

## GLOBAL DYNAMICS AND SENSITIVITY ANALYSIS OF SEIAR MATHEMATICAL MODEL FOR COVID-19 PANDEMIC

Saad Z. Rida<sup>a</sup>, Ahmed A. Farghaly<sup>b,c</sup>, Fatma Hussien<sup>b</sup>

<sup>a</sup> Department of Mathematics, Faculty of Science, South Valley University,  
Qena, Egypt

<sup>b</sup> Department of Mathematics, Faculty of Science, Assiut University,  
Assiut 71516, Egypt

<sup>c</sup> Department of Basic Science, Faculty of Computer and Information  
Science, Majmaah University, Majmaah 11952, KSA

Tel: 01114290505, ([szagloul1000@gmail.com](mailto:szagloul1000@gmail.com))

Tel: 01099386283, ([farghaly@aun.edu.eg](mailto:farghaly@aun.edu.eg))

Tel: 01063743390, ([fatma.hussin@science.aun.edu.eg](mailto:fatma.hussin@science.aun.edu.eg))

**Received:** 8/8/2021 **Accepted:** 29/11/2021 **Available Online:** 1/12/2021

In this paper, we introduce a mathematical *SEIAR* model for the ongoing outbreak of the COVID-19 pandemic. We use the reproduction number  $R_0$  to perform the sensitivity analysis and determine the most critical parameters from which we measure the volume of COVID-19 propagation. Global stability analysis of the model is investigated based on suitable Lyapunov functions. Based on facts to estimate the spread of COVID-19 throughout the world, numerical simulation is presented. Furthermore, we compare our simulation results to some real-world data collected by the World Health Organization over a period of time on reported infected cases.

**Keywords:** *Covid-19 pandemic, SEIAR Mathematical model, Basic reproduction number, Sensitivity analysis, Stability, Numerical simulation.*

## 1. INTRODUCTION

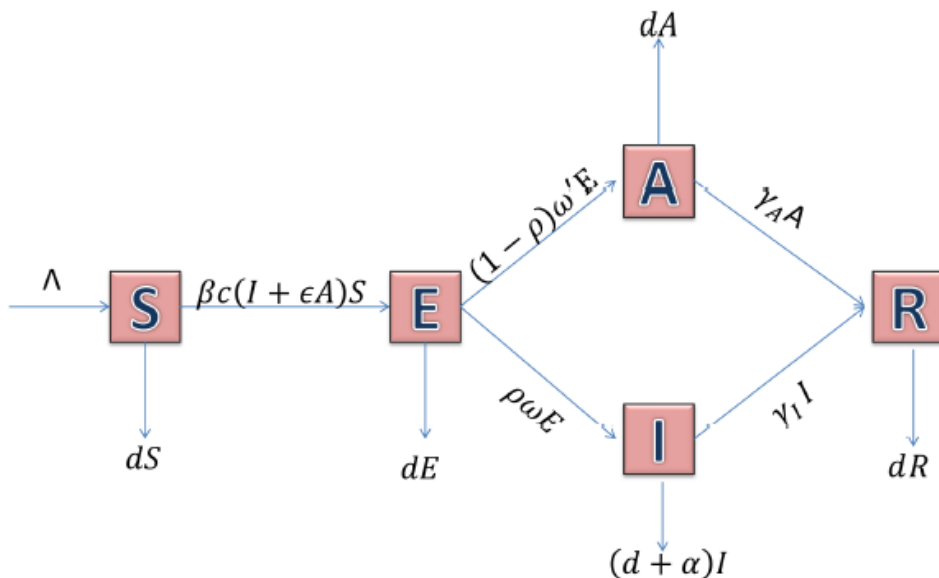
In Wuhan, China, the novel coronavirus appeared on 31 December 2019. A novel coronavirus (COVID-19), as announced by World Health Organization (WHO), was described, on 7 January 2020, by the Chinese authorities as the source agent for Wuhan pneumonia of undefined etiology. On 11 February 2020, as reported by the International Committee on Taxonomy of Viruses, the virus was renamed as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. This virus is from the family of coronavirus which includes other viruses such as SARS (Severe Acute Respiratory Syndrome identified in China in 2003) [2] and MERS (Middle East Respiratory Syndrome identified in Saudi Arabia in 2012), [3], as well as mild viruses causing the common flu. The possibility of human transmission was associated with the fast spreading of the novel coronavirus in other regions in China and in other countries. By 31 January, worldwide reported cases had registered 9,776 with a number of deaths of 213, and WHO announced the epidemic as an international public health emergency [4]. COVID-19 was declared as a pandemic on 11 March by WHO [5], leading to more than 118,000 outbreaks of coronavirus disease in more than 110 countries. More than 22.2 million confirmed cases were reported on 21 August 2020 and there were 782,500 deaths worldwide since the epidemic started [6]. In over 200 countries, confirmed cases were recorded with new infections and regular reports from countries. The propagation of COVID-19 is still ongoing and this virus was expanded faster worldwide. The severity of the outbreak of the disease has attracted scientists and researchers from all over the world who have studied COVID-19 from different perspectives. Mathematical modeling continues to have an important role in understanding the disease complex behavior [7, 8, 9, 10]. The calculation of the basic reproduction number  $R_0$  is vital in assessing the likelihood and extent of an outbreak. Several articles were recently published and released to evaluate  $R_0$  and the danger of outbreak [11, 12, 13]. Tian et al [14] introduced a transmission model for simulating the phase-based of COVID-19. According to Tian et al [14], we are developing the mathematical model for estimating the transmissibility and understanding the dynamical behavior of the virus transmission.

