Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2023, 2023:5
https://doi.org/10.28919/cmbn/7822
ISSN: 2052-2541

A SEIQRD EPIDEMIC MODEL TO STUDY THE DYNAMICS OF COVID-19 DISEASE

ISNANI DARTI[1], TRISILOWATI[1], MAYA RAYUNGSARI[1,2], RAQQASYI RAHMATULLAH MUSAFIR[1], AGUS SURYANTO[1,*]

[1]Department of Mathematics, Faculty of Mathematics and Natural Sciences, University of Brawijaya, Jl. Veteran Malang 65145, Indonesia

[2]Department of Mathematics Education, PGRI Wiranegara University, Pasuruan, Indonesia

Abstract. In this paper, we propose a COVID-19 epidemic model with quarantine class. The model contains 6 sub-populations, namely the susceptible (S), exposed (E), infected (I), quarantined (Q), recovered (R), and death (D) sub-populations. For the proposed model, we show the existence, uniqueness, non-negativity, and boundedness of solution. We obtain two equilibrium points, namely the disease-free equilibrium (DFE) point and the endemic equilibrium (EE) point. Applying the next generation matrix, we get the basic reproduction number (R_0). It is found that R_0 is inversely proportional to the quarantine rate as well as to the recovery rate of infected sub-population. The DFE point always exists and if $R_0 < 1$ then the DFE point is asymptotically stable, both locally and globally. On the other hand, if $R_0 > 1$ then there exists an EE point, which is globally asymptotically stable. Here, there occurs a forward bifurcation driven by R_0. The dynamical properties of the proposed model have been verified our numerical simulations.

Keywords: coronavirus disease 2019; dynamical analysis; SEIQRD epidemic model; local and global stability; forward bifurcation.

2020 AMS Subject Classification: 37N25, 92B05, 92D30.

*Corresponding author

E-mail address: suryanto@ub.ac.id

Received November 14, 2022

1. INTRODUCTION

Coronavirus Disease-2019 or COVID-19, which was first found in Wuhan, China in December 2019, is an infectious disease caused by a newly discovered coronavirus. COVID-19 virus is primarily transmitted through droplets produced when an infected person coughs, sneezes, or exhales. Fever, coughing, and breathing problems are the earliest symptoms of this disease. In the following stages, the infection can cause pneumonia, serious acute respiratory syndrome, kidney damage, and even death [1]. COVID-19 is currently receiving great attention among researchers, governments and the general public due to its high rate of spread of infection and significant number of death [2]. As a result, many researchers in the Mathematics field are investigating and developing mathematical models of the spread of COVID-19 disease [3–5], as well as forecasting the time required for the disease to be eradicated [6–8].

When modelling the spread of an epidemic, population is divided into several fundamental disease states. One of simple epidemic models is the susceptible-infected-recovered (SIR) model, which consists of the susceptible class, the infected class, and the recovered class [9]. Individuals in susceptible state are at risk of getting the disease. Infected individuals are contagious and may spread the disease to others through contact with susceptible individuals. Recovered state consists of individuals who have immunity from a vaccine or acquired immunity and died people. Several investigating of COVID-19 SIR model are studied in [10–12]. The models are in the form of system of three nonlinear ordinary differential equations (ODEs).

It is well known that COVID-19 has latency or incubation period. Hence, some researchers have considered SEIR COVID-19 epidemic model by including an exposed (E) class, those who have been infected but have not exhibited any disease symptoms and cannot transmit the disease [13–16]. By taking into account the further characteristics of COVID-19, Peng et al. [18] extended the SEIR model by involving class of protected inviduals, quarantined individuals and closed (or death due to COVID-19 disease). In this case, Peng et al. [18] divides the population into 7 sub-population classes, namely Susceptible (S), Insusceptible, i.e. susceptible but protected because they always apply health protocols in an orderly manner (P), exposed (infected but not yet be infectious) (E), infected population (I), infected and quarantined privately

or in hospital (Q), recovered (R) and closed cases (or dead) from COVID-19 (D). The quarantined class can't spread the disease to others because they have lost contact with susceptible people [17]. It is expected that the COVID-19 outbreak will be controlled by the presence of a quarantined sub-population [5]. Peng et al. [18] obtained a SEIQRD epidemic model. The SEIQRD model in [18] does not consider demographic parameters such as birth and death rates. Therefore López and Rodó [19] modified the model (2.6) by including those two demographic parameters. However, they ignored the deaths of infected individuals caused by the COVID-19 disease and assumed that the infected individuals cannot recover from the disease unless they quarantine first. Zeb et al. [2] have also proposed a COVID-19 epidemic model which includes S, E, I, Q and R classes, but without considering the closed class (D). Zeb et al. [2] assumes that disease transmission occurs through contact between susceptible individuals and exposed individuals or infected individuals where the rate of transmission follows the bilinear incidence rate.

According to Postavaru et al. [20], the exposed individuals are infected individuals but not yet infective. Thus, in this article, we propose a SEIQRD COVID-19 epidemic model by assuming that COVID-19 transmission occurs only when there is contact between susceptible individuals and infected individuals, with the transmission rate being the standard incident rate. We also consider deaths due to COVID-19 disease and recovery in both infected and quarantined sub-populations into the model, which are not considered by Zeb et al [2] nor by López and Rodó [19].

The outline of this study is given as follows. In Section 2, we present the SEIQRD model with some assumptions. We provide the basic properties of proposed model in Section 3 and 4. In Section 5 and 6, we study the equilibrium points and basic reproduction number. The stability of equilibrium points, both locally and globally, are provided in Section 7. The numerical simulation was carried out in Section 8. Finally, some conclusion will be given in Section 9.

2. Model Development

Based on the assumptions described previously, we formulate a SEIQRD COVID-19 model by considering the model of Zeb et al. [2] and the model of López and Rodó [19], but without

including the confined (C) compartment. The compartment diagram of the proposed model can be seen in Figure 1.

