SIARD MODEL AND EFFECT OF LOCKDOWN ON THE DYNAMICS OF COVID-19 DISEASE WITH NON TOTAL IMMUNITY

M.A. Aziz-Alaoui[1,2,3], F. Najm[4] and R. Yafia[4,*]

Abstract. We propose a new compartmental mathematical model describing the transmission and the spreading of COVID-19 epidemic with a special focus on the non-total immunity. The model *(called SIARD)* is given by a system of differential equations which model the interactions between five populations "susceptible", "reported infectious", "unreported infectious", "recovered with/without non total immunity" and "death". Depending on the basic reproduction number, we prove that the total immunity induces local stability-instability of equilibria and the epidemic may disappear after a first epidemic wave and more epidemic waves may appear in the case of non-total immunity. Using the sensitivity analysis we identify the most sensitive parameters. Numerical simulations are carried out to illustrate our theoretical results. As an application, we found that our model fits well the Moroccan epidemic wave, and predicts more than one wave for French case.

1. Introduction and mathematical model

For the first infection, the body responds quickly to the threat and activates the first defence called innate immune system. This system is quick-acting but is not targeted to the specific threat, and distracts the infection while the body produces a more targeted but slower response against the infection, via the adaptive and "specific" immune system. The adaptive immune system produces antibodies to fight infections. These are what we measure in the blood when trying to determine who has been exposed to SARS-CoV-2 (the virus that causes COVID-19 epidemic). In this case, the body produces different types and specific antibodies to respond to different parts of the virus. But only some have the ability to stop the virus entering cells. These are called neutralizing antibodies. According to the WHO, people recovered from COVID-19 develop antibodies in their blood. But some people appear to have low levels of neutralising antibodies and scientific communities still do not know how the human immune system responds to SARS-CoV-2 and whether or not people develop long-term immunity.

[1] Normandie Univ, 76600 Le Havre, France.
[2] ULH, LMAH, 76600 Le Havre. France
[3] FR-CNRS-3335, ISCN, 25 rue Ph. Lebon, 76600 Le Havre, France.
[4] Laboratory of Analysis, Geometry and Applications (LAGA), Department of Mathematics Faculty of Sciences, Ibn Tofail University, Campus Universitaire BP 133, Kenitra, Morocco.

* Corresponding author: radouane.yafia@uit.ac.ma

TABLE 1. Table of reinfection confirmed cases.

	Sex	Age (year)	Infection date	Reinfection date	Interval (days)	References
Nevada, USA	Male	25	April, 2020	June 2020	48	[15]
Hong Kong	Male	33	April, 2020	August, 2020	142	[16]
Belgium	Female	51	March, 2020	June, 2020	93	[17]
Ecuador	Male	46	May, 2020	July, 2020	63	[12]
India	Male	25	May, 2020	August, 2020	108	[7]
India	Femele	28	May, 2020	September, 2020	111	[7]

The question is, can immunity protect recovered individuals from reinfection of COVID-19? Biologically, the answer is no, because:

A man (25-year-old) from Nevada (USA) [15] presented to health authorities on two times with symptoms of viral infection, first one was in April, 2020, and a second time at the end of May and beginning of June, 2020. In each time, the patient had a positive test for SARS-CoV-2, the two tests separated by two negative tests done during follow-up in May, 2020.

In Hong Kong, during routine airport checking, a man (33 years) presented with symptoms of cough, sore throat, fever and headache for three days during the first episode and was tested positive to SARS-Cov-2 and hospitalised (see [16]). Although his symptoms had mostly disappeared upon hospitalisation. Two weeks later and after two subsequent negative tests, the patient was discharged. During the reinfection, the patient had a high viral load which decreased over time.

In Belgium, a case of reinfection which is a women in a 51 year old who presented with headache (see [17]), fever, myalgia, cough, chest pain, dyspnoea and anosmia to hospital on 9 March 2020. The patient was self-isolated at home and reported persistent symptoms for nearly five weeks. Three months (10 June 2020) after her initial symptoms, the patient presented with headache, cough, fatigue and rhinitis and was tested again positive.

In Ecuador, Prado-Vivar *et al.* [12], report a case (46 year) of reinfection who presented on 12 May 2020 to hospital and was tested positive.. In July, the same patient presented with symptoms including headache, fever, cough and shortness of breath. On 22 July 2020, he was tested positive to SARS-Cov-2.

In India, Gupta *et al.* [7] report two cases (25 year old man and 28 year old woman) of reinfection. During routine surveillance of health workers, the two patients were tested positive, the first on on 5 May 2020 and the second on 17 May 2020. They continued working thereafter and were tested again PCR-positive, the first on 17 August and the second on 5 September. We summarize these finding in Table 1.

In a research letter published in "JAMA Network" (see [9]), the authors stated that, four individuals tested positive for a period of $5-13$ days after showing clinical signs of recovery from COVID-19. Another study published in "American Journal of Respiratory and Critical Care Medicine" (see [3]) confirmed that, by collecting throat swabs from 16 people recovered from COVID-19, one person had a false negative result, and half of the these people tested positive up to 8 days after their symptoms resolved. In this paper, we focus our study on the effect of lockdown and non total immunity via mathematical modeling.

Mathematical models of the dynamics of infectious disease transmission are useful for forecasting and controlling epidemics. In compartmental epidemic models, each individual of the population is categorized based on their disease status in addition to, possibly, their attributes and/or the treatment they received. The dynamics of disease transmission are then typically modeled with differential equations that describe the interaction of individuals between the compartments as the population mixes, the disease spreads, infected individuals progress through the stages of the disease, and public health interventions are implemented. The classical Susceptible-Infectious-Removed (SIR) compartmental model was introduced by Kermack and McKendrick [8]. Based on the idea of the SIR framework, numerous types of mathematical models using compartmental approach have been developed in the meanwhile, all incorporating more structure and details of the transmission process and infectious disease dynamics [1, 2, 4]. Other authors use stochastic approach to models the dynamics and
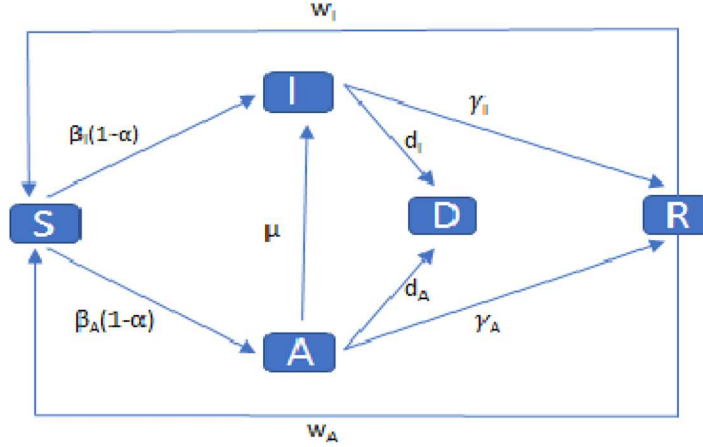
FIGURE 1. Schematic diagram of SIARD model.

transmission of emergent and re-emergent infectious diseases [13, 14]. In [13], the authors develop and study a stochastic approach to model the spread of COVID-19 epidemic by taking into account the cross immunity and time delay of transmission. They prove the existence and uniqueness of positive global solution of proposed SIRC model and they deduce that; when the white noise is relatively large, the infectious diseases will become extinct; Re-infection and periodic outbreaks can occur due to the existence of feedback time-delay (or memory) in the transmission terms.

