

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 approach 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 analysis 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:

$$N(t) = S(t) + S_c(t) + Q(t) + E(t) + I_s(t) + I_a(t) + C_t(t) + C_s(t) + R(t) \quad (1)$$

- 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 , $i \in \{1, 2, \dots, n\}$, 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:

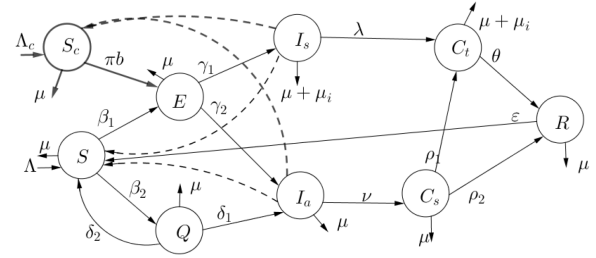


Figure 1. COVID-19 transmission diagram.

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 standart incidence:

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

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

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

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

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

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

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

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

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

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

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

whith the initial condition (12):

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

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

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

3.2. Existence and Uniqueness of Solution

Let

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

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

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

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

is positively invariant.

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

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

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

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

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

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

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

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

$$E(t) \geq 0$$

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

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

$$Q(t) \geq 0$$

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, $N(t) = \frac{\Lambda + \Lambda_c}{\mu}$.

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

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

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

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

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

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

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

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

where

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

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

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

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

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

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

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

and

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

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

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.

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:

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

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

with

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

and

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

Then

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

and

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

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

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

Then the matrix of the next generation is

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

Then

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

3.4. Sensitivity Study

The stability indices are as follows:

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

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

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

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

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

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

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

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

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

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:

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

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

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 $(x_1, 0) \in \mathbb{R}^5 \times \mathbb{R}^4$ 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:

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

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

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

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

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

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-à-dire au point d'équilibre \mathcal{E}_0 . Soit J_2 cette matrice maximale. Alors J_2 n'est rien d'autre la matrice jacobienne de (28) en \mathcal{E}_0 :

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

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:

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

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	$b_1 = \frac{-1}{a_2} (a_0 - a_1 a_2)$	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	estimated
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]	estimated
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	estimated
ε_c	0.25	estimated

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 $\mathcal{R}_0 = 0.61$.

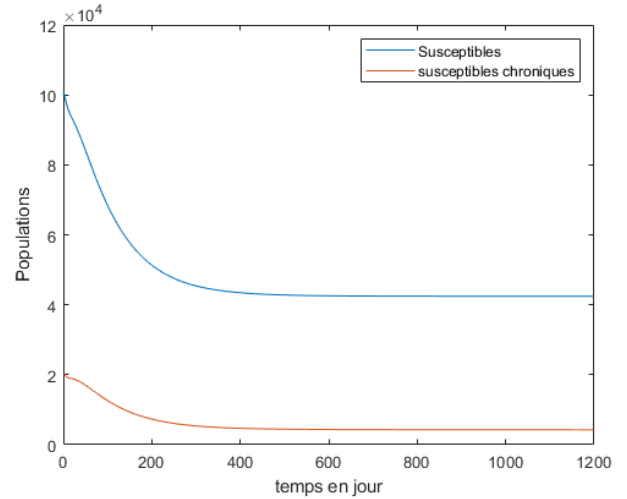


Figure 2. Density of susceptible individuals .

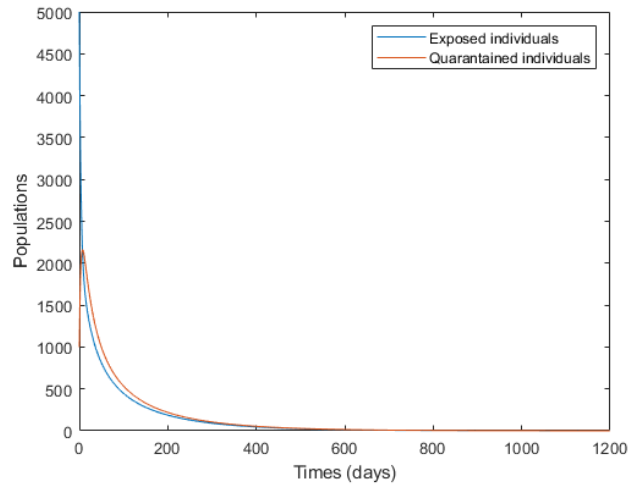


Figure 3. Density of exposed and quarantined individuals.