The rest of this paper is structured according to the following. In Section 2, we formulate proposed model and produce a detailed mathematical analysis. Simulation analysis using real data is discussed in Section 3. Section 4 ends the paper of conclusions and discussion.

## 2. MATHEMATICAL MODELING AND ANALYSIS

### 2.1. The model

Mathematical modeling is an essential method to measure and forecast the severity, size and time period of infectious diseases. Based on the spreading mechanism of COVID-19 and Tian et al [14], we proposed the basic *SEIAR* compartmental model. The population is partitioned into five groups namely susceptible ( $S(t)$ ), exposed ( $E(t)$ ), symptomatic ( $I(t)$ ), asymptomatic ( $A(t)$ ) and recovered ( $R(t)$ ). In the model, we assumed the recovered people has immunity during this time of epidemic. Figure 1 shows the flow diagram transmission of the proposed model.



**Figure 1:** The diagram of the proposed COVID-19 model.

Based on Figure 1., the nonlinear system of the *SEIAR* model to explain the transmission mechanism of a novel COVID-19 are given as follows:

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - \beta c S(t)(I(t) + \varepsilon A(t)) - dS(t), \\
 \frac{dE}{dt} &= \beta c S(t)(I(t) + \varepsilon A(t)) - (\rho \omega + (1 - \rho) \omega' + d)E(t), \\
 \frac{dI}{dt} &= \rho \omega E(t) - (d + \alpha + \gamma_I)I(t), \\
 \frac{dA}{dt} &= (1 - \rho) \omega' E(t) - (d + \gamma_A)A(t), \\
 \frac{dR}{dt} &= \gamma_A A(t) + \gamma_I I(t) - dR(t).
 \end{aligned}
 \tag{1}$$

The parameters  $\Lambda, \beta, c, \varepsilon, d, \rho, \omega, \omega', \alpha, \gamma_I,$  and  $\gamma_A$  are positive constants, where  $\Lambda$  is the birth rate,  $\beta$  is the transmission rate per contact,  $c$  is the contact rate,  $\varepsilon$  is the multiple of the transmissibility of  $A$  to that of  $I$ , where  $0 \leq \varepsilon \leq 1$ ,  $d$  is the rate of natural death,  $\rho$  is the symptomatic infection rate,  $\frac{1}{\omega}$  is the incubation period,  $\frac{1}{\omega'}$  is the latent period,  $\alpha$  is the death rate triggered by disease,  $\gamma_I$  is the recovery rate in patients with symptoms, and  $\gamma_A$  is the recovery rate among asymptomatic patients. The initial conditions of system (1) are

$$S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, A(0) \geq 0, R(0) \geq 0.$$

The total size  $N(t)$  of the population is given by  $N(t) = S(t) + E(t) + I(t) + A(t) + R(t)$ , and satisfies

$$\frac{dN}{dt} = \Lambda - dN(t) - \alpha I(t) \leq \Lambda - dN(t),$$

hence,  $N(t) \rightarrow \frac{\Lambda}{d}$  as  $t \rightarrow \infty$ .

Then, biologically feasible region

$\Omega = \{(S, E, I, A, R) : 0 \leq S, E, I, A, R, N \leq \frac{\Lambda}{d}\}$  is bounded and invariant positively corresponding to the proposed model (1).

### 2.2. Equilibrium points and basic reproduction number

In the current subsection, we compute the equilibrium points and the basic reproduction number of system (1). The system (1) has two equilibrium points:

1. Disease-free point  $E_0 = (\frac{\Lambda}{d}, 0, 0, 0, 0)$
2. Endemic point  $E_1 = (S^*, E^*, I^*, A^*, R^*)$ ,  $S^* \in (0, S_0)$

where,

$$S^* = \frac{(\rho\omega + (1-\rho)\omega' + d)}{\beta c \left( \frac{\rho\omega}{d + \alpha + \gamma_I} + \frac{\varepsilon(1-\rho)\omega'}{d + \gamma_A} \right)}, E^* = \frac{\Lambda - dS^*}{(\rho\omega + (1-\rho)\omega' + d)}, I^* = \frac{\rho\omega}{d + \alpha + \gamma_I} E^*,$$

$$A^* = \frac{(1-\rho)\omega'}{d + \gamma_A} E^*, R^* = \frac{1}{d} \left( \frac{\rho\omega\gamma_I}{d + \alpha + \gamma_I} + \frac{(1-\rho)\omega'\gamma_A}{d + \gamma_A} \right) E^*.$$

Letting  $X = (E, I, A)^T$ , then, it follows from system (1)

$$\frac{dX}{dt} = \Phi - \Psi$$

where

$$\Phi = \begin{pmatrix} \beta c S(t)(I(t) + \varepsilon A(t)) \\ 0 \\ 0 \end{pmatrix}, \Psi = \begin{pmatrix} (\rho\omega + (1-\rho)\omega' + d)E(t) \\ -\rho\omega E(t) + (d + \alpha + \gamma_I)I(t) \\ -(1-\rho)\omega' E(t) + (d + \gamma_A)A(t) \end{pmatrix}.$$