Based on the SIR and SIRC models described above, we propose a new compartmental mathematical model (called SIARD) which takes into account the role of lockdown and the effect of fails of immunity and reinfection by COVID-19 after recovery. The SIARD model describes the interaction between susceptible population S, reported infectious population I, unreported asymptomatic infectious population A, recovered population R and death population D (see Fig. 2).

In this work, we suppose that our system is a closed system and the proposed model follows the schematic diagram described in Figure 1. We assume that α is the rate of susceptible and asymptomatic populations which obey to lockdown rules and $1 - \alpha$ is the rate of susceptible and asymptomatic populations which does not obey to lockdown rules. So with a contagion rate β_I (resp. β_A) an infected person infects on average $\beta_I(1 - \alpha)S$ (resp. $\beta_A(1 - \alpha)S$) of susceptible population. Moreover, if the COVID-19 epidemic immunity does not exist or is very low, a part of recovered population R becomes susceptible and re-enters again into the susceptible population which obey to the lockdown rules with a rate w_I if the recovered population comes from infectious population and with a rate w_A if the recovered population comes from asymptomatic population. The second compartment I will be incremented by infected individuals leaving the susceptible compartment and by asymptomatic individuals which become symptomatic with a rate μ. The recovered compartment receives all recovered individuals and loses those who become susceptible due to their non-immunity w_I and w_A. The model is given as follows:

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = -\beta_I(1 - \alpha)SI - \beta_A(1 - \alpha)^2 SA + \omega_I R + \omega_A R \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \beta_I(1 - \alpha)SI - (\gamma_I + d_I)I + \mu A \\ \frac{\mathrm{d}A}{\mathrm{d}t} = \beta_A(1 - \alpha)^2 SA - (\gamma_A + d_A)A - \mu A \\ \frac{\mathrm{d}R}{\mathrm{d}t} = \gamma_I I + \gamma_A A - \omega_I R - \omega_A R \end{cases} \tag{1.1}$$

Remark 1.1. As the system (1.1) is a closed system, we can consider also the equation of death population

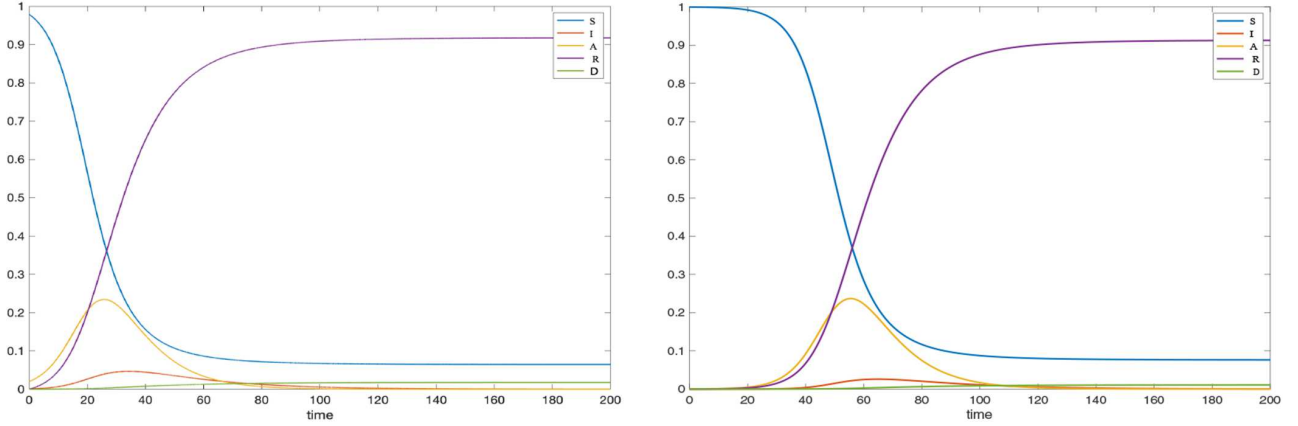$$\frac{\mathrm{d}D}{\mathrm{d}t} = d_I I + d_A A$$

FIGURE 2. Numerical illustrations showing the SIARD dynamics for different values of transmission rate β_I. The right plot illustrates how a lower value of β_I flattens the curve of infectious population (red line) while also significantly delaying the infection peak. Further, this picture suggests that social distancing and lockdown measures may have to be imposed for a very long time to be effective. β_I in the left plot is greater than the right one and with total immunity.

and we have $N = S + I + A + R + D = cste = 1$.

The parameters meaning is summarized in the following table.

Parameters	Epidemiological interpretation
β_I	The averaged contact rate between S and I
β_A	The averaged contact rate between S and A
$\alpha \in [0,1]$	Lockdown rate of susceptible and asymptomatic populations
γ_I	Self-recovery rate for symptomatic cases
γ_A	Self-recovery rate for asymptomatic cases
d_I	Death rate for symptomatic cases
d_A	Death rate for asymptomatic cases
μ	Transition rate from asymptomatic to symptomatic compartments
w_I	Non-immunized rate for symptomatic cases
w_A	Non-immunized rate for asymptomatic cases

For epidemiological reasons, we assume $\beta_A \leq \beta_I$ which means that, the symptomatic population is more infectious than the asymptomatic one. $d_A \leq d_I$, the infectious populations died more than the asymptomatic one. $\mu \ll 1$, the asymptomatic individuals become symptomatic rarely. $\gamma_I \leq \gamma_A$, the asymptomatic population recovered more than the symptomatic one. $w_A \leq w_I$, asymptomatic population is more immunized than the infectious one.

As $S + I + A + R + D = cste$, we can easily prove that, all solutions of system (1.1) starting from a positive initial conditions are bounded and positives. The current work is organized as follows: In Section 2 we study the SIARD model with lockdown rule ($\alpha > 0$) and total immunity $w_I = w_A = 0$. In Section 3, we study the SIARD model with lockdown rule ($\alpha > 0$) and non total immunity $w_I > 0$, $w_A > 0$. Section 4 is devoted to detecting the most critical parameters by using sensitivity analysis. We end our paper by a conclusion. We carry out some numerical simulations to illustrate our theoretical results and an application of the SIARD model to Moroccan and French COVID-19 epidemic data.

2. Dynamics with total immunity

In this section, we suppose all recovered population is immune $w_I = w_A = 0$.

