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Commun. Math. Biol. Neurosci. 2020, 2020:41

<https://doi.org/10.28919/cmbn/4551>

ISSN: 2052-2541

STABILITY ANALYSIS OF MATHEMATICAL MODEL NEW CORONA VIRUS (COVID-19) DISEASE SPREAD IN POPULATION

ABDERRAHIM LABZAI^{1,*}, ABDELFAH KOUIDERE¹, OMAR BALATIF², MOSTAFA RACHIK¹

¹Laboratory of Analysis Modeling and Simulation, Department of Mathematics and Computer Science, Faculty of Sciences Ben M'Sik, Sidi Othman, Hassan II University, Casablanca, Morocco

²Laboratory of Dynamical Systems, Mathematical Engineering Team (INMA), Department of Mathematics, Faculty of Sciences El Jadida, Chouaib Doukkali University, El Jadida, Morocco

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Abstract. The New Corona Virus epidemic is the most serious epidemic that the international community had known in 2019 and this is manifested by the deaths it claimed and in a short time. Its risk is much greater than (MERS) disease that emerged in the Republic of Korea and it is spreading largely more than SARS disease which appeared in Saudi Arabia and the Middle East. This deadly disease caught the public's attention and caused terror in many societies around the world. We are building a dynamic model based on the detailed data of mortality from the World Health Organization (WHO) and the actual spread of the epidemic. By using Routh-Hurwitz criteria and constructing Lyapunov functions, the local and the global stability of the disease-free equilibrium and the disease equilibrium are obtained. We also study the sensitivity analysis of model parameters to know the parameters that have a high impact on the reproduction number R_0 . Finally, numerical simulations are performed to verify the theoretical analysis using Matlab.

Keywords: mathematical modeling; stability; sensitivity; optimal control; disease COVID-19.

2010 AMS Subject Classification: 93A30, 49J15.

*Corresponding author

E-mail address: labzaiabdo1977@gmail.com

Received March 1, 2020

INTRODUCTION

The New Corona Virus 2019 was considered at the time of its emergence in late 2019 a dangerous disease to the international community and this is manifested by the deaths it claimed in a short time in China and the whole world. On 31st December 2019, the World Health Organization (WHO) was informed of cases of idiopathic pulmonary infections in Wuhan, Hubei Province, China. Then, on January 7th, the Chinese authorities identified the cause of these cases with a new virus, the newly created Corona virus (2019-nCoV). Since the first cases of the virus were declared up to now, globally, 87137 of infected individuals have been confirmed including 79968 cases in China. The death toll reached 2873 deaths globally according to Coronavirus disease 2019 (COVID-19) Situation Report – 41 [1].

Investigations are still ongoing to assess the source of the disease, the mode or modes of transmission and the extent of infection. Currently, available evidence of the emerging Corona virus and past experiences with other Corona viruses (Middle East Respiratory Syndrome (MERS) and SARS virus) and other respiratory symptoms viruses (such as bird flu) indicate the possibility of the new virus transmission from an animal source[2, 3, 4].

The organization (WHO) adds that Corona viruses are a group of viruses that are known to cause diseases ranging from common cold to more severe diseases, such as Middle East Respiratory Syndrome (MERS) and severe acute respiratory syndrome (SARS). The Corona virus (nCoV) is a new strain of the virus that has not been previously discovered in humans. Detailed investigations have concluded that the SARS-CoV virus transmitted from civet cats to humans and that the Middle East respiratory syndrome (MERS-CoV) virus has passed from camels to humans. There are several known types of Corona viruses that circulate among animals without infecting humans so far. Common signs of infection include: respiratory symptoms, fever, cough, shortness of breath, and breathing difficulties. In more severe cases, infection may cause pneumonia, severe acute respiratory syndrome, kidney failure and even death [8].

Creating a mathematical model for the corona virus (COVID-19) is of great importance as it helps to explain the extent of the disease taking into consideration that it is an invisible and infectious virus. Based on this mathematical model, we can judge whether approved measures such as quarantine are sufficient to limit the spread of the virus. The spread of the Corona

virus started in China the most populous country in the world with a population of 1.4 billion according to recent statistics. The density of population in this country surely will make the cases of infection and deaths greater compared to other countries. Our research takes into account the sensitivity of these previous characteristics of this country and for that reason we chose it as a case study.

Many studies and research of mathematical models can be used to analyze the spread of infectious diseases or the social behavior of people [5, 6, 9, 10, 14, 16, 19, 24, 25]. As regard to Corona virus, several different mathematical models have been formulated and studied to help in reducing the number of people who have an infectious disease [2, 3, 9, 10]. M. Tahir et al [2] developed a non-linear mathematical model of MERS-CoV and studied the global stability analysis and also they introduced Lyapunov function. Zhi-Qiang Xia et al [18] constructed two dynamical models to simulate the propagation processes and found out that the basic reproduction number R_0 reaches up to 4.422. They showed that the reasons of the quick spread of the disease are the lack of self-protection and control measures. A. Naheed et al's study [19] of a population model based on the epidemic of (SARS) examined the effect of the diffusion on the spread of the disease and analysed the stability of the numerical solutions.

We will propose a mathematical model that defines and describes the spread of the new Corona virus (COVID-19). The discrete modeling is more realistic but since the data on (COVID-19) is collected daily, we rely on a continuous model in particular because it is less complicated to be processed. Majority cases of (COVID-19) virus spread from human-to-human connection. The virus is transmitted by direct contact with an infected person. So, the use of a boiler model for an infectious disease is very appropriate in this case. We will first test the local stability of the model in both disease-free model and in endemic equilibrium, then we will test the global stability of the model.