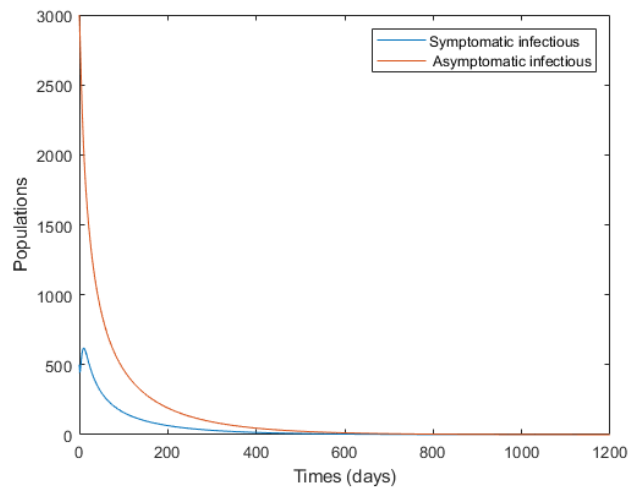


Figure 4. Density of infectious individuals.

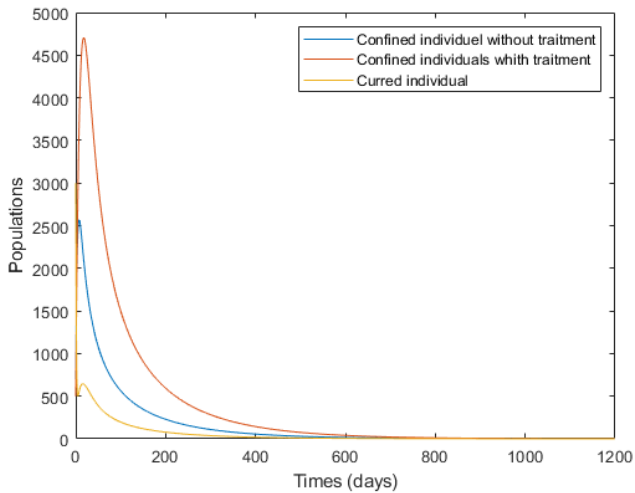


Figure 5. Density of confined and recovered individuals.

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 chronic 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.

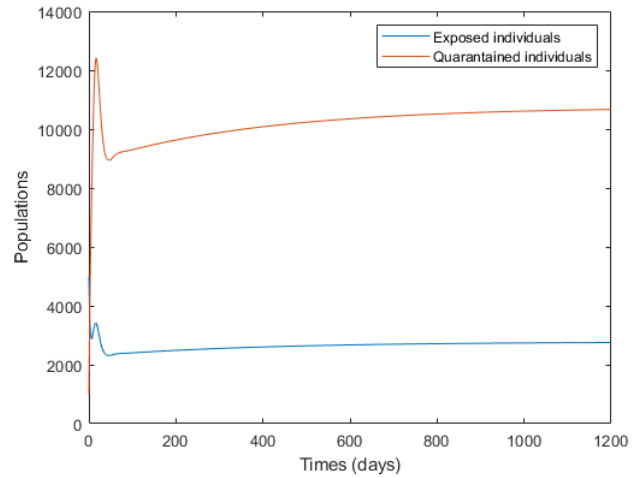


Figure 7. Density of exposed and quarantined individuals.

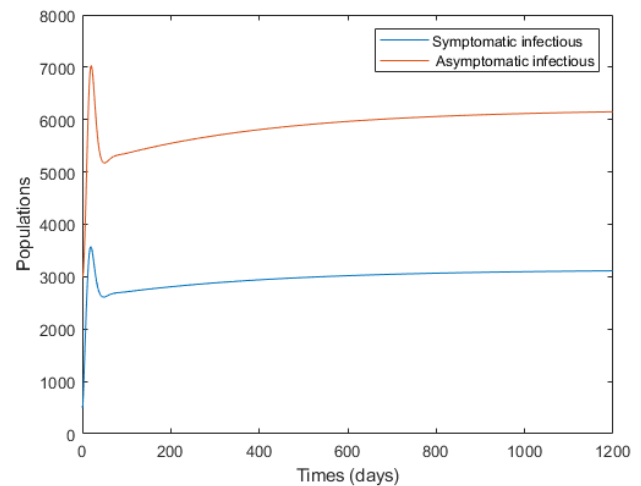


Figure 8. Density of infectious individuals.

