SABA Publishing

Journal of Mathematical Analysis and Modeling
jmam.sabapub.com
ISSN 2709-5924

J Math Anal & Model
(2024)5(3) : 36-49
doi:10.48185/jmam.v1i3.1316

The importance of quarantine: A bifurcation analysis and modeling of the transmission dynamics of Covid-19

FATMA N. A. MOHAMED [a,*], MOHAMED H. AMSAAD [b],

[a] Department of Mathematics, College of Education, University of Benghazi, Benghazi, Libya
[b] Department of Mathematics, College of Arts and Sciences, University of Benghazi, Qaminis, Libya

• *Received: 24 August 2024* • *Accepted: 06 October 2024* • *Published Online: 03 November 2024*

Abstract

The research aims to construct a mathematical model for COVID-19 that includes features six compartments to evaluate the positive effects of quarantine measures. The model categorizes individuals into the following classes: susceptible, exposed, quarantined, asymptomatic cases, symptomatic cases, and recovered ($SEQI_1I_2R$). Several assumptions regarding positivity and boundness are identified to ensure that the solution originated within a certain class and that the basic reproduction number is analyzed. Of course, the existence of an endemic equilibrium is argued, which provides an understanding of the long-term persistence of the disease. More precisely, to enhance our understanding of the model's dynamics, we have analyzed both the local and global asymptotic stability of the disease-free equilibrium. Moreover, to assess the global stability of the system, we employ a Lyapunov function which provides a comprehensive mathematical evaluation. At the same time, our findings show evidence of a backward bifurcation which is recognized as a possible result of the clinical transition from an asymptomatic state to symptom one.

Keywords: Quarantine, Basic reproduction number, Stability, Backward bifurcation, Covid-19 model.
2010 MSC: 92B05, 92D30.

1. Introduction

Covid-19 is an infectious disease caused by the *SARS-CoV*-2 virus. To effectively prevent infection and limit transmission, it is essential to understand the virus and its modes of spread from WHO [24]. Researchers have developed various models to study the dynamics of the *Covid*-19 epidemic, which emerged in 2019 [1, 6, 9, 15].

The initial models in mathematical epidemiology, such as the *Susceptible-Exposed-Infectious-Recovered* (*SEIR*) and *Susceptible-Infectious-Recovered* (*SIR*) models, were established on 1920s. These ordinary differential equation models have proven to be valuable

*Corresponding author: fatma.mohammed@uob.edu.ly

tools for analyzing the dynamics of various real-world phenomena [2, 11, 16, 17]. However, there is a growing trend towards the utilization of fractional calculus models in exhibiting the mathematics of several factors including the transmission index of *Covid*-19 [4, 8, 12, 20].

An additional examination of parameters was conducted through bifurcation analysis, which offers insights into the structural characteristics of a system as parameters change. This approach proved to be particularly beneficial in the investigation of virus transmission. The clinical progression from asymptomatic to mild and severe stages has been associated with backward bifurcation. Furthermore, the dynamics of the two equilibria endemic and disease-free were also taken into account, along with the concept of backward bifurcation [25]. The examination of bifurcation and the modeling of *Covid*-19 transmission dynamics have revealed that backward bifurcation often takes place. This occurrence complicates disease control efforts, as a stable disease-free equilibrium can coexist with a stable endemic equilibrium, even when the *basic reproduction number* ($\mathcal{R}_o$) is less than one. It is well-known that such number is one of the most fundamental metrics employed in assessing transmissibility within the field of epidemiology [23]. If $\mathcal{R}_o$ exceeds one, each infection has the potential to propagate within the population density; conversely, if $\mathcal{R}_o$ is below one, the incidence of the disease diminishes [21]. Therefore, while it is crucial to reduce $\mathcal{R}_o$ to below one, this measure alone is insufficient for the total elimination of the disease [7, 13, 14, 17, 22].

Given the widespread nature of transmission, it is crucial to implement a variety of interventions, including vaccines, quarantine protocols, and protective measures. The models indicate that employing all these strategies simultaneously is the most effective approach to mitigate the further progression of *Covid*-19 [13, 14, 22]. The addition of comorbidity into these models demonstrates a significant boost in risk which supports the significance of well-rounded treatment strategies [7]. The pandemic has shown that it is crucial to understand how the diseases spread and how to protect from them, and that the tools of managing exist and need to be followed [19].

One major problem in fighting *Covid*-19 is the fact that there are infected individuals who do not show symptoms of the virus; these often go in quarantined. To evaluate the effects of quarantine on the behavior of the *SEIR* model, we have expanded the model by the addition of a quarantine compartment (Q) for non-infected but exposed persons. Some of the researchers have use quarantine factor within epidemic models, see [3, 10, 25]. The *SEQIR* model in [18] categorizes the infected individuals into distinct classes based on their symptoms, specifically those who have no exhibiting or mild symptoms and those who experiencing severe symptoms.

In this study, our endeavor is to develope an epidemic model referred to as $\mathrm{SEQI_1I_2R}$. This model addresses various elements, including the positivity and boundedness of the system, the identification of equilibrium points, and the calculation and estimation of the basic reproduction number. Additionally, our main contribution is to demonstrate the presence of backward bifurcation and establishing the global stability of the system. The paper is organized as follows: Section 2 begins by formulation of essential constructions to our model. Section 3 presentes brief recalling of some basic properties to the model. Section 4 focuse on precisely quantified the basic reproduction number, whereas Section 5 is adebted to analyzes the cases where the steady states enjoy the basic reproduction

number. We conclude with Section 6, where we discussed backward bifurcation and it is possible occourance.

2. Methodology

2.1. Model Formulation

Our model is based on the SEIR model transmission, we will investigate the effects of quarantine. The model formulation $SEQI_1I_2R$ used in this study focuses on human-to-human transmission of the *Covid*-19 within a closed population. The total population size at time t is denoted by $N(t)$, which subdivided into six different classes, namely, the susceptible individuals $S(t)$, the exposed individuals $E(t)$, the infected class can be subdivided into two cases, the asymptomatic infections $I_1(t)$, symptomatic infections $I_2(t)$, the quarantined individuals $Q(t)$, and the recovered individuals $R(t)$. In this paper, we assume that some exposed persons may not be quarantined, and thus move directly into the infected compartment at the rate $\sigma_j\, E\ (j = 1, 2)$ where $\sigma_1 + \sigma_2 = \sigma$, and those quarantined, tested and found negative move back into the susceptible population at the rate $\rho\, Q$. Similarly, recovered persons who eventually loose immunity become susceptible again at the rate $\tau\, R$.