The paper is organized as follows: In section 1, the formulation of the model and some basic properties are derived. In section 2, equilibria of the proposed model are obtained and their stability is discussed. In section 3, the global stability of the equilibrium point is discussed. The problem of sensitivity's parameters is discussed in section 4. Some numerical simulations and discussions are given in section 5. Lastly, we give the conclusion of the paper.

1. A MATHEMATICAL MODEL AND BASIC PROPERTIES

1.1. A Mathematical Model. We propose a continuous model $SEIHR$ to describe the interaction within a population where the disease $COVID-19$ exists. The population is divided into five compartments: Susceptible individuals exposed to have new Corona virus $S(t)$, Asymptomatic infected cases or cases with mild symptoms $E(t)$, Infected people with symptoms and carriers of the virus $I(t)$, Hospitalized cases $H(t)$, The recovered cases $R(t)$. The total number of the population at time t is given by $N(t) = S(t) + E(t) + I(t) + H(t) + R(t)$.

The graphical representation of the proposed model is shown in Figure (1).

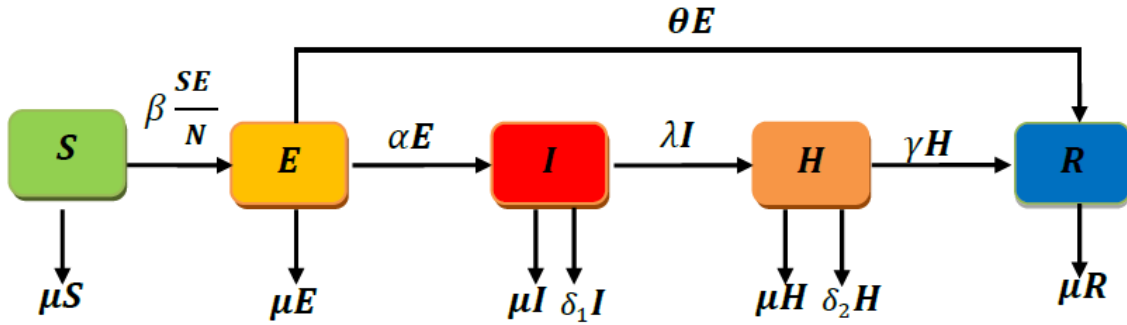


FIGURE 1.

We consider the following system of five non-linear differential equations:

$$(1) \quad \begin{cases} \frac{dS(t)}{dt} = \Lambda - \beta \frac{SE}{N} - \mu S \\ \frac{dE(t)}{dt} = \beta \frac{SE}{N} - (\mu + \alpha + \theta) E \\ \frac{dI(t)}{dt} = \alpha E - (\mu + \alpha + \delta_1) I \\ \frac{dH(t)}{dt} = \lambda I - (\mu + \gamma + \delta_2) H \\ \frac{dR(t)}{dt} = \gamma H + \theta E - \mu R \end{cases}$$

where $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, H(0) \geq 0$, and $R(0) \geq 0$ are the given initial states.

Where Λ represents new birth rate in susceptible human population, β represents the transmission coefficient from susceptible individuals to asymptomatic infected cases or cases with mild symptoms due to the movement and contact that occur among them. μ represents the natural death rate in all compartments. α represents the rate of transmission of asymptomatic

infected cases or cases with mild symptoms to infected individuals with symptoms. λ is the transmission coefficient of the infected people with symptoms to the hospitalized cases. γ is the transmission coefficient of the hospitalized cases to the recovered cases. θ represents the rate of transmission of asymptomatic infected cases or cases with mild symptoms to the recovered cases due the strong immunity of these individuals. δ_1 and δ_2 respectively represent the death rate of the infected individuals and the death rate of the hospitalized cases.

1.2. Basic Properties of the model.

1.2.1. Invariant Region. It is necessary to prove that all solutions of system (1) with positive initial data will remain positive for all times $t > 0$. This will be established by the following lemma.

Lemma 1. *All feasible solutions $S(t), E(t), I(t), H(t)$ and $R(t)$ of system equation (1) are bounded by the region*

$$(2) \quad \Omega = \left\{ (S, E, I, H, R) \in IR_+^5 : S + E + I + H + R \leq \frac{\Lambda}{\mu} \right\}.$$

Proof. From the system equation (1)

$$(3) \quad \frac{dN(t)}{dt} = \Lambda - \mu N(t) - \delta_1 I - \delta_2 H$$

Implies that

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N(t)$$

It follows that

$$(4) \quad N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t}.$$

Where $N(0)$ is the initial value of total number of people, thus

$$(5) \quad \limsup_{t \rightarrow +\infty} N(t) \leq \frac{\Lambda}{\mu}$$

then

$$S(t) + E(t) + I(t) + H(t) + R(t) \leq \frac{\Lambda}{\mu}$$

Hence, for the analysis of model (1), we get the region which is given by the set:

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

which is a positively invariant set for (1). So, we only need to consider the dynamics of the system (1) on the set Ω non-negative of solutions. \square

1.2.2. Positivity of solutions of the model.

Theorem 2. *If $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, H(0) \geq 0$ and $R(0) \geq 0$, then the solutions of system equation (1) $S(t), E(t), I(t), H(t)$ and $R(t)$ are positive for all $t > 0$.*