The Jacobian matrices of  $\Phi$  and  $\Psi$  at disease-free equilibrium are given by

$$F = \begin{pmatrix} 0 & \frac{\beta c \Lambda}{d} & \frac{\beta c \varepsilon \Lambda}{d} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} (\rho \omega + (1-\rho)\omega' + d) & 0 & 0 \\ -\rho \omega & (d + \alpha + \gamma_I) & 0 \\ -(1-\rho)\omega' & 0 & (d + \gamma_A) \end{pmatrix},$$

then, the next generation matrix  $FV^{-1}$  for system (1) is

$$FV^{-1} = \begin{pmatrix} \frac{\beta c \Lambda}{d(\rho \omega + (1-\rho)\omega' + d)} \left( \frac{\rho \omega}{d + \alpha + \gamma_I} + \frac{\varepsilon(1-\rho)\omega'}{d + \gamma_A} \right) & \frac{\beta c \Lambda}{d(d + \alpha + \gamma_I)} & \frac{\beta c \varepsilon \Lambda}{d(d + \gamma_A)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The spectral radius of  $FV^{-1}$  is the basic reproduction number; i.e.,

$$R_0 = \rho(FV^{-1}) = \frac{\beta c \Lambda}{d(\rho \omega + (1-\rho)\omega' + d)} \left( \frac{\rho \omega}{d + \alpha + \gamma_I} + \frac{\varepsilon(1-\rho)\omega'}{d + \gamma_A} \right).$$

Accordingly, the endemic equilibrium  $E_1 = (S^*, E^*, I^*, A^*, R^*)$ , which keep a live of the novel coronavirus propagation can be reformulated as follows:

$$S^* = \frac{(\rho \omega + (1-\rho)\omega' + d)}{\beta c \left( \frac{\rho \omega}{d + \alpha + \gamma_I} + \frac{\varepsilon(1-\rho)\omega'}{d + \gamma_A} \right)}, E^* = \frac{d(R_0 - 1)S^*}{(\rho \omega + (1-\rho)\omega' + d)}, I^* = \frac{\rho \omega d(R_0 - 1)S^*}{(d + \alpha + \gamma_I)(\rho \omega + (1-\rho)\omega' + d)},$$

$$A^* = \frac{(1-\rho)\omega' d(R_0 - 1)S^*}{(d + \gamma_A)(\rho \omega + (1-\rho)\omega' + d)}, R^* = \frac{(R_0 - 1)S^*}{(\rho \omega + (1-\rho)\omega' + d)} \left( \frac{\rho \omega \gamma_I}{d + \alpha + \gamma_I} + \frac{(1-\rho)\omega' \gamma_A}{d + \gamma_A} \right),$$

which exists only if  $R_0 > 1$ .

### 2.3. Sensitivity analysis of $R_0$

In this subsection, we examine the sensitivity analysis of the model

parameters to identify the impact of different parameters on the spread of Covid-19. Sensitivity analysis is carried out on  $R_0$  in order to determine the most important parameters that have a high impact on the disease transmission. This helps to assess where measures are involved to reduce disease deaths and morbidity. One of the ways to calculate the sensitivity is by finding the partial derivatives of  $R_0$  for each parameter. Based on Chitnis et al. [15], the formula of the normalized forward sensitivity index is defined by:

$$\Upsilon_h^{R_0} := \frac{\partial R_0}{\partial h} \times \frac{h}{R_0},$$

where  $\Upsilon_h^{R_0}$  is the  $R_0$  sensitivity index in relation to parameter  $h$ . For each parameter in  $R_0$ , we calculate the sensitivity indices. For example, the  $R_0$  sensitivity index for parameter  $c$  is referred to

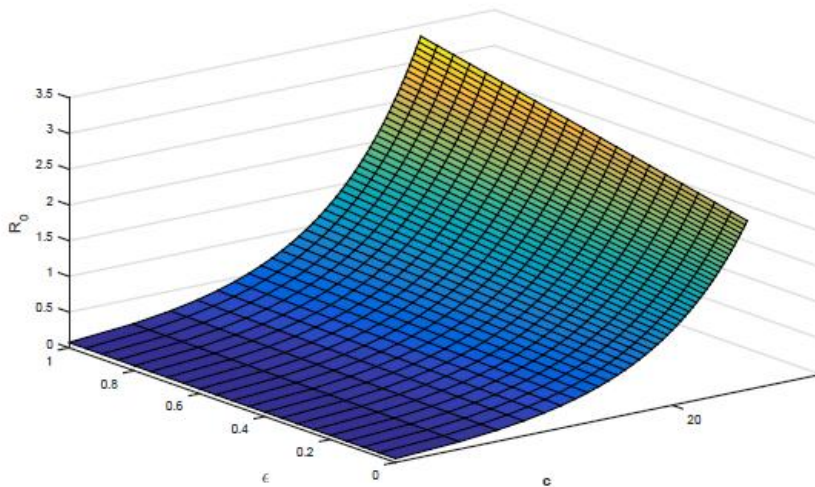
$$\Upsilon_c^{R_0} = \frac{\partial R_0}{\partial c} \times \frac{c}{R_0} = +1.$$

For the other parameters in  $R_0$ , the  $R_0$  sensitivity indices and the values of their parameters are shown in Table 1.

