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Research paper

Mathematical Modelling and Numerical Simulation of Transmission Dynamics of COVID-19 Pandemic

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Abstract

In this paper we have conceived an original deterministic model for the propagation of Covid-19 dynamics. Mathematical analysis of the model has been done and reveals the existence of a single disease-free equilibrium witch is locally and asymptotically stable. The basic reproduction number $\mathscr{R}_0$ has also been evaluated and gives an idea on the disease evolution in the world. This is because if $\mathscr{R}_0 < 1$, the disease disappears whereas if $\mathscr{R}_0 > 1$, the disease remains in the population. Numerical results are consistent with the theoretical results and highlight the effect of the infectious contact rate α on the evolution of the pandemic.

Keywords: *modeling, transmission, simulation, pandemic, COVID-19*

1. Introduction

The coronavirus epidemic known as Covid-19, has been discovered in the end of December 2019 in Wuhan region of China. It quickly spread in worldwide [1, 2, 16, 26]. As early as January 2020, Europe was already affected, whereas in Africa the first cases were recorded in Egypt in February 2020 [2, 16, 14, 26]. The causal virus was identified by WHO on February 11, 2020 as the new coronavirus SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) [1, 15, 16, 24]. On March 11, 2020, the WHO declared this epidemic a pandemic [1, 2, 26].
After one year, this flu pandemic has caused more than one hundred million victims with more than two million deaths in the end of January 2021 [27, 30, 31]. By its speed of propagation and its capacity to cause harm, it created panic, thus arousing enough reactions in the scientific world, as witnessed by the number of scientific productions and activities carried out. Many scientific researches including mathematical models have been done in the world but the best solution is not yet found. In this paper, we propose an original deterministic model using compartimental modelling to explain its propagation dynamics. Our work includes three parts. The first one aimed for the development of the model, the second one is devoted to the mathematical analysis of the model and the last one is based on numerical simulation.

2. Formulation of the model

2.1. Assumptions

Our model is built on the following assumptions in order to find number $\mathscr{R}_o$:

H1) Transmission is from man to man through direct or indirect contacts[30, 20]
H2) We note N the size of the population who is divided into 5 compartments S , E, I_r, I_u and R respectively including susceptible individuals, exposed individuals, treated infectious individuals, asymptomatic infectious individuals and recovered individuals.
H3) The only vectors are the infectious individuals in the compartiments I_r et I_u [20]
H4) Vertical transmission is neglected
H5) Deaths do not contribute to the chain of disease transmission
H6) The recovered individuals are healthy

H7) Asymptomatic patients are discovered over time
H8) There is no acquired immunity.
H9) The total population density is governed by a logistic law [22, 10].

2.2. Transmission Diagram

Under the assumptions H1) to H9), we can build the following conceptual compartimental[5] transmission diagram:

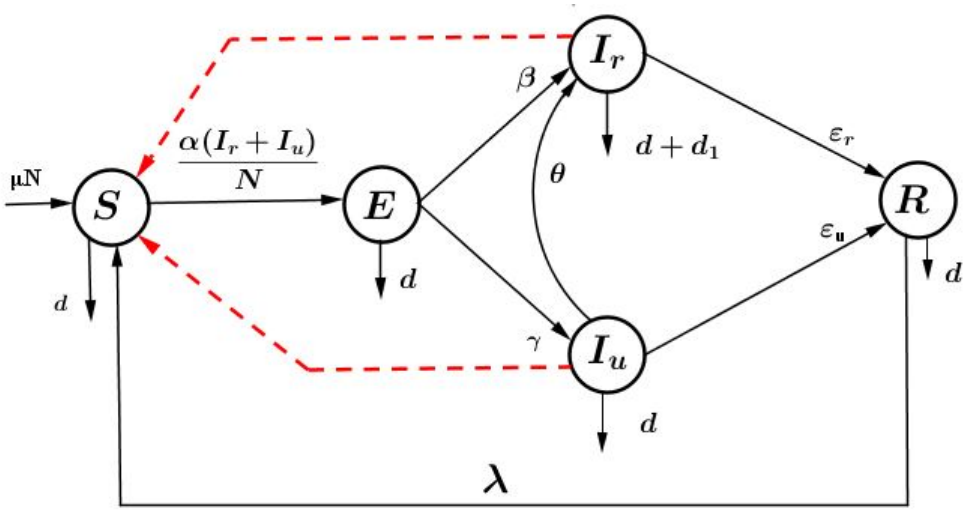


Figure 1: Diagram of covid-19 transmission

2.3. Description and estimation of parameters

The estimation of parameters has been made through the existing literature and current events on covid-19. The values are recorded in the table below:

Table 1: Estimation of parameters

Parameters	Description	Values per day
α	infectious contacts rate	estimed
μ	birth rate	0.04 [13, 22]
d_1	mortality rate induced by the disease	0.03 [30]
ω	natural mortality rate independent of population density	0.000016 [22]
ω_1	natural mortality rate depending on population density	0.0000003 [22]
β	Patient care rates	$\frac{1}{7}$ [23]
θ	rate of asymptomatic patients under treatment	$\frac{1}{14}$ [23]
γ	asymptomatic patient rate	$\frac{1}{7}$ [23]
ε_r	cure rate of patients under treatment	$\frac{1}{14}$ [23]
ε_u	cure rate of asymptomatic patients	$\frac{1}{14}$ [23]
λ	vulnerability rate of recovered individuals	0.75 estimated

2.4. Interactions between compartments

The Covid-19 pandemic spreads within a population through direct or indirect contacts with droplets emitted by infectious individuals during conversation, sneezing or coughing [19, 20, 30].
By contacting infectious individuals, $\alpha\frac{S(I_r+I_u)}{N}$ susceptible individuals become potential suspect cases and join latent individuals in the compartment E .
A proportion β of latent individuals will undergo biological tests, after which they will be declared positive and placed in compartment I_r while others in a proportion γ, although carriers of the virus won't show any symptoms and are registered in compartment I_u.
In the case of other diseases, a proportion θ of asymptomatic patients will test positive and join the compartment I_r.
A proportion ε_r of I_r and another ε_u of I_u will evolve towards healing and form the compartment R. Recovered individuals become vulnerable after a short period of time because there is no acquired immunity.

In all compartments we consider a probable natural mortality with a proportion d. In compartment I_r individuals undergo in addition to natural death, the one induced by the disease with a rate d_1.

2.5. Conceptual Model

From the above diagram of the figure1, we can formulate the following model (using the compartimental modelling technic [5]):

$$\begin{cases} \dfrac{dS}{dt} = \mu N + \lambda R - dS - \alpha \dfrac{S(I_r + I_u)}{N} \\ \dfrac{dE}{dt} = \alpha \dfrac{S(I_r + I_u)}{N} - (d + \beta + \gamma)E \\ \dfrac{dI_r}{dt} = \beta E + \theta I_u - (d + d_1 + \varepsilon_r)I_r \\ \dfrac{dI_u}{dt} = \gamma E - (\theta + d + \varepsilon_u)I_u \\ \dfrac{dR}{dt} = \varepsilon_r I_r + \varepsilon_u I_u - (\lambda + d)R \\ \dfrac{dN}{dt} = (\mu - \omega)\left(1 - \dfrac{\omega_1 N}{\mu - \omega}\right)N - d_1 I_r \end{cases} \tag{1}$$

3. Mathematical Analysis

3.1. Existence and unicity of the solution

To show that the problem (1) is mathematical well posed, we defined the following:
Taking $X = (X_1, X_2, X_3, X_4, X_5, X_6)^T = (S, E, I_r, I_u, R, N)^T \in \mathbb{R}^6$ the state of the system at the instant t with $X_0 = (S_0, E_0, I_{r_0}, I_{u_0}, R_0, N_0)^T \in \mathbb{R}^6$ its initial state.
Let $f = (f_1, f_2, f_3, f_4, f_5, f_6)^T$ defined from $\mathbb{R}^6$ to $\mathbb{R}^6$ by:

$$f(X) = \begin{pmatrix} f_1(X) \\ f_2(X) \\ f_3(X) \\ f_4(X) \\ f_5(X) \\ f_6(X) \end{pmatrix} = \begin{pmatrix} \mu N + \lambda R - dS - \alpha \dfrac{S(I_r + I_u)}{N} \\ \alpha \dfrac{S(I_r + I_u)}{N} - (d + \beta + \gamma)E \\ \beta E + \theta I_u - (d + d_1 + \varepsilon_r)I_r \\ \gamma E - (\theta + d + \varepsilon_u)I_u \\ \varepsilon_r I_r + \varepsilon_u I_u - (\lambda + d)R \\ (\mu - \omega)\left(1 - \dfrac{\omega_1 N}{\mu - \omega}\right)N - d_1 I_r \end{pmatrix}$$

Then the above system(1) can be written as:
$\dfrac{dX_i(t)}{dt} = f_i(X)$, with $i = 1, 2, ..., 6$
Taking the initial conditions in the compact Ω defined by:
$\Omega = \Omega_1 \times \Omega_2$ with
$\Omega_1 = \left\{(S, \quad E, \quad I_r, \quad I_u, \quad R)^t \in \mathbb{R}_+^5\right\}$ and
$\Omega_2 = \left\{N \in \mathbb{R} \quad | \quad 0 < N \leq \dfrac{\mu - \omega}{\omega_1}\right\}$.
For any $X \in \Omega$, the system(1) can be written as a Cauchy problem:

$$\begin{cases} \dfrac{dX_i(t)}{dt} = f_i(X), \qquad i = 1, 2, ..., 6 \\ X_i(0) = X_{i0}, \end{cases}$$

We obtain the following results:

Proposition 3.1. *For any initial condition $X_0 \in \Omega$, the model (1) admits a single solution X defined for any $t \geq 0$.*

Proof 3.1. *The components f_i $(i = 1, 2, ..., 6)$ of function f are distinctively polynomial functions or sums of polynomial and rational functions defined in Ω. Then the function f is of class $C^\infty(\Omega)$ and in particular of class $C^1(\Omega)$. So f is locally lipschitzian. Thus by using Cauchy-Lipschitz's existence and uniqueness theorem [17, 11], for any initial condition $X_0 \in \Omega$;the model (1) admits a single maximum solution defined for any $t \geq 0$.*

Theorem 3.1. ***For any initial condition $X_0 \in \Omega$, the model (1) admits a single solution X that remains in Ω for any $t \geq 0$.***

Proof 3.2. *The existence and unity of the solution has been proved by the proposition 3.1.*
It remains to show that Ω is positively invariant by the model(1) that is to say that for any initial condition $X_0 \in \Omega$, the state of the system $X \in \Omega$ at any time $t > 0$.
To do this, we adopt a similar approach to the one used in [3, 4, 10, 25, 29].
Let us first assume that the initial condition $X_0 = (S_0, E_0, I_{r_0}, I_{u_0}, R_0, N_0)^t \in \Omega$, that is $S_0 \geq 0$; $E_0 \geq 0$; $I_{r_0} \geq 0$; $I_{r_0} \geq 0$; $R_0 \geq 0$ and $0 < N_0 \leq \dfrac{\mu - \omega}{\omega_1}$. To show the positivity of the solution, we rewrite each equation of the model(1) as inequations that we integrate using the variable separation method.

- *Positivity of the number of susceptible:*
  we have: $\mu N + \lambda R \geq 0$ *so*

$$\frac{dS}{dt} \geq -dS - \alpha \frac{S(I_r + I_u)}{N} \tag{2}$$

$$\Longrightarrow S(t) \geq S_0.exp\left[-\int_0^t \left(d + \alpha \frac{I_r + I_u}{N}\right) dt\right] \tag{3}$$