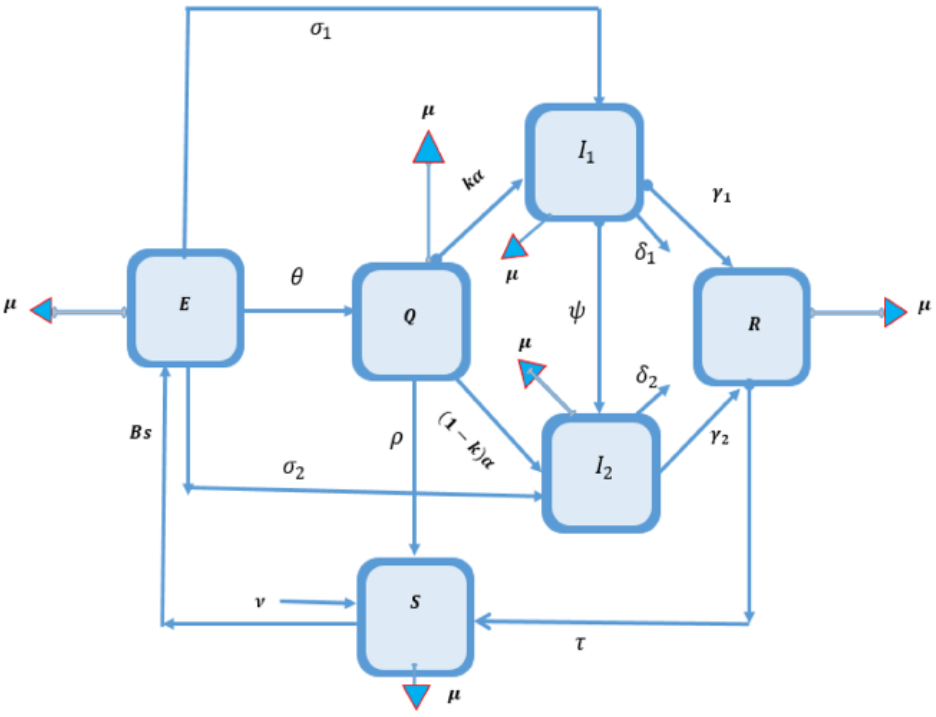

Figure 1: A schematic diagram illustrating the transmission dynamics of *Covid*-19.

Table 1: Description of the variables and the parameters for the model

Variables for the model		**Parameters for the model**	
Variable	**Description**	**Parameter**	**Description**
$S(t)$	Susceptible people	δ_1	Death rate by disease from asymptomatic people
$E(t)$	Exposed people	δ_2	Death rate by disease from symptomatic people
$Q(t)$	Quarantined people	α	Progression rate from Q to either I_1 or I_2
$I_1(t)$	Asymptomatic infections	σ_j	The rate of propagation from the exposed class directly into the infected classes $I_j, j = 1, 2$. $\sigma = \sigma_1 + \sigma_2$
$I_2(t)$	Symptomatic infections	ψ	The rate of propagation from the asymptomatic to symptomatic
	$I(t) = I_1(t) + I_2(t)$	ν	The recruitment rate of susceptible individuals
$R(t)$	Recovered people	ρ	The rate of those quarantined tested and found negative move back into the susceptible population
		τ	The rate of recovered person who lose immunity become susceptible again
		β_1	Rate of transmission from S to E due to contract with I_1
		β_2	Rate of transmission from S to E due to contract with I_2
			$\beta_S = \beta_1 + \beta_2$
		θ	The rate of propagation from the exposed class directly into the quarantined class
		γ_1	Rate of recovery of people from I_1
		γ_2	Rate of recovery of people from I_2
		μ	Natural death rate

This study investigates a homogeneous distribution of individuals, facilitating the creation of a mathematical model to depict the spread of *Covid*-19 among a population. An outline of the parameters employed in the construction of the model is provided in Table 1. These parameters are essential for characterizing the system's dynamics and are crucial for understanding the disease's behavior. Based on Figure 1, we establish the following model:

2.2. Model Constructs

The governing equations for the model are subsequently expressed as nonlinear ODE's:

$$\begin{aligned}
\dot{S}(t) &= \nu - (\mu + \beta_S)\, S + \rho\, Q + \tau\, R \\
\dot{E}(t) &= \beta_S\, S - (\mu + \sigma + \theta)\, E \\
\dot{Q}(t) &= \theta\, E - (\mu + \rho + \alpha)\, Q \\
\dot{I_1}(t) &= \kappa\, \alpha\, Q + \sigma_1 E - (\mu + \gamma_1 + \psi + \delta_1)\, I_1 \\
\dot{I_2}(t) &= (1 - \kappa)\, \alpha\, Q + \psi\, I_1 + \sigma_2\, E - (\mu + \gamma_2 + \psi + \delta_2)\, I_2 \\
\dot{R}(t) &= \gamma_1\, I_1 + \gamma_2\, I_2 - (\mu + \tau)\, R
\end{aligned} \tag{2.1}$$

with the initial conditions

$$S(0) \geqslant 0,\ E(0) \geqslant 0,\ Q(0) \geqslant 0,\ I_1(0) \geqslant 0,\ I_2(0) \geqslant 0,\ R(0) \geqslant 0. \tag{2.2}$$

3. Model analysis

In this section, a detailed and rigorous analysis of the model is presented to gain insight into the dynamical features of the model.

3.1. Basic properties of the model

In this subsection, we will discuss some fundamental properties of the proposed model. First, we establish the existence and boundedness of solutions to demonstrate that the system (2.1) has well-defined solutions. Furthermore, these solutions are unique and confined within a positive invariant region. These properties ensure the well-posedness and epidemiological significance of the developed model.