**Table 1:**  $R_0$  sensitivity indices and values of their parameters for the COVID-19 model estimated in the world.

Parameter	Value	Source	Sensitivity index
$c$	40.319	[16]	+1
$\beta$	0.0267	Fitted	+1
$\omega$	1/5.2	[11]	+0.1113
$\omega'$	1/5.2	Assumed	-0.0732
$\rho$	0.6628	[16]	+0.2551
$\varepsilon$	0.8098	[16]	+0.2512
$\gamma_I$	0.7418	[6]	-0.7201
$\gamma_A$	0.94	Assumed	-0.2491
$\alpha$	0.022	[6]	-0.0214

From Table 1, the  $R_0$  value decreases with the increase of the the sensitivity index with negative sign, while  $R_0$  increases with the increase of the the sensitivity index with positive sign. The results of sensitivity analysis indicate that the most sensitive parameters are  $c, \beta, \gamma_1, \rho, \varepsilon, \gamma_A$  and so on, taken in descending order. This means that by reducing the rate of  $c, \beta, \rho$  and  $\varepsilon$ , the spread of Covid-19 would be halted. Figure 2 shows the effect of  $c, \varepsilon$  on  $R_0$ . It implies the importance of asymptomatic individuals on the control parameters that provide valuable theoretical reference for a better estimation of Covid-19. In reducing  $c, \beta$ , and  $\varepsilon$ , the behavioral change in the rate of transmission may be considered for further study.



**Figure 2:** The effective of  $c, \varepsilon$  on  $R_0$ . Other parameters are fixed here.

#### 2.4. Global stability of the model

Since the variable  $R(t)$  does not exist in the first four equations of system (1). Therefore, the dynamics of system (1) are the the same dynamics of the following system:



$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - \beta c S(t)(I(t) + \varepsilon A(t)) - dS(t), \\
 \frac{dE}{dt} &= \beta c S(t)(I(t) + \varepsilon A(t)) - (\rho \omega + (1 - \rho)\omega' + d)E(t), \\
 \frac{dI}{dt} &= \rho \omega E(t) - (d + \alpha + \gamma_I)I(t), \\
 \frac{dA}{dt} &= (1 - \rho)\omega' E(t) - (d + \gamma_A)A(t).
 \end{aligned}
 \tag{5}$$

We investigate the global stability of the equilibrium points of model (5) by Lyapunov direct method [17]. We will implement the following function for simplicity and mathematical convenience:  $F : R > 0 \rightarrow R \geq 0$  as  $F(y) = y - 1 - \ln(y)$ , we note that  $F(y) \geq 0$  for any  $y > 0$ .

**Theorem 1.** If  $R_0 \leq 1$ , then the disease free equilibrium  $E_0$  of the model (5) is globally stable.

**Proof:** Consider the following Lyapunov function:

$$V_1(t) = \left( S - S^0 - S^0 \ln \frac{S}{S^0} \right) + E + \frac{\beta c S^0}{d + \alpha + \gamma_I} I + \frac{\varepsilon \beta c S^0}{d + \gamma_A} A,
 \tag{6}$$

where,  $V_1(t) > 0$  for all  $(S, E, I, A) > 0$ , and  $V_1(t) = 0$  at the disease free equilibrium  $E_0 = \left( \frac{\Lambda}{d}, 0, 0, 0 \right)$ .

The derivative of  $V_1(t)$  with respect to  $t$  is:

$$\begin{aligned}
 \frac{dV_1}{dt} &= \left( 1 - \frac{S^0}{S} \right) \frac{dS}{dt} + \frac{dE}{dt} + \frac{\beta c S^0}{d + \alpha + \gamma_I} \frac{dI}{dt} + \frac{\varepsilon \beta c S^0}{d + \gamma_A} \frac{dA}{dt} \\
 &= \left( 1 - \frac{S^0}{S} \right) (\Lambda - dS - \beta c SI - \beta c \varepsilon SA) + (\beta c SI + \beta c \varepsilon SA - (\rho \omega + (1 - \rho)\omega' + d)E) \\
 &\quad + \frac{\beta c S^0}{d + \alpha + \gamma_I} (\rho \omega E - (d + \alpha + \gamma_I)I) + \frac{\varepsilon \beta c S^0}{d + \gamma_A} ((1 - \rho)\omega' E - (d + \gamma_A)A).
 \end{aligned}$$

Substituting  $\Lambda = dS^0$  and cancelling the same terms, yields

$$\begin{aligned}
\frac{dV_1}{dt} &= \frac{(S-S^0)}{S} (dS^0 - dS) - (\rho\omega + (1-\rho)\omega' + d)E + \frac{\beta c S^0 \omega \rho E}{d + \alpha + \gamma_I} + \frac{\beta c S^0 \varepsilon (1-\rho)\omega' E}{d + \gamma_A} \\
&= \frac{-d(S-S^0)^2}{S} - (\rho\omega + (1-\rho)\omega' + d)E \left[ 1 - \frac{\beta c S^0}{(\rho\omega + (1-\rho)\omega' + d)} \left( \frac{\rho\omega}{d + \alpha + \gamma_I} + \frac{\varepsilon(1-\rho)\omega'}{d + \gamma_A} \right) \right] \\
&= \frac{-d(S-S^0)^2}{S} - (\rho\omega + (1-\rho)\omega' + d)(1-R_0)E.
\end{aligned} \tag{7}$$

Obviously,  $\frac{dV_1}{dt} \leq 0$  when  $R_0 \leq 1$  and  $\frac{dV_1}{dt} = 0$  if and only if  $S = S^0$ ,  $E = 0$ ,  $I = 0$ , and  $A = 0$ . By LaSalle's Invariance Principle [18], the proof is done.