Proof. From the first equation of the system (1), we have

$$(6) \quad \frac{dS(t)}{dt} = \Lambda - A(t)S(t)$$

where

$$(7) \quad A(t) = \beta \frac{E(t)}{N} + \mu$$

We multiply equation (6) by $\exp\left(\int_0^t A(s)ds\right)$, we find

$$(8) \quad \frac{dS(t)}{dt} * \exp\left(\int_0^t A(s)ds\right) = [\Lambda - A(t)S(t)] * \exp\left(\int_0^t A(s)ds\right)$$

Implies that

$$(9) \quad \frac{dS(t)}{dt} * \exp\left(\int_0^t A(s)ds\right) + A(t)S(t) * \exp\left(\int_0^t A(s)ds\right) = \Lambda * \exp\left(\int_0^t A(s)ds\right)$$

Therefore

$$(10) \quad \frac{d}{dt} \left[S(t) * \exp\left(\int_0^t A(s)ds\right) \right] = \Lambda * \exp\left(\int_0^t A(s)ds\right).$$

Taking integral with respect to s from 0 to t , we get

$$(11) \quad S(t) * \exp\left(\int_0^t A(s)ds\right) - S(0) = \Lambda * \int_0^t \left(\exp\left(\int_0^w A(s)ds\right) \right) dw.$$

Multiplying the equation (11) by $\exp(-\int_0^t A(s)ds)$, we get

$$(12) \quad S(t) - S(0) * \exp(-\int_0^t A(s)ds) = \Lambda * \exp(-\int_0^t A(s)ds) * \int_0^t \left(\exp(\int_0^w A(s)ds) \right) dw$$

Then,

$$(13) \quad S(t) = S(0) * \exp(-\int_0^t A(s)ds) + \Lambda * \exp(-\int_0^t A(s)ds) * \int_0^t \left(\exp(\int_0^w A(s)ds) \right) dw \geq 0.$$

So, the solution $P(t)$ is positive.

Similarly, from the others equations of system (1), we have

$$(14) \quad E(t) \geq E(0) \exp(-\int_0^t \left(\beta \frac{S(s)}{N} - (\mu + \alpha + \theta) \right) ds) \geq 0$$

$$(15) \quad I(t) \geq I(0) \exp[-(\mu + \lambda + \delta_1)t] \geq 0$$

$$(16) \quad H(t) \geq H(0) \exp[-(\mu + \gamma + \delta_2)t] \geq 0$$

$$(17) \quad R(t) \geq R(0) \exp[-(\mu + \theta)t] \geq 0$$

Therefore, we can see that the solutions $S(t)$, $E(t)$, $I(t)$, $H(t)$ and $R(t)$ of the system (1) are positive for all $t \geq 0$. This completes the proof. \square

The first three equations in system (1) are independents of the variables H and R . Hence, the dynamics of equation system (1) is equivalent to the dynamics of equation system:

$$(18) \quad \begin{cases} \frac{dS(t)}{dt} = \Lambda - \beta \frac{SE}{N} - \mu S \\ \frac{dE(t)}{dt} = \beta \frac{SE}{N} - (\mu + \alpha + \theta)E \\ \frac{dI(t)}{dt} = \alpha E - (\mu + \lambda + \delta_1)I \end{cases}$$

2. STABILITY ANALYSIS AND SENSITIVITY OF THE MODEL

2.1. Equilibrium Points: In this model, there are two equilibrium points, that is, COVID-19 disease-free equilibrium point and COVID-19 disease present equilibrium point. The equilibrium points are found by setting the right hand side of the system (18) equal to zero.

The COVID-19 disease-free equilibrium $E_{eq}^0 \left(\frac{\Lambda}{\mu}, 0, 0 \right)$ is achieved in the absence of virus ($E = I = 0$).

The COVID-19 disease present equilibrium $E_{eq}^* (S^*, E^*, I^*)$ is achieved when the disease exists ($E \neq 0$ and $I \neq 0$).

Where:

$$(19) \quad S^* = \frac{N}{\beta}(\mu + \alpha + \theta),$$

$$(20) \quad E^* = \frac{\mu N}{\beta}(R_0 - 1),$$

$$(21) \quad I^* = \frac{\mu N \alpha}{\beta(\mu + \lambda + \delta_1)}(R_0 - 1),$$

$$(22) \quad R_0 = \frac{\Lambda \beta}{\mu N(\mu + \alpha + \theta)}.$$

R_0 is the basic reproduction number that measures the average number of the new infected individuals generated by a single infected individual in a population of susceptible individuals. The value of R_0 will indicate whether the epidemic could occur or not. The reproduction basic number can be determined by using the next generation matrix method formulated in [7].

2.2. Local stability analysis. Now we proceed to study the stability behavior of equilibria E_{eq}^0 and E_{eq}^* .

2.2.1. The disease-free equilibrium. In this section, we analyze the local stability of the COVID-19 disease-free equilibrium.

Theorem 3. *The COVID-19 disease-free equilibrium $E_{eq}^0 \left(\frac{\Lambda}{\mu}, 0, 0 \right)$ of the system (18) is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix at E_{eq} is given by

$$(23) \quad J(E_{eq}) = \begin{pmatrix} -\beta \frac{E}{N} - \mu & -\beta \frac{S}{N} & 0 \\ \beta \frac{E}{N} & \beta \frac{S}{N} - (\mu + \alpha + \theta) & 0 \\ 0 & \alpha & -(\mu + \lambda + \delta_1) \end{pmatrix}$$

The Jacobian matrix for the disease-free equilibrium is given by

$$(24) \quad J(E_{eq}^0) = \begin{pmatrix} -\mu & -(\mu + \alpha) & 0 \\ 0 & \beta \frac{\Lambda}{\mu N} - (\mu + \alpha + \theta) & 0 \\ 0 & \alpha & -(\mu + \lambda + \delta_1) \end{pmatrix}$$

The characteristic equation of this matrix is given by $\det (J(E_{eq}^0) - \lambda I_3) = 0$, where I_3 is a square identity matrix of order 3.