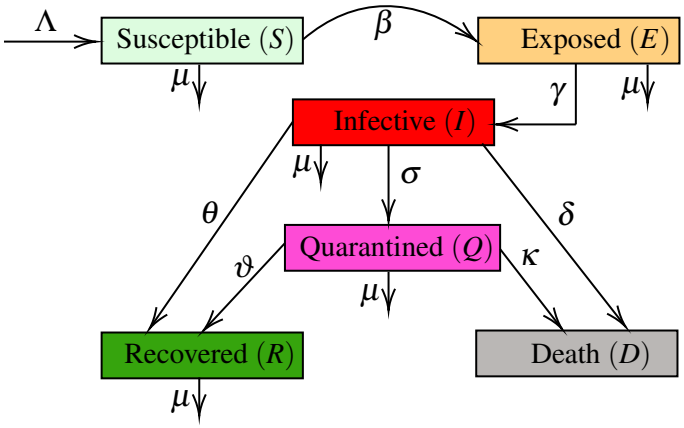

FIGURE 1. Compartment diagram.

Based on the diagram, the developed model is a system of

$$
\begin{aligned}
\frac{dS(t)}{dt} &= \Lambda - \frac{\beta S(t)I(t)}{N(t)} - \mu S(t),\\
\frac{dE(t)}{dt} &= \frac{\beta S(t)I(t)}{N(t)} - (\gamma+\mu)E(t),\\
\frac{dI(t)}{dt} &= \gamma E(t) - (\sigma+\theta+\delta+\mu)I(t),\\
\frac{dQ(t)}{dt} &= \sigma I(t) - (\vartheta+\kappa+\mu)Q(t),\\
\frac{dR(t)}{dt} &= \theta I(t) + \vartheta Q(t) - \mu R(t),\\
\frac{dD(t)}{dt} &= \delta I(t) + \kappa Q(t),
\end{aligned}
\tag{1}
$$

where S represents the size of the susceptible sub-population, i.e. all people who are at risk of contracting COVID-19; E represents the size of the exposed sub-population, i.e. people who have been infected and have not shown symptoms of disease, I represents the size of the infected sub-population, i.e. people who have shown symptoms of the disease, Q represents the size of the quarantined sub-population, i.e. people who are infected and self-isolated or hospitalized for treatment, R represents the size of the sub-population who have fully recovered from COVID-19, and D represents the size of death sub-population caused by COVID-19. The parameters of system (1) are described in the Table 1.

For dynamical analysis, the last equation in the model (1) is not considered because other equations do not involve variable D in the model. Hence, we only consider first five equations in model (1).

TABLE 1. Description of Parameters

Parameter	Description
Λ	recruitment rate
β	infection rate
μ	natural death rate
γ	incubation rate
σ	quarantine rate
θ	recovery rate of I
δ	death rate of I induced by the disease
ϑ	recovery rate of Q
κ	death rate of Q induced by the disease

3. THE NON-NEGATIVITY AND BOUNDEDNESS OF SOLUTION

In this section, we prove the non-negativity and boundedness of solution of model (1) to show that the model is epidemiologically meaningful.

Theorem 1. *All solutions of model (1) subject to non-negative initial values are non-negative and ultimately bounded.*

Proof. We first show the non-negativity of S. By assuming that this not the case, the intermediate value theorem in [21] guarantees the existence of $\tau > 0$ such that

$$S(\tau^-) \geq 0,\ S(\tau) = 0,\ S(\tau^+) < 0.$$

From model (1) we have

$$\left.\frac{dS}{dt}\right|_{t=\tau} = \Lambda > 0.$$

This means that $S > 0$ in $(\tau, \tau+\varepsilon)$ for arbitrary small positive constant ε. This leads to a contradiction. Hence, $S \geq 0$ for all $t > 0$. The non-negativity of E, I, Q, and R can be proven analogously. Therefore, all solutions of model (1) are non-negative.

Total population is generally defined as the number of living humans, so that the total population $(N(t))$ is obtained by adding up all sub-populations in the model (1) except the sub-population D, i.e. $N(t) = S(t) + E(t) + I(t) + Q(t) + R(t)$. If all equations in the system (1) are summed up, then the following differential equation is obtained.

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - \delta I(t) - \kappa Q(t) \leq \Lambda - \mu N(t). \tag{2}$$

The solution of the equation (2) satisfies

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) \exp(-\mu t),$$

where $N(0)$ is the initial value. It is clear that

$$\lim_{t \to \infty} N(t) \leq 0,$$

and thus $N(t)$ is bounded with $N(t) \leq \frac{\Lambda}{\mu}$. Hence, we can see that the feasible region of model (1) is

$$\Omega = \left\{ (S,E,I,Q,R) \in \mathbb{R}^5_+ \cup \vec{0} \ : \ N = S+E+I+Q+R \leq \frac{\Lambda}{\mu} \right\},$$

which is positively invariant region. □

4. THE EXISTENCE AND UNIQUENESS OF SOLUTION

The existence and uniqueness of solution of model (1) can be proven by Derrick and Groosman theorem in [22], which states that if Lipchitz's condition as in Definition 1 is satisfied, then the solution of model (1) exists and is unique.

Definition 1. [23] $\vec{f}$ in system (1) satisfies Lipchitz's condition in $\Omega \subset \mathbb{R}^5$ if there is a positive constant k such as

$$||\vec{f}(\vec{X_1}) - \vec{f}(\vec{X_2})|| < k||\vec{X_1} - \vec{X_2}||, \forall \vec{X_1}, \vec{X_2} \in \Omega.$$

The following theorem guarantee the existence and uniqueness of solution of model (1).

Theorem 2. *The model (1) subject to non-negative initial values has a unique solution in* Ω *for all* $t \geq 0$.

