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Mathematical Modeling of COVID-19 with Chronic Patients and Sensitivity Analysis

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Abstract: Human health is constantly threatened by the appearance and resurgence of several diseases, as shown by recent epidemics. COVID-19 was one of the epidemics that left its mark on the world in terms of economic and human damages. In the search for solution to this pandemic, the scientific community is involved in all its diversity. Mathematicians are taking part in the fight through mathematical modeling in various approaches. Ordinary derivative compartmental modeling approache is one of the techniques widely used in epidemiological modeling. This paper presents a mathematical contribution to fight against COVID-19 using a compartmental SQEICRS model. This model takes into account five stages. In particular, the role of chronic diseases on the dynamique of COVID-19, is focused. A mathematical analysis of the model has been carried out, and shows that the model is well-posed in the biological and mathematical sense. Aspects such as existence, equilibrium points and their stability, the basic reproduction number $\mathcal{R}_0$ and sensitivity anlysis have been discussed. Sensitivity analysis allowed us to identify the parameters which contribute to the spread of the disease, including the chronicity rate due to chronic diseases. The direction of disease propagation was also determined according to $\mathcal{R}_0$. Finally, the numerical results with Matlab are in conformity with theoretical results.

Keywords: COVID-19, Chronic Disease, Mathematical Model, Stability, Sensitivity Analysis, Numerical Simulation

1. Introduction

The COVID-19 pandemic is one of the world's most worrying public health issues. It is a respiratory syndrome caused by the SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) virus, which occurs in various forms, including the Delta variant which is more infectious than the others (Alpha, Beta, Gamma, etc.) [2]. The complexity of this virus lies in its ability to mutate rapidly, not only to counteract the measures taken against it, but also to produce more virulent variants. This disease is potentially dangerous for the elderly and chronically patient, whose bodies are already weakened, and to a lesser extent for young people. It is spreading rapidly and has spared no region in the globe [30]. The impact of barrier measures taken to stem the epidemic's spread has been felt in all sectors of activity, causing a general recession [20, 21]. The search for solutions to this pandemic is therefore an imperative for the scientific community, involving multi-disciplinary skills. Mathematics is playing its part in this challenge through modeling. Several papers have been published on the dynamics of disease transmission and its impact on society. Mathematical models with ordinary derivatives have been formulated on the dynamics of propagation [1, 3, 5, 6, 9, 10, 12, 13]. F. Sulayman et al, M. Manaqib et al, Vijayalakshmi et al and many others have analysed the impact of vaccination on the transmission

dynamics of COVID-19. In other models, the impact of barrier measures in controlling the epidemic has been studied. Kiesha Prem et al. [7], Sasmina et al. [8], Ankamah et al. [29] have performed optimal control on COVID-19 models. Fractional models have been developed by other authors : Shahram, Paul, Jian, Vijayalakshmi. Stochastic models have also been developed by Tomas, Zizhen and Shah. All these publications highlight control mechanisms aimed at stemming the spread of the epidemic. However, there are few articles dealing with the impact of chronic diseases and asymptomatic COVID-19 patients on its transmission dynamics. Therefore this paper aims to study the impact of chronic patients on COVID-19 transmission dynamics, using a compartmental SQEICRS model with ordinary derivatives. Once developed, the model will be subjected to mathematical analysis, during which aspects of existence, uniqueness, equilibrium points and their stability, and sensitivity will be examined. In support to the theoretical results, numerical simulations will be implemented with Matlab.

2. Elaboration of the COVID-19 Model

2.1. Hypotheses

The following assumptions are necessary for model development:

H1) Transmission is human-to-human via direct or indirect contact with symptomatic and asymptomatic infectious individuals [17, 18, 24, 26–28, 30]

H2) In the disease development chain, one has identified 9 stages at each time t. The population of density $N(t)$ is thus divided into 9 compartments whose densities $S(t)$, $S_c(t)$, $Q(t)$, $E(t)$, $I_s(t)$, $I_a(t)$, $C_t(t)$, $C_s(t)$ and $R(t)$ are described in Table 1. On alors:

$$\begin{aligned} N(t) =& S(t) + S_c(t) + Q(t) + E(t) + I_s(t) + I_a(t) \\ &+ C_t(t) + C_s(t) + R(t) \end{aligned} \tag{1}$$

H3) Vertical transmission is neglected ;

H4) Deaths are not part of the disease transmission chain: assuming that COVID-19-related deaths are well managed up to burial, therefore one can consider that they are no longer in the epidemic's transmission chain.

H5) There is no immunity [1, 4]: after a brief immunity, cured individuals fall back into the susceptible compartment.

H7) When a patient has several chronical diseases with vulnerability rates b_i, $i \in \{1, 2, ..., n\}$, then the vulnerability rate b to COVID-19 is given by $b = \max(b_i)$. Assume that $b_i \geq 1$.

Under the above assumptions, the dynamics of dengue propagation can be described by the diagram of figure 1:

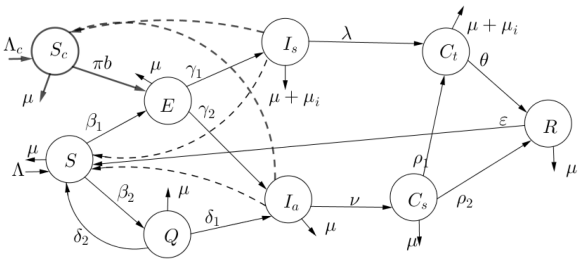

Figure 1. *COVID-19 transmission diagram.*

2.2. Description of Variables and Parameters

Variables and parameters of the model are described in tables 1 and 2 respectively.