3.1.1. Positively-invariant

Let Λ be the region of the model defined by

$$\Lambda = \{(S, E, Q, I_1, I_2, R) \in \mathbb{R}^6_+ \ :\ S + E + Q + I_1 + I_2 + R \leqslant \nu/\mu\}$$

which is positively-invariant according to the following statement.

Lemma 3.1. *The region Λ is positively-invariant which indicates that all solutions of the system (2.1) with the intial conditions (2.2) in Λ remain in Λ for all positive times.*

Proof. We begin by adding the both sides of the system (2.1) to get

$$\dot{N}(t) = \nu - \mu N(t) - \delta_1 I_1(t) - \delta_2 I_2(t)$$

and it follows that

$$\dot{N}(t) \leqslant \nu - \mu N(t).$$

Multiplying the last inequality by the integrating factor $\exp(\mu t)$ and integrate the both sides from 0 to t to obtain

$$N(t) \leqslant N(0)\ \exp(-\mu t) + (\nu/\mu)(1 - \exp(-\mu t)).$$

Passing to the limit as t goes to ∞ yields $\exp(-\mu t)$ tends to zero and hence $0 \leqslant N(t) \leqslant \nu/\mu$. Observe that whenever $N(t) > \nu/\mu$, then $\dot{N} < 0$ which means that the population size $N(t)$ approaches ν/μ. On the other hand, whenever $N(t) \leqslant \nu/\mu$ every solution of the system (2.1) with the intial conditions (2.2) in $\mathbb{R}^6_+$ will stay in the region Λ for all $t > 0$. Thus, the region Λ is positively-invariant. □

3.1.2. Positivity and boundedness

For the system (2.1) to be epidemiologically meaningful, it pertinent to show that all its state variables are non-negative for all times.

Lemma 3.2. *For all* $t > 0$*, the solution* $(S(t), E(t), Q(t), I_1(t), I_2(t), R(t))$ *of the system* (2.1) *with the initial conditions* (2.2) *are positive.*

Proof. Set $t^* = \sup\{t > 0 : S(0) \geqslant 0, E(0) \geqslant 0, Q(0) \geqslant 0, I_1(0) \geqslant 0, I_2(0) \geqslant 0, R(0) \geqslant 0\}$. From the first equation of the system (2.1) we have

$$\dot{S}(t) = \nu - (\mu + \beta_S)\, S(t) + \rho\, Q(t) + \tau\, R(t)$$

which implies

$$\dot{S}(t) \geqslant \nu - (\mu + \beta_S)\, S(t).$$

Then, multiplying the last inequality by the integrating factor $\exp\left(\mu t + \int_0^t \beta_S(z)\, dz\right)$ and integrate the both sides from 0 to t^* to obtain

$$S(t^*) \geqslant S(0) \exp\left(-\mu t^* - \int_0^{t^*} \beta_S(z) dz\right) + \nu \exp\left(-\int_0^{t^*} \beta_S(z) dz\right) \int_0^{t^*} \exp\left(\int_0^t \beta_S(z) dz\right) dt$$

which indicates that $S(t^*) \geqslant 0$ when $S(0) \geqslant 0$. Similarly, we can obtain $E(t) \geqslant 0, Q(t) \geqslant 0, I_1(t) \geqslant 0, I_2(t) \geqslant 0, R(t) \geqslant 0$ for all times $t > 0$. Therefore, the solution of the system (2.1) with the initial conditions (2.2) are positive. □

Remark 3.3. Boundedness of the solution holds as a conclusion of Lemmas 3.1, 3.2 above.

3.2. Equilibria

This subsection focuses on existence of equilibria of the system (2.1).

3.2.1. Disease-Free Equilibrium

The system (2.1) has a unique *disease-free equilibrium* (*DFE*), represented as

$$\chi_o(S, E, Q, I_1, I_2, R) = (\nu/\mu, 0, 0, 0, 0, 0) \tag{3.1}$$

which can be easily achieves by setting the left-hand side of the system (2.1) to zero and solving the resulting equations simultaneously. Epidemiologically, it means that the disease infection can not be transmission, or equivalently the avrage of spread infection equals to zero.

3.2.2. Disease-Endemic Equilibrium

The disease-endemic equilibrium (*DEE*) of the system (2.1) can be expressed as

$$S^* = \frac{\nu}{\mu + H_4 \sum_{i=1}^{2} \beta_i I_i^*}$$

$$E^* = \frac{\nu \sum_{i=1}^{2} \beta_i I_i^*}{A \left(\mu + H_4 \sum_{i=1}^{2} \beta_i I_i^*\right)}$$

$$Q^* = \frac{\theta\,\nu\,\sum_{i=1}^{2}\beta_i I_i^*}{A\,B\,\left(\mu + H_4\sum_{i=1}^{2}\beta_i I_i^*\right)}$$

$$I_1^* = \frac{\nu\,H_1\,\sum_{i=1}^{2}\beta_i I_i^*}{\mu + H_4\sum_{i=1}^{2}\beta_i I_i^*} \tag{3.2}$$

$$I_2^* = \frac{\nu\,H_2\,\sum_{i=1}^{2}\beta_i I_i^*}{\mu + H_4\sum_{i=1}^{2}\beta_i I_i^*} \tag{3.3}$$

$$R^* = \frac{\nu\,H_3\,\sum_{i=1}^{2}\beta_i I_i^*}{\mu + H_4\sum_{i=1}^{2}\beta_i I_i^*}$$

where

$$H_1 = \frac{\kappa\,\alpha\,\theta + \sigma_1\,A}{A\,B\,C} > 0,$$
$$H_2 = \frac{(1-\kappa)\,\alpha\,\theta\,C + \sigma_2\,B\,C + \psi\kappa\,\alpha\,\theta + \psi\,\sigma_1\,B}{A\,B\,C\,D} > 0,$$
$$H_3 = \frac{1}{\mu+\tau}[\gamma_1\,H_1 + \gamma_2\,H_2] > 0,$$
$$H_4 = 1 - \frac{\rho\,\theta}{A\,B} - \tau\,H_3 > 0 \quad \text{if } 1-(\rho\,\theta/A\,B) > \tau\,H_3.$$

