Bol. Soc. Paran. Mat.
©SPM – E-ISSN-2175-1188
SPM: www.spm.uem.br/bspm

(3s.) **v. 2025 (43)** : 1–14.
ISSN-0037-8712
doi:10.5269/bspm.67224

Modeling two-strain COVID-19 infection dynamics with vaccination strategy

Zakaria Yaagoub and Karam Allali

ABSTRACT: In this paper, we will study five differential equations describing two-strain COVID-19 infection dynamics with vaccination strategy. The variables of our model will represent the susceptible, the two strain infected sub-populations and the vaccinated individuals. First, we will study the well-posedness of our model. Next, we will give the different equilibria of our model. After that, we will study the global stability of each equilibrium. Finally, we will give different numerical simulations in order to illustrate the convergence of the solutions toward the equilibria. In addition, the comparison between the numerical tests and COVID-19 clinical data is conducted.

Key Words: Two-strain infection; COVID-19; vaccination; quarantine.

Contents

1. Introduction

Modeling infection dynamics have shown its great importance in understanding how infectious diseases spread, it also helps to take to right decisions to reduce the infection propagation [1]. The first epidemiological mathematical model have been established by Mckendrick and Kermack in 1927 that study the spread of epidemics by direct contact [2]. This model is abbreviated by SIR, with S meaning the susceptible individuals, I the infected individuals and R the removed individuals. The vaccination strategy is considered the most effective use in combating infectious disease and limiting its spread [3]. When this strategy is taking account, another compartment will be added, denoted by V and represents the vaccinated individuals, there are many works dealing with $SVIR$ models [4,5,6,7,8,9,10,11,12].

Coronavirus disease 2019, also called COVID-19 is a contagious disease caused by the virus SARS-CoV-2. This disease affects all ages and causes symptoms ranging from mild to severe, some infected individuals can recover without any medical intervention; meanwhile, others need intensive medical attention [13]. This virus has infected 562 million of people in the world [14]. This virus is characterized by its rapid spread and multiplicity of strains [15]. For example, the Alpha variant was discovered in the UK in October 2020, this virus drew attention after it spread rapidly, causing a high incidence of infection and causing a severe pandemic, this variant is 30 to 50% more spread than the first SARS-CoV-2 strain [16]. The Delta variant found in India at the end of 2020, this variant is 80 to 90% more contagious than the Alpha variant, so it caused more than twice the number of infected individuals compared to

2020 *Mathematics Subject Classification:* 92D30.
Submitted February 21, 2023. Published March 12, 2025

the previous variants [17]. The Omicron sub-variants appeared in November 2021 in South Africa and Botswana, these variants have more than 30 mutations are more contagious than the Delta variant [18].

The mathematical literature contains an important number of works that have studied COVID-19 [19,20,21,22,23,24,25,26,27,28,29,30]. However, as COVID-19 is distinguished by the multiplicity of its variants, so we can use multi-strain models to describe the dynamics of COVID-19. In the case of two-strain, the infected population will be divided into two subpopulations I_1 and I_2, with the first one, denoting the population of the COVID-19 first strain infected individuals and the second standing for the population of the infected individuals by the second strain. These models use two incidence functions, the first one for the strain-1 and the second stands for the strain-2 infection. Several works use two-strain models to study the dynamics of COVID-19 [31,32,33,34,35,36,37]. Recently Baba and Hincal [38] have suggested a two-strain epidemic with two incidence functions, the first one is bilinear for the strain-1 and the second is non-monotonic for the strain-2, they demonstrated the global stability of equilibrium point using some Lyapunov functions. This last work will be improved by the same authors [39] by choosing two non-monotonic incidence functions. Bentaleb and Amine [40] proposed a two-strain epidemic model with bilinear and non-monotonic incidence functions, they add a new compartment E for the exposed individuals and demonstrated the global stability of the equilibria. This last work will be improved by Meskaf et al. [41] by proposing two non-monotonic incidence rates.