2.1. Equilibria, stability and R_0

The equilibria of system (1.1) are: the extinction equilibrium (EE) $E_0 = (0,0,0,0)$ and the disease free equilibrium (DFE) $(S^*, 0, 0, 0)$. The biological meaningful of disease free equilibrium (DFE) is the case when $S^* = S_0 = N = 1$ (because the other components are zeros). Next, we compute the basic reproduction number by using the next generation matrix introduced by Van Den Driessche et $al.$ [18] and by Diekmann et $al.$ [5] and we have:

$$F = \begin{pmatrix} \beta_I(1-\alpha)S^* & 0 \\ 0 & \beta_A(1-\alpha)^2 S^* \end{pmatrix}$$

and

$$V = \begin{pmatrix} -(\gamma_I + d_I) & \mu \\ 0 & -(\gamma_A + d_A + \mu) \end{pmatrix}.$$

The eigenvalues of $-FV^{-1}$ are $\lambda_1 = \dfrac{\beta_I(1-\alpha)}{\gamma_I + d_I} = R_I(1-\alpha)$ and $\lambda_2 = \dfrac{\beta_A(1-\alpha)^2}{\gamma_A + d_A + \mu} = R_A(1-\alpha)$, with $R_I = \dfrac{\beta_I}{\gamma_I + d_I}$ and $R_A = \dfrac{\beta_A}{\gamma_A + d_A + \mu}$.

Then,

$$R_0 = \rho(-FV^-1) = max(R_I, R_A(1-\alpha))(1-\alpha). \tag{2.1}$$

Remark 2.1. From equation (2.1), R_0 depends on the lockdown rate α (see Fig. 4). If the lockdown rate is increasing the basic reproduction number R_0 is decreasing. In addition, noting R_I (resp. R_A) the basic reproduction number of infectious population I (resp. asymptomatic population A), according to a study given by Petersen et $al.$ [11] of the 115 people who tested positive with SARS-CoV-2, 16 or 13.9% reported symptoms, while 99 people or 86.1% of the patients, did not report any specific symptoms on the day of the test. In Morocco, according to the health authority $70 - 98\%$ of tested people are asymptomatic Based on this study, we can suppose $R_A > R_I$.

By linearizing system (1.1) around the DFE $E_0 = (S_0, 0, 0, 0)$, we have the corresponding jacobian matrix:

$$J_{E_0} = \begin{pmatrix} 0 & \beta_I(1-\alpha) & \beta_A(1-\alpha)^2 \\ 0 & \beta_I(1-\alpha) - (\gamma_I + d_I) & \mu \\ 0 & 0 & \beta_A(1-\alpha)^2 - (\gamma_A + d_A + \mu) \end{pmatrix}$$

and the characteristic equation is

$$\Delta = \lambda(\lambda - \beta_I(1-\alpha) + (\gamma_I + d_I))(\lambda - \beta_A(1-\alpha)^2 + (\gamma_A + d_A + \mu)) = 0. \tag{2.2}$$

The corresponding eigenvalues are 0, $\beta_I(1-\alpha)-(\gamma_I+d_I)$ and $\beta_A(1-\alpha)^2-(\gamma_A+d_A+\mu)$. Therefore, we obtain the following result.

Proposition 2.2. *The equilibrium DFE E_0 is stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Remark 2.3. The non-trivial equilibrium of (1.1) is given by $E^* = (S^*, 0, 0, R^*)$, where $(S^*, R^*) \in \{(S, R) \in \mathbb{R}^2_{+*}\}$.

Next we consider a non-trivial equilibrium $E^* = (S^*, 0, 0, R^*)$, then the jacobian matrix is as follows:

$$J_{E^*} = \begin{pmatrix} 0 & \beta_I(1-\alpha)S^* & \beta_A(1-\alpha)^2 S^* \\ 0 & \beta_I(1-\alpha)S^* - (\gamma_I + d_I) & \mu \\ 0 & 0 & \beta_A(1-\alpha)^2 S^* - (\gamma_A + d_A + \mu) \end{pmatrix}$$

and the corresponding eigenvalues are 0, $\beta_I(1-\alpha)S^* - (\gamma_I + d_I)$ and $\beta_A(1-\alpha)^2 S^* - (\gamma_A + d_A + \mu)$. Let $\mathcal{R}_0(S) = SR_0$, therefore, we obtain the following result.

Proposition 2.4. *E^* is stable if $\mathcal{R}_0(S^*) < 1$ and unstable otherwise.*

Remark 2.5. Note $\alpha = \alpha^I_{pic}$, the critical value of the lockdown rate such that for all $\alpha > \alpha^I_{pic}$, the curve of I has a pic and no pic elsewhere. That is; there exists T^I_{pic} et α^I_{pic}, $\frac{dI}{dt} < 0$ for all $t < T^I_{pic}$, $\frac{dI}{dt} = 0$ for $t = T^I_{pic}$ and $\frac{dI}{dt} > 0$ for $t > T^I_{pic}$. At $t = T^I_{pic}$, R_0 becomes < 1 and at $t = T^I_{pic}$, $\frac{dR_0}{dt} < 0$. To compute α^I_{pic}, from equation $(1.1)_2$, we have $\frac{dI}{dt}(T^I_{pic}) = 0$ and

$$\alpha^I_{pic} = 1 - \frac{1}{R_I S(T^I_{pic})} + \frac{\mu A(T^I_{pic})}{\beta_I S(T^I_{pic}) I_{max}}. \tag{2.3}$$

By the same method we compute $\alpha = \alpha^A_{pic}$ the critical value of the lockdown rate such that, the curve of A has a pic and no pic elsewhere. From equation $(1.1)_3$, we have $\frac{dA}{dt}(T^A_{pic}) = 0$ and

$$\alpha^A_{pic} = 1 - \frac{1}{R_A S(T^A_{pic})}. \tag{2.4}$$

From Figure 3, the peaks values of the populations I and A are decreasing and go to initial conditions when the temporal solutions of I and A start to decreasing immediately from initial conditions (*i.e.* $\alpha > \alpha^I_{pic}$ and $\alpha > \alpha^A_{pic}$).

Remark 2.6. With total immunity $w_1 = w_2 = 0$ and by changing the values of lockdown rate α and the transition rate μ, the infectious and asymptomatic populations have only one peak over time see Figures 4 (right) and 5. In this case there is only one wave COVID-19 epidemic.

3. Dynamics with non total immunity

Suppose now, there is a fraction of recovered population which become infected after recovery (*i.e.* $w_I \neq 0$ and $w_A \neq 0$). In this case the equilibria are of the form $E^{**} = (S^{**}, 0, 0, 0)$ ($S^{**} > 0$) and the jacobian is as

TABLE 2. Parameters values.

