i	ϵ_q	ϵ_u	ϵ_v	ϵ_w
Value	0.1	0.1	0.1	0.01	0.001	0.001

Utilizing parameters from Table Ib along with the weights in Table II, we analyze the dynamics of the control variables as depicted in Figure 4. The results reveal that initially, non-pharmaceutical interventions (represented by the red graph) require intensive implementation to mitigate disease spread. However, this need diminishes rapidly, requiring only mini-

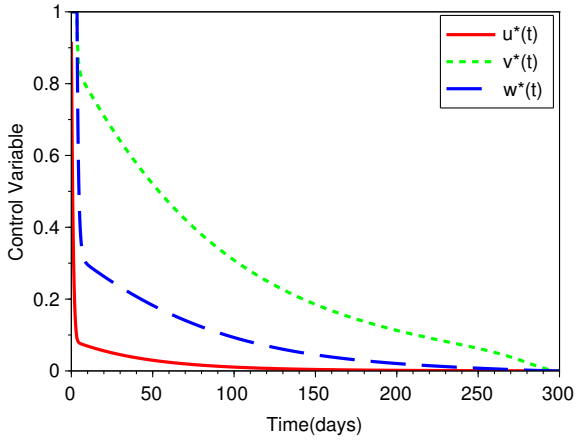

Fig. 4: Dynamics of control variables aimed at achieving optimal conditions.

mal intervention over time. Conversely, vaccination efforts do not experience a swift decline in intensity, as illustrated by the green graph, which indicates a gradual reduction until around day 300, at which point vaccination is no longer necessary. The blue graph, representing quick response measures, follows a trend similar to that of non-pharmaceutical interventions. Both control strategies necessitate a relatively

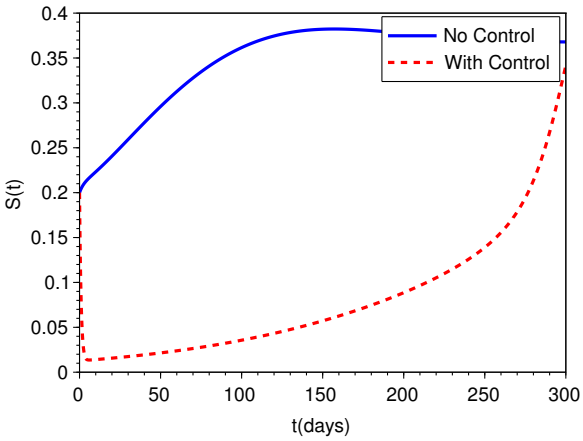

Fig. 5: Susceptible population dynamics with and without control.

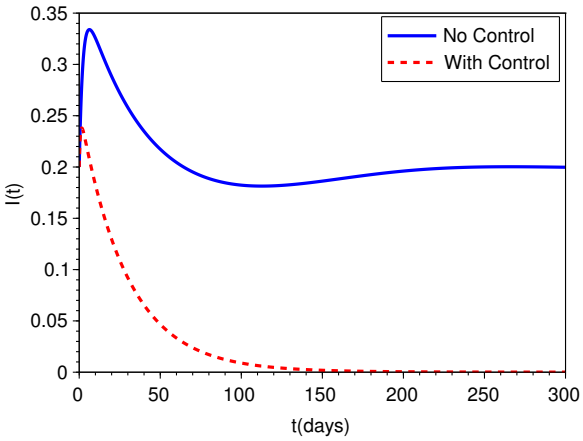

Fig. 7: Infected population dynamics with and without control.

brief period of high intensity to effectively manage disease transmission, although the level of quick response is slightly higher than that of non-pharmaceutical interventions.