Therefore, we see that the characteristic equation $\varphi(\zeta)$ of $J(E_{eq}^0)$ are:

$$(25) \quad \varphi(\zeta) = -(\mu + \zeta) \left[\left(\beta \frac{\Lambda}{\mu N} - (\mu + \alpha + \theta) - \zeta \right) \left(-(\mu + \lambda + \delta_1) - \zeta \right) \right]$$

Where, eigenvalues of the characteristic equation of $J(E_{eq}^0)$ are:

$$(26) \quad \begin{aligned} \zeta_1 &= -\mu \\ \zeta_2 &= (\mu + \alpha + \theta) (R_0 - 1) \\ \zeta_3 &= -(\mu + \lambda + \delta_1) \end{aligned}$$

And

$$(27) \quad R_0 = \frac{\Lambda \beta}{\mu N (\mu + \alpha + \theta)}.$$

Therefore, all the eigenvalues of the characteristic equation $J(E_{eq}^0)$ are clearly real and negative if $R_0 < 1$. □

We conclude that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

2.2.2. Disease present equilibrium. In this section, we analyze the local stability of the disease present equilibrium. We consider $\frac{dS(t)}{dt} = 0$, $\frac{dE(t)}{dt} = 0$ and $\frac{dI(t)}{dt} = 0$.

We have

$$(28) \quad S^* = \frac{N}{\beta}(\mu + \alpha + \theta)$$

$$(29) \quad = \frac{\Lambda}{\mu R_0}$$

From the second equation in the system (18), we have

$$(30) \quad E^* = \frac{\mu N}{\beta}(R_0 - 1),$$

Also, the third equation in the system (18) gives

$$(31) \quad I^* = \frac{\mu N \alpha}{\beta(\mu + \lambda + \delta_1)}(R_0 - 1),$$

Let the following theorem analysis the local stability of the disease present equilibrium.

Theorem 4. *The COVID-19 disease present equilibrium E_{eq}^* is locally asymptotically stable if $R_0 > 1$, and unstable if $R_0 \leq 1$.*

Proof. We present $E_{eq}^*(S^*, E^*, I^*)$ as the COVID-19 disease present equilibrium of system (18) and $S^* \neq 0, E^* \neq 0, I^* \neq 0$. □

The Jacobian matrix at E_{eq}^* is given by

$$(32) \quad J(E_{eq}^*) = \begin{pmatrix} -\mu R_0 & -(\mu + \alpha + \theta) & 0 \\ \mu(R_0 - 1) & 0 & 0 \\ 0 & \alpha & -(\mu + \lambda + \delta_1) \end{pmatrix}$$

We see that the characteristic equation $\varphi(\zeta)$ of $J(E_{eq}^*)$ is:

$$(33) \quad \varphi(\zeta) = \zeta^3 + a_1 \zeta^2 + a_2 \zeta + a_3$$

where

$$(34) \quad a_1 = \mu R_0 + \mu + \lambda + \delta_1 > 0 ;$$

$$(35) \quad a_2 = \mu(R_0 - 1)(\mu + \alpha + \theta) + \mu R_0(\mu + \lambda + \delta_1) > 0,$$

$$(36) \quad a_3 = \mu(R_0 - 1)(\mu + \alpha + \theta)(\mu + \lambda + \delta_1)$$

By routh- Hurwitz Criterion, the system (18) is locally asymptotically stable if $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1 a_2 > a_3$.

Thus, the present equilibrium E_{eq}^* of system (18) is locally asymptotically stable if $R_0 > 1$.

3. GLOBAL STABILITY

3.1. Global stability of the COVID-19 disease-free equilibrium. To show that the system (18) is globally asymptotically stable, we use the Lyapunov function theory for both the COVID-19 disease free equilibrium and the COVID-19 disease present equilibrium. First, we present the global stability of the COVID-19 disease-free equilibrium E_{eq}^0 .

Theorem 5. *The COVID-19 disease free equilibrium E_{eq}^0 is globally asymptotically stable Ω If $R_0 \leq 1$ and unstable otherwise.*

Proof. Let the following Lyapunov function:

$$(37) \quad \begin{aligned} V & : \quad \Gamma \rightarrow IR \\ V(S, E) & = \frac{1}{2} [(S - S_0) + E]^2 + \frac{N}{\beta} (2\mu + \alpha + \theta) E \end{aligned}$$

where

$$\Gamma = \{(S, E) \in \Gamma / S > 0, E > 0\}$$

Then, the time derivative of the Lyapunov function is given by:

$$(38) \quad \frac{dV(S, E)}{dt} = [\Lambda - \mu S - (\mu + \alpha + \theta)E] [(S - S_0) + E] + \left(\frac{N}{\beta} (2\mu + \alpha + \theta) \right) \frac{dE}{dt}$$

Using $\Lambda = \mu S_0$ to rewrite this, we get

$$\begin{aligned} \frac{dV(S, E)}{dt} & = -\mu(S - S_0)^2 - (\mu + \alpha + \theta)E^2 - (2\mu + \alpha + \theta)(S - S_0)E + \left(\frac{N}{\beta} (2\mu + \alpha + \theta) \right) \frac{dE}{dt} \\ & = -\mu(S - S_0)^2 - (\mu + \alpha + \theta)E^2 + \frac{N}{\beta} (2\mu + \alpha + \theta)(\mu + \alpha + \theta)(R_0 - 1)E \end{aligned}$$

Thus, $\frac{dV(S, E)}{dt} \leq 0$ for $R_0 \leq 1$.