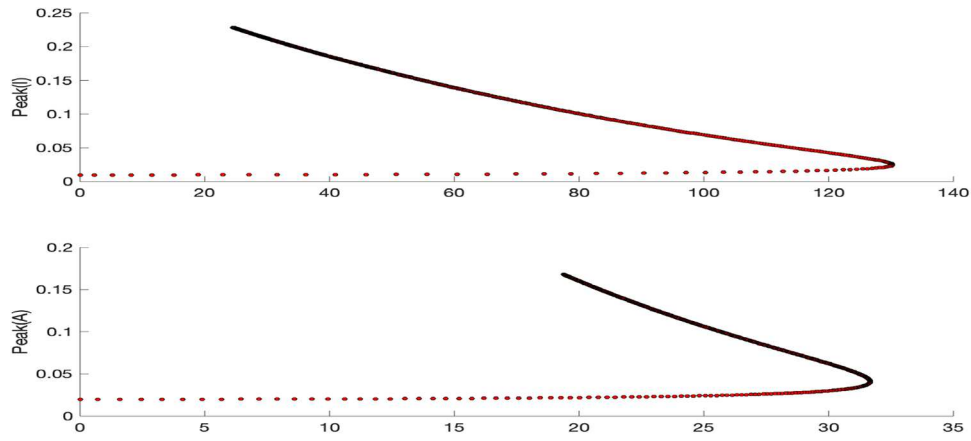| Parameter	Estimation
β_I	0.294
β_A	0.29
α	0–1
γ_I	0.05
γ_A	0.1
d_I	0.007
d_A	0.0001
μ	0–0.2

FIGURE 3. Peak time versus peak value of populations I and A for different values of lockdown parameter α varying from 0 to 1 with 0.001 step and $w_1 = 0$, $w_2 = 0$.
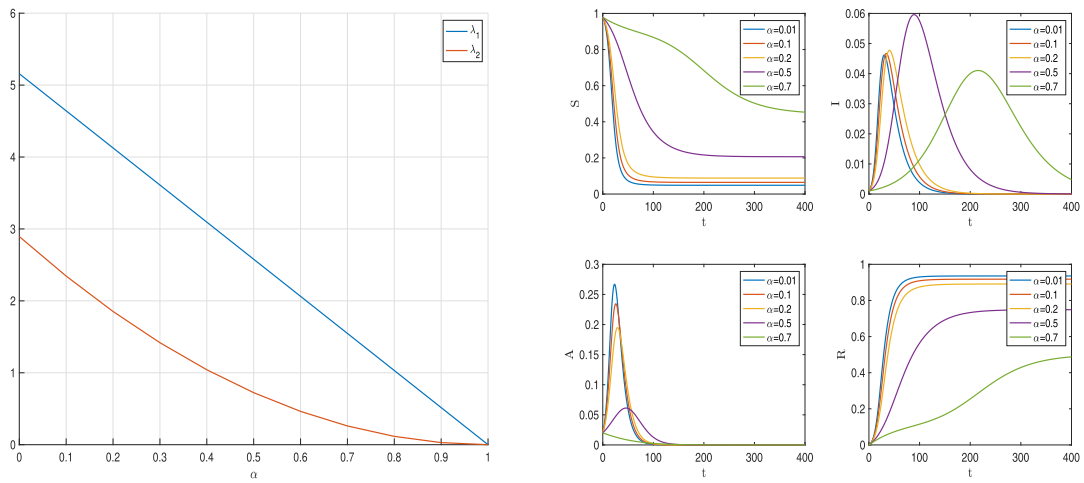

FIGURE 4. *Left*: Curve of $R_0 = max(\lambda_1, \lambda_2)$ with respect to α. *Right*: Temporal evolution of different populations S, I, A, R for $\mu = 0.0001$. We have plotted many solutions for different values of α and $w_1 = 0$, $w_2 = 0$.
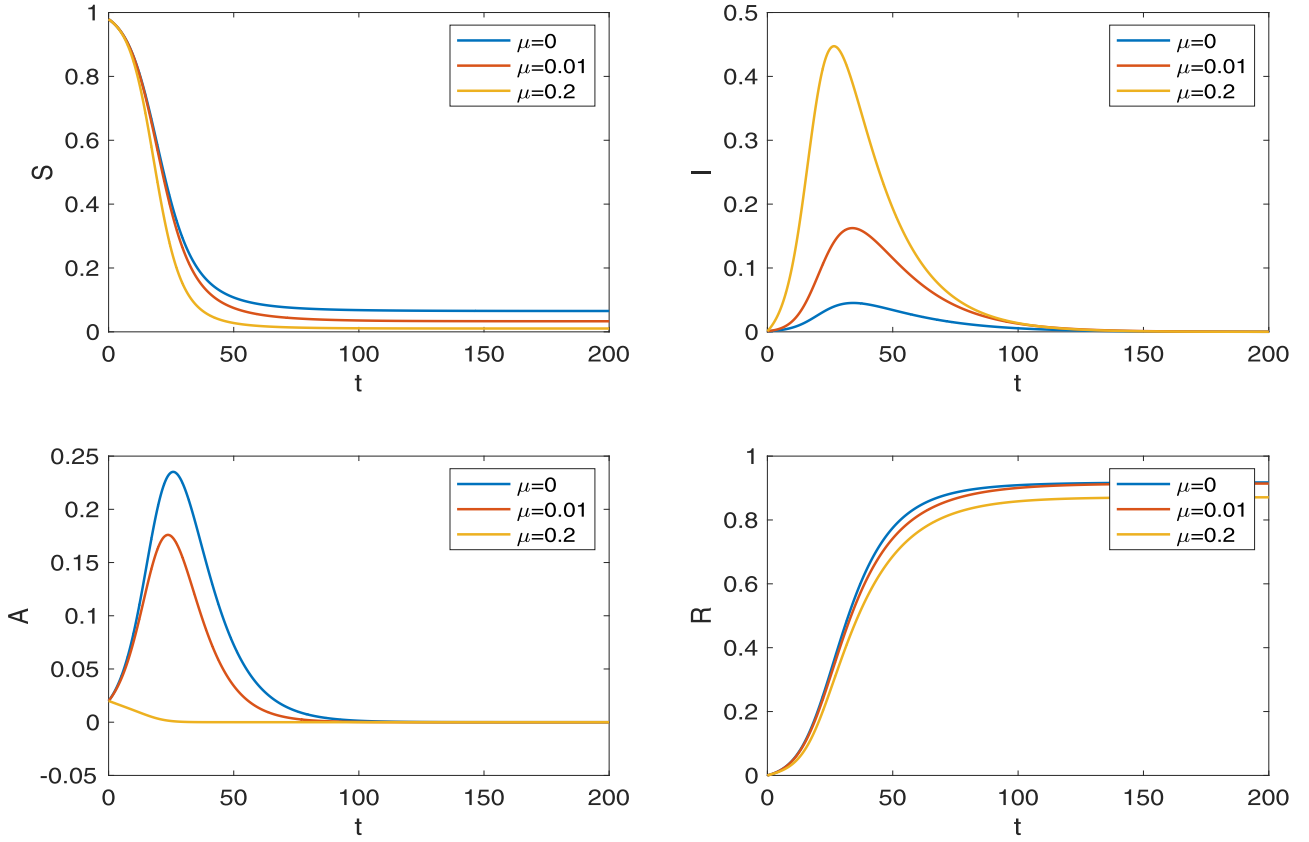
FIGURE 5. Temporal evolution of different populations S, I, A and R for $\mu = 0, 0.001, 0.2$ and $w_1 = 0$, $w_2 = 0$.