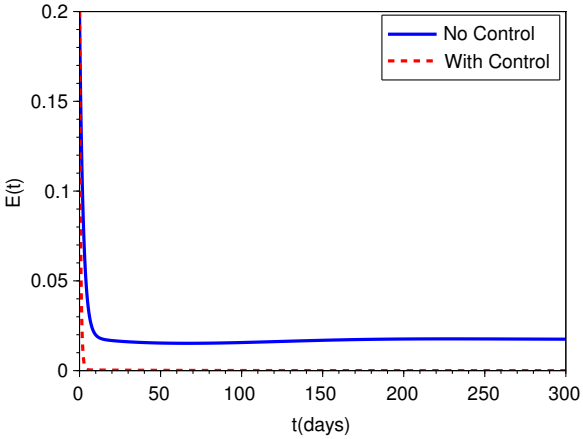

Fig. 6: Exposed population dynamics with and without control.

Another simulations were conducted to compare population dynamics with and without control strategies. Figures 5 and 6 display the dynamics of the susceptible and exposed populations, respectively. When no controls are implemented, the susceptible population gradually increases toward its limiting value. In contrast, the implementation of controls results in a marked decrease in susceptible individuals within approximately five days, although this population begins to rise again thereafter.

The analysis reveals that the control measures, particularly vaccination, significantly reduce the exposed and infected populations, as seen in Figures 6 and 7. Conversely, quick response efforts notably influence the quarantined population, where prompt quarantining leads to an increase in this compartment initially, before it eventually declines as disease transmission is effectively curtailed, as seen in Figure 8.

For the recovered population, as shown in Figure 9, the administration of vaccination directly facilitates a rapid increase in the number of individuals who recover, due to the immediate transfer of susceptible individuals to the recovered category, bypassing the exposed and infected stages.

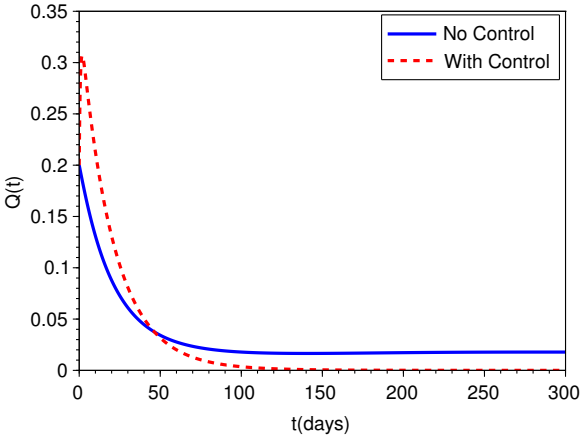

Fig. 8: Quarantined population dynamics with and without control.

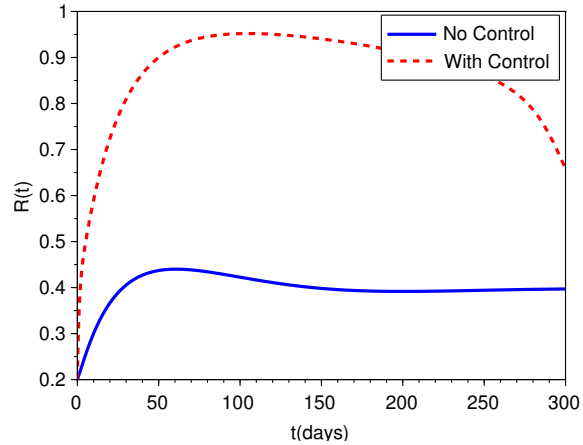

Fig. 9: Recovered population dynamics with and without control.

C. Parameter Sensitivity

The next simulation seeks to identify the parameters that significantly impact the level of virus transmission, including the parameters influencing the level of control measures aimed at mitigating the disease's spread while minimizing costs.

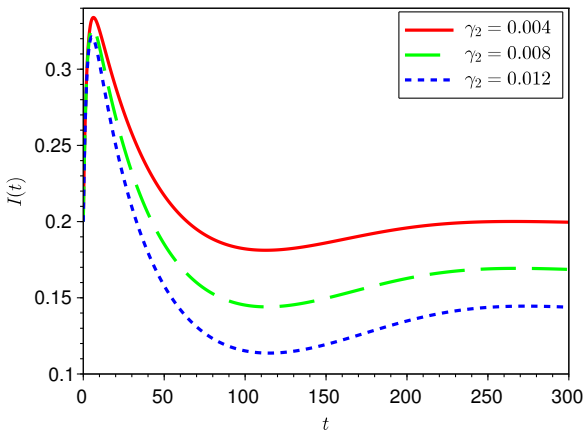

Fig. 10: Comparisons the infected population dynamics with varying γ_2.