Proof. The right side of model (1) can be written as follows.

$$\begin{aligned} f_1 &= \Lambda - \frac{\beta S(t)I(t)}{N(t)} - \mu S(t), \\ f_2 &= \frac{\beta S(t)I(t)}{N(t)} - (\gamma+\mu)E(t), \\ f_3 &= \gamma E(t) - (\sigma+\theta+\delta+\mu)I(t), \\ f_4 &= \sigma I(t) - (\vartheta+\kappa+\mu)Q(t), \\ f_5 &= \theta I(t) + \vartheta Q(t) - \mu R(t), \\ f_6 &= \delta I(t) + \kappa Q(t), \end{aligned}$$

Suppose that $x_1 = S$, $x_2 = E$, $x_3 = I$, $x_4 = Q$, and $x_5 = R$. Then, it can be shown that $\frac{\partial f_i}{\partial x_j}$ is continuous and $\left|\frac{\partial f_i}{\partial x_j}\right| < \infty$ for all $i, j = 1, 2, .., 6$. Based on Derrick and Groosman theorem in [22], system (1) satisfies Lipchitz's condition, meaning that the model (1) has a unique solution.

□

5. EQUILIBRIUM POINTS

Let $X = (S, E, I, Q, R)^T$. By setting

$$\frac{dX(t)}{dt} = \vec{0}, \tag{3}$$

we get $E = \frac{(\sigma+\theta+\delta+\mu)}{\gamma}I$, $Q = \frac{\sigma}{(\vartheta+\kappa+\mu)}I$, $R = \frac{\theta I+\vartheta Q}{\mu}$, and $N = \frac{\Lambda-\delta I-\kappa Q}{\mu} = \frac{\Lambda-\left(\delta+\frac{\kappa\sigma}{\theta+\kappa+\mu}\right)I}{\mu}$. Then, by substituting E to the second equation of (3), we obtain

$$\frac{\beta SI}{N} - \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\gamma}I = 0 \implies I = 0 \vee S = \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\beta\gamma}N.$$

For $I = 0$, it is clear that $E = 0, Q = 0, R = 0, N = \frac{\Lambda}{\mu}, S = \frac{\Lambda}{\mu}$. Meanwhile, for $S = \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\beta\gamma}N$, based on the first equation of (3), we get

$$\begin{aligned} 0 &= \Lambda - \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\gamma}I - \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\Lambda}{\beta\gamma} + \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\left(\delta+\frac{\kappa\sigma}{\theta+\kappa+\mu}\right)I}{\beta\gamma}, \\ I &= \frac{\left[\frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\Lambda}{\beta\gamma}\right]-\Lambda}{\left[\frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\left(\delta+\frac{\kappa\sigma}{\theta+\kappa+\mu}\right)}{\beta\gamma} - \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\gamma}\right]} \\ &= \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\Lambda-\beta\gamma\Lambda}{\left[(\gamma+\mu)(\sigma+\theta+\delta+\mu)\left(\delta+\frac{\kappa\sigma}{\theta+\kappa+\mu}\right)-(\gamma+\mu)(\sigma+\theta+\delta+\mu)\right]}. \end{aligned}$$

As a result, we obtain the endemic equilibrium point $(S^*, E^*, I^*, Q^*, R^*)$ with

$$
\begin{aligned}
I^* &= \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\Lambda-\beta\gamma\Lambda}{\left[(\gamma+\mu)(\sigma+\theta+\delta+\mu)\left(\delta+\frac{\kappa\sigma}{\theta+\kappa+\mu}\right)-(\gamma+\mu)(\sigma+\theta+\delta+\mu)\right]}, \\
S^* &= \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\Lambda}{\beta\gamma\mu} - \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)\left(\delta+\frac{\kappa\sigma}{\theta+\kappa+\mu}\right)I^*}{\beta\gamma\mu}, \\
E^* &= \frac{(\sigma+\theta+\delta+\mu)}{\gamma}I^*, \\
Q^* &= \frac{\sigma}{\vartheta+\kappa\mu}I^*, \\
R^* &= \frac{\theta I^*+\vartheta Q^*}{\mu}.
\end{aligned} \tag{4}
$$

Theorem 3. *The model (1) has two equilibrium points as follows.*

(1) The disease-free equilibrium point $\varepsilon^0 = (S^0, 0, 0, 0, 0)$ *with* $S^0 = \frac{\Lambda}{\mu}$

(2) The endemic equilibrium point $\varepsilon^* = (S^*, E^*, I^*, Q^*, R^*)$ *with* S^*, E^*, I^*, Q^*, R^* *are given by equations (4)*

6. Basic Reproduction Number

One of important epidemiologic metric is the basic reproduction number ($\mathscr{R}_0$), which measures the contagiousness or transmissibility of infectious agents. The basic reproduction number can be determined by the next generation matrix method. For that aim, we consider $Z = (E, I, Q)^T$. Then, we have

$$\frac{dZ}{dt} = \mathscr{F}(Z) - \mathscr{V}(Z),$$

where

$$\mathscr{F}(Z) = \begin{pmatrix} \frac{\beta SI}{N} \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathscr{V}(Z) = \begin{pmatrix} (\gamma+\mu)E \\ -\gamma E + (\sigma+\theta+\delta+\mu)I \\ -\sigma I + (\vartheta+\kappa+\mu)Q \end{pmatrix}.$$

The Jacobian matrices of $\mathscr{F}$ and $\mathscr{V}$ evaluated at ε^0 are respectively given by F and V as follows

$$F = \begin{pmatrix} 0 & \beta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \mu+\gamma & 0 & 0 \\ -\gamma & \sigma+\theta+\delta+\mu & 0 \\ 0 & -\sigma & \vartheta+\kappa+\mu \end{pmatrix}.$$

Hence, the next generation matrix is given by

$$FV^{-1} = \begin{pmatrix} \frac{\beta\gamma}{(\mu+\gamma)(\sigma+\theta+\delta+\mu)} & \frac{\beta}{(\sigma+\theta+\delta+\mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The basic reproduction number ($\mathscr{R}_0$) is a spectral radius of the next generation matrix, that is,

$$\mathscr{R}_0 = \rho(FV^{-1}) = \frac{\beta\gamma}{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}.$$

We observe that $\mathscr{R}_0$ is inversely proportional to the the recovery rate of the infected sub-population (θ) as well as to the quarantine rate σ. The larger the value of θ or σ, the smaller the $\mathscr{R}_0$ value. We also notice that I^* in endemic point can be written in term of $\mathscr{R}_0$ as follows

$$I^* = \frac{\Lambda(1-\mathscr{R}_0)}{\delta + \frac{\kappa\sigma}{\theta+\kappa+\mu} - \beta}.$$

If $\mathscr{R}_0 > 1$, then

$$\beta > \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\gamma} > (\sigma+\theta+\delta+\mu) > \delta+\sigma > \delta+\sigma\frac{\kappa}{\theta+\kappa+\mu}.$$

Thus, the endemic equilibrium point exists if $\mathscr{R}_0 > 1$.