Note that $\frac{dV}{dt} = 0$ if and only if $S = S_0$ and $E = 0$. Hence, by Lasalle's invariance principle [17], E_{eq}^0 is globally asymptotically stable in Ω . \square

3.2. Global stability of the COVID-19 disease present equilibrium. The final result of the global stability of E_{eq}^* in this section is as follows:

Theorem 6. *The disease of COVID-19 disease present equilibrium point E_{eq}^* is globally asymptotically stable if $R_0 > 1$.*

Proof. Let the Lyapunov function V :

$$(39) \quad \begin{aligned} V & : \Gamma \rightarrow \mathbb{R} \\ V(S, E) & = S - S^* \ln\left(\frac{S}{S^*}\right) + E - E^* \ln\left(\frac{E}{E^*}\right) \end{aligned}$$

Where

$$\Gamma = \{(S, E) \in \Gamma / S > 0, E > 0\}$$

Then, the time derivative of the Lyapunov function is

$$(40) \quad \frac{dV(S, E)}{dt} = -\Lambda(S - S^*) \left[\frac{S - S^*}{SS^*} - \frac{\beta}{N}(E - E^*) \right] + \frac{\beta}{N}(E - E^*)(S - S^*)$$

Then

$$(41) \quad \frac{dV(S, E)}{dt} = -\Lambda \frac{[S - S^*]^2}{SS^*} \leq 0$$

Also, we obtain

$$(42) \quad \frac{dV(P, M)}{dt} = 0 \Leftrightarrow S = S^*.$$

Hence, by LaSalle's invariance principle [17] the COVID-19 disease present equilibrium point E_{eq}^* is globally asymptotically stable on Γ . \square

4. SENSITIVITY ANALYSIS OF R_0

Sensitivity analysis is commonly used to determine the model robustness to parameter values, that is, to help us know the parameters that have a high impact on the reproduction number R_0 (because there are usually errors in data collection and assumed parameter values).

Using the approach in Chitnis et al [5], we calculate the normalized forward sensitivity indices of R_0 . Let

$$(43) \quad \Upsilon_n^{R_0} = \frac{\partial R_0}{\partial n} * \frac{n}{R_0},$$

Denote the sensitivity index of R_0 with respect to the parameter n , we get

$$(44) \quad R_0 = \frac{\Lambda\beta}{\mu N(\mu + \alpha + \theta)},$$

$$(45) \quad \Upsilon_\beta^{R_0} = 1,$$

$$(46) \quad \Upsilon_\alpha^{R_0} = -\frac{\alpha}{\mu + \alpha + \theta},$$

$$(47) \quad \Upsilon_\theta^{R_0} = -\frac{\theta}{\mu + \alpha + \theta},$$

$$(48) \quad \Upsilon_\mu^{R_0} = -\frac{\mu}{\mu + \alpha + \theta} - 1,$$

From the above discussion we observe that the basic reproduction number R_0 is most sensitive to changes in β . If β increases R_0 will also increase with the same proportion and if β decreases in the same proportion, μ , α and θ will have an inversely proportional relationship with R_0 . So, an increase in any one of them will bring about a decrease in R_0 . However, the size of the decrease will be proportionally smaller. Recall that μ is the natural death rate of the population. Given R_0 's sensitivity to β , it seems sensible to focus efforts on the reduction of β . In other words, this sensitivity analysis tells us that prevention is better than cure. Efforts to increase prevention are more effective in controlling the spread of habitual COVID-19 disease than efforts to increase the numbers of individuals accessing quarantine.

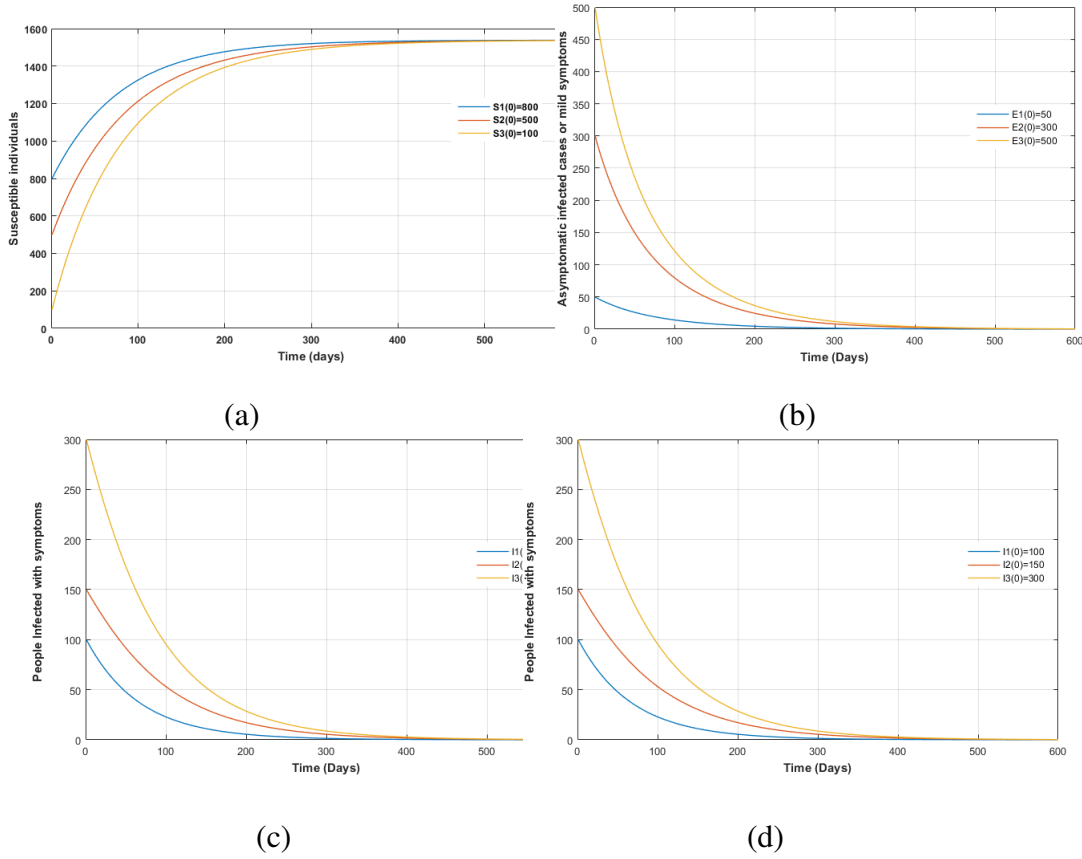
Parameter	Description	Sensitivity index
β	The effective contact rate	+1
α	Coefficient of transmission from E to I	-0.2326
θ	Coefficient of transmission from E to R	-0.0116
μ	The natural death rate	-1.7558