The first parameter under examination is γ_2, the isolation rate constant for the infected population. This value is varied to assess its effect on the number of infected individuals. As demonstrated in Figure 10, an increase in γ_2, which corresponds to enhanced isolation of the infected population, leads to a reduction in disease transmission, as evidenced by the decreasing number of infected individuals. This finding aligns with the relationship between γ_2 and R_0, as illustrated in equation (4), where both parameters exhibit an inverse relationship.

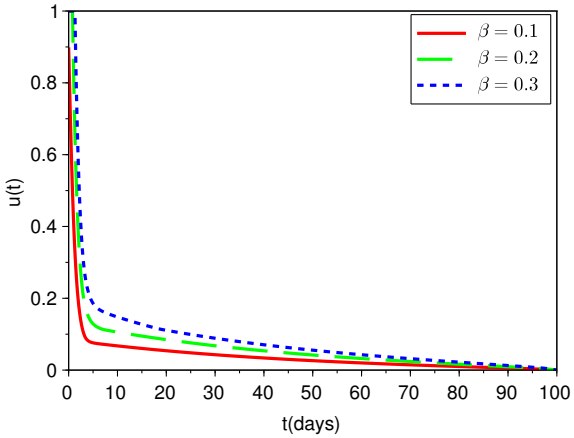

Fig. 11: Comparisons the non-pharmaceutical intervention dynamics when β are varied.

The next parameters considered are ϕ, β, and γ_1, which are varied to evaluate their influence on the control variables u, v, and w. The parameter β, which denotes the direct interactions between the infected and susceptible populations, is analyzed in relation to the implementation of non-pharmaceutical interventions u. Figure 11 highlights that non-pharmaceutical activities u, such as advisories and strict regulations for social distancing, should be intensified in response to the increasing number of susceptible individuals who, either knowingly or unknowingly, come into direct contact with the infected population.

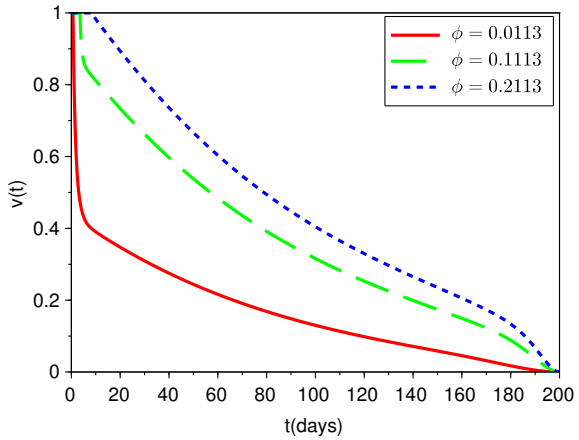

Fig. 12: Comparisons the non-pharmaceutical intervention dynamics when ϕ are varied.

Regarding the parameter ϕ, which represents the growth rate constant for the susceptible population, an increase in its value necessitates an enhancement of vaccination efforts v to prevent or mitigate the spread of the virus. Figure 12 indicates that during the initial 10 days of vaccination implementation, optimal efforts are required when ϕ is raised to 0.2113. Subsequently, vaccination efforts can be gradually reduced to minimize costs and can be discontinued after day 200.

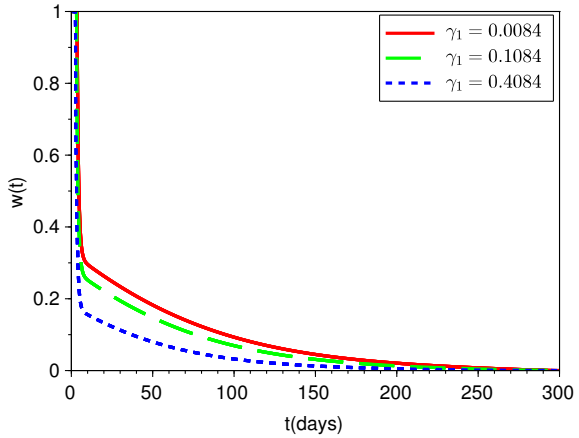

Fig. 13: Comparisons the non-pharmaceutical intervention dynamics when γ_1 are varied.