follows

$$J_{E^{**}} = \begin{pmatrix} 0 & \beta_I(1-\alpha)S^{**} & \beta_A(1-\alpha)^2 S^{**} & w_I + w_A \\ 0 & \beta_I(1-\alpha)S^{**} - (\gamma_I + d_I) & \mu & 0 \\ 0 & 0 & \beta_A(1-\alpha)^2 S^{**} - (\gamma_A + d_A + \mu) & 0 \\ 0 & \gamma_I & \gamma_A & -(w_I + w_A) \end{pmatrix}.$$

The corresponding characteristic equations takes the form

$$\lambda(\lambda + w_I + w_A)(\lambda - (\beta_I(1-\alpha)S^{**} - (\gamma_I + d_I)))(\lambda - (\beta_A(1-\alpha)^2 S^{**} - (\gamma_A + d_A + \mu))) = 0,$$

and the stability of E^{**} is deduced from the sign of $\beta_I(1-\alpha)S^{**} - (\gamma_I + d_I)$ and $\beta_A(1-\alpha)^2 S^{**} - (\gamma_A + d_A + \mu)$.
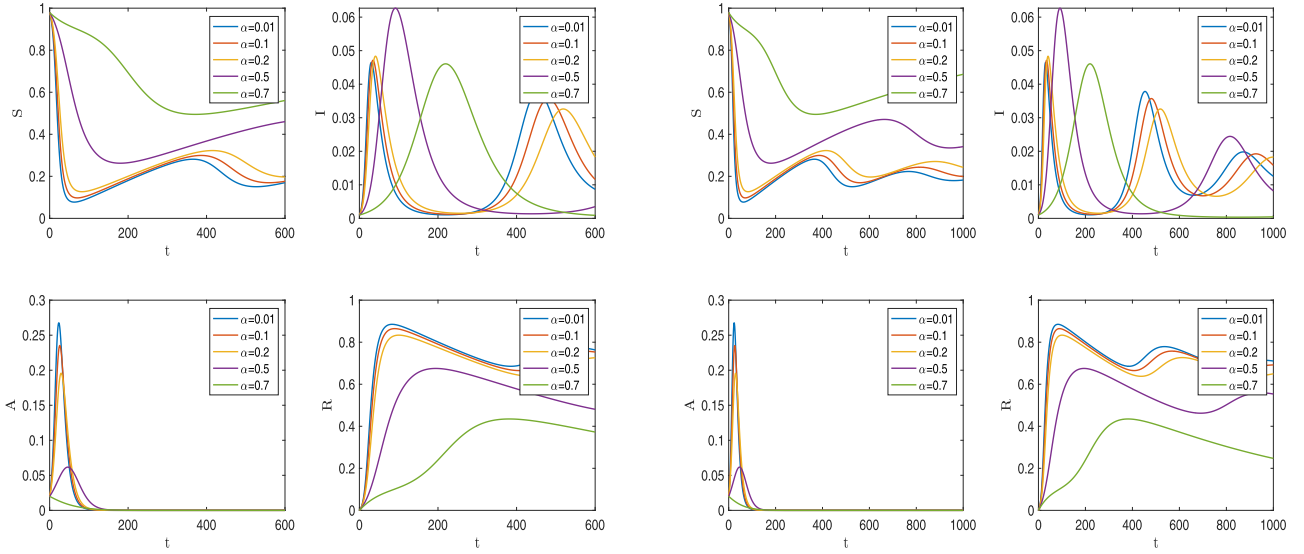
Proposition 3.1. *If $\mathcal{R}_0(S^{**}) < 1$, the equilibrium E^{**} is stable and unstable if $\mathcal{R}_0(S^{**}) > 1$.*

FIGURE 6. Temporal evolution of different populations S, I, A, R for diffrent values of $t = 600$ (*left*) and 1000 (*right*) for $0 < w_1 \ll 1$, $0 < w_2 \ll 1$.

Remark 3.2. With non total immunity $0 < w_1 \ll 1$, $0 < w_2 \ll 1$, (*i.e.* recovered population from SARS-Cov-2 may be re-infected for second time) the infectious population has more than one wave as we noticed from Figures 6 and 7 and the amplitude of the second wave is smaller than the first one, but with appropriate parameters values the amplitude of the second wave may be greater than the first one (see Fig. 8) and the asymptomatic population has only one wave over time Figures 6 and 7. Then, we conclude that the non total immunity can lead to more one wave of COVID-19 epidemic.

3.1. Application to real data

In what follows, we apply our results to real epidemic data of France and Morocco countries collected from the website www.statista.com (see Fig. 9). With matlab software and using the "lsqcurvefit" function to fit curves to real data, we get the estimated values of unknown parameters which are summarized in Table 3 for moroccan total population $N = 35.000.000$ and french total population $N = 50.000.000$. From Figure 9, we see that our model presents a good fit for real statistical data of infectious population with only one wave for moroccan case and more one wave for french case with a slight phase shift to the right due to high transmission value of β_I and low values of β_A, γ_A, low transition rate μ and low non-total immunity.