Table 1. *Description of model variables.*

Variables	Description
$S(t)$	Density of susceptible individuals
$S_c(t)$	Density of susceptible individuals with chronical diseases
$E(t)$	Density of exposed individuals
$Q(t)$	Density of quarantined individuals
$I_s(t)$	Density of symptomatic infectious individuals
$I_a(t)$	Density of asymptomatic infectious individuals
$C_t(t)$	Density of individuals confined under treatment
$C_s(t)$	Density of individuals confined without treatment
$R(t)$	Density of recovered individuals

Table 2. *Description of model parameters.*

Parameters	Description
Λ	recruitment rate
Λ_c	recruitment rate of chronical individuals
b	rate of vulnerability induced by chronic disease
μ	natural death rate
β_1	adequate contacts rate of susceptible individuals without chronic diseases
π	adequate contact rates of chronic patients
β_2	rate of quarantined individuals
δ_1	infection rates of quarantined individuals
δ_2	rate of vulnerability of quarantined individuals
γ_1	rate of symptomatic infections
γ_2	rate of asymptomatic infections
θ	cure rate of symptomatic infections
ρ_2	cure rate for asymptomatic infections
ρ_1	aggravation rate of asymptomatic confined patients
ε	rate of vulnerability of recovered individuals
ε_c	vulnerability rate of chronic patients cured of COVID-19
u_i	rate induced by COVID-19

3. Results

3.1. Model

Using a stantard incidence:

$$f(t, I_s(t), I_a(t)) S(t) = \frac{I_s(t) + I_a(t)}{N(t)} S(t) \tag{2}$$

on diagram 1, the model of COVID-19 is formulate by system (3)-(11):

$$\frac{dS(t)}{dt} = \Lambda + \delta_2 Q(t) + \varepsilon R(t) - \mu S(t) - (\beta_1 + \beta_2)\, f\,(t, I_s(t), I_a(t))\, S(t) \tag{3}$$

$$\frac{dS_c(t)}{dt} = \Lambda_c + \varepsilon_c R(t) - \pi b f\,(t, I_s(t), I_a(t))\, S_c(t) - \mu S_c(t) \tag{4}$$

$$\frac{dQ(t)}{dt} = \beta_2 f\,(t, I_s(t), I_a(t))\, S(t) - (\delta_1 + \delta_2 + \mu)\, Q(t) \tag{5}$$

$$\frac{dE(t)}{dt} = \beta_1 f\,(t, I_s(t), I_a(t))\, S(t) + \pi b f\,(t, I_s(t), I_a(t))\, S_c(t) - (\mu + \gamma_1 + \gamma_2) E(t) \tag{6}$$

$$\frac{dI_s(t)}{dt} = \gamma_1 E(t) - (\mu + \mu_i + \lambda)\, I_s(t) \tag{7}$$

$$\frac{dI_a(t)}{dt} = \delta_1 Q(t) + \gamma_2 E(t) - (\mu + \nu)\, I_a(t) \tag{8}$$

$$\frac{dC_s(t)}{dt} = \nu I_a(t) - (\mu + \rho_1 + \rho_2)\, C_s(t) \tag{9}$$

$$\frac{dC_t(t)}{dt} = \lambda I_s(t) + \rho_2 C_s(t) - (\mu + \mu_i + \theta)\, C_t(t) \tag{10}$$

$$\frac{dR(t)}{dt} = \theta C_t(t) + \rho_2 C_s(t) - (\mu + \varepsilon + \varepsilon_c)\, R(t) \tag{11}$$

whith the initial condition (12):

$$x_0 = (S(0), S_c(0), Q(0), E(0), I_s(0), I_a(0), C_s(0),\ \ C_t(0), R(0))^T \in \mathbb{R}_+^9 \tag{12}$$

The total density is governed by the equation (13):

$$\frac{dN(t)}{dt} = \Lambda + \Lambda_c - \mu N(t) - \mu_i I_s(t) - \mu_i C_t(t) \tag{13}$$

3.2. Existence and Uniqueness of Solution

Let

$$x(t) = (S(t), S_c(t), Q(t), E(t), I_s(t), I_a(t), C_s(t),\ \ C_t(t), R(t))^T \tag{14}$$

be the variable state of system (3)-(11) for any $t \geq 0$.

Lemma 3.1. The domain Ω of the solutions defined by:

$$\Omega = \{x(t) \in \mathbb{R}_+^9 \quad | \quad 0 < N(t) \leq \frac{\Lambda + \Lambda_c}{\mu}\} \tag{15}$$

is positively invariant.

Proof. According to the system (3)-(11):

$$\frac{dS(t)}{dt} \geq -\left((\beta_1 + \beta_2)\, f\,(I_s, I_a) + \mu\right) S(t)$$

$$\frac{\dot{S(t)}}{S(t)} \geq -\left(\left(\beta_1+\beta_2\right) f\left(I_s, I_a\right)+\mu\right)$$

$$S(t) \geq S(0) \exp\left(-\mu t-\int_0^t\left(\beta_1+\beta_2\right) f\left(I_s, I_a\right) dt\right) \geq 0$$

$$\frac{dS_c(t)}{dt} \geq -\left(\pi b f\left(I_s, I_a\right)+\mu\right) S_c(t)$$

$$S_c(t) \geq S_c(0) \exp\left(-\mu t-\pi b \int_0^t f\left(I_s, I_a\right) dt\right) \geq 0$$

$$\frac{dE(t)}{dt} \geq -(\mu+\gamma_1+\gamma_2)E(t)$$

$$E(t) \geq E(0) \exp\left(\mu+\gamma_1+\gamma_2\right) t$$

$$E(t) \geq 0$$

$$\frac{dQ(t)}{dt} \geq -(\mu+\delta_1+\delta_2)E(t)$$

$$Q(t) \geq Q(0) \exp\left(\mu+\delta_1+\delta_2\right) t$$

$$Q(t) \geq 0$$

By analogy: $I_s(t) \geq 0$, $I_a(t) \geq 0$, $C_s(t) \geq 0$, $C_t(t) \geq 0$, $R(t) \geq 0$ and consequently $N(t) \geq 0$. In the absence of the disease, $N(t)=\dfrac{\Lambda+\Lambda_c}{\mu}$.

Hence $0 \leq N(t) \leq \dfrac{\Lambda+\Lambda_c}{\mu}$.