7. THE STABILITY OF EQUILIBRIUM POINTS

In this section, we first provide the local asymptotic stability of disease-free equilibrium point ε^0 as follow.

Theorem 4. *The disease-free point ε^0 is locally asymptotically stable in domain Ω if $\mathscr{R}_0 < 1$.*

Proof. The Jacobian matrix of system (1) evaluated at ε^0 is given by

$$J(\varepsilon^0) = \begin{pmatrix} -\mu & 0 & \beta & 0 & 0 \\ 0 & -(\gamma+\mu) & \beta & 0 & 0 \\ 0 & \gamma & -(\sigma+\theta+\delta+\mu) & 0 & 0 \\ 0 & 0 & \sigma & -(\vartheta+\kappa+\mu) & 0 \\ 0 & 0 & \theta & \vartheta & -\mu \end{pmatrix}.$$

The first three eigenvalues of the Jacobian matrix $J(\varepsilon^0)$ are $\lambda_1 = \lambda_2 = \mu < 0$, $\lambda_3 = -(\vartheta + \kappa + \mu) < 0$, while the other two eigenvalues are the eigenvalues of the following matrix

$$J_R = \begin{pmatrix} -(\gamma+\mu) & \beta \\ \gamma & -(\sigma+\theta+\delta+\mu) \end{pmatrix}.$$

Notice that $Trace(J_R) = -(\gamma+\sigma+\theta+\delta+2\mu) < 0$ and $Det(J_R) = (\gamma+\mu)(\sigma+\theta+\delta+\mu) - \beta\gamma$. It is clear that $Det(J_R) > 0$ if $\mathscr{R}_0 < 1$. Thus, if $\mathscr{R}_0 < 1$, then the real parts of λ_4 and λ_5 are negative. Consequently, the disease-free point ε^0 is locally asymptotically stable if $\mathscr{R}_0 < 1$. □

In the following theorems, we show the global asymptotic stability of the disease-free equilibrium point ε^0 and the endemic equilibrium point ε^*.

Theorem 5. *The disease-free equilibrium point ε^0 is globally asymptotically stable in the domain Ω if $\mathscr{R}_0 < 1$.*

Proof. To prove this theorem, we follow the method of Castilo-Chavez et al. [24]. First, we rewrite the model (1) as

$$\begin{aligned} \frac{dY}{dt} &= F_1(Y,Z) = \begin{pmatrix} \Lambda - \frac{\beta SI}{N} - \mu S \\ \theta I - \vartheta Q - \mu R \end{pmatrix}, \\ \frac{dZ}{dt} &= F_2(Y,Z) = \begin{pmatrix} \frac{\beta S(t)I(t)}{N(t)} - (\gamma+\mu)E(t) \\ \gamma E(t) - (\sigma+\theta+\delta+\mu)I(t) \\ \sigma I(t) - (\vartheta+\kappa+\mu)Q(t) \end{pmatrix}; \; F_2(Y,\vec{0}) = \vec{0}, \end{aligned}$$

where the elements of $Y = (S,R) \in \mathbb{R}^2_+$ describe the number of non-infected individuals while the components of $Z = (E,I,Q) \in \mathbb{R}^3_+$ indicate the number of non-infected individuals. Let $\varepsilon^0 = \left(Y^0, \vec{0}\right)$ with $Y^0 = \left(\frac{\Lambda}{\mu}, 0\right)$. Based on the theorem in [24], the ε^0 is globally asymptotically stable if $\mathscr{R}_0 < 1$ and the following conditions hold:

(**H.1**) Y^0 is globally asymptotically stable for system $\frac{dY}{dt} = F_1(Y,\vec{0})$.

(**H.2**) $F_2(Y,\vec{0}) = \vec{0}$ and $F_2(Y,Z) = CZ - \hat{F}_2(Y,Z)$ where $\hat{F}_2(Y,Z) \geq 0$ for any $(Y,Z) \in \Omega$ and C is the Jacobian matrix $\left(\frac{\partial F_2}{\partial Z}\right)$ evaluated at ε^0.

We notice that

$$C = \begin{pmatrix} -(\gamma+\mu) & -\beta & 0 \\ \gamma & -(\sigma+\theta+\delta+\mu) & 0 \\ 0 & \sigma & -(\varepsilon+\kappa+\mu) \end{pmatrix} \text{ and } \hat{F}_2(Y,Z) = \begin{pmatrix} \frac{\beta I}{N}\left(\frac{\lambda}{\mu} - S\right) \\ 0 \\ 0 \end{pmatrix}.$$

It is clearly seen that the elements of $\hat{F}_2(Y,Z)$ are non-negative, and therefore condition (**H.2**) is satisfied.