Lastly, the parameter γ_1, or the isolation rate constant for the exposed population, is investigated for its impact on the control variable w, which pertains to rapid response measures. As a growing number of exposed individuals become aware of their infection, for instance through PCR testing, and promptly isolate themselves, the simulations

illustrated in Figure 13 suggest that health authorities may not need to intensify rapid response measures for isolating these individuals. Rather, it may be adequate to reduce such measures, thereby minimizing the associated costs.

VI. Conclusion

In this article, we have developed and analyzed the spread of COVID-19 using the SEIQRS model, which incorporates the possibility that individuals who have recovered may become susceptible again. This indicates that there is a potential for reinfection among recovered individuals. From this model, two equilibrium states are identified: disease-free and endemic.

To achieve the disease-free state, the basic reproduction number must be maintained below one. Conversely, when the basic reproduction number exceeds one, and the conditions outlined in Theorem 4 are satisfied, an endemic state emerges. One simulation conducted in this study demonstrates that the disease can be effectively suppressed by increasing the isolation rate of the infected population. This outcome is attributed to heightened awareness and responsibility among infected individuals regarding self-isolation. This means that direct contact with the susceptible population is reduced, resulting in a decrease in the number of infected individuals.

We anticipated that the endemic scenario can be transformed into a disease-free situation. Therefore, this paper discussed three control variables: non-pharmaceutical interventions, vaccination, and quick response measures aimed at mitigating the spread of the virus. The implementation these controls has proven effective in suppressing the disease's transmission. Utilizing Pontryagin's minimum principle, these controls facilitate the prevention of virus spread at minimal cost.

Furthermore, we have investigated three parameters concerning their impact on the control variables. When the direct contact rate between susceptible and infected individuals rises, a simulation suggests that the implementation of non-pharmaceutical interventions, such as social distancing, should be intensified to prevent virus transmission. Similarly, an increase in the growth rate of the susceptible population necessitates a substantial enhancement in vaccination efforts. Conversely, when the exposed population demonstrates a high level of awareness regarding self-isolation, a simulation indicates that health authorities need not intensify quick response measures; instead, these measures can be scaled back while still effectively preventing disease transmission.

References

[1] A. Lee, "Wuhan novel coronavirus (covid-19): why global control is challenging?" *Public health*, vol. 179, pp. A1–A2, 2020.

[2] J. Zhong, J. Tang, C. Ye, and L. Dong, "The immunology of covid-19: is immune modulation an option for treatment?" *The Lancet Rheumatology*, vol. 2, no. 7, pp. e428–e436, 2020.

[3] J. Cuevas-Maraver, P. G. Kevrekidis, G. A. Kevrekidis, and Y. Drossinos, "Vaccination compartmental epidemiological models for the delta and omicron sars-cov-2 variants," *Mathematical Biosciences*, vol. 367, no. 6, p. 109109, 2023.

[4] E. A. Iboi, C. N. Ngonghala, and A. B. Gumel, "Will an imperfect vaccine curtail the covid-19 pandemic in the us?" *Infectious Disease Modelling*, vol. 5, pp. 510–524, 2020.

[5] A. Awasthi, "A mathematical model for transmission dynamics of covid-19 infection," *Eur. Phys. J. Plus*, vol. 138, no. 3, p. 285, 2023.

[6] P. Samui, J. Mondal, and S. Khajanchi, "A mathematical model for covid-19 transmission dynamics with a case study of india," *Chaos, Solitons & Fractals*, vol. 140, p. 110173, 2020.

[7] M. A. A. Pratama, A. K. Supriatna, and N. Anggriani, "A mathematical model of covid-19 transmission between regions with symptomatic and asymptomatic cases," *IAENG International Journal of Applied Mathematics*, vol. 52, no. 2, pp. 404–410, 2022.