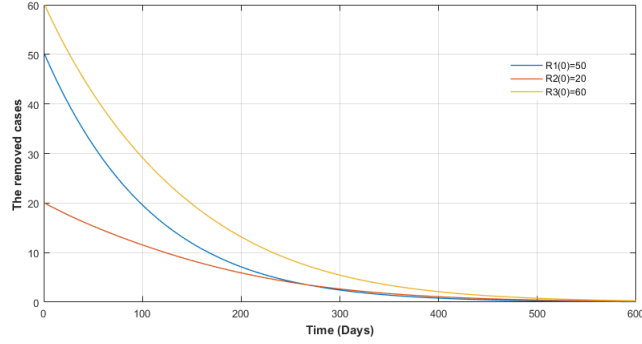
5. NUMERICAL SIMULATIONS :

In this section, we illustrate some numerical solutions of model (1) for different values of the parameters. The resolution of the system (1) was created using the Gauss-Seidel-like implicit finite-difference method developed by Gumel et al [9] presented in [15] and denoted GSS1 method. We use the following different initial values such that $S + E + I + H + R = 1000$.

We use and present some numerical simulations of the system (1) to illustrate our results. By choosing $\Lambda = 100, \beta = 0.025, \alpha = 0.022, \lambda = 0.024, \gamma = 0.015, \mu = 0.065, \delta_1 = 0.001, \delta_2 = 0.004,$

$\theta = 0.001, t_f = 600$ and different initial values for each variable of state, we have the COVID-19 disease free equilibrium $E^0 = (1.5 \times 10^3, 0, 0)$ and $R_0 = 0.437 < 1$. In this case, according to theorem (5), the COVID-19 disease free equilibrium E^0 of the system (1) is globally asymptotically stable on Ω . (See Figure 2).





(e)

FIGURE 2.

We start by a graphic representation of the COVID-19 disease-free equilibrium E^0 and we use the same parameters and different initial values given in table 1. $R_0 = 0.437$ and $R_0 < 1$.

From these figures, using the different values of initial variables S_0, E_0, I_0, H_0 and R_0 , we obtained the following remarks:

The number of potential individuals increases and approaches the number $S_0 = 1536$ (see Figure 2 (a)). Also, the number of the asymptomatic infected cases or cases with mild symptoms decreases and converges to zero (see Figure 2 (b)). The number of the infected people with symptoms and carriers of the virus increases at first, after that it decreases and approaches zero (see Figure 2 (c)). The number of the hospitalized cases decreases and approaches zero (see Figure 2 (d)). The number of the recovered cases decreases and approaches zero (see Figure 2 (e)). Therefore, the solution curves to the equilibrium $E_{eq}^0(S_0, 0, 0, 0, 0)$ when $R_0 < 1$. Hence, model (1) is globally asymptotically stable.

Also, for $\Lambda = 10^2, \beta = 0.25, \alpha = 0.022, \lambda = 0.024, \gamma = 0.015, \mu = 0.065, \delta_1 = 0.001, \delta_2 = 0.004, t_f = 600$. We have the COVID-19 disease equilibrium $E_{eq}^* = (3.52 \times 10^2, 8.76 \times 10^2, 2.14 \times 10^2)$ and $R_0 = 4.37 > 1$. In this case, according to theorem (6), the COVID-19 disease equilibrium E_{eq}^* of the system (1) is globally asymptotically stable on Ω . (See Figure 3).

Also, we begin with a graphic representation of the COVID-19 disease present equilibrium E_{eq}^* and we use the same parameters and different initial values given in table 2, $R_0 = 4.3706$ and $R_0 > 1$.