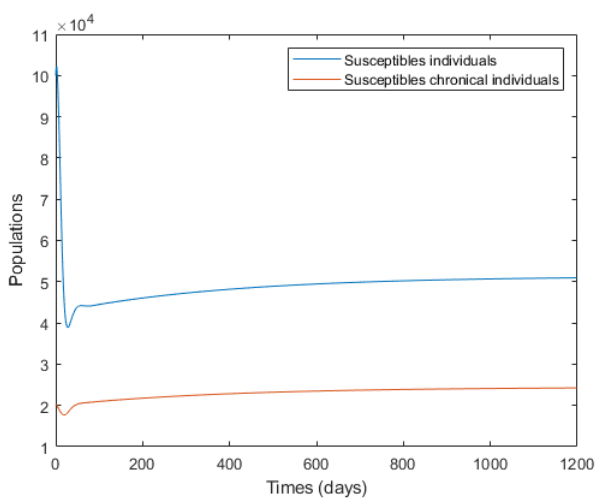


Figure 6. Density of susceptible individuals .

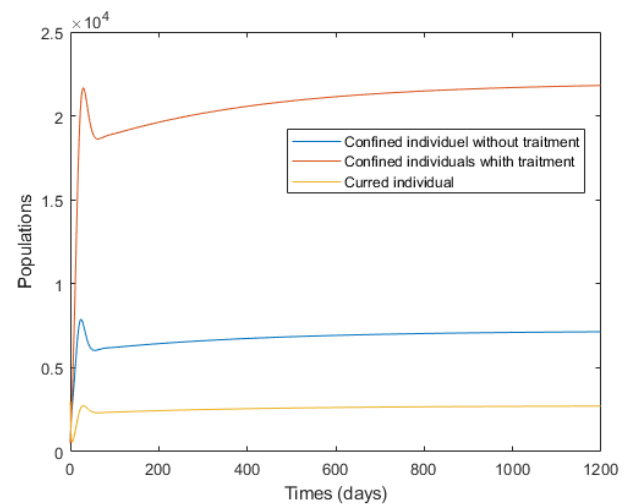


Figure 9. Density of confined and recovered individuals.

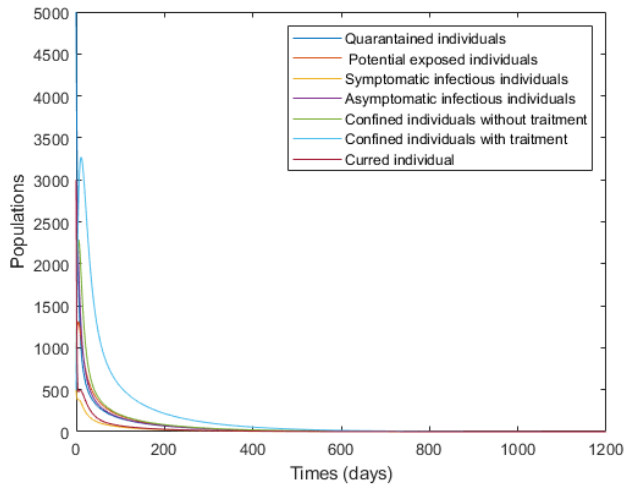


Figure 10. $b = 1$, $\mathcal{R}_0 = 0.76$.

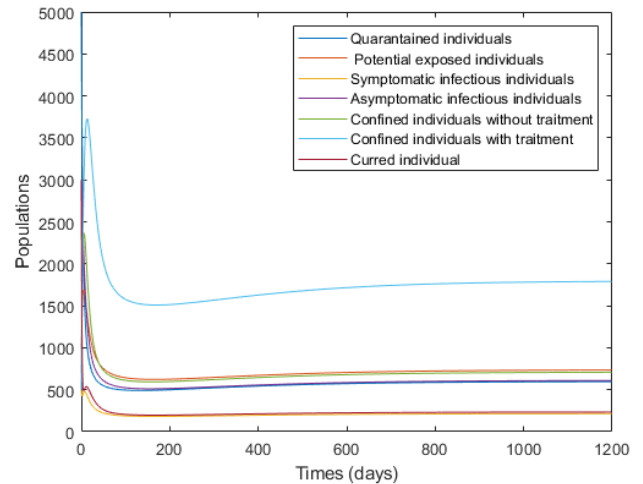


Figure 13. $b = 3.75$, $\mathcal{R}_0 = 2.32$.