Motivated by the previous publications, we will add to this work the effect of vaccination and quarantine strategies in order to study our new two-strain COVID-19 infection model:

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \alpha(1-u_1)SI_1 - \beta(1-u_2)SI_2 - (\sigma+\mu)\,S, \\ \dfrac{dV}{dt} = \sigma S - \eta V I_2 - \mu V, \\ \dfrac{dI_1}{dt} = \alpha(1-u_1)SI_1 - (\gamma_1+\mu)\,I_1, \\ \dfrac{dI_2}{dt} = \beta(1-u_2)SI_2 + \eta I_2 V - (\gamma_2+\mu)\,I_2, \\ \dfrac{dR}{dt} = \gamma_1 I_1 + \gamma_2 I_2 - \mu R. \end{cases} \tag{1.1}$$

With S, V, I_1, I_2 and R represent respectively the compartment of susceptible, vaccinated, strain-1 infected, strain-2 infected and recovered individuals. All of the parameters are given in Table 1. The infection dynamics of our two-strain epidemic model are represented in Fig 1.

Table 1: Description of parameters of the model (1.1).

Parameters	Description
Λ	Recruitment rate
$1/\mu$	Average life of the population
α	Strain-1 infection rate
β	Strain-2 infection rate
u_1	Strain-1 quarantine effect
u_2	Strain-2 quarantine effect
σ	Strain 1 vaccination rate
η	Transmission rate of vaccinated individuals to strain 2
$1/\gamma_1$	Strain-1 average infection period
$1/\gamma_2$	Strain-2 average infection period

The present work is organized as follows. In Section 2, we will prove the existence, positivity and boundedness of model (1.1) solutions. In Section 3, we will give the different equilibrium points depending on the basic reproduction numbers, in the same section we will prove the global stability of these equilibria.

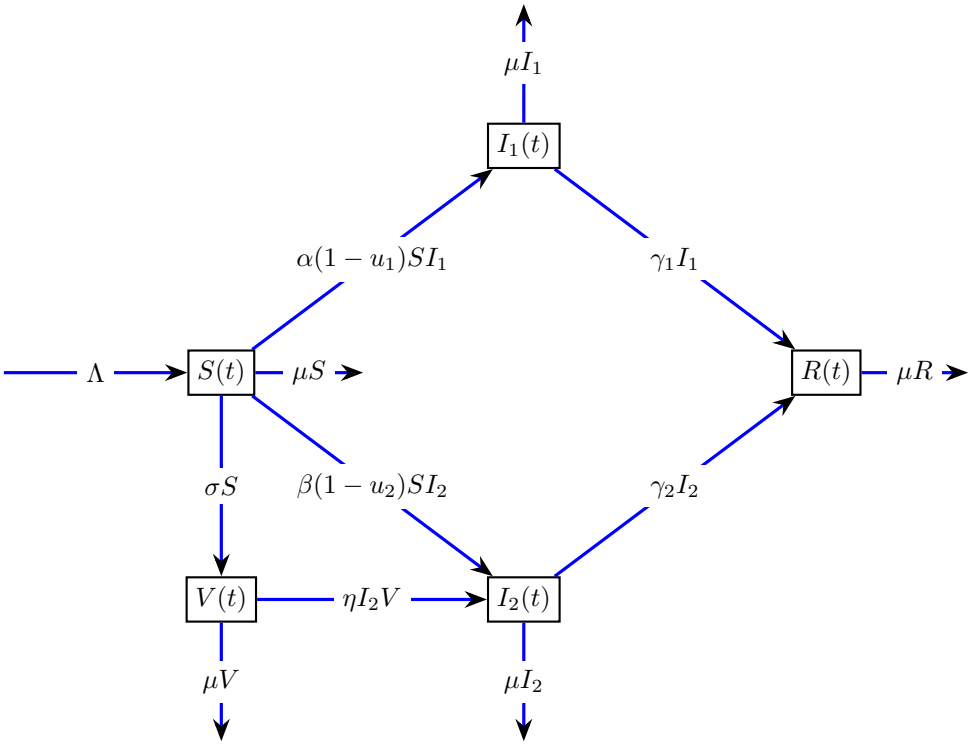

Figure 1: The diagram two-strain model.