**Theorem 2.** Suppose  $R_0 > 1$  for the endemic existing, then endemic equilibrium point  $E_1$  of model (5) is globally stable.

**Proof:** Consider Lyapunov function as follows:

$$\begin{aligned}
V_2(t) &= \left( S - S^* - S^* \ln \frac{S}{S^*} \right) + \left( E - E^* - E^* \ln \frac{E}{E^*} \right) + \frac{\beta c S^0}{d + \alpha + \gamma_I} \left( I - I^* - I^* \ln \frac{I}{I^*} \right) \\
&\quad + \frac{\beta c \varepsilon S^0}{d + \gamma_A} \left( A - A^* - A^* \ln \frac{A}{A^*} \right).
\end{aligned} \tag{8}$$

For all  $S, E, I, A > 0$ , it clear that  $V_2(t)$  is positive and continuous. It is also clear that  $V_2(t) = 0$  at  $S = S^*, E = E^*, I = I^*, A = A^*$ .

Taking the derivative of  $V_2(t)$ , then

$$\begin{aligned}
 \frac{dV_2(t)}{dt} &= \left(1 - \frac{S^*}{S}\right) \frac{dS}{dt} + \left(1 - \frac{E^*}{E}\right) \frac{dE}{dt} + \frac{\beta c S^0}{d + \alpha + \gamma_I} \left(1 - \frac{I^*}{I}\right) \frac{dI}{dt} + \frac{\beta c \varepsilon S^0}{d + \gamma_A} \left(1 - \frac{A^*}{A}\right) \frac{dA}{dt} \\
 &= \left(1 - \frac{S^*}{S}\right) (\Lambda - dS - \beta c SI - \beta c \varepsilon SA) + \left(1 - \frac{E^*}{E}\right) (\beta c SI + \beta c \varepsilon SA - (\rho\omega + (1-\rho)\omega') + d) \\
 &\quad + \frac{\beta c S^0}{d + \alpha + \gamma_I} \left(1 - \frac{I^*}{I}\right) (\rho\omega E - (d + \alpha + \gamma_I)I) + \frac{\beta c \varepsilon S^0}{d + \gamma_A} \left(1 - \frac{A^*}{A}\right) ((1-\rho)\omega' E - (d + \alpha + \gamma_I)A)
 \end{aligned}$$

By substituting  $\Lambda = \beta c S^* I^* + \beta c \varepsilon S^* A^* + dS^*$  and collecting the same terms, we get

$$\begin{aligned}
 \frac{dV_2(t)}{dt} &= \frac{-d(S - S^*)^2}{S} + 3\beta c S^* I^* + 3\beta c \varepsilon S^* A^* - \beta c S^* I^* \frac{S^*}{S} \\
 &\quad - \beta c \varepsilon S^* A^* \frac{S^*}{S} - (\rho\omega + (1-\rho)\omega' + d)E - \beta c SI \frac{E^*}{E} - \beta c \varepsilon SA \frac{E^*}{E} \\
 &\quad + \beta c S^* \frac{\rho\omega}{d + \alpha + \gamma_I} E - \beta c S^* I^* \frac{EI^*}{E^* I} + \beta c \varepsilon S^* \frac{(1-\rho)\omega'}{d + \gamma_A} E - \beta c \varepsilon S^* A^* \frac{EA^*}{E^* A} \\
 &= \frac{-d(S - S^*)^2}{S} - \beta c S^* I^* \left[ -3 + \frac{S^*}{S} + \frac{SE^* I}{S^* EI^*} + \frac{EI^*}{E^* I} \right] - \beta c \varepsilon S^* A^* \left[ -3 + \frac{S^*}{S} + \frac{SE^* A}{S^* EA^*} \right] \\
 &\quad - (\rho\omega + (1-\rho)\omega' + d)E \left[ 1 - \frac{\beta c S^*}{(\rho\omega + (1-\rho)\omega' + d)} \left( \frac{\rho\omega}{d + \alpha + \gamma_I} + \frac{\varepsilon(1-\rho)\omega'}{d + \gamma_A} \right) \right].
 \end{aligned}$$