  Hence $S(t) \geq 0$ *because* $S_0 \geq 0$ *and* $exp\left[-\int_0^t \left(d + \alpha \frac{I_r + I_u}{N}\right) dt\right] > 0$.
- *Positivity of the number of exposed individuals E:*
  We have: $\alpha \frac{S(I_r + I_u)}{N} \geq 0$ *thus*

$$\frac{dE}{dt} \geq -(d + \beta + \gamma)E \tag{4}$$

$$\Longrightarrow E(t) \geq E_0.exp[-(d + \beta + \gamma)t] \tag{5}$$

  Thus $E(t) \geq 0$ *because* $E_0 \geq 0$ *and* $exp[-(d + \beta + \gamma)t] > 0$.
  By repeating this approach to the other variables, we obtain $I_r \geq 0, \quad I_u \geq 0, \quad R \geq 0$. *So we have shown that for any initial condition* $X_0 \in \Omega$, $(S, \quad E, \quad I_r, \quad I_u, \quad R)^T \in \Omega_1$.
- *Positivity of the density N:*
  Given that $N(t) = S(t) + E(t) + I_r(t) + I_u(t) + R(t)$, *we have* $N \geq 0$.
  Let us now show that $N(t) \leq \frac{\mu - \omega}{\omega_1}$.
  Such as $d_1 I_r \geq 0$ *then*
  $\frac{dN}{dt} \leq (\mu - \omega)\left(1 - \frac{\omega_1 N}{\mu - \omega}\right) N \Longrightarrow N(t) = \frac{N_0 exp[(\mu - \omega)t]}{1 - \frac{\omega_1 N_0}{\mu - \omega}[1 - exp(\mu - \omega)t]}$
  When $t \longrightarrow +\infty$, *we have:* $\lim_{t \longrightarrow +\infty} N(t) \leq \frac{\mu - \omega}{\omega_1}$
  So for every $t \geq 0$, $0 < N(t) \leq \frac{\mu - \omega}{\omega_1}$ *hence* $N \in \Omega_2$
  Thus we conclude that ω *is positively invariant for the model (1).*
  Therefore for any initial condition $X_0 \in \Omega$, *the X solution remains in* Ω *for any* $t \geq 0$, *which shows that the model (1) is mathematically correct.*

3.2. Disease-free equilibrium

Theorem 3.2. ***The*** (1) ***model assumes a single disease-free equilibrium.***

Proof 3.3. *If there is no disease, we have* $I_r = I_u = 0$. *The disease free equilibrium is obtained by solving the system* $\frac{dX_i}{dt} = 0, i.e. the equation f(X) = 0$. *The only solution* X_{dfe} *is given by the relation (6).*

$$X_{dfe} = \left(\frac{\mu - \omega}{\omega_1}, \quad 0, \quad 0, \quad 0, \quad \frac{\mu - \omega}{\omega_1}\right)^T \tag{6}$$

3.3. Basic reproduction number

Commonly noted $\mathscr{R}_0$, the basic reproduction number is the average number of new cases generated by an average infectious individual. during its infectious period ; in a population composed entirely by susceptible individuals Van, Famane, Sallet.The number $\mathscr{R}_0$ is a very important parameter in epidemiology that provides information on the evolution of the disease according to the following theorem:

Theorem 3.3. *[23, 10]* ***If the basic reproduction number*** $\mathscr{R}_0 < 1$ ***then the epidemic will disappear while if*** $\mathscr{R}_0 > 1$***, the epidemic will persist in the population if no response measures are taken.***

For $\mathscr{R}_0$ calculation in the model (1), we use P. van den Driessche's method [23] which is based on the next generation matrix .This method is also used by several authors such as those of [22, 23, 10, 25, 4].
By posing $x = (x_1, x_2, x_3) = (E, I_r, I_u)$, we define the matrix F (of new infections) and V (of transition) as it follows:
$F = \left[\frac{\partial \mathscr{F}_i(X_{dfe})}{\partial x_j}\right]_{1 \leq i,j \leq 3}$ and $V = \left[\frac{\partial \mathscr{V}_i(X_{dfe})}{\partial x_j}\right]_{1 \leq i,j \leq 3}$ with
$\mathscr{F}(X) = \begin{pmatrix} \alpha \frac{S(I_r + I_u)}{N} \\ 0 \\ 0 \end{pmatrix}$ and $\mathscr{V}(X) = \begin{pmatrix} -(d + \beta + \gamma)E \\ \beta E - (d + d_1 + \varepsilon_r)I_r - \theta I_u \\ \gamma E - (d + \theta + \varepsilon_u)I_u \end{pmatrix}$.
Then we have:

$$F=\begin{pmatrix}0&\alpha&\alpha\\0&0&0\\0&0&0\end{pmatrix} \text{ and } V=\begin{pmatrix}-(d+\beta+\gamma)&0&0\\\beta&-(d+d_1+\varepsilon_r)&\theta\\\gamma&0&-(d+\theta+\varepsilon_u)\end{pmatrix}$$

The next generation matrix is:

$$FV^{-1}=\frac{1}{det(V)}\begin{pmatrix}A&B&C\\0&0&0\\0&0&0\end{pmatrix} \tag{7}$$

where $A=\alpha\beta(d+\theta+\varepsilon_u)+\alpha\gamma(d+d_1+\varepsilon_r+\theta)$; $\quad B=\alpha(d+\beta+\gamma)(d+\theta+\varepsilon_u)$
$C=\alpha(d+\beta+\gamma)(d+d_1+\theta+\varepsilon_r)$

$$\mathscr{R}_0=\rho\left(FV^{-1}\right) \tag{8}$$

$$\mathscr{R}_0=\alpha\left[\frac{\beta(d+\theta+\varepsilon_u)+\gamma(d+\theta+d_1+\varepsilon_r)}{(\beta+\gamma+d)(d+d_1+\varepsilon_r)(d+\theta+\varepsilon_u)}\right] \tag{9}$$

3.4. Stability of the disease-free equilibrium

The local stability of the disease-free equilibrium depends upon the basic reproduction number $\mathscr{R}_0$ according to the theorem below:

Theorem 3.4. ***The disease-free equilibrium, X_{dfe}, is locally and asymptotically stable when $\mathscr{R}_0<1$ while it is unstable if $\mathscr{R}_0>1$ [22].***

Proof 3.4. *To establish the proof of the theorem3.4, we write the system1 as follows:*

$$\begin{cases}\dfrac{dE(t)}{dt}=\alpha\dfrac{S(I_r+I_u)}{N}-(d+\beta+\gamma)E\\[2mm]\dfrac{dI_r(t)}{dt}=\beta E+\theta I_u-(d+d_1+\varepsilon_r)I_r\\[2mm]\dfrac{dI_u(t)}{dt}=\gamma E-(\theta+d+\varepsilon_u)I_u\\[2mm]\dfrac{dS(t)}{dt}=\mu N+\lambda R-dS-\alpha\dfrac{S(I_r+I_u)}{N}\\[2mm]\dfrac{dR(t)}{dt}=\varepsilon_r I_r+\varepsilon_u I_u-(\lambda+d)R\end{cases} \tag{10}$$

The Jacobian matrix J of the model (10) at the disease-free equilibrium X_{dfe} is given by the relation (11):

$$J=\begin{pmatrix}-(d+\beta+\gamma)&\alpha&\alpha&0&0\\\beta&-(d+d_1+\varepsilon_r)&\theta&0&0\\\gamma&0&-(d+\theta+\varepsilon_u)&0&0\\0&-\alpha&-\alpha&-d&0\\0&\varepsilon_r&\varepsilon_u&0&-d\end{pmatrix}=\begin{pmatrix}J_{11}&0\\J_{21}&J_{22}\end{pmatrix} \tag{11}$$

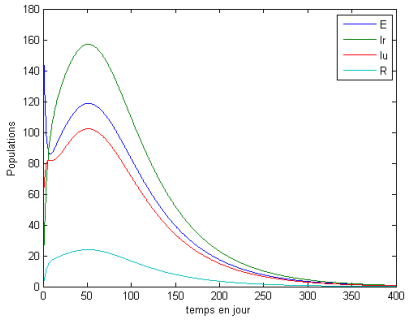
where

$$J_{11}=F-V \qquad J_{21}=\begin{pmatrix}0&-\alpha&-\alpha\\0&\varepsilon_r&\varepsilon_u\end{pmatrix} \qquad J_{22}=\begin{pmatrix}-d&0\\0&-d\end{pmatrix}$$