We next consider that

$$\frac{dY}{dt} = F_1(Y,\vec{0}) = \begin{pmatrix} \Lambda - \mu S \\ -\mu R \end{pmatrix},$$

from which we get

$$Y(t) = \begin{pmatrix} S(t) \\ R(t) \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} + \left(S(0) - \frac{\Lambda}{\mu}\right)\exp(-\mu t) \\ R(0)\exp(-\mu t) \end{pmatrix}.$$

It is observed that $S(t) \to \frac{\Lambda}{\mu}$ and $R(t) \to 0$ as $t \to \infty$, showing that Y^0 is globally asymptotically stable. Since condition (**H.1**) is also satisfied, the disease-free point ε^0 is globally asymptotically stable in domain Ω. □

Theorem 6. *Assume that $\mathscr{R}_0 > 1$, and thus the endemic equilibrium ε^* exists. Then, the point ε^* is globally asymptotically stable in the domain Ω.*

Proof. Consider a Lyapunov function W, which is defined by

$$W(S,E,I) = S - S^* - S^*\ln\left(\frac{S}{S^*}\right) + E - E^* - E^*\ln\left(\frac{E}{E^*}\right) + \frac{\gamma+\mu}{\gamma}\left(I - I^* - I^*\ln\left(\frac{I}{I^*}\right)\right).$$

The Lyapunov function W is a positive definite function in region Ω. The derivative of W with respect to t is

$$\begin{aligned} \frac{dW}{dt} &= \left(1 - \frac{S}{S^*}\right)\frac{dS}{dt} + \left(1 - \frac{E}{E^*}\right)\frac{dE}{dt} + \frac{\gamma+\mu}{\gamma}\left(1 - \frac{I}{I^*}\right)\frac{dI}{dt} \\ &= \left(1 - \frac{S}{S^*}\right)\left(\Lambda - \frac{\beta S(t)I(t)}{N(t)} - \mu S(t)\right) + \left(1 - \frac{E}{E^*}\right)\left(\frac{\beta S(t)I(t)}{N(t)} - (\gamma+\mu)E(t)\right) \\ &\quad + \frac{\gamma+\mu}{\gamma}\left(1 - \frac{I}{I^*}\right)(\gamma E(t) - (\sigma+\theta+\delta+\mu)I(t)) \end{aligned}$$

$$
\begin{aligned}
&= -\frac{\mu}{S}(S-S^*)^2 + \frac{\beta S^* I^*}{N} - \frac{\beta SI}{N} - \frac{\beta (S^*)^2 I^*}{N} + \frac{\beta S^* I}{N} + \frac{\beta SI}{N} - \frac{\beta SIE^* S^* I^*}{NES^* I^*} - (\gamma+\mu)E \\
&\quad +(\gamma+\mu)E^* + (\gamma+\mu)E - \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\gamma} I - \frac{(\gamma+\mu)I^* EE^*}{IE^*} \\
&\quad + \frac{(\gamma+\mu)(\sigma+\theta+\delta+\mu)}{\gamma} I^* \\
&= -\frac{\mu}{S}(S-S^*)^2 + \frac{\beta S^* I^*}{N}\left(3 - \frac{S^*}{S} - \frac{E^* SI}{ES^* I^*} - \frac{EI^*}{E^* I}\right)
\end{aligned}
$$

Since the geometric mean is less than or equal to the arithmetic mean [25], we have

$$3 \leq \left(\frac{S^*}{S} + \frac{E^* SI}{ES^* I^*} + \frac{EI^*}{E^* I}\right).$$

It is clear that $\frac{dW}{dt} \leq 0$. Furthermore, $\frac{dW}{dt} = 0$ is satisfied if and only if $S = S^*$, $E = E^*$, and $I = I^*$. The LaSalle's Invariance Principle in [25] guarantees that $S \to S^*$, $E \to E^*$, and $I \to I^*$ as $t \to \infty$. Hence, $Q \to Q^*$ and $R \to R^*$ as $t \to \infty$. In other words, ε^* is globally asymptotically stable. □

From discussion above, we know that the disease-free equilibrium ε^0 always exists. If $\mathscr{R}_0 < 1$ then the endemic equilibrium point ε^* does not exist and ε^0 is globally asymtotically stable. However, if $\mathscr{R}_0 > 1$ then ε^* co-exists and is globally asymtotically stable. Hence, based on Martcheva [25], there occurs a forward bifurcation driven by $\mathscr{R}_0$. Because $\mathscr{R}_0$ is directly proportional to the infection rate β, the forward bifurcation may also caused by β. For example, we perform simulation using parameters value as in Table 2 and varying the value of β. Based on this simulation, we plot the bifurcation diagram as shown in Figure 2. It can be seen that the model (1) exhibits a forward bifurcation, where the bifurcation occurs when $\mathscr{R}_0 = 1$ or $\beta = 0.2314$.

8. Numerical Simulations

To confirm our previous analytical results, we solve the SEIQRD epidemic model (1) using the fourth-order Runge-Kutta method. If not stated otherwise, our simulation uses parameter values as in Table 2. First, we take infection rate $\beta = 1.7$ and initial value $S(0) = 9{,}000, E(0) = 45, I(0) = 5, Q(0) = 40, R(0) = 17{,}500, D(0) = 1{,}000$. Using these parameter values, we get $\mathscr{R}_0 = 3.0245 > 1$, and therefore the endemic equilibrium point exists and it

TABLE 2. Parameters values

Parameter	Value	Unit	Source
Λ	3.52	$\frac{\text{individual}}{\text{day}}$	[26]
μ	0.0001	$\frac{1}{\text{day}}$	[26]
γ	0.052	$\frac{1}{\text{day}}$	[26]
σ	0.5	$\frac{1}{\text{day}}$	Assumed
θ	0.041	$\frac{1}{\text{day}}$	[26]
δ	0.2–0.0001	$\frac{1}{\text{day}}$	[26]
ϑ	0.041	$\frac{1}{\text{day}}$	[27]
κ	0.2–0.0001	$\frac{1}{\text{day}}$	Assumed

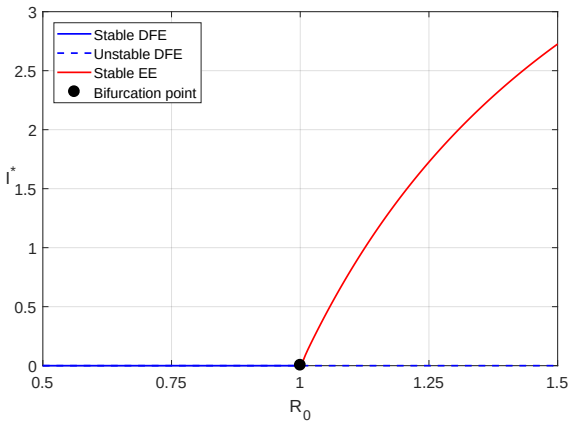

FIGURE 2. Forward bifurcation driven by $\mathscr{R}_0$.

is globally asymptotically stable. This stability properties is confirmed by our simulation depicted in Figure 3, that is the numerical solution is convergent to the endemic equilibrium point $\varepsilon^* = (8795.7718, 50.6799, 4.6976, 38.5049, 17713.0407)$.