Theorem 3.1. For any initial condition $x_0 \in \Omega$, the model (3)-(11) admits a unique solution which remains in Ω for all $t \geq 0$.

Proof Let $g=\left(g_1, g_2, g_3, g_4, g_5, g_6, g_7, g_8, g(9)\right)^T$ be the function defined from in $\mathbb{R}^9$ by 16:

$$g(x):=\begin{pmatrix}\Lambda+\delta_2 Q(t)+\varepsilon R(t)-\left(\beta_1+\beta_2\right) f\left(I_s, I_a\right) S(t)-\mu S(t) \\ \Lambda_c+\varepsilon_c R(t)-\pi b f\left(t, I_s, I_a\right) S_c(t)-\mu S_c(t) \\ \beta_2 f\left(I_s, I_a\right) S(t)-\left(\delta_1+\delta_2+\mu\right) Q(t) \\ (\pi b+\beta_1) f\left(I_s, I_a\right) S(t)-(\mu+\gamma_1+\gamma_2)E(t) \\ \gamma_1 E(t)-\left(\mu+\mu_i+\lambda\right) I_s(t) \\ \delta_1 Q(t)+\gamma_2 E(t)-\left(\mu+\nu\right) I_a(t) \\ \nu I_a(t)-\left(\mu+\rho_1+\rho_2\right) C_s(t) \\ \lambda I_s(t)+\rho_1 C_s(t)-\left(\mu+\mu_i+\theta\right) C_t(t) \\ \theta C_t(t)+\rho_2 C_s(t)-\left(\mu+\varepsilon\right) R(t)\end{pmatrix} \tag{16}$$

By using (14), the system (3)-(11) can be written in the equivalent form of Cauchy problem (17):

$$\begin{cases}\dfrac{dx(t)}{dt}=g(t, x); \quad t>0 \\ x(0)=x_0 \in \Omega\end{cases} \tag{17}$$

The function g is $C^{\infty}(\Omega)$ and $C^1(\Omega)$ in particular. Therefore g is locally lipschitzian with respect to its second variable. Thus the Cauchy-Lipschitz theorem ensures the existence and uniqueness of the solution $x(t)$ of (17) for any initial condition $x_0 \in \Omega$ and any $t \geq 0$. According to the lemma 3.1, $x(t) \in \Omega$ for all $t \geq 0$.

Remark 3.1. Any solution of the model (3)-(11) verifying the initial condition $x_0 \in \Omega$ is global in Ω.

Proof By the lemma 3.1, the domain Ω is a closed subset in $\mathbb{R}^8$ which is positively invariant by (3)-(11). Therefore it is a compact set. By theorem 3.1 and Proposition 1.1 [25] lead to the conclusion.

Theorem 3.2. The model (3)-(11) admits:

a) an unique disease-free equilibrium
$\mathcal{E}_0 = \left(\frac{\Lambda}{\mu}, \frac{\Lambda_c}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0, 0\right)$

b) an unique endemic equilibrium
$\mathcal{E}_e = (S^*, S_c^*, Q^*, E^*, I_s^*, I_a^*, C_s^*, C_t^*, R^*)$

where

$$^* = \frac{\Lambda}{\mu + (\beta_1 + \beta_2) G} + \left[\frac{a_1 a_2 a_3}{\nu \delta_1} + \frac{\varepsilon a_4}{\mu + \varepsilon + \varepsilon_c}\right] C_t^* - \left[\frac{\lambda a_1 a_1}{\nu \rho_2 \delta_1} + \frac{a_5}{\gamma_1 \delta_1}\gamma_2 + \frac{\lambda \varepsilon_c}{a_6}\right] I_s^*$$

$$S_c^* = \frac{\Lambda_c}{\mu + \pi b G} + \frac{\varepsilon_c a_4}{a_6} C_t^* - \frac{\rho_2 \rho_2 \varepsilon_c}{\lambda a_6} I_s^*$$

$$Q^* = \frac{(\mu + \mu_i + \theta)\, a_2}{\nu \rho_2} \frac{a_3}{\delta_1} C_t^* - \left[\frac{a_5}{\gamma_1 \delta_1}\delta_2 + \frac{\lambda a_2 a_3}{\nu \rho_2 \delta_1}\right] I_s^*$$

$$E^* = \frac{a_5}{\gamma_1} I_s^*$$

$$I_a^* = \frac{a_1 a_2}{\nu \rho_2} C_t^* - \frac{\lambda a_2}{\nu \rho_2} I_s^*$$

$$C_s^* = \frac{a_1}{\rho_2} C_t^* - \frac{\lambda}{\rho_2} I_s^*$$

$$N^* = \frac{\Lambda + \Lambda_c}{\mu} - \frac{\mu_i}{\mu}\left(I_s^* + C_t^*\right)$$

and

$$G = \frac{\dfrac{a_1 a_2}{\nu \rho_2} C_t^* + \left(1 - \dfrac{\lambda a_2}{\nu \rho_2}\right) I_s^*}{\dfrac{\Lambda + \Lambda_c}{\mu} - \dfrac{\mu_i}{\mu}\left(I_s^* + C_t^*\right)}$$

$$a_1 = \mu + \mu_i + \theta \quad a_2 = \mu + \rho_1 + \rho_2 \quad a_3 = \mu + \nu \quad a_4 = \mu + \mu_i + 2\theta \quad a_5 = \mu + \mu_i + \lambda \quad a_6 = \mu + \varepsilon + \varepsilon_c$$

Proof

a) Equilibrium $\mathcal{E}_0$ is obtained by solving the system $g(x) = 0$ under the constraints $I_s(t) = I_a(t) = 0$.

b) Equilibrium $\mathcal{E}_e$ is obtained by solving the system $g(x) = 0$ without constraints.
Assuming that the numbers of symptomatic patients and patients confined to treatment are more manageable at each instant, and express the densities of the other compartments in terms of the latter.