Some numerical simulations are suggest in Section 4 in order to confirm and value our theoretical results. The last section concludes the work.

2. Existence, positivity and boundedness of solutions

In this section, we will prove the existence, positivity and boundedness of solutions.

Proposition 2.1 *For any positive initial condition $S(0)$, $V(0)$, $I_1(0)$, $I_2(0)$ and $R(0)$, the system (1.1) has a unique, positive and bounded solution $(S(t), I_1(t), I_2(t), R(t))$ for all $t \geq 0$.*

Proof: We can rewrite the system in the form

$$\dot{X} = f(X),$$

with

$$X = \begin{pmatrix} S \\ V \\ I_1 \\ I_2 \\ R \end{pmatrix}, \text{ and } f(X) = \begin{pmatrix} \Lambda - \alpha(1-u_1)SI_1 - \beta(1-u_2)SI_2 - (\sigma+\mu)\,S \\ \sigma S - \eta V I_2 - \mu V \\ \alpha(1-u_1)SI_1 - (\gamma_1+\mu)\,I_1 \\ \beta(1-u_2)SI_2 + \eta I_2 V - (\gamma_2+\mu)\,I_2 \\ \gamma_1 I_1 + \gamma_2 I_2 - \mu R \end{pmatrix}.$$

For the uniqueness of solution, it is clear that the function F is Lipschitz function, moreover we have

$$\| F(X) - F(Y) \|_1 \leq k \| X - Y \|_1, \ \forall X, Y \in \mathbb{R}^5_+$$

with

$$k = \max\{A, D, E, F\}$$

and

$$A = \alpha(1-u_1)I_1 + \beta(1-u_2)I_2 + \mu,\ D = \gamma_1 + \mu,\ E = \gamma_2 + \mu,\ F = \mu.$$

So, the system (1.1) has a unique solution $\in \mathbb{R}^5_+$.
For the positivity, we have

$$\dot{S}\ |_{S=0} = \Lambda \geq 0,$$
$$\dot{I}_1\ |_{I_1=0} = 0 \geq 0,$$
$$\dot{I}_2\ |_{I_2=0} = 0 \geq 0,$$
$$\dot{V}\ |_{V=0} = \sigma S \geq 0.$$

So, we can deduce that solution remain positive for all $t \geq 0$..
Now we will prove that that the biologically feasible region

$$H = \{(S,\, I_1,\, I_2,\, V) \in \mathbb{R}^5_+ \, such\, that\, S + I_1 + I_2 + V \leq \frac{\Lambda}{\mu}\}$$

is positively invariant. Let

$$N(t) = S(t) + I_1(t) + I_2(t) + V(t),$$

By adding the equations of system (1.1), we will have

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

then,

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

hence,

$$\lim_{t\to+\infty} N(t) = \frac{\Lambda}{\mu},$$

hence, $\exists t_0 > 0$ such as $\forall t \geq t_0$ we have

$$N(t) \leq \frac{\Lambda}{\mu},$$

so its clear that H is positively invariant. We conclude that the system (1.1) has a unique, positive and bounded solution in $\in \mathbb{R}^5_+$. □

3. Analysis of the model

In this section, we show there exists a disease-free equilibrium point and three equilibrium points, we will study the global stability of these steady state using the Lyapunov functional method's, the first note to notice is that the first four equations of the system (1.1) are independent to the last equation and as the total number of the population N verifies the Eq. (3.2). so we can reduce the system (1.1) to the system (3.1)