For the second numerical simulation, we take a larger value of the quarantine rate, i.e. $\sigma = 2$. The basic reproduction number in this case is $\mathscr{R}_0 = 0.8233 < 1$. Hence, our previous analysis says that the disease-free equilbrium point $\varepsilon^0 = (35200, 0, 0, 0, 0)$ is globally asymtotically stable. The numerical solution using initial value $S(0) = 38,000, E(0) = 2000, I(0) =$

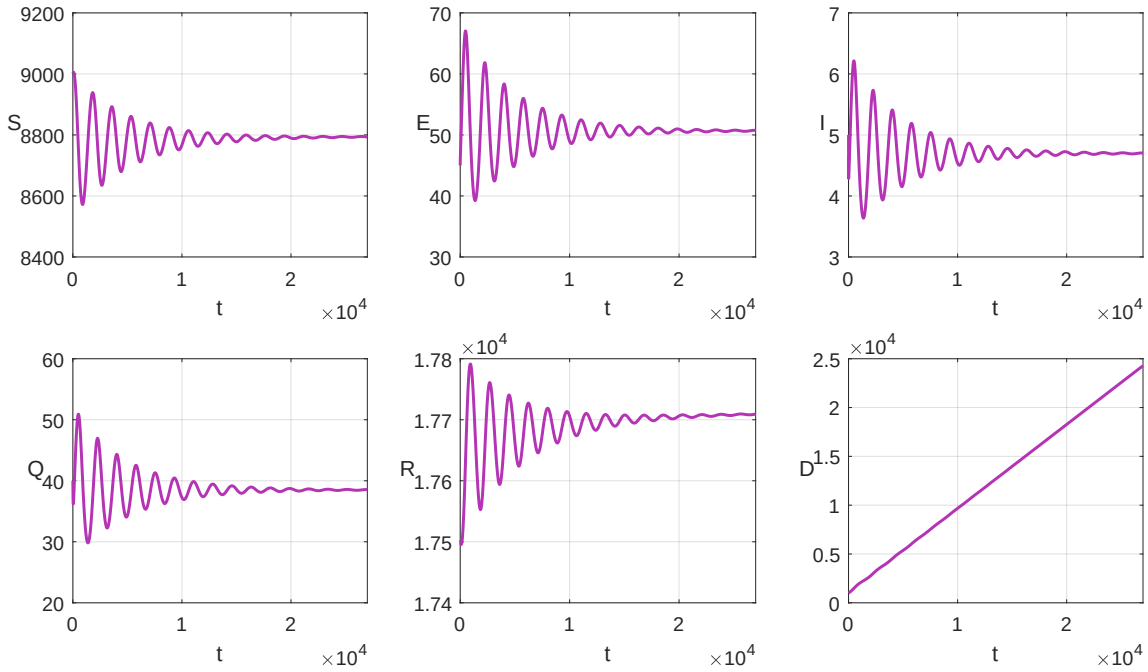

FIGURE 3. Numerical solution of the model (1) with parameters value as in Table 2 and $\beta = 1.7$.

$1000, Q(0) = 3000, R(0) = 50, D(0) = 1,000$ is shown in Figure 4. This picture shows that $\varepsilon^0 = (35200, 0, 0, 0, 0)$ is indeed asymtotically stable.

To see the effect of the recovery rate of the infected class (θ), we perform simulation using parameters value as for the first simulation but with $\theta = 1.2$. The basic reproduction number in this case is $\mathscr{R}_0 = 0.8233 < 1$ and hence we can expect that the disease-free equilibrium point $\varepsilon^0 = (35200, 0, 0, 0, 0)$ is globally asymptotically stable. This is confirmed by our numerical result shown in Figure 5. Here we take initial value $S(0) = 38,000, E(0) = 2000, I(0) = 1000, Q(0) = 3000, R(0) = 50, D(0) = 1,000$.