4. Sensitivity analysis

Sensitivity analysis is commonly used to determine the robustness of model predictions to parameters values, since there are usually errors in collected data and presumed parameters values. It is used to discover parameters that have a high impact on the threshold R_0 and should be targeted by intervention strategies. More accurately, sensitivity indices allows us to measure the relative changes in a variable when a parameter changes. For that purpose, we use the normalized forward sensitivity index of a variable with respect to a given parameter, which is defined as the ratio of the relative change in the variable to the relative change in the parameter. If such variable is differentiable with respect to the parameter, then the sensitivity index is defined as follows.
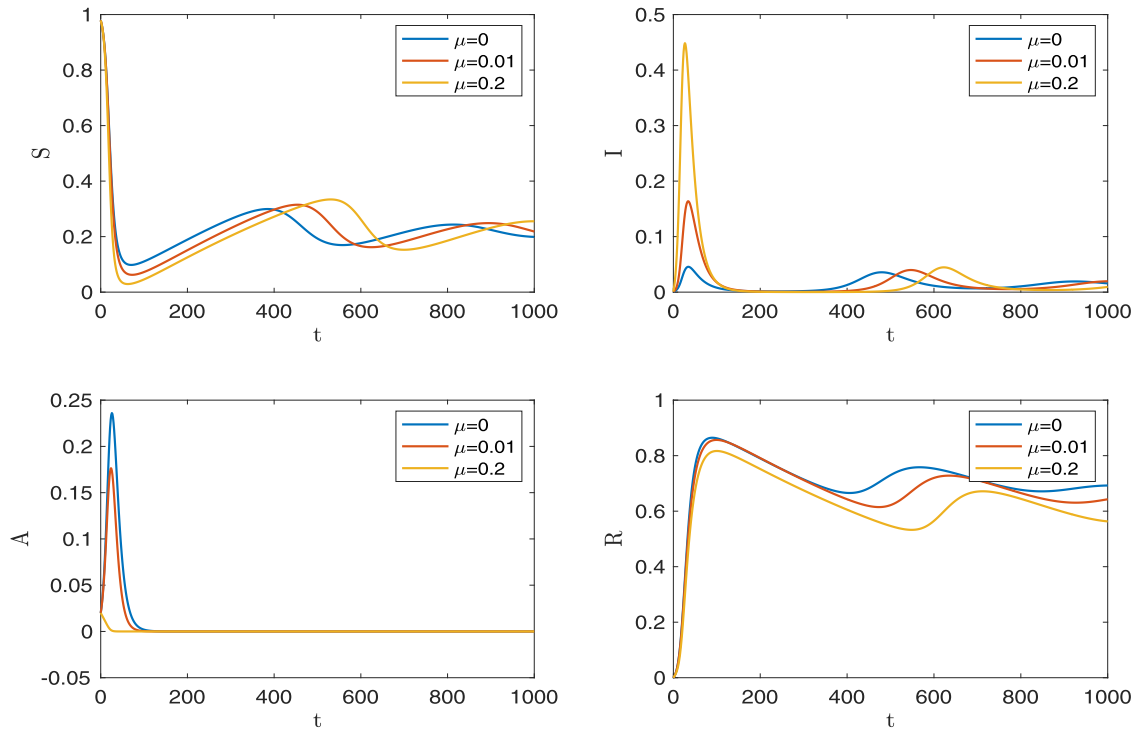
FIGURE 7. Temporal evolution of different populations S, I, A and R for $\mu = 0, 0.001, 0.2$ and $0 < w_1 \ll 1$, $0 < w_2 \ll 1$.
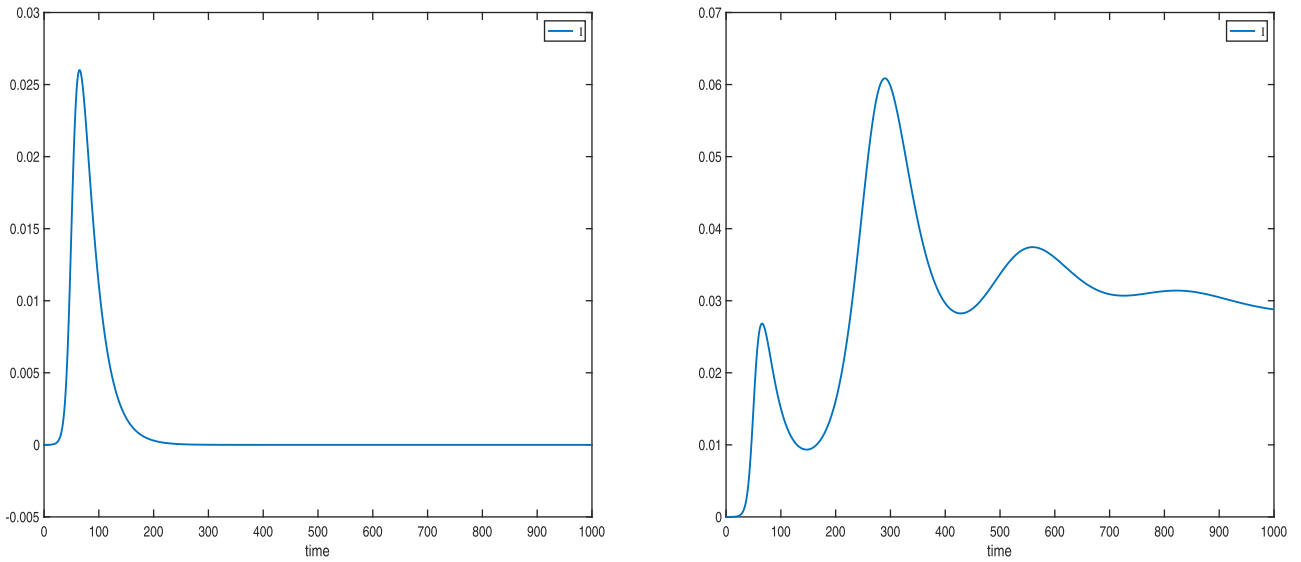

FIGURE 8. Temporal evolution of infectious population I with one wave, $w_1 = w_2 = 0$ (*left*) and two waves $w_1 = 0.003$, $w_2 = 0.00001$ (*right*), for $\alpha = 0.1$; $\beta_I = 0.294$; $\beta_A = 0.29$; $\gamma_I = 0.05$; $\gamma_A = 0.1$; $\mu = 0.0001$; $d_I = 0.007$; $d_A = 0.0001..$

TABLE 3. Estimating parameters values with "lsqcurvefit" function in Matlab.

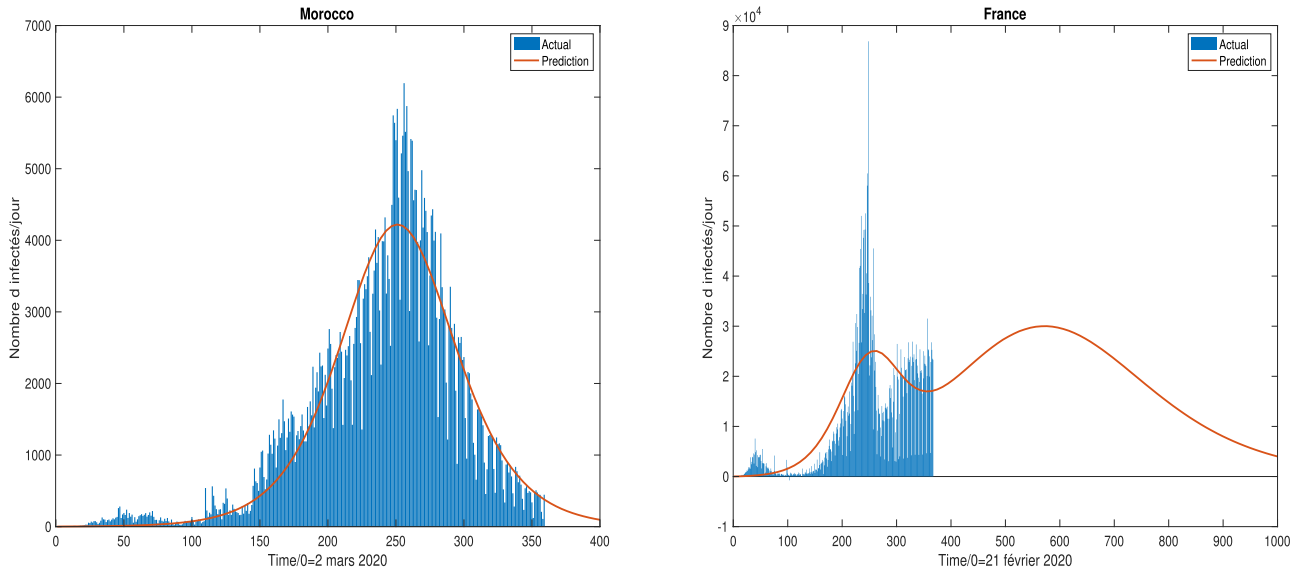| Parameters	France	Morocco	Reference
β_I	0.89730	0.38407	Estimated
β_A	0.07578	0.56392	Estimated
α	0.06205	0.05881	Estimated
γ_I	0.73	0.953	Health ministry
γ_A	0.5702×10^{-6}	0.00334	Estimated
d_I	0.09	0.017	Health ministry
d_A	$0.254.10^{-6}$	0.01182	Estimated
μ	0.04960	0.44828	Estimated
w_I	0.22×10^{-13}	0.31×10^{-13}	Estimated
w_A	0.22×10^{-13}	0.31×10^{-13}	Estimated