[8] J. Tan, Y. Ge, L. Martinez, J. Sun, C. Li, A. Westbrook, E. Chen, J. Pan, Y. Li, W. Cheng, F. Ling, Z. Chen, Y. Shen, and H. Huang, "Transmission roles of symptomatic and asymptomatic covid-19 cases: a modelling study," *Epidemiol Infect.*, vol. 150, p. e171, 2022.

[9] R. Ramesh and G. A. Joseph, "The optimal control methods for the covid-19 pandemic model's precise and practical siqr mathematical model," *IAENG International Journal of Applied Mathematics*, vol. 54, no. 8, pp. 1657–1672, 2024.

[10] T. Hussain, M. Ozair, F. Ali, S. ur Rehman, T. A. Assiri, and E. E. Mahmoud, "Sensitivity analysis and optimal control of covid-19 dynamics based on seiqr model," *Results in Physics*, vol. 22, p. 103956, 2021.

[11] M. Sinan, A. Ali, K. Shah, T. A. Assiri, and T. A. Nofal, "Stability analysis and optimal control of covid-19 pandemic seiqr fractional mathematical model with harmonic mean type incidence rate and treatment," *Results in Physics*, vol. 22, p. 103873, 2021.

[12] P. Das, R. K. Upadhyay, A. K. Misra, F. A. Rihan, P. Das, and D. Ghosh, "Mathematical model of covid-19 with comorbidity and controlling using non-pharmaceutical interventions and vaccination," *Nonlinear Dynamics*, vol. 106, no. 2, pp. 1213–1227, 2021.

[13] G. Webb, "A covid-19 epidemic model predicting the effectiveness of vaccination in the us," *Infectious Disease Reports*, vol. 13, no. 3, pp. 654–667, 2021.

[14] G. Webb and X. E. Zhao, "An epidemic model with infectioin age and vaccination age structure," *Infectious Disease Reports*, vol. 16, no. 1, pp. 35–64, 2024.

[15] M. Makhoul, H. H. Ayoub, H. Chemaitelly, S. Seedat, G. R. Mumtaz, S. Al-Omari, and L. J. Abu-Raddad, "Epidemiological impact of sars-cov-2 vaccination: Mathematical modeling analyses," *Vaccines*, vol. 8, no. 4, p. 668, 2020.

[16] W. Ogana, V. O. Juma, and W. D. Bulimo, "Mathematical modelling of non-pharmaceutical interventions to control infectious disease: application to covid-19 in kenya," *Frontiers in Applied Mathematics and Statistics*, vol. 10, pp. 1–12, 2024.

[17] S. Ullah and M. A. Khan, "Modeling the impact of non-pharmaceutical intervention on the dymanics of novel coroanvirus with optimal control analysis with a case study," *Chaos, Solitons & Fractals*, vol. 139, p. 110075, 2020.

[18] Marsudi, N. Shofianah, and U. Habibah, "Analysis of optimal cost-effectiveness in the covid-19 epidemic model by considering quarantine, hospitalization, and the environment," *IAENG International Journal of Applied Mathematics*, vol. 53, no. 4, pp. 1375–1388, 2023.

[19] H. Song, R. Wang, S. Liu, Z. Jin, and D. He, "Global stability and optimal control for a covid-19 model with vaccination and isolation delays," *Results in physics*, vol. 42, p. 106011, 2022.

[20] A. I. Abioye, O. J. Peter, H. A. Ogunseye, F. A. Oguntolu, K. Oshinubi, A. A. Ibrahim, and I. Khan, "Mathematical model of covid-19 in nigeria with optimal control," *Results in Physics*, vol. 28, p. 104598, 2021.

[21] C. Castillo-Chavez, Z. Feng, and W. Huang, "On the computation of r_0 and its role on global stability," *IMA*, vol. 125, pp. 229–250, 2002.

[22] M. W. Hirsch, S. Smale, and R. L. Devaney, *Differential equations, dynamical systems, and an introduction to chaos*. Academic press, 2012.

[23] J. D. Murray, *Mathematical Biology: I. An Introduction*. Springer, 2002.