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \alpha(1-u_1)SI_1 - \beta(1-u_2)SI_2 - (\sigma+\mu)\,S, \\ \dfrac{dV}{dt} = \sigma S - \eta V I_2 - \mu V, \\ \dfrac{dI_1}{dt} = \alpha(1-u_1)SI_1 - (\gamma_1+\mu)\,I_1, \\ \dfrac{dI_2}{dt} = \beta(1-u_2)SI_2 + \eta I_2 V - (\gamma_2+\mu)\,I_2. \end{cases} \tag{3.1}$$

$$R = N - S - I_1 - I_2. \tag{3.2}$$

3.1. The basic reproduction number

The basic reproduction number R_0 is defined biologically as the number of secondary cases caused by one typical infected individual in a population containing only susceptible individuals [43], mathematically R_0 is the spectral radius of the next generation matrix FV^{-1} with F is the non-negative matrix of new infection cases and V noted the matrix of the transition of the infections associated with the system (3.1) Let

$$F = \begin{pmatrix} \frac{\beta\Lambda}{\sigma+\mu} & 0 \\ 0 & \frac{\alpha\Lambda}{\sigma+\mu} + \frac{\eta\sigma\Lambda}{\mu(\mu+\sigma)} \end{pmatrix},$$

and

$$V = \begin{pmatrix} \gamma_1+\mu & 0 \\ 0 & \gamma_2+\mu \end{pmatrix}.$$

So, we have

$$FV^{-1} = \begin{pmatrix} \frac{\alpha(1-u_1)\Lambda}{(\gamma_1+\mu)(\sigma+\mu)} & 0 \\ 0 & \frac{\beta(1-u_2)\Lambda}{(\gamma_2+\mu)(\sigma+\mu)} + \frac{\eta\sigma\Lambda}{\mu(\gamma_2+\mu)(\mu+\sigma)} \end{pmatrix}.$$

So the basic reproduction number of the model (3.1) is

$$R_0 = \max R_0^1,\ R_0^2,$$

with

$$R_0^1 = \frac{\alpha(1-u_1)\Lambda}{(\gamma_1+\mu)(\sigma+\mu)},$$

and

$$R_0^2 = \frac{\beta(1-u_2)\Lambda}{(\gamma_2+\mu)(\sigma+\mu)} + \frac{\eta\sigma\Lambda}{\mu(\gamma_2+\mu)(\mu+\sigma)}.$$

As

$$d = \gamma_1+\mu \text{ and } e = \gamma_2+\mu,$$

then, we have

$$R_0^1 = \frac{\alpha(1-u_1)\Lambda}{d(\sigma+\mu)},$$

and

$$R_0^2 = \frac{\beta(1-u_2)\Lambda}{e(\sigma+\mu)} + \frac{\eta\sigma\Lambda}{\mu e(\mu+\sigma)}$$

3.2. Steady states

The system (3.1) has one disease-free equilibrium point and three endemic equilibrium points given by:

1. The disease-free equilibrium point $E_0(S_0,\ V_0,\ 0,\ 0)$, where

$$S_0 = \frac{\Lambda}{\sigma+\mu},\ V_0 = \frac{\sigma\Lambda}{\mu(\mu+\sigma)}.$$

2. The strain 1 endemic equilibrium point $E_1(S_{s_1}^*,\ V_{s_1}^*,\ I_{1,s_1}^*,\ I_{2,s_1}^*)$, where

$$S_{s_1}^* = \frac{d}{\alpha(1-u_1)},\ V_{s_1}^* = \frac{\sigma d}{\mu\alpha(1-u_1)},$$

$$I_{1,s_1}^* = \frac{\sigma+\mu}{\alpha(1-u_1)}\left(R_0^1-1\right),\ I_{2,s_1}^* = 0.$$

3. The strain 2 endemic equilibrium point $E_2(S^*_{s_2},\, V^*_{s_2},\, I^*_{1,s_2},\, I^*_{2,s_2})$, where

$$S^*_{s_2} = \frac{e-\eta V^*_{s_2}}{\beta(1-u_2)},\; V^*_{s_2} = \frac{\sigma S^*_{s_2}}{\eta I^*_{2,s_2}+\mu},$$

$$I^*_{1,s_2} = 0,\; I^*_{2,s_2} = \frac{\sigma+\mu}{\beta(1-u_2)}\left(\frac{\Lambda}{(\sigma+\mu)S^*_{s_2}}\right).$$