FIGURE 9. Curves fitting to Moroccan and French COVID-19 epidemic data.

Definition 4.1. [10] The normalized forward sensitivity index of R_0, which is differentiable with respect to a given parameter θ, is defined by

$$\Upsilon_\theta^{R_0} = \frac{\partial R_0}{\partial \theta} \frac{\theta}{R_0}.$$

From the definition of $R_0 = \rho(-FV^{-1}) = max(\lambda_1, \lambda_2)$, we discuss its sensitivity analysis for each case. Note that, the sensitivity index may depend on several parameters of the system, but also can be constant, independent of any parameter. For example, $\Upsilon_\theta^{R_0} = +1$ means that increasing (decreasing) θ by a given percentage increases (decreases) always R_0 by that same percentage. The estimation of a sensitive parameter should be carefully done, since a small perturbation in such parameter leads to relevant quantitative changes. On the other hand, the estimation of a parameter with a rather small value for the sensitivity index does not require as much
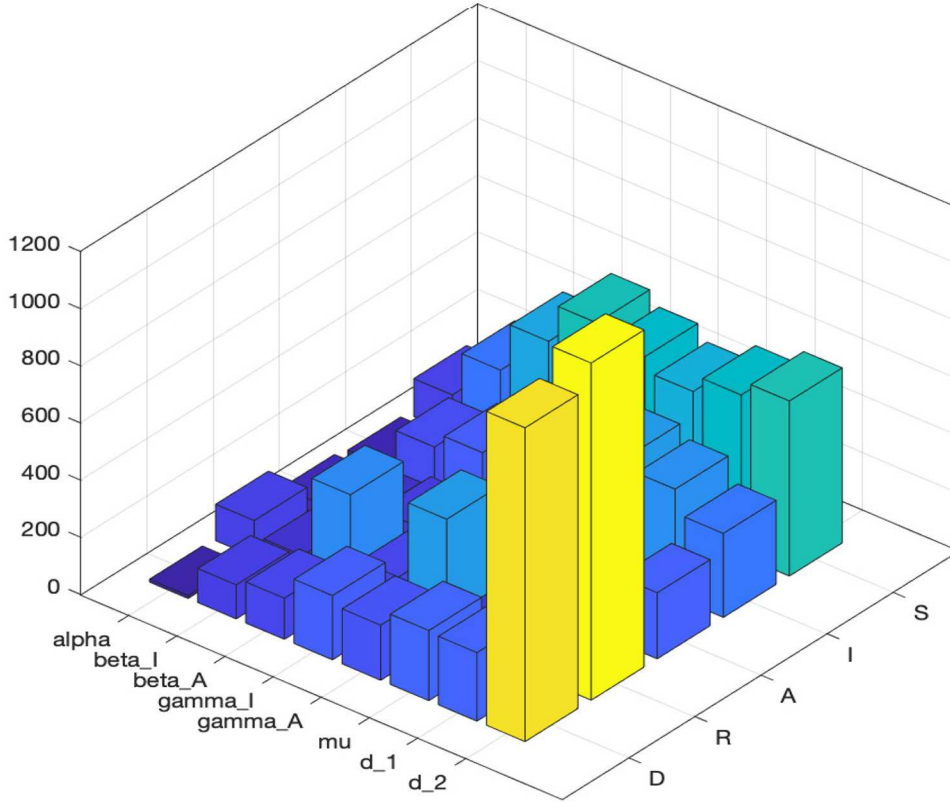
FIGURE 10. Sensitivity analysis with no normalization, $w_1 = w_2 = 0$, for $\alpha = 0.2$; $\beta_I = 0.294$; $\beta_A = 0.29$; $\gamma_I = 0.05$; $\gamma_A = 0.1$; $\mu = 0.002$; $d_I = 0.007$; $d_A = 0.0001$..

TABLE 4. Sensitivity of $R_0 = \lambda_1 = \dfrac{\beta_I(1-\alpha)}{\gamma_I + d_I}$ evaluated for the parameters values given in Table 2.

Parameter	Sensitivity index $R_0 = \lambda_1$	Index value "Morocco"	Index value "France"
α	$-\dfrac{\alpha}{1-\alpha}$	-0.0624	-0.066
β_I	$+1$	$+1$	$+1$
γ_I	$-\dfrac{\gamma_I}{\gamma_I + d_I}$	-0.982	-0.92
d_I	$-\dfrac{d_I}{\gamma_I + d_I}$	-0.017	-0.109

attention to estimate, because a small perturbation of parameters leads to small changes. We conclude that the most sensitive parameters to the basic reproduction number R_0 of COVID-19 are β_A, β_I, γ_A and γ_I. Then, to control the propagation of COVID-19 needs to control these sensitive parameters (Tab. 4).

Using non normalization, normalization relative to numerator and full dedimentialization techniques in Sim-Biology Toolbox for MATLAB to calculate the local sensitivity of each model state with respect to model constants, we can detect the most critical and sensitive parameters(see Figs. 10–12). In Figure 10, we see that

TABLE 5. Sensitivity of $R_0 = \lambda_2 = \dfrac{\beta_A(1-\alpha)^2}{\gamma_A + d_A + \mu}$ evaluated for the parameters values given in Table 2.