3.3. Basic Reproduction Number $\mathcal{R}_0$

The reproduction number $\mathcal{R}_0$ is a very important parameter (a threshold) in epidemiology, providing information on the evolution of the disease [15]. The most widely used method for estimating the basic reproduction number is that of P. Van den Driessche et al. [15].

Assuming $x = (x_1, x_2, x_3) = (E, I_s, I_a)$ then the matrices F (of new infections) and V (of transition) are defined as follows:

$$F = \left[\frac{\partial \mathcal{F}_i(x_e^*)}{\partial x_j}\right]_{1 \le i,j \le 4} \quad and \quad V = \left[\frac{\partial \mathcal{V}_i(x_e^*)}{\partial x_j}\right]_{1 \le i,j \le 4}$$

whith

$$\mathcal{F}(X) = \begin{pmatrix} \beta_1 \dfrac{I_s + I_a}{N} S + \pi b \dfrac{I_s + I_a}{N} S_c \\ 0 \\ 0 \end{pmatrix} \tag{18}$$

and

$$\mathcal{V}(X) = \begin{pmatrix} (\mu + \gamma_1 + \gamma_2) E \\ -\gamma_1 E + (\mu + \mu_i + \lambda) I_s \\ -\delta_1 Q - \gamma_2 E + (\mu + \nu) I_a \end{pmatrix} \tag{19}$$

Then

$$F = \begin{pmatrix} 0 & \beta_1 + \pi b & \beta_1 + \pi b \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{20}$$

and

$$V = \begin{pmatrix} \mu + \gamma_1 + \gamma_2 & 0 & 0 \\ -\gamma_1 & \mu + \mu_i + \lambda & 0 \\ -\gamma_2 & 0 & \mu + \nu \end{pmatrix} \tag{21}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{a_7} & 0 & 0 \\ \frac{1}{a_2 a_7}\gamma_1 & \frac{1}{a_2} & 0 \\ \frac{1}{a_3 a_7}\gamma_2 & 0 & \frac{1}{a_3} \end{pmatrix} \tag{22}$$

with $a_7 = \mu + \gamma_1 + \gamma_2$, $a_8 = \beta_1 + \pi b$.

Then the matrix of the next generation is

$$FV^{-1} = \begin{pmatrix} \frac{1}{a_2 a_3 a_7}(\beta_1 \gamma_1 a_3 + a_8 \gamma_2 a_2) & \frac{1}{a_2} a_8 & \frac{1}{a_3} a_8 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{23}$$

Then

$$\begin{aligned} \mathcal{R}_0 &= \rho\left(FV^{-1}\right) \\ &= (\beta_1 + \pi b) \frac{(\mu + \mu_i + \lambda)\gamma_2 + \gamma_1 (\mu + \nu)}{(\mu + \gamma_1 + \gamma_2)(\mu + \mu_i + \lambda)(\mu + \nu)} \end{aligned} \tag{24}$$

3.4. Sensitivity Study

The stability indices are as follows:

$$\Upsilon_{\beta_1}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_1} \times \frac{\beta_1}{\mathcal{R}_0} = 1 > 0$$

$$\Upsilon_{\pi}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \pi} \times \frac{\pi}{\mathcal{R}_0} = b > 0$$

$$\Upsilon_{b}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial b} \times \frac{b}{\mathcal{R}_0} = \pi > 0$$

$$\Upsilon_{\gamma_2}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \gamma_2} \times \frac{\gamma_2}{\mathcal{R}_0} = \frac{(\mu + \gamma_2)(\mu + \nu) - \gamma_2 (\mu + \mu_i + \lambda)}{(\mu + \gamma_1 + \gamma_2)(\gamma_2 (\mu + \mu_i + \lambda) + \gamma_1 (\mu + \nu))} > 0$$

$$\Upsilon_{\gamma_1}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \gamma_1} \times \frac{\gamma_1}{\mathcal{R}_0} = \frac{(\mu + \gamma_1)(\mu + \mu_i + \lambda) - \gamma_1 (\mu + \nu)}{(\mu + \gamma_1 + \gamma_2)(\gamma_2 (\mu + \mu_i + \lambda) + \gamma_1 (\mu + \nu))} < 0$$

$$\Upsilon_{\lambda}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \lambda} \times \frac{\lambda}{\mathcal{R}_0} = \frac{-\lambda \gamma_1 (\mu + \nu)}{(\mu + \mu_i + \lambda)(\gamma_2 (\mu + \mu_i + \lambda) + \gamma_1 (\mu + \nu))} < 0$$

$$\Upsilon_{\nu}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \nu} \times \frac{\nu}{\mathcal{R}_0} = \frac{-\nu \gamma_2 (\mu + \mu_i + \lambda)}{(\mu + \nu)(\gamma_2 (\mu + \mu_i + \lambda) + \gamma_1 (\mu + \nu))} < 0$$

$$\Upsilon_{\mu_i}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mu_i} \times \frac{\mu_i}{\mathcal{R}_0} = \frac{-\mu_i \gamma_1 (\mu + \nu)}{(\mu + \mu_i + \lambda)(\gamma_2 (\mu + \mu_i + \lambda) + \gamma_1 (\mu + \nu))} < 0$$

$$\Upsilon_{\mu}^{\mathcal{R}_0} = -\frac{\gamma_2 \left(a_7^2 a_3 + a_2^2 a_3\right) + \gamma_1 a_3^2 (a_2 + a_7)}{a_2^2 a_3^2 a_7^2} < 0$$

Then the parameters β_1, π, b and γ_2, whose sensitivity indices are positive, potentially contribute to the spread of the disease, while the parameters μ, μ_i, ν, θ and λ, whose sensitivity indices are negative, have no influence on its spread. Therefore, a slight increase in β_1, π, b and γ_2 can turn into a highly endemic situation. Then $\mathcal{R}_0$ can be expressed as it follows:

$$\mathcal{R}_0 = k (\beta_1 + \pi b) \tag{25}$$

avec $k = \frac{(\mu+\mu_i+\lambda)\gamma_2+\gamma_1(\mu+\nu)}{(\mu+\gamma_1+\gamma_2)(\mu+\mu_i+\lambda)(\mu+\nu)}$

One can see that $\mathcal{R}_0$ is an increasing function in b, β_1 and π.