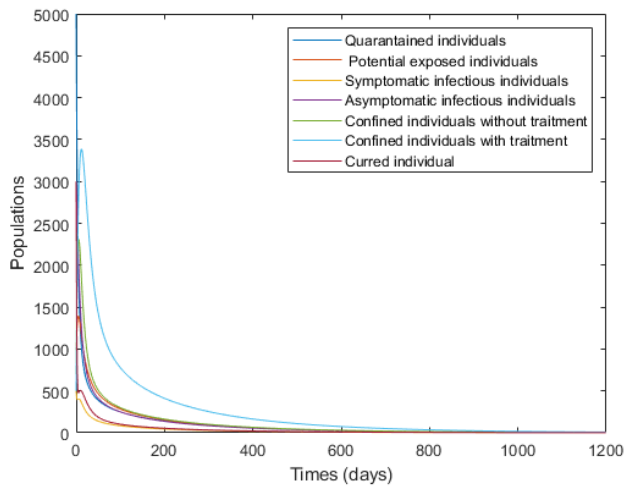


Figure 11. $b = 1.75$, $\mathcal{R}_0 = 0.98$.

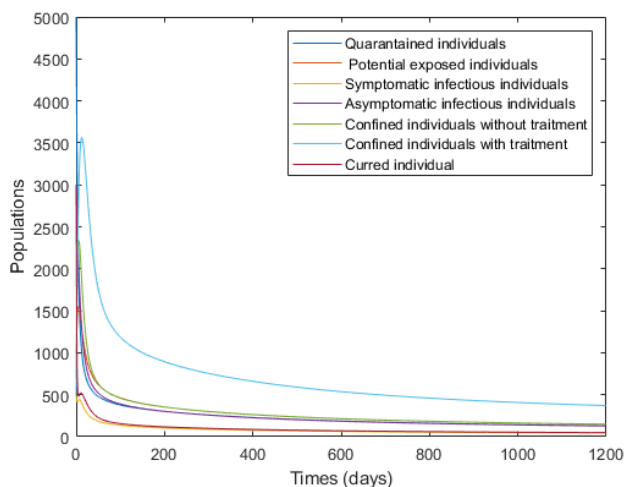


Figure 12. $b = 2.75$, $\mathcal{R}_0 = 1.45$.

4. Conclusion

In this paper, a mathematical S_QEICRS 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.

References

- [1] Abba Mahamane Oumarou, Saley Bisso, *Modelling and simulating a transmission of COVID-19 disease: Niger Republic case*, EJPAM, Vol. 13, No. 3, 2020, 549-566.
- [2] WHO, *COVID-19 Weekly Epidemiological Update*.
- [3] Wahid, B. K. A., Moustapha, D., Rabi, H.G. and Bisso, S. (2020), *Contribution to the Mathematical Modeling of COVID-19 in Niger*. Applied Mathematics , 11, 427-435. <https://doi.org/10.4236/am.2020.116030>