From these figures, using the different values of initial variables S_0, E_0, I_0, H_0 , and R_0 , we obtained the following remarks:

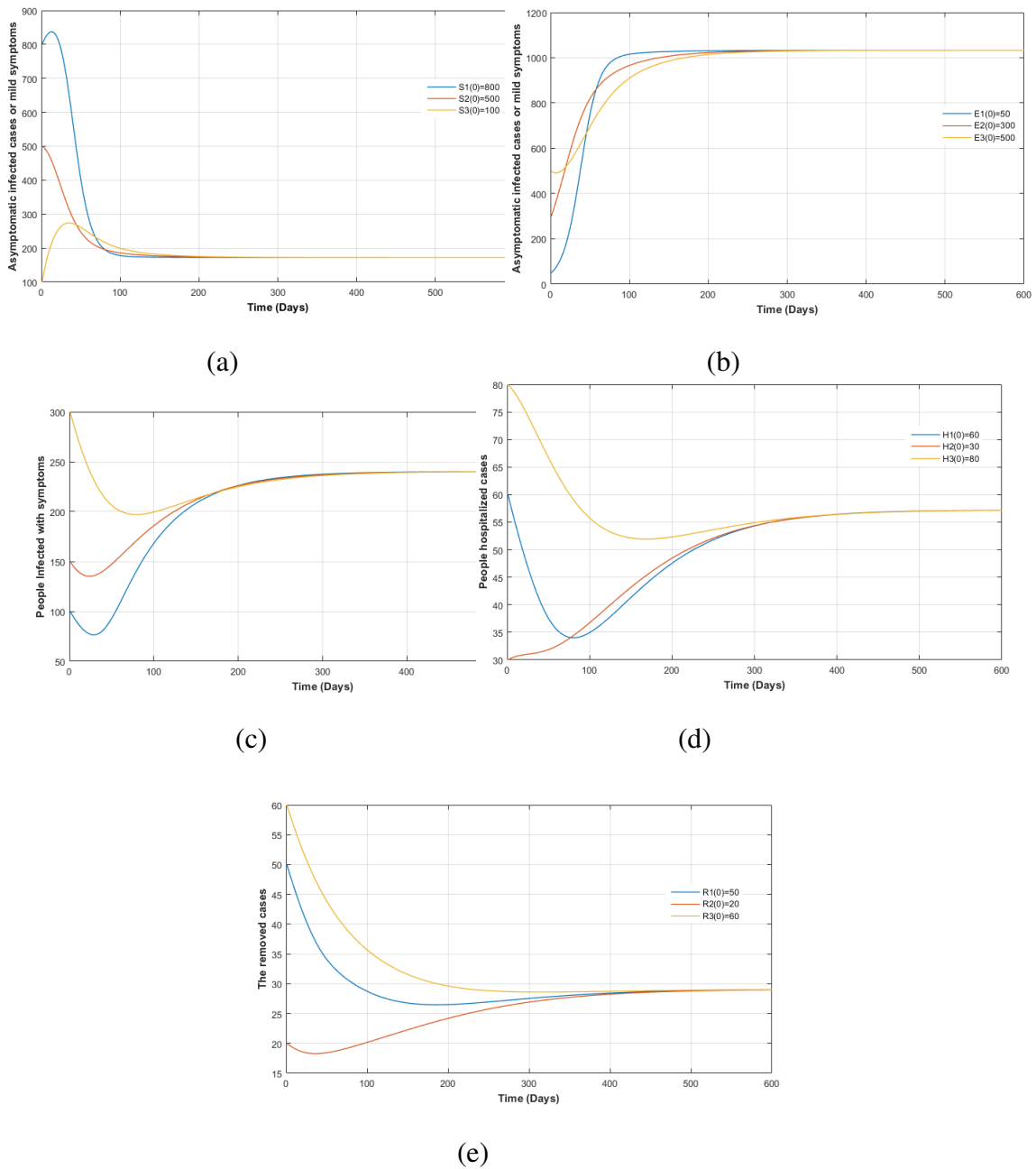


FIGURE 3.

The number of potential individuals increases at first, then it decreases slightly and approaches the value $S^* = 352$ (see Figure 3 (a)). Concerning the number of the asymptomatic infected cases or cases with mild symptoms, it decreases rapidly at first, then it increases slightly and converges the value $E^* = 876$ (see Figure 3 (b)). The number of the infected people with symptoms and carriers of the virus increases and converges the value $I^* = 219$ (see Figure 3 (c)). Also, the number of the hospitalized cases decreases and converges the value $H^* = 62$ (see

Figure 3 (d)). The number of the recovered cases decreases and converges the value $R^* = 28$ (see Figure 3 (e)). Therefore, the solution curves to the equilibrium $E_{eq}^*(S^*, E^*, I^*, H^*, R^*)$ when $R_0 > 1$. Hence, the model (1) is globally asymptotically stable.

6. CONCLUSION

In this work, we formulated and presented a continuous mathematical model *SEIHR* of COVID-19 disease that describes the dynamics of citizens who were infected by this disease. We have found $R_0 = \frac{\Lambda\beta}{\mu N(\mu + \alpha + \theta)}$, as basic reproduction number of the system (1), which helps us to determine the dynamical of the system. We also studied the sensitivity analysis of model parameters to know the parameters that have a high impact on the reproduction number R_0 . We used the stability analysis theory for nonlinear systems to analyze the mathematical COVID-19 disease model and to study both the local and global stability of COVID-19 disease. Local asymptotic stability for the COVID-19 disease-free equilibrium E_{eq}^0 can be obtained if the threshold quantity $R_0 \leq 1$. On the other hand, if $R_0 > 1$, then the COVID-19 disease present equilibrium E_{eq}^* is locally asymptotically stable. A Lyapunov function was used to show that E_{eq}^0 is globally asymptotically stable if $R_0 \leq 1$. Also, a Lyapunov function was used to show that E_{eq}^* is globally asymptotically stable if $R_0 > 1$.