3.5. Stability of Disease-free Equilibrium

The local stability of the disease-free equilibrium depends on the number of basic reproductions $\mathcal{R}_0$ according to the theorem below:

Theorem 3.3. The disease-free equilibrium state $\mathcal{E}_0$ is locally asymptotically stable when $0 \leq \mathcal{R}_0 < 1$ and unstable otherwise.

Proof The proof can be obtained using Liapunov's theorem and Routh's criterion.

Theorem 3.4. The disease-free equilibrium $\mathcal{E}_e$ is globally asymptotically stable (GAS) when $\mathcal{R}_0 < 1$.

Proof Our approach is similar to those developed by J. C. Kamgang et al. [23]. Let us check whether the model (3)-(11) satisfies the assumptions H_1 to H_5 of Theorem 4.3 [23]. Let $x_1 = (S, S_c, Q, C_s, C_t, R)$ and $x_2 = (E, I_s, I_a)$. For simplicity's sake, x_1 can be denoted by $(x_1, 0) \in R^5 \times \mathbb{R}^4$ and x_2 by $(0, x_2) \in \mathbb{R}^5 \times \mathbb{R}^4$. With these notations, the system (3)-(11) can then be written:

$$\begin{cases} \dot{x}_1 = A_1(x).(x_1 - \mathcal{E}_0) + A_{12}(x).x_2 \\ \dot{x}_2 = A_2(x).x_2 \end{cases} \tag{26}$$

The system (26) is of course defined on the positively invariant compact $\Omega \subset \mathbb{R}^9_+$ and satisfies the assumption H_1.

The subsystem $\dot{x}_1 = A_1(x).(x_1 - \mathcal{E}_0)$ is expressed by (27) and the subsystem $\dot{x}_2 = A_2(x).x_2$ by (28):

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda + \delta_2 Q(t) + \varepsilon R(t) - \mu S \\ \dfrac{dS_c(t)}{dt} = \Lambda_c + \varepsilon_c R(t) - \mu S_c(t) \\ \dfrac{dQ(t)}{dt} = -(\delta_1 + \delta_2 + \mu) Q(t) \\ \dfrac{dC_s(t)}{dt} = -(\mu + \rho_1 + \rho_2) C_s(t) \\ \dfrac{dC_t(t)}{dt} = \rho_2 C_s(t) - (\mu + \mu_i + \theta) C_t(t) \\ \dfrac{dR(t)}{dt} = \theta C_t(t) + \rho_2 C_s(t) - (\mu + \varepsilon) R(t) \end{cases} \tag{27}$$

$$\begin{cases} \dfrac{dE(t)}{dt} = \beta_1 f(I_s(t), I_a(t)) S(t) - (\mu + \gamma_1 + \gamma_2) E(t) \\ \dfrac{dI_s(t)}{dt} = \gamma_1 E(t) - (\mu + \mu_i + \lambda) I_s(t) \\ \dfrac{dI_a(t)}{dt} = \gamma_2 E(t) - (\mu + \nu) I_a(t) \end{cases} \tag{28}$$

$$A_2(x) = \begin{pmatrix} -(\mu + \gamma_1 + \gamma_2) & \dfrac{\beta_1 S + \pi b S_c}{N} & \dfrac{\beta_1 S + \pi b S_c}{N} \\ \gamma_1 & -(\mu + \mu_i + \lambda) & 0 \\ \gamma_2 & 0 & -(\mu + \nu) \end{pmatrix} \tag{29}$$

Comme le stipule l'hypothèse H_3, $A_2(x)$ est une matrice de Metzler irréductible pour tout $x \in \Omega$

Pour tout $x \in \Omega$, la matrice $A_2(x)$ admet un maximum atteint pour $S = N$ c'est-?dire au point d'équilibre $\mathcal{E}_0$. Soit J_2 cette matrice maximale. Alors J_2 n'est rien d'autre la la matrice jacobienne de (28) en $\mathcal{E}_0$:

$$J_2 = \begin{pmatrix} -(\mu + \gamma_1 + \gamma_2) & \beta_1 + \pi b & \beta_1 + \pi b \\ \gamma_1 & -(\mu + \mu_i + \lambda) & 0 \\ \gamma_2 & 0 & -(\mu + \nu) \end{pmatrix} \tag{30}$$

In addition, $J_2 = A_2(x_1^*, 0)$, which satisfies assumption H_4.

Since the first four hypotheses hold, then according to hypothesis H_5, one must have $\alpha(J_2) \leq 0$.

Let us look for the sign of the real parts of the eigenvalues of J_2 by the Routh-Hurwitz criterion. The characteristic polynomial $P(y)$ of J_2 is:

$$P(y) = y^3 - (J_{33} + J_{44} + J_{55}) y^2 + (J_{55}(J_{33} + J_{44}) - (\beta_1 + \pi b)(\gamma_1 + \gamma_2) + J_{33} J_{44}) y - J_{33} J_{44} J_{55}(1 - \mathcal{R}_0) \tag{31}$$

avec $J_{33} = -(\mu + \gamma_1 + \gamma_2)$, $J_{44} = -(\mu + \mu_i + \lambda)$, $J_{55} = -(\mu + \nu)$

The Routh-Hurwitz coefficients of P(y) are computed in table 3:

Table 3. *Routh-Hurwitz coefficients of P(y).*

y^3	$a_3 = 1$	$a_1 = J_{55}(J_{33} + J_{44}) - (\beta_1 + \pi b)(\gamma_1 + \gamma_2) + J_{33} J_{44}$
y^2	$a_2 = -J_{33} - J_{44} - J_{55}$	$a_0 = -J_{33} J_{44} J_{55}(1 - \mathcal{R}_0)$
y^1	$b_1 = \dfrac{-1}{a_2}(a_0 - a_1 a_2)$	0
y^0	$c_0 = a_0$	0