From (3.2) and (3.3) we get

$$\beta_1\,H_4\,(I_1^*)^2 + \beta_2\,H_4\,I_1^*\,I_2^* + (\mu - \nu\,\beta_1\,H_1)\,I_1^* - \nu\,\beta_2\,H_1 I_2^* = 0 \tag{3.4}$$

and

$$\beta_2\,H_4\,(I_2^*)^2 + \beta_1\,H_4\,I_1^*\,I_2^* + (\mu - \nu\,\beta_2\,H_2)\,I_2^* - \nu\,\beta_1\,H_2 I_1^* = 0. \tag{3.5}$$

By taking equation (3.4) and multiplying it by $\beta_1\,H_4$, and then multiplying equation (3.5) by $\beta_2\,H_4$, we can subtract the two resulting expressions to derive a quadratic equation involving the two variables, I_1^* and I_2^*, as demonstrated below

$$\beta_1^2 H_4 (I_1^*)^2 - \beta_2^2 H_4 (I_2^*)^2 + \left[\beta_1\mu - \nu\beta_1^2 H_1 + \nu H_2\beta_1\beta_2\right] I_1^* - \left[\nu\beta_1\beta_2 H_1 + \beta_2\mu - \nu\beta_2^2 H_2\right] I_2^* = 0.$$

Consequently, since the discriminant is expressed as $\Delta = -4[\beta_1^2 H_4][-\beta_2^2 H_4]$ greater than zero, the system defined by equations (2.1) has two unequal endemic equilibria, which can be denoted by $\chi^* = (S^*, E^*, Q^*, I_1^*, I_2^*, R^*)$ and $\chi^{**} = (S^{**}, E^{**}, Q^{**}, I_1^{**}, I_2^{**}, R^{**})$.

Remark 3.4. The system (2.1) has two differnt disease-endemic equilibria.

4. Basic Reproduction Number

We utilize the next generation matrix method to calculate the basic reproduction number for the *Covid*-19 infection [23]. Our main focus is on the infected subsystem of the system (2.1), which can be represented as follows:

$$\frac{d\chi_i}{dt} = \mathcal{F}_i(\chi) - \mathcal{V}_i(\chi) \quad (i = 1, \cdots, 4),$$

where $\chi = (E, Q, I_1, I_2)^t$, $\mathcal{F}_i$ denotes the rate of new infections occurring in compartment i, while $\mathcal{V}_i$ signifies the rates at which infections are transferred into and out of compartment i, we obtain

$$\begin{aligned}
&\mathcal{F}_1 = \beta_S\, S\,, \quad \mathcal{F}_2 = \mathcal{F}_3 = \mathcal{F}_4 = 0,\\
&\mathcal{V}_1 = (\mu+\sigma+\theta)\, E,\\
&\mathcal{V}_2 = (\mu+\rho+\alpha)\, Q - \theta\, E,\\
&\mathcal{V}_3 = (\mu+\gamma_1+\psi+\delta_1)\, I_1 - \kappa\,\alpha\, Q - \sigma_1\, E,\\
&\mathcal{V}_4 = (\mu+\gamma_2+\psi+\delta_2)\, I_2 - \psi\, I_1 - \sigma_2\, E - (1-\kappa)\,\alpha\, Q.
\end{aligned}$$

Hence, the Jacobian matrices for $\mathcal{F}$ and $\mathcal{V}$ evaluated at *DFE*; χ_o, are

$$J[\mathcal{F}](\chi_o) = \begin{bmatrix} 0 & 0 & \beta_1\nu/\mu & \beta_2\nu/\mu \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$J[\mathcal{V}](\chi_o) = \begin{bmatrix} \mu+\sigma+\theta & 0 & 0 & 0 \\ 0 & \mu+\rho+\alpha & 0 & 0 \\ -\sigma_1 & -\kappa\alpha & \mu+\gamma_1+\psi+\delta_1 & 0 \\ -\sigma_2 & -(1-\kappa)\,\alpha & -\psi & \mu+\gamma_2+\psi+\delta_2 \end{bmatrix}$$

The inverse of the matrix $\mathcal{V}$ is obtained as

$$\mathcal{V}^{-1} = \begin{bmatrix} 1/A & 0 & 0 & 0 \\ \theta/AB & 1/B & 0 & 0 \\ [\kappa\alpha\theta+\sigma_1 B]/ABC & \kappa\alpha/BC & 1/C & 0 \\ ([\kappa\psi+(1-\kappa)C]\alpha\theta+[\sigma_1\psi+\sigma_2 C]B)/ABCD & [\kappa\psi+(1-\kappa)C]\alpha/BCD & \psi/CD & 1/D \end{bmatrix}$$

where $|\mathcal{V}| = ABCD$ and $A = \mu+\sigma+\theta$, $B = \mu+\rho+\alpha$, $C = \mu+\gamma_1+\psi+\delta_1$, $D = \mu+\gamma_2+\psi+\delta_2$. Therefore, the basic reproduction number, denoted by $\mathcal{R}_0$, is given by the spectral radius of the next generation matrix $\mathcal{F}\mathcal{V}^{-1}$ for system (2.1), i.e,

$$\mathcal{R}_o = \left(\frac{\nu}{\mu}\right)\left[\frac{\beta_1(\kappa\alpha\theta+\sigma_1 B)D + \beta_2\,([\kappa\psi+(1-\kappa)C]\alpha\theta + [\sigma_1\psi+\sigma_2 C]B)}{A\,B\,C\,D}\right]. \tag{4.1}$$

5. Steady State Analysis

This section addresses the concepts pertinent to stability. We will investigate that in the case $E = Q = I_1 = I_2 = R = 0$, i.e when there is no infection, the model will have a steady state at the *DFE*, χ_o. It is essential to employ the direct method of Lyapunov alongside the Routh-Hurwitz criteria to formulate sufficient conditions for both global stability and asymptotic stability concerning χ_o. The Routh-Hurwitz criteria serve as a crucial instrument for determining sufficient conditions regarding the roots of the characteristic polynomial. This criteria will be utilized to ascertain the local stability of χ_o.