Because  $S^* = \frac{(\rho\omega + (1-\rho)\omega' + d)}{\beta c \left( \frac{\rho\omega}{d + \alpha + \gamma_I} + \frac{\varepsilon(1-\rho)\omega'}{d + \gamma_A} \right)}$ , then we obtain

$$\begin{aligned}
 \frac{dV_2(t)}{dt} &= \frac{-d(S - S^*)^2}{S} - \beta c S^* I^* \left[ \left( \frac{S^*}{S} - 1 - \ln \frac{S^*}{S} \right) + \left( \frac{EI^*}{E^* I} - 1 - \ln \frac{EI^*}{E^* I} \right) + \left( \frac{SE^* I}{S^* EI^*} - 1 - \ln \frac{SE^* I}{S^* EI^*} \right) \right] \\
 &\quad - \beta c S^* I^* \left[ \ln \frac{S^*}{S} + \ln \frac{EI^*}{E^* I} + \ln \frac{SE^* I}{S^* EI^*} \right] - \beta c \varepsilon S^* A^* \left[ \ln \frac{S^*}{S} + \ln \frac{EA^*}{E^* A} + \ln \frac{SE^* A}{S^* EA^*} \right] \\
 &\quad - \beta c \varepsilon S^* A^* \left[ \left( \frac{S^*}{S} - 1 - \ln \frac{S^*}{S} \right) + \left( \frac{EA^*}{E^* A} - 1 - \ln \frac{EA^*}{E^* A} \right) + \left( \frac{SE^* A}{S^* EA^*} - 1 - \ln \frac{SE^* A}{S^* EA^*} \right) \right].
 \end{aligned}$$

Since,

$$\ln \frac{S^*}{S} + \ln \frac{EI^*}{E^*I} + \ln \frac{SE^*I}{S^*EI^*} = \ln \frac{S^*}{S} + \ln \frac{EA^*}{E^*A} + \ln \frac{SE^*A}{S^*EA^*} = 0,$$

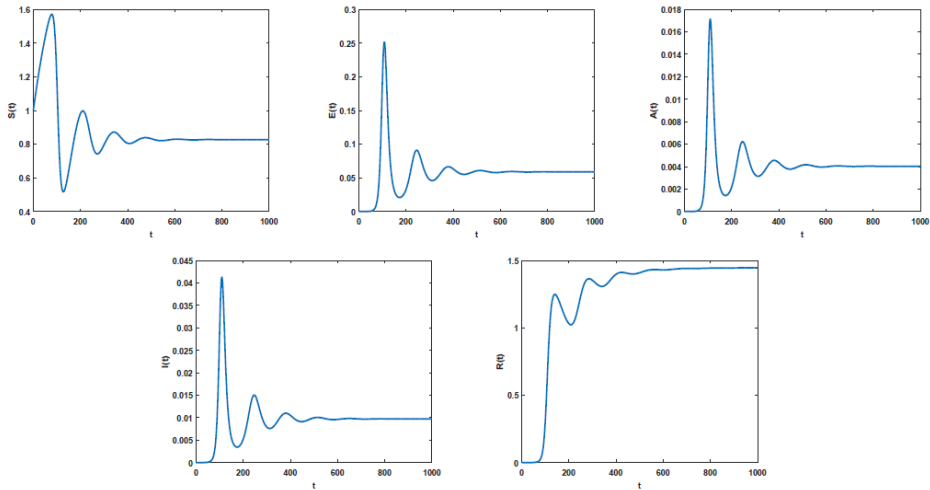
it follows that,

$$\begin{aligned} \frac{dV_2(t)}{dt} = & \frac{-d(S-S^*)^2}{S} - \beta c S^* I^* \left[ \left( \frac{S^*}{S} - 1 - \ln \frac{S^*}{S} \right) + \left( \frac{EI^*}{E^*I} - 1 - \ln \frac{EI^*}{E^*I} \right) + \left( \frac{SE^*I}{S^*EI^*} - 1 - \ln \frac{SE^*I}{S^*EI^*} \right) \right] \\ & - \beta c \varepsilon S^* A^* \left[ \left( \frac{S^*}{S} - 1 - \ln \frac{S^*}{S} \right) + \left( \frac{EA^*}{E^*A} - 1 - \ln \frac{EA^*}{E^*A} \right) + \left( \frac{SE^*A}{S^*EA^*} - 1 - \ln \frac{SE^*A}{S^*EA^*} \right) \right] \end{aligned}$$

Since the function  $F(y) = y - 1 - \ln y \geq 0$  for all  $y > 0$  and  $F(y) = 0$  if  $y = 1$ , then  $\frac{dV_2(t)}{dt} < 0$ . We can also observe that,  $\frac{dV_2(t)}{dt} = 0$  iff  $S = S^*$ ,  $E = E^*$ ,  $I = I^*$  and  $A = A^*$ . The endemic equilibrium point  $E_1$  is globally stable with LaSalle's Invariance Principle [18], and this concludes the proof.