The Routh coefficients a_3, a_2 and b_1 are all positive whereas $c_0 > 0 \Leftrightarrow \mathcal{R}_0 < 1$ So when $\mathcal{R}_0 < 1$, then all eigenvalues of J_2 have strictly negative real parts. It follows that $\alpha(J_2) \leq 0$. Consequently, the hypothesis H_5 is verified, which completes the proof. The global stability of the disease-free equilibrium means that, for any initial condition, the solution of the system always returns to this equilibrium.
According to the global stability condition of the disease-free equilibrium $\mathcal{E}_0$, the condition $\beta_1 + \pi b < \frac{(\mu+\gamma_1+\gamma_2)(\mu+\mu_i+\lambda)(\mu+\nu)}{(\mu+\mu_i+\lambda)\gamma_2+\gamma_1(\mu+\nu)}$ is sufficient for the disease extinction.

Theorem 3.5. The endemic equilibrium $\mathcal{E}_e$ is globally asymptotically stable when $\mathcal{R}_0 > 1$.

3.6. Numerical Simulation

It has been proven earlier that the model (3)-(11) has a unique solution in Ω. However, its analytical solving is very difficult. Therefore one turned to numerical solution using the 4 Runge-Kutta algorithm in Matlab [19, 22].

The parameters have been estimated in table 4:

Table 4. *Routh-Hurwitz coefficients of P(y).*

Parameters	Values	Sources
Λ	1000	estimed
μ	0.0118	[11]
μ_i	0.03	[30]
β_1	$[0.0805; 0.25]$	[13, 14, 16]
β_2	$[0.0805; 0.25]$	[13, 14, 16]
π	$[0.0805; 0.25]$	[13, 14, 16]
b	$[1; 5]$	estimed
δ_1	$\frac{1}{7}$	[13]
δ_2	$\frac{1}{7}$	[13]
γ_1	$\frac{1}{7}$	[13]
λ	0.45	[14]
γ_2	$\frac{1}{7}$	[13]
θ	$\frac{1}{14}$	[13]
ρ_1	0.175	[13]
ρ_2	$\frac{1}{14}$	[13]
ε	0.75	estimed
ε_c	0.25	estimed

3.6.1. Disease Extinction

In this section, curves are obtained for $\mathcal{R}_0 < 1$ under the initial condition $(S(0), S_c(0), Q(0), E(0), I_s(0), I_a(0), C_s(0), C_t(0), R(0)) = (100000; 20000; 5000; 1000; 500; 3000; 700; 500; 3000)$. The dynamics of the infection are illustrated in figures 2 to 5 for $\mathcal{R}_0 = 0.61$.