5.1. Local stability of the DFE

The following theorem lies at the core of our approach which demonstrates that a limited size of infected pepole will not result in widespread outbreaks, and the disease will eventually disappear over time.

Theorem 5.1. *The disease-free equilibrium* χ_o *of* (2.1) *is locally-asymptotically stable if* $\mathcal{R}_0 < 1$*, and unstable otherwise.*

Proof. In order to assess the local stability of the *DFE*, we analyze the Jacobian matrix of the system (2.1) at the DFE point, χ_o. Therefore,

$$J_6(\chi_o) = \begin{bmatrix} -\mu & 0 & \rho & -\beta_1\nu/\mu & -\beta_2\nu/\mu & \tau \\ 0 & -A & 0 & \beta_1\nu/\mu & \beta_2\nu/\mu & 0 \\ 0 & \theta & -B & 0 & 0 & 0 \\ 0 & \sigma_1 & \kappa\alpha & -C & 0 & 0 \\ 0 & \sigma_2 & (1-\kappa)\alpha & \psi & -D & 0 \\ 0 & 0 & 0 & \gamma_1 & \gamma_2 & -(\mu+\tau) \end{bmatrix}.$$

Consequently, the local stability of χ_o is ascertained by the eigenvalues derived from the equations for E, Q, I_1, and I_2, which are represented by the Jacobian matrix

$$J_4(\chi_o) = \begin{bmatrix} -A & 0 & -\beta_1\nu/\mu & -\beta_2\nu/\mu \\ \theta & -B & 0 & 0 \\ \sigma_1 & \kappa\alpha & -C & 0 \\ \sigma_2 & (1-\kappa)\alpha & \psi & -D \end{bmatrix}.$$

The characteristic polynomial of the matrix is then defined by

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + ABCD(1-\mathcal{R}_0) = 0$$

where $a_1 = A+B+C+D$, $a_2 = AB+(A+B)C+(A+B+C)D-(\beta_1\sigma_1+\beta_2\sigma_2)\nu/\mu$, and

$a_3 = AD(B+C)+BC(A+D)-(\nu/\mu)(\beta_1[\sigma_1(D+B)-\theta\kappa\alpha]-\beta_2[\alpha\theta(1-\kappa)-\sigma_1\psi-\sigma_2(B+C)])$.

According to the Routh-Hurwitz criterion, the roots of the characteristic polynomial possess negative real parts, thereby indicating the stability of χ_o, provided that the following hypotheses are satisfied:

$a_1 > 0,\ a_2 > 0,\ a_1a_2 > a_3$,

$a_4 > 0$ if and only if $ABCD(1-\mathcal{R}_0) > 0$ if $\mathcal{R}_0 < 1$,

$a_1a_2a_3 > 0$, and

$a_1a_2a_3 - a_3^2 > a_1ABCD(1-\mathcal{R}_0)$, where $A > 0, B > 0, C > 0, D > 0$. □

5.2. Global stability of the DFE

Now, we show that the system (2.1) has a globally asymptotic stability at χ_o to ensure that the diseases extinction is not influenced by the restricted size of the infected population.

Theorem 5.2. *The disease-free equilibrium χ_o of (2.1) is globally asymptotically stable if $\mathcal{R}_o < 1$.*

Proof. First, we consider a Lyapunov function $V(t) = c_1 E(t) + c_2 Q(t) + c_3 I_1(t) + c_4 I_2(t)$ for any nonnegative real numbers c_i $(i = 1,2,3,4)$. Consequently, we have its time-derivative

$$\begin{aligned}\dot{V}(t) &= c_1 \dot{E}(t) + c_2 \dot{Q}(t) + c_3 \dot{I_1}(t) + c_4 \dot{I_2}(t)\\ &= c_1[\beta_S S - AE] + c_2[\theta E - BQ] + c_3[\kappa\alpha Q + \sigma_1 E - CI_1] + c_4[(1-\kappa)\alpha Q + \sigma_2 E + \psi I_1 - DI_2]\\ &\leqslant c_1[(\nu/\mu)(\beta_1 I_1 + \beta_2 I_2) - AE] + c_2[\theta E - BQ] + c_3[\kappa\alpha Q + \sigma_1 E - CI_1] + c_4[(1-\kappa)\alpha Q + \sigma_2 E\\ &\quad + \psi I_1 - DI_2].\end{aligned}$$

We will now choose the coefficients c_1, c_2, c_3 and c_4, setting the coefficients of E, I_1 and I_2 to zero. Then, we conclude the following

$$\begin{aligned}&c_1 = \nu/\mu, \qquad c_2 = (\nu/\mu\theta)\,[A - (\sigma_1\nu/\mu C)\,[\beta_1 + \beta_2(\psi/D)] - (\sigma_2\nu\beta_2/\mu D)]\,,\\ &c_3 = (1/C)\,[\beta_1 + \beta_2(\psi/D)]\,(\nu/\mu)^2\,, \quad c_4 = (\beta_2/D)\,(\nu/\mu)^2\,.\end{aligned}$$

By substituting the values of c_1, c_2, c_3 and c_4 into $V(t)$, the derivative of $V(t)$ can be represented as follows

$$\dot{V}(t) \leqslant (\nu/\mu)(A\,B/\theta)\,[\mathcal{R}_o - 1]\,Q.$$

Clearly, $\dot{V}(t) \leqslant 0$ when $\mathcal{R}_o < 1$. Furthermore

$$\dot{V}(t) = 0 \quad \text{if and only if} \quad E = Q = I_1 = I_2 = 0$$

which complets the proof. □

In the stability analysis of the *DFE*, we will illustrate the occurrence of backward bifurcation in the next section.