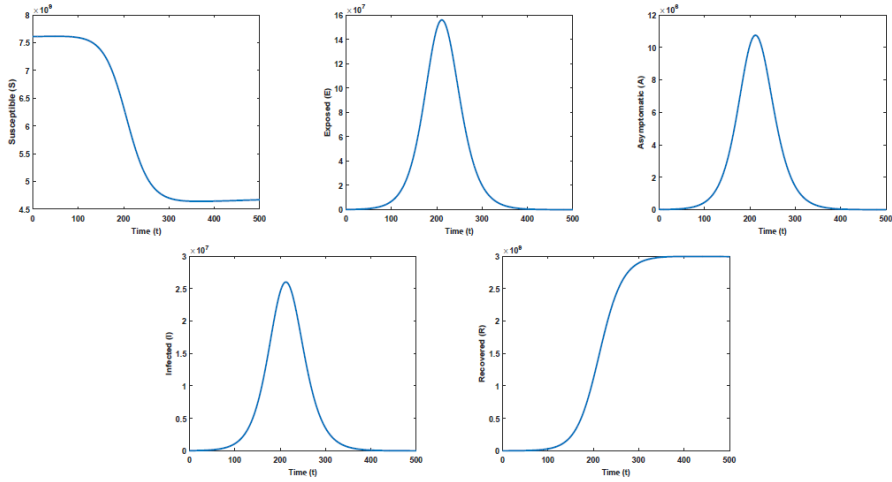
### 3. SIMULATIONS AND RESULTS

In this section, we present numerical simulations of system (1) with the real data set out in Table 1 to investigate the dynamics and the effects of proposed model. The data listed in Table 1 are used for simulation in the world from 4 February 2020. The population of the world was around  $N = 7610105452$  on 4 February 2020 with a birth rate of  $\Lambda = 0.018077$  and a death rate of  $d = 0.007612$  [19]. According to world meter and WHO reports [6], the initial conditions on 4 February are  $I(0) = 24545$  and  $R(0) = 907$ . We assume  $A(0) = 50000$ ,  $E(0) = 80000$  and since  $N(0) = S(0) + E(0) + A(0) + I(0) + R(0)$ , then  $S(0) = 7609950000$ . Real data for a novel coronavirus around the world with a fitting curve are used to estimate the transmission rate for each contact  $\beta$  listed in Table 1. According to these data with simple computations, we have found that the value of  $R_0$  is 2.8609 and system (1) has a unique endemic equilibrium point  $E_1 = (0.8297, 0.0588, 0.0040, 0.0097, 1.4433)$ . Obviously, Theorems 2 holds, thus the endemic equilibrium  $E_1$  is globally asymptotically stable as shown in Figure 3.

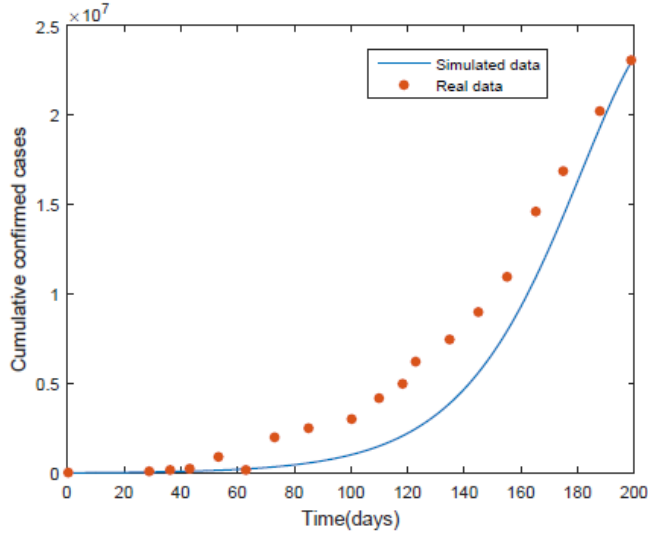


**Figure 3:** Dynamics of system (1) corresponding to Table 1 with  $\Lambda = 0.018077$  and  $d = 0.007612$ : Pointing to asymptotic stability of  $E_1 = (0.8297, 0.0588, 0.0040, 0.0097, 1.4433)$

In Figure 4, we show the dynamics of susceptible, exposed, asymptomatic, infected and recovered population of the proposed model of COVID-19 outbreak in the world within 300 days. Figure 5 displays the cumulative recorded cases by WHO of the Covid-19 pandemic between 4 February and 21 August for 200 days with our simulated results. Matlab program version (2015) is used to plot the total number of cases reported by WHO reached 22,256,220 on 21 August 2020 [6]. The results of our simulations show that our model is well adapted and in good agreement with the data recorded.



**Figure 4:** Time series of the model states defined in system (1) for Table 1.



**Figure 5:** Comparison of simulated and real data for the cumulative reported cases of our proposed COVID-19 model from 4 February to 21 August in the world.

#### 4. CONCLUSIONS AND DISCUSSION

In this paper, we have formulated a *SEIAR* mathematical model for the novel coronavirus (COVID-19) transmission that illustrates the spreading mechanism and evaluates the dynamics of model states and parameters. Equilibrium points were calculated for the model, also by applying the next generation method, we have derived the basic reproduction number  $R_0$  which is a significant factor for estimating the probability of the outbreak of COVID-19. The sensitivity analysis of the model was performed to find parameters that have a significant impact on  $R_0$  and lead to outbreak of infection mostly in the population. It has been shown that  $c, \beta, \rho, \varepsilon$  are the most sensitive factors that have a major role in the rapid speed of disease. In addition, the global stability requirements for the two equilibrium points have been studied. Our studies show that disease-free and endemic equilibria are stable globally if  $R_0 < 1$  and  $R_0 > 1$  respectively. Furthermore, computational simulations were provided to confirm the analytical results. We applied our findings to investigate the COVID-19 outbreak using recorded data by WHO and world meter. Simulated infected class outcomes have been matched with actual evidence for reported cases of infection. We observed that our results were well adapted to the recorded data. The results in our simulations indicate that, we must be ready for fight COVID-19 for a long period to decrease endemic risk and likely disease eradication. In conclusion, to achieve a rapid end to the COVID-19 outbreak, the existing comprehensive control action and self-protection strategies, including isolation and contact reduction, must be maintained.