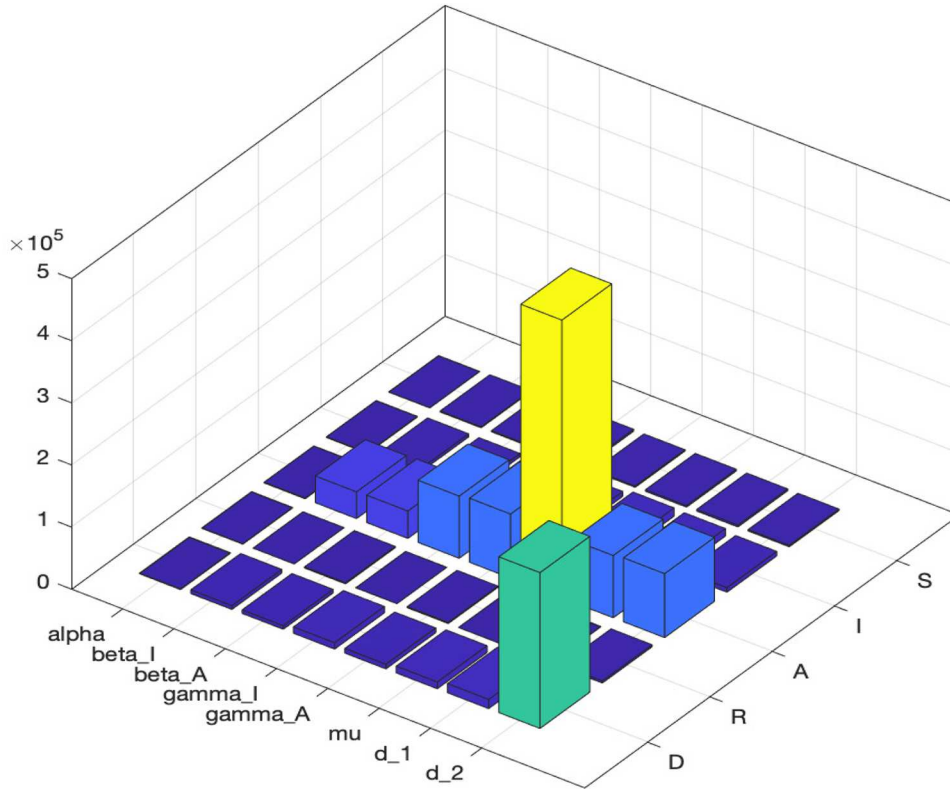| Parameter	Sensitivity index $R_0 = \lambda_2$	Index value "Morocco"	Index value "France"
α	$-2\dfrac{\alpha}{1-\alpha}$	-0.124	-0.132
β_A	$+1$	$+1$	$+1$
γ_A	$-\dfrac{\gamma_A}{\gamma_A + d_A + \mu}$	-0.0072	$-1.149. \times 10^{-5}$
d_A	$-\dfrac{d_A}{\gamma_A + d_A + \mu}$	-0.0255	-5.12×10^{-6}
μ	$-\dfrac{\mu}{\gamma_A + d_A + \mu}$	-0.96	-0.999

FIGURE 11. Sensitivity analysis with normalization relative to numerator, $w_1 = w_2 = 0$, for $\alpha = 0.2$; $\beta_I = 0.294$; $\beta_A = 0.29$; $\gamma_I = 0.05$; $\gamma_A = 0.1$; $\mu = 0.002$; $d_I = 0.007$; $d_A = 0.0001..$

d_2 is the most critical parameter followed by β_I and the other parameters. In Figure 11, we see that μ is the most critical parameter followed by d_2 and the other parameters. In Figure 12, we see that β_I and β_A are the most critical parameters followed by γ_I and γ_A and the other parameters. Then we deduce that the third technique agreed the results given in Tables 2 and 5.
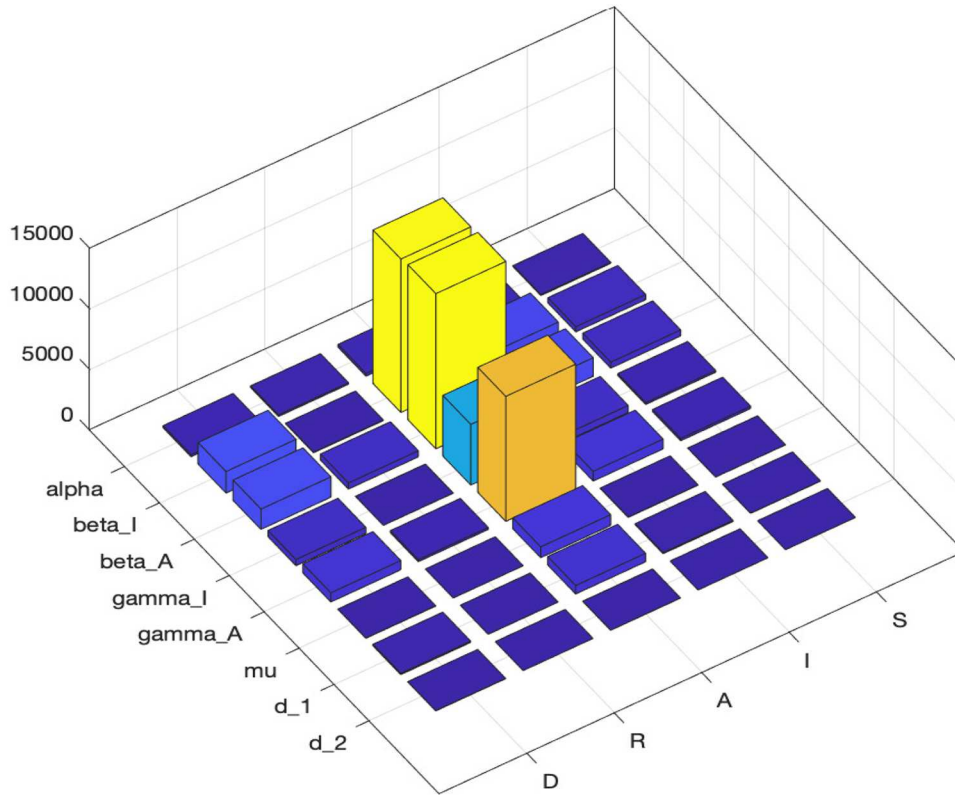
FIGURE 12. Sensitivity analysis with full dedimentialization, $w_1 = w_2 = 0$, for $\alpha = 0.2$; $\beta_I = 0.294$; $\beta_A = 0.29$; $\gamma_I = 0.05$; $\gamma_A = 0.1$; $\mu = 0.002$; $d_I = 0.007$; $d_A = 0.0001$..

5. CONCLUSION

In the presence of vaccines, some infectious diseases like measles have achieved herd immunity. But in the absence of vaccines, our mathematical finding confirm the answer of our question **"can immunity protect recovered individuals from reinfection of COVID-19?"**. The mathematical analysis given in this paper proves that, the immunity cannot protect humans from a second infection, in this case herd immunity is not the best concept to fight SARS-Cov-2 because we do not how long immunity lasts after an infection, and how much that varies from person to person and we do not know whether ending the pandemic through herd immunity is even possible. Actually, a vaccines against SARS-Cov-2 virus is the ideal way to achieve herd immunity. By the same way for each country, we have identified the parameters having an impacts on the basic reproduction number. This sensitivity analysis has been performed by using the numerical values of the parameters obtained through fitting of the model with actual number of infectious cases.

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