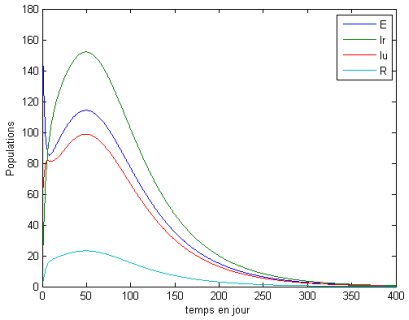
The equilibrium X_{dfe} is asymptotically stable if all eigenvalues of J have a strictly negative real part and unstable if at least one of the eigenvalues of J has a real part positive or zero.
J is a triangular block matrix so its eigenvalues are those of the diagonal blocks J_{11} and J_{22}. J_{22} has only one double real eigenvalue which is $-d<0$.So the stability of X_{dfe} depends only on the eigenvalues of the $F-V$ matrix.
We notice that F is a non-negative matrix.
The V matrix has its negative extradiagonal elements and the sum of the components of each of its column vectors is positive. Therefore V is non-singular a M-matrix [22], theorem A.1]. Thus, referring to the proof of theorem 2 in [23], we have the following equivalence:s $\left(FV^{-1}\right)<0$ $\quad(resp\quad s\left(FV^{-1}\right)>0)\quad\Longleftrightarrow\quad\rho\left(FV^{-1}\right)<1\quad(resp\qquad\rho\left(FV^{-1}\right)>1)$
Where the number s $\left(FV^{-1}\right)$ designates the spectral abscissa of the matrix FV^{-1}. According to the previous section, we have $\mathscr{R}_0=\rho\left(FV^{-1}\right)$. So we can conclude that X_{dfe} is locally asymptotically stable if $\mathscr{R}_0<1$ and unstable if $\mathscr{R}_0>1$.

4. Numerical Simulation

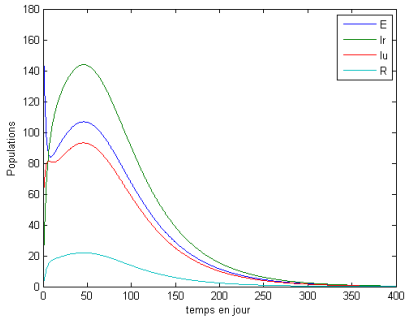
In this section, we solve numerically the model1 by the Runge-Kutta method in order 4 using MATLAB [7, 9]. This section aims to verify the conformity of the theoretical and numerical results. We also highlight the effect of the infectious contact rate α on the evolution of the epidemic.
Using the values of the parameters in the table 1, we obtain the following graphs:



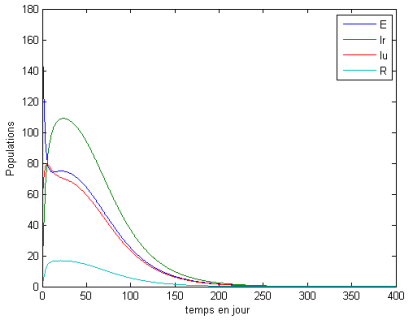
(a) $\alpha = 0.1421; \mathscr{R}_0 = 1$.



(b) $\alpha = 0.141; \mathscr{R}_0 = 0.9923$.



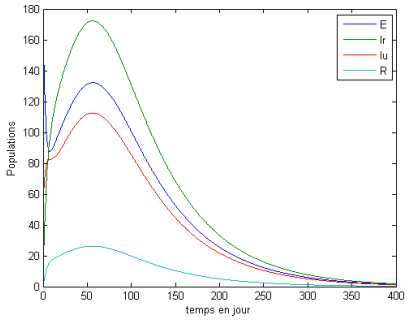
(c) $\alpha = 0.139; \mathscr{R}_0 = 0.9782$.