4. The total endemic equilibrium point $E_t(S^*_t,\, V^*_t,\, I^*_{1,t},\, I^*_{2,t})$,

$$S^*_t = \frac{d}{\alpha(1-u_1)} = \frac{e-\eta V^*_t}{\beta(1-u_2)},\; V^*_t = \frac{d\beta(1-u_2)}{\alpha(1-u_1)\eta}\left(\frac{R^1_0}{R^2_0}-1\right),$$

$$I^*_{1,t} = \frac{\Lambda}{\alpha(1-u_1)S^*_t} - \frac{\beta(1-u_2)I^*_{2,t}}{\alpha(1-u_1)} - \frac{\sigma+\mu}{\alpha(1-u_1)},\; I^*_{2,t} = \frac{\mu}{\eta}\left(\frac{\sigma S^*_t}{\mu V^*_t}-1\right).$$

Remark 3.1 The equilibrium points E_1, E_2 and E_t exists when $R^1_0 > 1$ and $R^2_0 > 1$.

3.3. Global stability

Theorem 3.1 *If $R^1_0 \le 1$ and $R^2_0 \le 1$. Then the disease-free equilibrium point E_0 is globally asymptotically stable.*

Proof: We consider the Lyapunov function in $\in \mathbb{R}^4_+$

$$L_f(S,\, V,\, I_1,\, I_2) = S_0\left(\frac{S}{S_0} - \ln\left(\frac{S}{S_0}\right) - 1\right) + V_0\left(\frac{V}{V_0} - \ln\left(\frac{V}{V_0}\right) - 1\right) + I_1 + I_2.$$

The time derivative of L_f is given by

$$\begin{aligned}
\dot{L_f}(S,\, V,\, I_1,\, I_2) &= \dot{S} - \frac{\dot{S}}{S}S_0 - \dot{V} - \frac{\dot{V}}{V}V_0 + \dot{I}_1 + \dot{I}_2 \\
&= (\sigma+\mu)S_0 + (\sigma+\mu)S - (\sigma+\mu)S_0\frac{S_0}{S} + \alpha(1-u_1)S_0I_1 + \beta(1-u_2)S_0I_2 + (\sigma+\mu)S_0 \\
&+ \sigma S - \mu V - \sigma S\frac{V_0}{V} + \eta V_0 I_2 + \mu V_0 - dI_1 + eI_2 \\
&\le \mu S_0\left(2 - \frac{S}{S_0} - \frac{S_0}{S}\right) + \sigma S_0\left(3 - \frac{S_0}{S} - \frac{V}{V_0} - \frac{SV_0}{S_0V}\right) + I_1\left(\alpha(1-u_1)S_0 - d\right) \\
&+ I_2\left(\beta(1-u_2)S_0 - e\right) \\
&\le \mu S_0\left(2 - \frac{S}{S_0} - \frac{S_0}{S}\right) + \sigma S_0\left(3 - \frac{S_0}{S} - \frac{V}{V_0} - \frac{SV_0}{S_0V}\right) + dI_1\left(R^1_0 - 1\right) + eI_2\left(R^2_0 - 1\right).
\end{aligned}$$

As the arithmetic mean is greater than or equal to the geometric mean, we will have

$$2 - \frac{S}{S_0} - \frac{S_0}{S} \le 0$$

and

$$3 - \frac{S_0}{S} - \frac{V}{V_0} - \frac{SV_0}{S_0V} \le 0.$$

Then, when $R^1_0 \le 1$ and $R^2_0 \le 1$, we will have $\dot{L_f} \le 0$. Therefore the disease-free equilibrium point E_0 is globally asymptotically stable. □

Theorem 3.2 *If $R^1_0 > 1$ and $R^2_0 \le 1$, then the strain 1 endemic disease equilibrium E_1 is globally asymptotically stable.*