6. Bifurcation Analysis

This section analyzes the properties of the equilibrium solutions of the disease transmission model near the bifurcation point χ, particularly when $\mathcal{R}_o$ is equal to 1 in a neighborhood of the disease-free equilibrium (*DFE*), χ_o. To enhance clarity in our notation, we define a parameter β as $\mathcal{R}_o - 1$ and reformulate the system (2.1) for all β as a general form of ODE's in $\mathcal{R}^6 \times \mathcal{R}$:

$$\dot{\chi}(t) = \mathbf{f}(\beta, \chi(t)) \quad \text{under prescribed } \chi(0) = \chi_o, \tag{6.1}$$

where the vector field $\mathbf{f} : \mathcal{R}^6 \times \mathcal{R} \to \mathcal{R}^6$ is continuously differentiable at least twice with respect to both of β and χ [5]. In order to do this, let us begin by changing our standard

notations of the system (2.1) in the following simple variables: $\chi_1 = S, \chi_2 = E, \chi_3 = Q, \chi_4 = I_1, \chi_5 = I_2$, and $\chi_6 = I_2$, so that $\chi_1 + \chi_2 + \chi_3 + \chi_4 + \chi_5 + \chi_6 = N$. Additionally, employing the vector notation $\boldsymbol{\chi} = (\chi_1, \chi_2, \chi_3, \chi_4, \chi_5, \chi_6)^t$ allows the system (2.1) to be expressed in the form (6.1) with $(f_1, f_2, f_3, f_4, f_5, f_6)^t$ as follows

$$\begin{aligned}
\dot{\chi}_1 &= \nu - \mu\chi_1 - \beta_1\chi_1\chi_4 - \beta_2\chi_1\chi_5 + \rho\chi_3 + \tau\chi_6 := f_1,\\
\dot{\chi}_2 &= \beta_1\chi_1\chi_4 - \beta_2\chi_1\chi_5 - (\mu + \sigma + \theta)\chi_2 := f_2,\\
\dot{\chi}_3 &= \theta\chi_2 - (\mu + \rho + \alpha)\chi_3 := f_3,\\
\dot{\chi}_4 &= \kappa\alpha\chi_3 - \sigma_1\chi_2 - (\mu + \gamma_1 + \psi + \delta_1)\chi_4 := f_4,\\
\dot{\chi}_5 &= (1-\kappa)\alpha\chi_3 + \sigma_2\chi_2 + \psi\chi_4 - (\mu + \gamma_2 + \delta_2)\chi_5 := f_5,\\
\dot{\chi}_6 &= \gamma_1\chi_4 + \gamma_2\chi_5 - (\mu + \tau)\chi_6 := f_6.
\end{aligned} \tag{6.2}$$

We apply Theorem 4.1 presented in [5] to demonstrate that the system (6.2) has the potential to display a backward bifurcation when $\mathcal{R}_o$ equals 1. First, we treat the parameter β, specifically $\beta = \beta_1 = \beta_2$ as the bifurcation parameter. When $\beta^* = \beta$ in (4.1) and setting $\mathcal{R}_o$ to unity, we can deduce the new corresponding parameter

$$\beta^* = \left(\frac{\nu}{\mu}\right)\left[\frac{A\,B\,C\,D}{(D+\psi)(\kappa\,\alpha\,\theta + \sigma_1\,B) + [(1-\kappa)\,\alpha\,\theta + \sigma_2\,B]\;C}\right]. \tag{6.3}$$

Then, the Jacobian matrix of system (6.2) around the disease-free equilibrium χ_o is given by

$$J(\beta^*, \chi_o) = \begin{bmatrix}
-\mu & 0 & \rho & -\beta^*\nu/\mu & -\beta^*\nu/\mu & \tau\\
0 & -A & 0 & \beta^*\nu/\mu & \beta^*\nu/\mu & 0\\
0 & \theta & -B & 0 & 0 & 0\\
0 & \sigma_1 & \kappa\alpha & -C & 0 & 0\\
0 & \sigma_2 & (1-\kappa)\alpha & \psi & -D & 0\\
0 & 0 & 0 & \gamma_1 & \gamma_2 & -(\mu+\tau)
\end{bmatrix}.$$

Therefore, one can readily check that the eigenvalues of $J(\beta^*, \chi_o)$ are given by $\lambda_1 = -\mu, \lambda_2 = 0, \lambda_3 = -(\mu + \tau)$, and by Routh- Hurwitz criterion, the other eigenvalues of $J(\beta^*, \chi_o)$ are negative. Thus, we can use the *center manifold theory* [5] to discuss the dynamics of system (6.2) when $\mathcal{R}_o = 1$. Hence, it follows that the disease-free equilibrium χ_o is a nonhyperbolic equilibrium.

Now, we evaluate a right eigenvector $\boldsymbol{w} = (w_1, w_2, w_3, w_4, w_5, w_6)^t$ of the matrix $J(\beta^*, \chi_o)$ associated with the zero eigenvalue $\lambda_2 = 0$. It can be obtained by

$$\begin{aligned}
&-\mu\,w_1 + \rho\,w_3 - (\beta^*\nu/\mu)\;w_4 - (\beta^*\nu/\mu)\;w_5 + \tau\,w_6 = 0,\\
&-A\,w_2 + (\beta^*\nu/\mu)\;w_4 + (\beta^*\nu/\mu)\;w_5 = 0,\\
&\theta\,w_2 - B\,w_3 = 0,\\
&\sigma_1\,w_2 + \kappa\,\alpha\,w_3 - C\,w_4 = 0,\\
&\sigma_2\,w_2 + (1-\kappa)\,\alpha\,w_3 + \psi\,w_4 - C\,w_5 = 0,\\
&\gamma_1\,w_4 + \gamma_2\,w_5 - (\mu+\tau)\,w_6 = 0.
\end{aligned}$$

So, the components of right eigenvector are $w_1 = (1/\mu)(\rho w_3 - (\beta^*\nu/\mu)[w_4 + w_5] + \tau w_6)$, $w_2 = B/\theta$, $w_3 = 1$, $w_4 = (1/C)[(\sigma_1 B/\theta) + \kappa\alpha]$, $w_5 = (1/C)[(\sigma_2 B/\theta) + (1-\kappa)\alpha +$