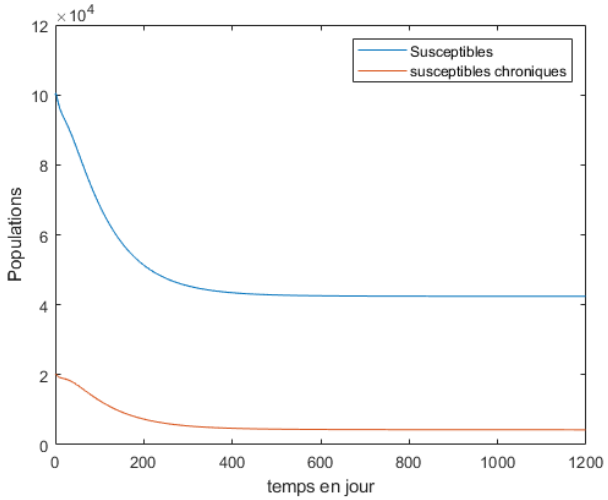

Figure 2. *Density of susceptible individuals .*

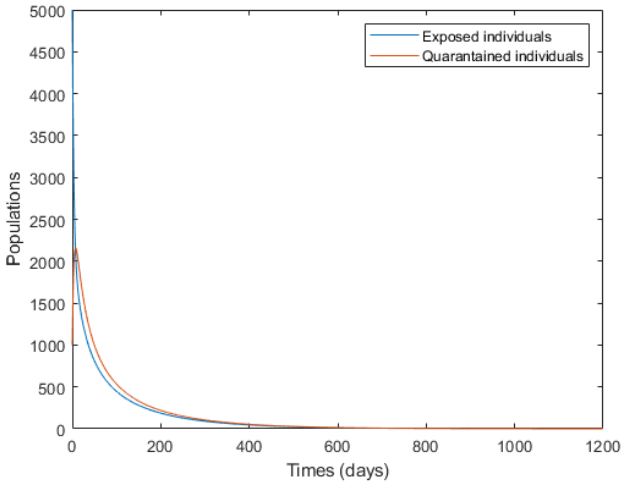

Figure 3. *Density of exposed and quarantained individuals.*

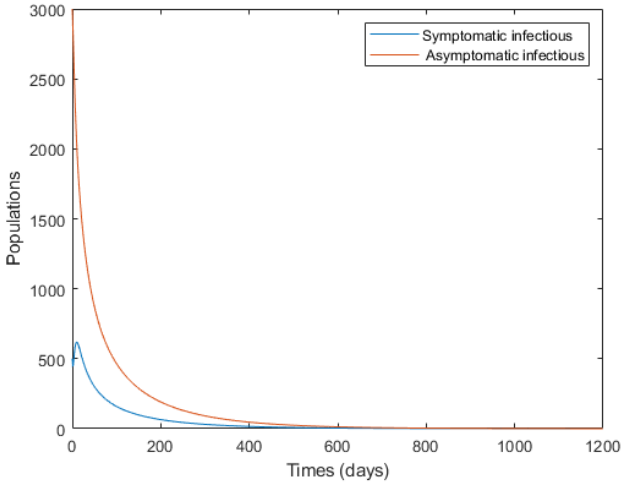

Figure 4. *Density of infectious individuals.*

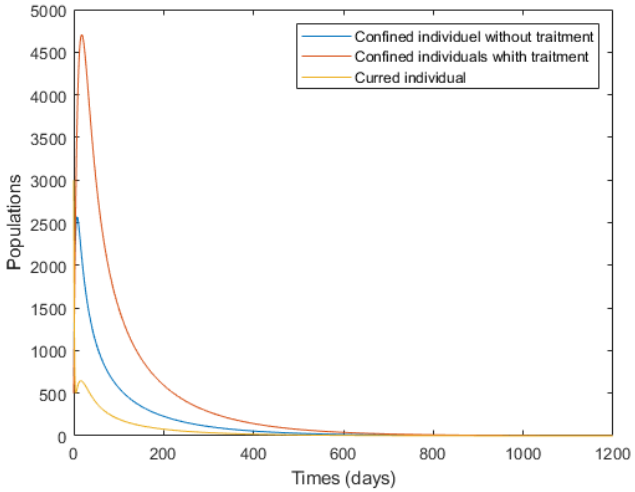

Figure 5. *Density of confined and recovered individuals.*

This figure shows the extinction of disease through the curves of figures 3, 4 and 5.

3.6.2. Persistence of the Disease

This section illustrate the epidemic dynamics for $\mathcal{R}_0 > 1$ under the initial condition $(S(0), S_c(0), Q(0), E(0), I_s(0), I_a(0), C_s(0), C_t(0), R(0)) = (100000; 20000; 500; 100; 50; 300; 70; 50; 300)$.

The dynamics of the disease are presented in figures 6 to 9 for $\mathcal{R}_0 = 2.06$.

The permanent occurrence of infectious individuals in the curves of the figure 8 attest the persistence of the disease.

The role of chronicity on COVID-19 dynamics can be perceived through the graphs in figures 7 to 9.

When $b = 1$, it means that there is no chronical disease. In this cas, all the susceptible individuals are vulnerable in the same degree.

In the otherwise i.e. if $b > 1$, then the vulnerability rate of chronic patients increases, which can sustain the disease within the population. See on figures 10 to 13.