CONFLICT OF INTERESTS

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

REFERENCES

- [1] <https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports/>
- [2] M. Tahir, IS. Ali Shah, G. Zaman, T. Khan, Prevention Strategies for Mathematical Model MERS-Corona Virus with Stability Analysis and Optimal Control. *J Nanosci. Nanotechnol. Appl.* 3 (2018), 101.
- [3] A.M. Zaki, S. van Boheemen, T.M. Bestebroer, A.D.M.E. Osterhaus, R.A.M. Fouchier, Isolation of a Novel Coronavirus from a Man with Pneumonia in Saudi Arabia, *N. Engl. J. Med.* 367 (2012), 1814–1820.
- [4] A. Assiri, J.A. Al-Tawfiq, A.A. Al-Rabeeah, F.A. Al-Rabiah, S. Al-Hajjar, A. Al-Barrak, H. Flemban, W.N. Al-Nassir, H.H. Balkhy, R.F. Al-Hakeem, H.Q. Makhdoom, A.I. Zumla, Z.A. Memish, Epidemiological, demographic, and clinical characteristics of 47 cases of Middle East respiratory syndrome coronavirus disease from Saudi Arabia: a descriptive study, *Lancet Infect. Dis.* 13 (2013), 752–761.

- [5] N. Chitnis, J.M. Hyman, J.M. Cushing, Determining Important Parameters in the Spread of Malaria Through the Sensitivity Analysis of a Mathematical Model, *Bull. Math. Biol.* 70 (2008), 1272–1296.
- [6] M. Tahir, S. Inayat Ali Shah, G. Zaman, S. Muhammad, Ebola virus epidemic disease its modeling and stability analysis required abstain strategies, *Cogent Biol.* 4 (2018). <https://doi.org/10.1080/23312025.2018.1488511>.
- [7] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.* 180 (2002), 29–48.
- [8] <https://www.who.int/health-topics/coronavirus>.
- [9] A.B. Gumel, P.N. Shivakumar, B.M. Sahai, A mathematical model for the dynamics of HIV-1 during the typical course of infection, *Nonlinear Anal., Theory Methods Appl.* 47 (3) (2001), 1773-1783.
- [10] A. Mwaia, J.M. Tchenche, Mathematical analysis of a cholera model with public health interventions, *Biosystems.* 105 (2011), 190–200.
- [11] H.-F. Huo, S.-R. Huang, X.-Y. Wang, H. Xiang, Optimal control of a social epidemic model with media coverage, *J. Biol. Dyn.* 11 (2017), 226–243.
- [12] H.-F. Huo, Y.-P. Liu, The analysis of the SIRS alcoholism models with relapse on weighted networks, *SpringerPlus.* 5 (2016), 722.
- [13] H.F. Huo, N.N. Song, Global Stability for a Binge Drinking Model with Two Stages, *Discrete Dyn. Nat. Soc.* 2012 (2012), Article ID 829386.
- [14] Z. Hu, Z. Teng, and H. Jiang, Stability Analysis in a Class of Discrete SIRS Epidemic Models, *Nonlinear Anal., Real World Appl.* 13 (5) (2012), 2017-2033.
- [15] J. Karrakchou, M. Rachik, S. Gourari, Optimal control and infectiology: Application to an HIV/AIDS model, *Appl. Math. Comput.* 177(2006), 807-818.
- [16] M. Kot, *Elements of Mathematical Ecology*, Cambridge University Press, Cambridge, 2000.
- [17] J.P. La Salle, *The Stability of Dynamical Systems*, Society for Industrial and Applied Mathematics, 1976.
- [18] Z.-Q. Xia, J. Zhang, Y.-K. Xue, G.-Q. Sun, Z. Jin, Modeling the Transmission of Middle East Respirator Syndrome Corona Virus in the Republic of Korea, *PLoS ONE.* 10 (2015), e0144778.
- [19] A. Naheed, M. Singh, D. Lucy, Numerical study of SARS epidemic model with the inclusion of diffusion in the system, *Appl. Math. Comput.* 229 (2014), 480–498.
- [20] X. Ma, Y. Zhou, H. Cao, Global stability of the endemic equilibrium of a discrete SIR epidemic model. *Adv. Difference Equations*, 2013 (2013), 42.
- [21] E.A.P. Poelen, R.H.J. Scholte, G. Willemsen, D.I. Boomsma, R.C.M.E. Engels, Drinking by parents, siblings, and friends as predictors of regular alcohol use in adolescents and young adults: a longitudinal twin-family study, *Alcohol Alcohol.* 42 (2007), 362–369.
- [22] B.A. Reboussin, E.-Y. Song, M. Wolfson, Social Influences on the Clustering of Underage Risky Drinking and Its Consequences in Communities, *J. Stud. Alcohol Drugs.* 73 (2012), 890–898.

- [23] R.H.J. Scholte, E.A.P. Poelen, G. Willemsen, D.I. Boomsma, R.C.M.E. Engels, Relative risks of adolescent and young adult alcohol use: The role of drinking fathers, mothers, siblings, and friends, *Addict. Behav.* 33 (2008), 1–14.
- [24] S. Sharma, G.P. Samanta, Drinking as an epidemic: a mathematical model with dynamic behaviour, *J. Appl. Math. Inform.* 31 (2013), 1–25.
- [25] X.Y. Wang, K. Hattaf, H.F. Huo, H. Xiang, Stability analysis of a delayed social epidemics model with general contact rate and its optimal control, *J. Ind. Manage. Optim.* 12 (4) (2016), 1267-1285.