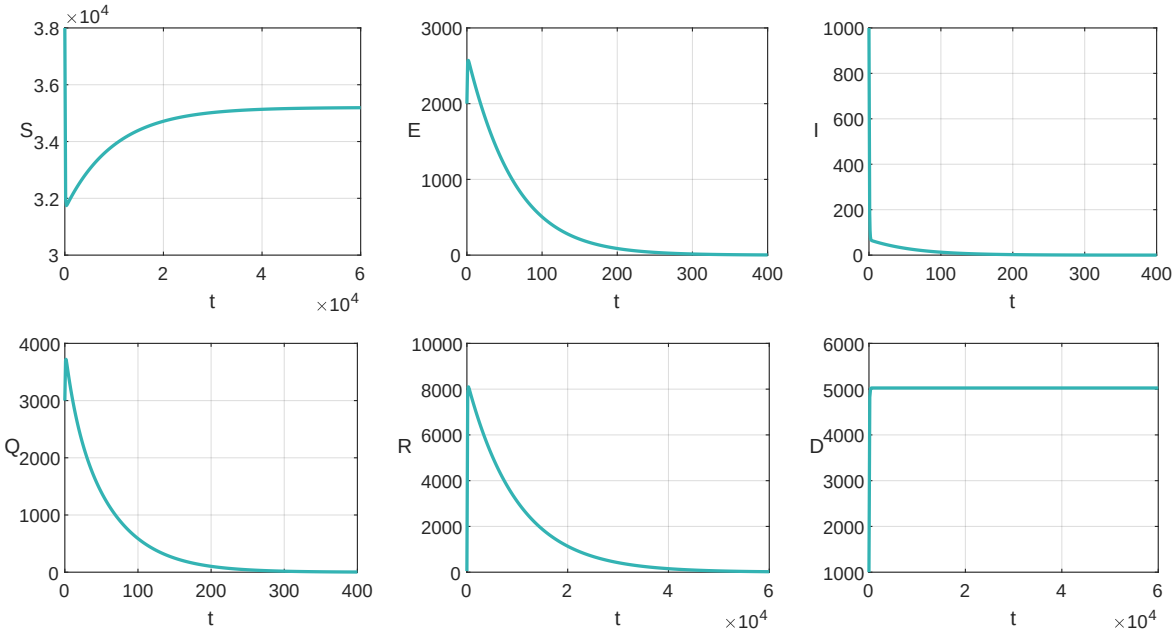

FIGURE 4. Numerical solution of the model (1) with $\beta = 1.7$, $\sigma = 2$, and other parameters value as in Table 2.

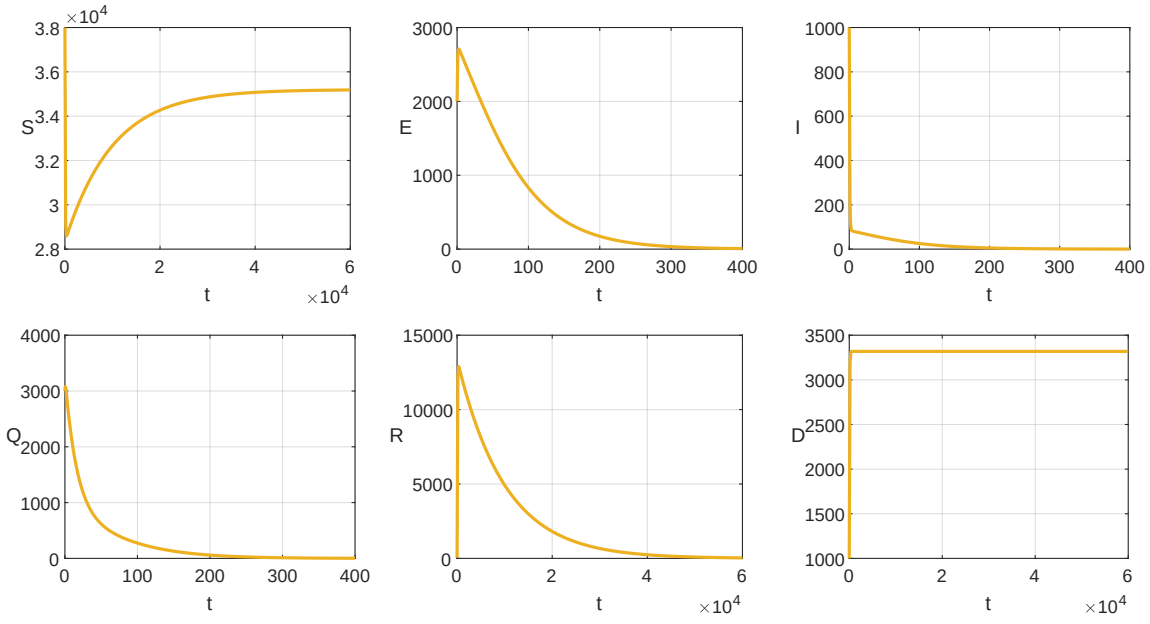

FIGURE 5. Numerical solution of the model (1) with $\beta = 1.7$, $\theta = 1.2$, and other parameters value as in Table 2.

9. CONCLUSIONS

A SEIQRD model to describe the spread of COVID-19 disease using the standard incidence rate has been developed in this paper. The model consists of susceptible (S), exposed (E), infected (I), quarantined (Q), recovered (R), and death caused by the COVID-19 disease(D) sub-populations. The existence, uniqueness, non-negativity, and boundedness of solution have been proven, showing that the proposed model is biologically feasible. The model has two equilibrium points, namely the disease-free equilibrium point and the endemic equilibrium point. Using the next generation matrix method, we have determined the basic reproduction number. The disease-free equilibrium point always exists and it is locally and globally asymptotically stable if the basic reproduction number is less than unity. If the endemic equilibrium point exists, i.e. when the basic reproduction number is greater than unity, then it is always globally asymptotically stable. The proposed model exhibits a forward bifurcation, where the basic reproduction number acts as a bifurcation parameter. The results of our dynamical analysis are confirmed by our numerical simulations. Furthermore, from the basic reproduction number formula and our numerical simulation results, the basic reproduction number can be reduced by increasing the rate of recovery or quarantine of the infected sub-population. This shows that COVID-19 disease can be controlled by treating infected individuals or by quarantining them.

ACKNOWLEDGEMENTS

This research is funded by Faculty of Mathematics and Natural Science (FMIPA) through Public Funds DPA (Dokumen Pelaksanaan Anggaran) Perguruan Tinggi Berbadan Hukum Pelaksanaan Anggaran) Perguruan Tinggi Berbadan Hukum (PTNBH) University of Brawijaya and based on FMIPA Professor Grant Contract No. 3084.19/UN10.F09/PN/2022.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

REFERENCES

[1] S. Rezapour, H. Mohammadi, M.E. Samei, SEIR epidemic model for COVID-19 transmission by Caputo derivative of fractional order, Adv. Differ. Equ. 2020 (2020), 490. https://doi.org/10.1186/s13662-020-02952-y.

[2] A. Zeb, E. Alzahrani, V.S. Erturk, et al. Mathematical model for coronavirus disease 2019 (COVID-19) containing isolation class, BioMed Res. Int. 2020 (2020), 3452402. https://doi.org/10.1155/2020/3452402.