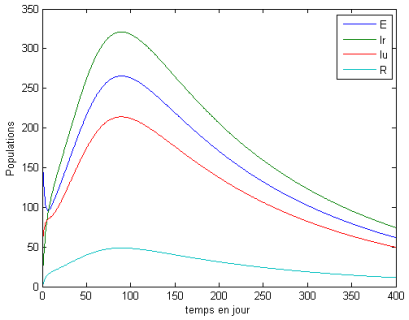
(d) $\alpha = 0.125; \mathscr{R}_0 = 0.8797$.

Figure 2: Evolution de la maladie pour $\mathscr{R}_0 < 1$.

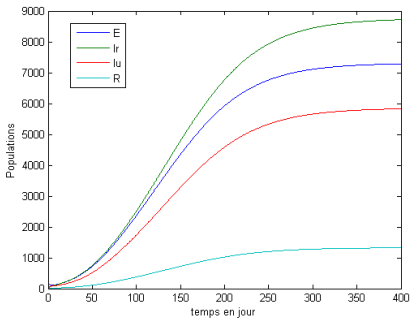
According to the graphs of figures 2(a), 2(b), 2(c), 2(d), we notice that the number of patients decreases over time with $\mathscr{R}_0$ and tends to zero.



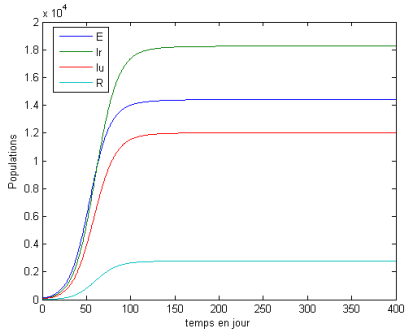
(a) $\alpha = 0.145; \mathscr{R}_0 = 1.0204$.



(b) $\alpha = 0.160; \mathscr{R}_0 = 1.1260$.



(c) $\alpha = 0.2; \mathscr{R}_0 = 1.4075$.



(d) $\alpha = 0.3; \mathscr{R}_0 = 2.1112$.

Figure 3: Evolution de la maladie pour $\mathscr{R}_0 > 1$.

According to the graphs of figures 3(a), 3(b), 3(c), 3(d), we see that the number of patients increases over time when $\mathscr{R}_0$ increases so that the total density N remains in Ω.

Interpretation

Numerical simulations were made for $\mathscr{R}_0 < 1$ (figures 2(a), 2(b), 2(c), 2(d)) and $\mathscr{R}_0 > 1$ (figure 3(a), 3(b), 3(c), 3(d)). The results are consistent with the theoretical results.
Indeed, the graphs of figures 2(a), 2(b), 2(c), 2(d) are obtained for $\mathscr{R}_0 < 1$ and show that the numbers of infectious individuals (in red and green) decrease and cancel over time.This shows that the pandemic will go away on its own.
The graphs of the figures 3(a), 3(b), 3(c), 3(d) are obtained for $\mathscr{R}_0 > 1$ and show that the numbers of infectious individuals (in red and green) increase over time. It means that the disease is raging in the population and therefore requires intervention.
For $\mathscr{R}_0 = 1$, we cannot conclude. However we notice that when $\mathscr{R}_0$ is neer to unit (1) by lower values (figures 2(a), 2(b)), the graphs move slightly off the abscissa axis over time. It means that there is eventually a bifurcation around the unit.

5. Conclusion

In this article we have developed a deterministic model for the dynamics of corovirus transmission. During the mathematical analysis of the model, we showed that the single disease-free equilibrium is locally stable and determined the basic reproductive number $\mathscr{R}_0$ wich is proportional to the infectious contacts rate. The formula of $\mathscr{R}_0$ is standard and can be applied to any data in the world. If $\alpha = 0$ then $\mathscr{R}_0 = 0$; it means that no contact can transmit the disease. The results of the numerical simulation highlighted the effect of the number α of infectious contacts on the evolution of the disease. Our analyses show that the pandemic becomes more and more severe when this parameter increases, hence the need to respect barrier measures that limit the number of contacts that could lead to new cases.
We also suspected the existence of a bifurcation around the unit value of $\mathscr{R}_0$ which is currently being studied.

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