- [4] Organisation Mondiale de la Santé “*Maladie à coronavirus 2019 (COVID-19): sérologie, anticorps et immunité*.” <https://www.who.int/fr/news-room/questions-and-answers/item/coronavirus-disease-COVID-19-serology>, consulter le 21 mars 2022.
- [5] Muhammad R. N. et al. Mathematical Model of COVID-19 with Quarantine and Vaccination. *Jurnal of matematika, statistika dan computasi*, Vol. 19, No. 2, January 2023, pp. 266-285. <https://doi.org/10.20956/j.v19i2.22301>
- [6] Sarbaz H. A et al. Mathematical Modelling for coronavirus disease (COVID-19) in predicting futur behaviours and sensitivity analysis. *Math. Model. Nat. Phenom.* 15 (2020) 33. <https://doi.org/10.1051/mmnp/2020020>
- [7] Kiesha Prem et al. The effect of control strategies to reduce social mixing on outcomes of the COVID-19 epidemic in Wuhan, China: a modelling study. [https://doi.org/10.1016/S2468-2667\(20\)30073-6](https://doi.org/10.1016/S2468-2667(20)30073-6)
- [8] Sasmita et al. Optimal control on a mathematical model to pattern the progression of coronavirus disease 2019 (COVID-19) in Indonesia. *Global Health Research and Policy* (2020) 5: 38. <https://doi.org/10.1186/s41256-020-00163-2>
- [9] Musyoka Kinyili et al. *Mathematical modeling and impact analysis of the use of COVID Alert SA app*. *AIMS Public Health*, 9(1): 106-128. <https://doi.org/10.3934/publichealth.2022009>
- [10] Wei Gao et al. *New investigation of bats-hosts-reservoir-people coronavirus model and application to 2019-nCoV system*. *Advances in Difference Equations* (2020) 2020: 391. <https://doi.org/10.1186/s13662-020-02831-6>
- [11] Bakary Traoré Ousmane Koutou, Boureima Sangaré A *Mathematical model of Malaria transmission dynamics with general incidence function and maturation delay in a periodic environment*, *Litters in Biomathematics an International Journal*, 2020.
- [12] Windjiré SOME et al., *Mathematical Modelling and Numerical Simulation of Transmission Dynamics of COVID-19 Pandemic*, *International Journal of Applied Mathematical Research*, 11 (1) (2022) 1-7.
- [13] Shweta Sankhwar, Narender Kumar, Ravins Dohare, *Mathematical model of transmission dynamics with mitigation and health measures for SARS-CoV-2 infection in European countries*, april 13, 2020.
- [14] Idris Ahmed and al., *A mathematical model of Coronavirus Disease (COVID-19) containing asymptomatic and symptomatic classes*.
- [15] P. Van den Driessche, James Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, *Mathematical Biosciences* 180 (2002) 29-48.
- [16] J.-C. Zhou, S. Salahshour, A. Ahmadian et al., *Modeling the dynamics of COVID-19 using fractal-fractional operator with a case study*. *Results in Physics* (2021). <https://doi.org/10.1016/j.rinp.2021.105103>
- [17] Tinku Joseph and al., *International pulmonologistes consensus on COVID-19*.
- [18] Noah C Peeri and al., *The SARS, MERS and novel coronavirus (COVID-19) epidemics, the newest and biggest global health threats: what lessons have we learned*, *International Journal of Epidemiology*, 2020, 1-10.
- [19] Eric Goucalvès, *Résolution numérique, discrétisation des EDP et EDO*, Institut National Polytechnique de Grenoble, septembre 2005.
- [20] Burkina Faso et partenaires, *COVID-19, Situation Analysis*.
- [21] Ministère de l'Éducation Nationale, de l'Alphabétisation et la Promotion des Langues Nationales (MENAPLN), *La maladie à coronavirus*, Burkina Faso.
- [22] David Sénéchal, *Méthodes numériques et simulations*, Université de Sherbrooke, mars 2020.
- [23] J. C. Kamgang and G. Sallet, *Computation of threshold conditions for epidemiological models and global stability of the disease-free equilibrium (dfe)*, *Mathematical biosciences*, vol. 213, no. 1, pp. 1-12, 2008.
- [24] Institut national de santé publique du Québec, “*COVID-19 : Modes de transmission et mesures de prévention et de protection contre les risques, incluant le rôle de la ventilation*”. <https://www.inspq.qc.ca/COVID-19/environnement/modes-transmission>, consulté le 15 mars 2022.
- [25] Famane KAMBIRE, *Modélisation mathématique par analyse compartimentale de la dynamique de transmission de deux maladies infectieuses: le chikungunya et le paludisme*.
- [26] Vidal, “*Coronavirus COVID-19*” <https://www.vidal.fr/maladies/voies-respiratoires/coronavirus-COVID-19.html>, consulté le 16 mars 2022.
- [27] Wang Zhou, *The coronavirus prevention Handbook, 101 science-based tips that could save your life*.
- [28] Michael Mosley, *COVID-19, what you need to know about the coronavirus and the race for the vaccine*.
- [29] Ankamah and al., *Nonlinear dynamics of COVID-19 SEIR infection model with optimal control analysis*, *Commun. Math. Biol. Neurosci.* 2021.
- [30] Worldometers, “*COVID-19 Coronavirus Pandemic*” <https://www.worldometers.info/coronavirus/> Consulté le 13/11/2021.