$(\psi/C)([\sigma_1 B/\theta] + \kappa\alpha)]$, and $w_6 = (1/C)([\gamma_1 + \gamma_2(\psi/C)][(\sigma_1 B/\theta) + \kappa\alpha] + \gamma_2[(\sigma_1 B/\theta) + (1-\kappa)\alpha])/(\mu+\tau)$. Moreover, the left eigenvector of the matrix $J(\beta^*, \chi_o)$, denoted by $\boldsymbol{\nu} = (\nu_1, \nu_2, \nu_3, \nu_4, \nu_5, \nu_6)^t$, which satisfies $\boldsymbol{\nu} \cdot \boldsymbol{w} = 1$ is given by

$$
\begin{aligned}
&-\mu\nu_1 = 0,\\
&-A\nu_2 + \theta\nu_3 + \sigma_1\nu_4 + \sigma_2\nu_5 = 0,\\
&\rho\nu_1 - B\nu_3 + \kappa\alpha\nu_4 + (1-\kappa)\alpha\nu_5 = 0,\\
&-(\beta^*\nu/\mu)\,\nu_1 + (\beta^*\nu/\mu)\,\nu_2 - C\nu_4 + \psi\nu_5 + \gamma_1\nu_6 = 0,\\
&-(\beta^*\nu/\mu)\,\nu_1 + (\beta^*\nu/\mu)\,\nu_2 - C\nu_5 + \gamma_2\nu_6 = 0,\\
&\tau\nu_1 - (\mu+\tau)\nu_6 = 0.
\end{aligned}
$$

So, the components of left eigenvector are $\nu_1 = 0$, $\nu_2 = (\theta\nu_3 + \sigma_1\nu_4 + \sigma_2\nu_5)/A$, $\nu_3 = (\alpha/B\,C)[(\kappa\psi/C) + 1](\beta^*\nu/\mu)\nu_2$, $\nu_4 = (1/C)[1+\psi](\beta^*\nu/\mu)\nu_2$, $\nu_5 = (1/C)(\beta^*\nu/\mu)\nu_2$ and $\nu_6 = 0$. Now, we compute all the partial derivatives of f_i with respect to χ_i $(i = 1, \cdots, 6)$ and β^* at the disease-free equilibrium, χ_o, to have

$$
\begin{aligned}
&\partial^2_{\chi_1\chi_4} f_1 = \partial^2_{\chi_4\chi_1} f_1 = \partial^2_{\chi_1\chi_5} f_1 = \partial^2_{\chi_5\chi_1} f_1 = -\beta^*,\\
&\partial^2_{\chi_1\chi_4} f_2 = \partial^2_{\chi_4\chi_1} f_1 = \partial^2_{\chi_1\chi_5} f_2 = \partial^2_{\chi_5\chi_1} f_2 = \beta^*,\\
&\partial^2_{\chi_4\beta^*} f_1 = \partial^2_{\chi_5\beta^*} f_1 = -\chi_1 = -\nu/\mu,\\
&\partial^2_{\chi_4\beta^*} f_2 = \partial^2_{\chi_5\beta^*} f_2 = \chi_1 = \nu/\mu,
\end{aligned}
$$

where all other second-order partial derivatives are equal to zero. Consequently, we are able to determine the coefficients $\mathbf{a}$ and $\mathbf{b}$ as outlined in Theorem 4.1 [5], i.e.

$$
\mathbf{a} = \sum_{k,i,j=1}^{6} \nu_k\, w_i\, w_j\, \partial^2_{\chi_i\chi_j} f_k(\beta^*, \chi_o) \quad \text{and} \quad \mathbf{b} = \sum_{k,i=1}^{6} \nu_k\, w_i\, \partial^2_{\chi_i\beta} f_k(\beta^*, \chi_o).
$$

Considering the system (6.2) and concentrating exclusively on the nonzero derivatives of the terms $\partial^2_{\chi_i\chi_j} f_k(\beta^*, \chi_o)$ and $\partial^2_{\chi_i\beta} f_k(\beta^*, \chi_o)$, one can deduce that

$$
\mathbf{a} = 2\nu_2 w_1 w_4\, \partial^2_{\chi_1\chi_4} f_2(\beta^*, \chi_o) + 2\nu_2 w_1 w_5\, \partial^2_{\chi_1\chi_5} f_2(\beta^*, \chi_o) = 2\nu_2 w_1 \beta^* (w_4 + w_5),
$$

and

$$
\mathbf{b} = \nu_2 w_4\, \partial^2_{\chi_4\beta} f_2(\beta^*, \chi_o) + \nu_2 w_5\, \partial^2_{\chi_5\beta} f_2(\beta^*, \chi_o) = \nu_2\,(\nu/\mu)\,(w_4 + w_5).
$$

Clearly, the coefficients $\mathbf{a}$ and $\mathbf{b}$ are always positive since ν_2 can be chosen to be positive. According to Theorem 4.1 of [5], one can conclude that, when $\mathbf{a} > 0$ and $\mathbf{b} > 0$, the bifurcation at $\mathcal{R}_o = 1$ is subcritical (backward), thus we have backward bifurcation when $\beta_1 = \beta_2 = \beta^*$.

Theorem 6.1. *Assume that the basic reproduction number $\mathcal{R}_o$ given by (4.1) equals to unity. If the signs of the parameters $\mathbf{a}$ and $\mathbf{b}$ are both positive, then the system (2.1) admits a backward bifurcation phenomena.*

7. Conclusion

In this study, we clarify the mechanisms of *Covid*-19 transmission through the examination of an epidemic model. We have established the existence of a disease-free equilibrium and demonstrated its asymptotic local and global stability for basic reproduction numbers $\mathcal{R}_o < 1$. We also examined the prerequisites for reaching an endemic equilibrium, or a consistent degree of infection throughout the community. The occurrence of a backward bifurcation has been observed, identified as a potential phenomenon resulting from the clinical transition from an asymptomatic state to one that presents symptoms. Adding more complicated variables to the model and assessing its efficacy could be the main goals of future research.

Acknowledments

The authors are grateful for invaluable contributions from collaborators to the successful completion of this work. The first author wishes to express her gratitude to Bothaina Bukhatwa for some stimulating discussions that led to the inception of this project. Last but not least, we thank the anonymous referee for indicating ways to clarify the presentation.

Conflicts of Interest

No conflict of interest to disclose.

Funding

The authors did not receive any financial support for this study.