#### REFERENCES

- [1] World Health Organization. *Coronavirus*. World Health Organization, cited January 19, (2020). Available: <https://www.who.int/health-topics/coronavirus>.
- [2] Hui DSC, Zumla A. *Severe acute respiratory syndrome: Historical, epidemiologic, and clinical features*. *Infect. Dis. Clin. North Am.* **33**, 869-889 (2019).
- [3] Killerby ME, Biggs HM, Midgley CM, Gerber SI, Watson JT. *Middle East*

*respiratory syndrome coronavirus transmission*. Emerg. Infect. Dis. **26**, 191-198 (2020).

[4] World Health Organization. *Statement on the second meeting of the International Health Regulations (2005) Emergency Committee regarding the outbreak of novel coronavirus (2019-ncov)* accessed on February 21, (2020).

[5] Cucinotta D, Vanelli M. *WHO Declares COVID-19 a Pandemic*. Acta Bio Med. Mar.19; **91**(1) (2020).

[6] Worldometer: COVID-19 coronavirus pandemic. American Library Association.

[7] Sarbaz H.A. K., Muhammad S., Mehboob A., Faisal S.. *A quantitative and qualitative analysis of the COVID-19 pandemic model*. Chaos, Solitons and Fractals **138** (2020).

[8] Kottakkaran S. N., Shabir A., Aman U., et al. *Mathematical analysis of SIRD model of COVID-19 with Caputo fractional derivative based on real data*. Results in Physics **21** (2021).

[9] Chinwendu E. M., Sambo D., Isaac O. O. *Controlling the Spread of COVID-19: Optimal Control Analysis*. Computational and Mathematical Methods in Medicine (2020).

[10] S. Olaniyi, O. S. Obabiyi, K. O. Okosun, A. T. Oladipo, S. O. Adewale. *Mathematical modelling and optimal cost-effective control of COVID-19 transmission dynamics* . Eur. Phys. J. Plus (2020).

[11] Li Q, Guan X, Wu P, Wang X, Zhou L, Tong Y, et al. *Early transmission dynamics in Wuhan, China, of novel coronavirus-infected pneumonia*. N Engl J Med. (2020).

[12] Wu JT, Leung K, Leung GM. *Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Chen et al*. Infectious Diseases of Poverty (2020).

[13] Zhao S, Lin Q, Ran J, Musa SS, Yang G, Wang W, et al. *Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCoV) in*



*China, from 2019 to 2020: a data-driven analysis in the early phase of the outbreak.* Int J Infect Dis. (2020).

[14] Chen TM, Rui J, Wang QP, Zhao ZY, Cui JA, Yin L. *A mathematical model for simulating the phase-based transmissibility of a novel coronavirus.* Infectious Diseases of Poverty. (2020).

[15] N. Chitnis, J. M. Hyman, J. M. Cushing. *Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model.* Bulletin of Mathematical Biology, **70**, 1272-1296 (2008).

[16] Hui W, Jing-an C, Guo-jing Y. *Risk estimation and prediction by modeling the transmission of the novel coronavirus (COVID-19) in mainland China excluding Hubei province.* medRxiv (2020).

[17] Hattaf K, Yousfi K N. *Global stability for reaction-diffusion equations in biology.* Comput. Math. Appl. **66**, 1488-1497 (2013).

[18] La Salle J, Lefschetz S. *Stability by Liapunov's Direct Method with Applications.* Academic Press, New York; (1961).

[19] Macrotrends. *The premier research platform for long term investors.* 2010â€“2020 Macrotrends LLC.

## العربي الملخص

في هذه الورقة ، نقدم نموذجًا رياضيًا فيما يتعلق بالتنفسي المستمر لوباء كوفيد-19 العالمي . تم بناء النموذج الرياضي من خلال الأخذ في الاعتبار العديد من معايير علم الأوبئة التي تتطابق بشكل وثيق مع الحالة الحقيقية . نستخدم رقم التكاثر الأساسي للفيروس ( $R_0$ ) لإجراء تحليل الحساسية وتحديد أهم البارامترات والعوامل التي نقيس من خلالها حجم انتشار جائحة كوفيد-19 . أجرت الدراسة أيضًا تحليلًا للنموذج وفحص الاستقرار لنقاط الإتران بناءً على دوال Lyapunov المناسبة. اعتماداً على الحقائق لتقدير انتشار كوفيد-19 في جميع أنحاء العالم ، يتم تقديم محاكاة عددية . علاوة على ذلك، قورنت نتائج المحاكاة الخاصة بنا لبعض البيانات الواقعية التي جمعتها منظمة الصحة العالمية على مدار فترة من الوقت على الحالات المصابة المبلغ عنها . خلصت الدراسة إلى أن علاجات كوفيد-19 يجب أن تركز عليها تقييد التفاعل بين الأفراد وتحسين الحجر الصحي.