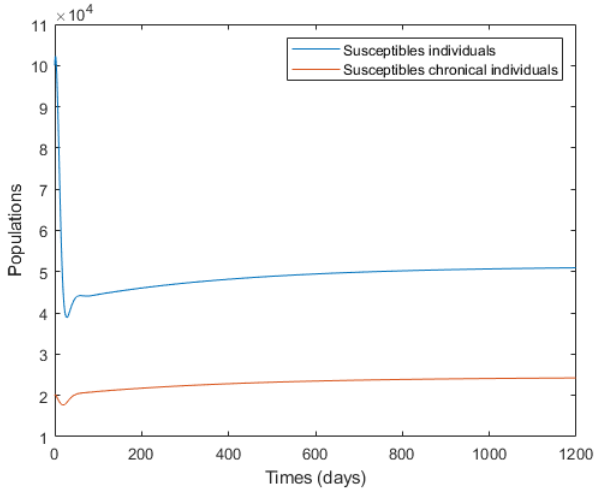

Figure 6. *Density of susceptible individuals .*

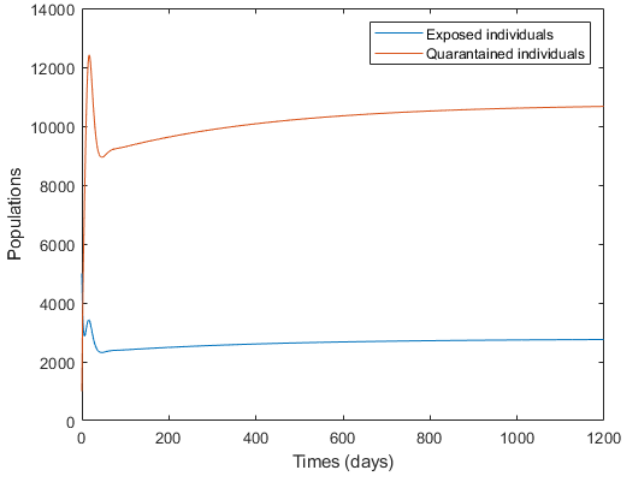

Figure 7. *Density of exposed and quarantained individuals.*

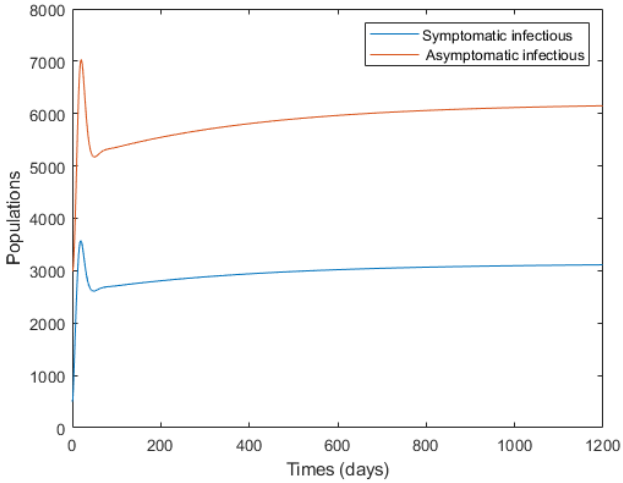

Figure 8. *Density of infectious individuals.*

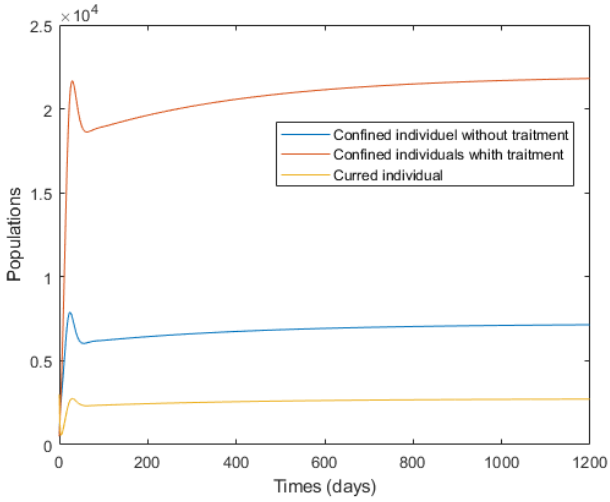

Figure 9. *Density of confined and recovered individuals.*

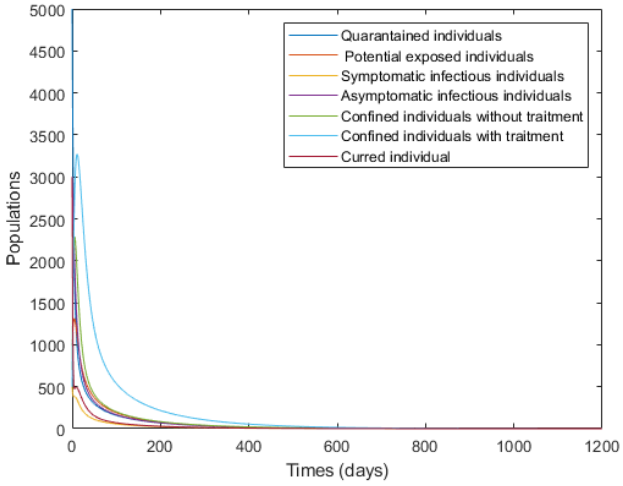

Figure 10. $b = 1$, $\mathcal{R}_0 = 0.76$.

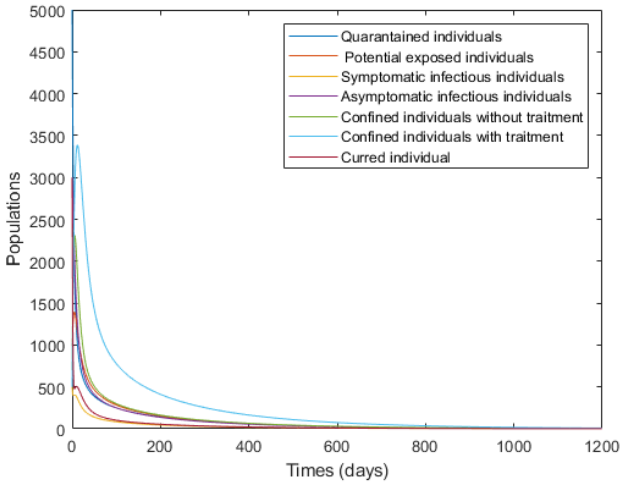

Figure 11. $b = 1.75$, $\mathcal{R}_0 = 0.98$.

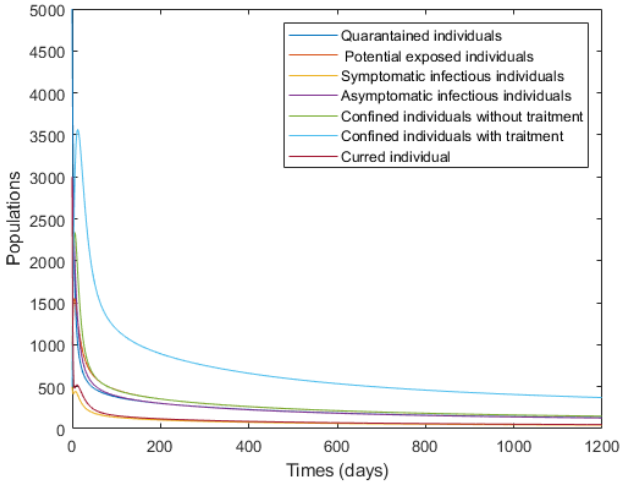

Figure 12. $b = 2.75$, $\mathcal{R}_0 = 1.45$.

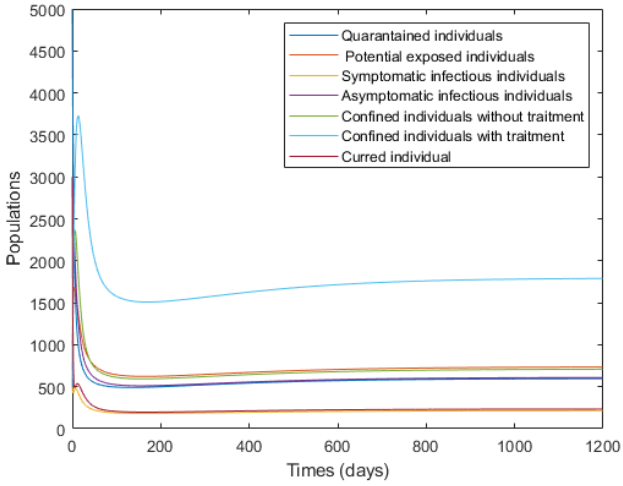

Figure 13. $b = 3.75$, $\mathcal{R}_0 = 2.32$.

4. Conclusion

In this paper, a mathematical SQEICRS model has been developed on COVID-19 transmission dynamics. Mathematical analysis revealed that the model is biologically and mathematically well-posed. The basic reproduction number has been evaluated and gives the direction of disease. The model has a single disease-free equilibrium which is globally stable disease if $\mathcal{R}_0 < 1$. It means that if $\mathcal{R}_0 < 1$, then COVID-19 will go to extinction and will be persistent in the otherwise (i.e $\mathcal{R}_0 > 1$). Sensitivity analysis highlighted the parameters that could potentially contribute to the propagation of the disease, such as adequate contacts rates and chronicity rate. This study clearly shows that people living with a chronic diseases are more vulnerable to COVID-19. Thus, this study confirmed the importance of special actions such as barrier measures required to protect the most vulnerable individuals and contribute the disease eradication. Finally, theoretical results are confirmed by numerical results.

Conflicts of Interest

The authors declare no conflicts of interest.

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