References

[1] Abdo, M. Shah , K. Wahash, H. & Panchal, S. (2020). On a comprehensive model of the novel coronavirus (COVID-19) under Mittag-Leffler derivative. *Chaos, Solitons & Fractals* 135, 109867. https://doi.org/10.1016/j.chaos.2020.109867

[2] Adebiyi, Ayodeji. O. (2016). Mathematical modeling of the population dynamics of tuberculosis. *A Master dissertation, University of the Western Cape, South Africa*.

[3] Ali, M., Shah, Syed Touqeer H., Imran, M. & Khan, A. (2020). The role of asymptomatic class, quarantine and isolation in the transmission of COVID-19. *Journal of biological dynamics*, 14(1), 389–408.

[4] Almalahi, M., Panchal, S., Shatanawi, W., Abdo, M., Shah, K. & Abodayeh, K. (2021). Analytical study of transmission dynamics of 2019-nCoV pandemic via fractal fractional operator. *Results in physics*, 24(2), 104045. https://doi.org/1010.1016/j.rinp.2021.104045

[5] Castillo-Chavez, C. & Song, B. (2004). Dynamical models of tubercolosis and their applications. *Mathematical Biosciences & Engineering*, 1(2), 361–404.

[6] Cherniha, R. & Davydovych, V. (2020). A mathematical model for the COVID-19 outbreak. *Symmetry*, 12(6), 990.

[7] Das, P., Nadim, S., Das, S., & Das, P. (2021). Dynamics of COVID-19 transmission with co-morbidity: a data driven modelling based approach. *Nonlinear Dynamics*, 106, 1197–1211. https://doi.org/10.1007/s11071-021-06324-3

[8] Eiman, Shah, K., Sarwar, M. & Abdeljawad, T. (2024). On rotavirus infectious disease model using piecewise modified ABC fractional order derivative. *Networks & Heterogeneous Media*, 19(1), 214–234. https://doi.org/10.3934/nhm.2024010

[9] Harapan, H. , Itoh, N., Yufika, A., Winardi, W., Keam, S., Te, H., Megawati, D., Hayati, Z. , Wanger, A. L. & Mudatsir, M. (2020). Coronavirus disease 2019 (COVID-19): A Literature review. *Journal of infection and public health*, 13(5), 667–673.

[10] Hethcote, H., Zhien, M., & Shengbing, L. (2002). Effects of quarantine in six endemic models for infectious diseases. *Mathematical biosciences*, 180(1–2), 141–160.

[11] Iannelli, Mimmo. (2005). The Mathematical Modelling of Epidemics. Lecture Notes on Mathematical Modelling in the Life Sciences: Theory and simulation *Unitext-La Matematica per il 3 piu 2*.

[12] Jeelani, M., Alnahdi, A., Abdo, M., Abdulwasaa, M., Shah, K. & Wahash, H. (2021). Mathematical modeling and forecasting of COVID-19 in Saudi Arabia under fractal-fractional derivative in caputo sense with power-law. *Axioms* 10(3), 228. https://doi.org/10.3390/axioms10030228

[13] Kassa, S., Njagarah, J., & Terefe, Y. (2020). Analysis of the mitigation strategies for COVID-19: From mathematical modelling perspective. *Chaos, Solitons, and Fractals*, 138, 109968. https://doi.org/10.1016/j.chaos.2020.109968

[14] Kotola, B., Teklu, S., & Abebaw, Y. (2023). Bifurcation and optimal control analysis of HIV/AIDS and COVID-19 co-infection model with numerical simulation. *PLOS ONE*, 18(5), e0284759. https://doi.org/10.1371/journal.pone.0284759

[15] Liu, Y., Kuo, R., & Shih, S. (2020). COVID-19: The first documented coronavirus pandemic in history. *Biomedical Journal*, 43(4), 328–333. https://doi.org/10.1016/j.bj.2020.04.007

[16] Luo, X., Feng, S., Yang, J., Peng, X. L., Cao, X., Zhang, J., Yao, M., Zhu, H., Li, M. Y., Wang, H. & al et. (2020). Analysis of potential risk of COVID-19 infections in China based on a pairwise epidemic model. *Preprints, Computer Science & Mathematics*, 2020020398. https://doi.org/10.20944/preprints202002.0398.v1

[17] Nadim, S., & Chattopadhyay, J. (2020). Occurrence of backward bifurcation and prediction of disease transmission with imperfect lockdown: A case study on COVID-19. *Chaos, Solitons, and Fractals*, 140, 110163. https://doi.org/10.1016/j.chaos.2020.110163

[18] NDAM, J.N. (2021). Mathematical modelling of the dynamics of COVID-19 pandemic. *Asia-Pacific Journal of Mathematics*, 8(12), 1–17.

[19] Paul, A., Bhattacharjee, J., Pal, A., & Chakraborty, S. (2021). Emergence of universality in the transmission dynamics of COVID-19. *Scientific Reports*, 11(1), 18891. https://doi.org/10.1101/2021.01.29.21250750

[20] Shah, K., Khana, A., Abdallaa, B., Khand,K. & Abdeljawad, T. (2024). A mathematical model for Nipah virus disease by using piecewise fractional order Caputo derivative. *Fractals*, 32(02), 2440013. https://doi.org/10.1142/S0218348X24400139

[21] Song, B. J. (2021). Basic reinfection number and backward bifurcation. *Mathematical Biosciences & Engineering*, 18(6), 8061–8083.

[22] Teklu, S. (2022). Mathematical analysis of the transmission dynamics of COVID-19 infection in the presence of intervention strategies. *Journal of Biological Dynamics*, 16(1), 640–664. https://doi.org/10.1080/17513758.2022.2111469

[23] Van den Driessche, P. (2017). Reproduction numbers of infection disease models. *Infectious disease modelling*, 2(3), 288–303.

[24] World Health Organisation. (2020). Coronavirus Disease 2019 (COVID-19) Situation Report-59, 19 March, 2020.

[25] Zhou, Xueyong & Shi, Xiangyun. (2022). Stability analysis and backward bifurcation on an SEIQR epidemic model with nonlinear innate immunity. *Electronic Research Archive*, 30(9), 3481–3508.