[3] D. Aldila, S.H.A. Khoshnaw, E. Safitri, et al. A mathematical study on the spread of COVID-19 considering social distancing and rapid assessment: The case of Jakarta, Indonesia, Chaos Solitons Fractals. 139 (2020), 110042. https://doi.org/10.1016/j.chaos.2020.110042.

[4] A.B. Gumel, E.A. Iboi, C.N. Ngonghala, et al. A primer on using mathematics to understand COVID-19 dynamics: Modeling, analysis and simulations, Infect. Dis. Model. 6 (2021), 148–168. https://doi.org/10.1016/j.idm.2020.11.005.

[5] R.R. Musafir, A. Suryanto, I. Darti, Dynamics of COVID-19 epidemic model with asymptomatic infection, quarantine, protection and vaccination, Commun. Biomath. Sci. 4 (2021), 106–124. https://doi.org/10.5614/cbms.2021.4.2.3.

[6] M. Rayungsari, M. Aufin, N. Imamah, Parameters estimation of generalized richards model for COVID-19 cases in Indonesia using genetic algorithm, Jambura J. Biomath. 1 (2020), 25–30. https://doi.org/10.34312/jjbm.v1i1.6910.

[7] I. Darti, A. Suryanto, H.S. Panigoro, et al. Forecasting COVID-19 epidemic in Spain and Italy using a generalized Richards model with quantified uncertainty, Commun. Biomath. Sci. 3 (2021), 90–100. https://doi.org/10.5614/cbms.2020.3.2.1.

[8] R.R. Musafir, S. Anam, Parameter estimation of COVID-19 compartment model in Indonesia using particle swarm optimization, J. Berkala Epidemiol. 10 (2022), 283–292. https://doi.org/10.20473/jbe.v10i32022.283-292.

[9] Y.C. Chen, P.E. Lu, C.S. Chang, et al. A time-dependent SIR model for COVID-19 with undetectable infected persons, IEEE Trans. Netw. Sci. Eng. 7 (2020), 3279–3294. https://doi.org/10.1109/tnse.2020.3024723.

[10] I. Cooper, A. Mondal, C.G. Antonopoulos, A SIR model assumption for the spread of COVID-19 in different communities, Chaos Solitons Fractals. 139 (2020), 110057. https://doi.org/10.1016/j.chaos.2020.110057.

[11] A. Atkeson, On using SIR models to model disease scenarios for COVID-19, Quart. Rev. 41 (2020), 1–35. https://doi.org/10.21034/qr.4111.

[12] N.A. Kudryashov, M.A. Chmykhov, M. Vigdorowitsch, Analytical features of the SIR model and their applications to COVID-19, Appl. Math. Model. 90 (2021), 466–473. https://doi.org/10.1016/j.apm.2020.08.057.

[13] S. He, Y. Peng, K. Sun, SEIR modeling of the COVID-19 and its dynamics, Nonlinear Dyn. 101 (2020), 1667–1680. https://doi.org/10.1007/s11071-020-05743-y.

[14] S. Annas, M.I. Pratama, M. Rifandi, et al. Stability analysis and numerical simulation of SEIR model for pandemic COVID-19 spread in Indonesia, Chaos Solitons Fractals. 139 (2020), 110072. https://doi.org/10.1016/j.chaos.2020.110072.

[15] S. Paul, A. Mahata, U. Ghosh, et al. Study of SEIR epidemic model and scenario analysis of COVID-19 pandemic, Ecol. Genet. Genom. 19 (2021), 100087. https://doi.org/10.1016/j.egg.2021.100087.

[16] M. Kamrujjaman, P. Saha, M.S. Islam, et al. Dynamics of SEIR model: A case study of COVID-19 in Italy, Results Control Optim. 7 (2022), 100119. https://doi.org/10.1016/j.rico.2022.100119.

[17] M.A. Bahloul, A. Chahid, T.M. Laleg-Kirati, Fractional-order SEIQRDP model for simulating the dynamics of COVID-19 epidemic, IEEE Open J. Eng. Med. Biol. 1 (2020), 249–256. https://doi.org/10.1109/ojemb.2020.3019758.

[18] L. Peng, W. Yang, D. Zhang, et al. Epidemic analysis of COVID-19 in China by dynamical modeling, (2020). https://doi.org/10.48550/ARXIV.2002.06563.

[19] L. López, X. Rodó, A modified SEIR model to predict the COVID-19 outbreak in Spain and Italy: Simulating control scenarios and multi-scale epidemics, Results Phys. 21 (2021), 103746. https://doi.org/10.1016/j.rinp.2020.103746.

[20] O. Postavaru, S.R. Anton, A. Toma, COVID-19 pandemic and chaos theory, Math. Computers Simul. 181 (2021), 138–149. https://doi.org/10.1016/j.matcom.2020.09.029.

[21] D.E. Varberg, E.J. Purcell, S.E. Rigdon, Calculus, Pearson Educación, 2007.

[22] W.R. Derrick, S.I. Grossman, A first course in differential equations with applications, West Publishing Company, 1987.

[23] R.C. Robinson, An introduction to dynamical systems: continuous and discrete, American Mathematical Society, 2012.

[24] C. Castillo-Chavez, Z. Feng, W. Huang, On the computation of R_0 and its role on global stability, in: C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, A.-A. Yakubu (Eds.), Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction, Springer New York, New York, NY, 2002: pp. 229–250. https://doi.org/10.1007/978-1-4757-3667-0_13.

[25] M. Martcheva, An introduction to mathematical epidemiology, Springer New York, 2015. https://doi.org/10.1007/978-1-4899-7612-3.

[26] L.P. Sinaga, H. Nasution, D. Kartika, Stability analysis of the corona virus (Covid-19) dynamics SEIR model in Indonesia, J. Phys.: Conf. Ser. 1819 (2021), 012043. https://doi.org/10.1088/1742-6596/1819/1/012043.

[27] M.A.B. Masud, M. Ahmed, M.H. Rahman, Optimal control for COVID-19 pandemic with quarantine and antiviral therapy, Sensors Int. 2 (2021), 100131. https://doi.org/10.1016/j.sintl.2021.100131.