Proof: We consider the Lyapunov function in $\mathbb{R}^4_+$

$$L_1(S,\,V,\,I_1,\,I_2) = S^*_{s_1}\left(\frac{S}{S^*_{s_1}} - \ln\left(\frac{S}{S^*_{s_1}}\right) - 1\right) + V^*_{s_1}\left(\frac{V}{V^*_{s_1}} - \ln\left(\frac{V}{V^*_{s_1}}\right) - 1\right) + I^*_{1,s_1}\left(\frac{I_1}{I^*_{1,s_1}} - \ln\left(\frac{I_1}{I^*_{1,s_1}}\right) - 1\right) + I_2.$$

The time derivative of L_1 is given by

$$\begin{aligned}\dot{L}_1(S,\,V,\,I_1,\,I_2) &= \left(1 - \frac{S^*_{s_1}}{S}\right)\dot{S} + \left(1 - \frac{V^*_{s_1}}{V}\right)\dot{V} + \left(1 - \frac{I^*_{s_1}}{I}\right)\dot{I}_1 + \dot{I}_2\\ &= \Lambda - \mu S - \mu V - dI_1 - eI_2 - \Lambda\frac{S^*_{s_1}}{S} + \alpha(1-u_1)S^*_{s_1}I_1 + \beta(1-u_2)S^*_{s_1}I_2\\ &+ (\sigma+\mu)S^*_{s_1} - \sigma S\frac{V^*_{s_1}}{V} + \eta V^*_{s_1}I_2 + \mu V^*_{s_1} - \alpha(1-u_1)SI^*_{1,s_1} + dI^*_{1,s_1}\end{aligned}$$

As E_1 is an equilibrium point of system (3.1),
we will have:

$$\begin{cases}\Lambda = \alpha(1-u_1)S^*_{s_1}I^*_{1,s_1} + (\sigma+\mu)S^*_{s_1},\\ \mu V^*_{s_1} = \sigma S^*_{s_1},\\ \alpha(1-u_1)S^*_{s_1}I^*_{1,s_1} = dI^*_{1,s_1}.\end{cases}$$

Therefore

$$\begin{aligned}\dot{L}_1 &= \sigma S^*_{s_1}\left(3 - \frac{S^*_{s_1}}{S} - \frac{SV^*_{s_1}}{S^*_{s_1}V} - \frac{V}{V^*_{s_1}}\right) + (\mu S^*_{s_1} + dI^*_{1,s_1})\left(2 - \frac{S^*_{s_1}}{S} - \frac{S}{S^*_{s_1}}\right) + I_1(\alpha(1-u_1)S^*_{s_1} - d)\\ &+ I_2(\eta V^*_{s_1} + \beta(1-u_2)S^*_{s_1} - e)\\ &\le \sigma S^*_{s_1}\left(3 - \frac{S^*_{s_1}}{S} - \frac{SV^*_{s_1}}{S^*_{s_1}V} - \frac{V}{V^*_{s_1}}\right) + (\mu S^*_{s_1} + dI^*_{1,s_1})\left(2 - \frac{S^*_{s_1}}{S} - \frac{S}{S^*_{s_1}}\right) + eI_2(R_0^2 - 1).\end{aligned}$$

We replace $S^*_{s_1}$ we obtain

$$\alpha(1-u_1)S^*_{s_1} - d = 0.$$

As the arithmetic mean is greater than or equal to the geometric mean, we will have

$$2 - \frac{S^*_{s_1}}{S} - \frac{S}{S^*_{s_1}} \le 0$$

and

$$3 - \frac{S^*_{s_1}}{S} - \frac{SV^*_{s_1}}{S^*_{s_1}V} - \frac{V}{V^*_{s_1}} \le 0.$$

Then, when $R_0^2 \le 1$, we will have $\dot{L}_1 \le 0$. Therefore the strain 1 endemic equilibrium point E_1 is globally asymptotically stable. □

Theorem 3.3 *If $R_0^2 > 1$ and $R_0^1 \le 1$, then the strain 2 endemic equilibrium point E_2 is globally asymptotically stable.*

Proof: We consider the Lyapunov function in $\mathbb{R}^4_+$

$$L_2(S,\,V,\,I_1,\,I_2) = S^*_{s_2}\left(\frac{S}{S^*_{s_2}} - \ln\left(\frac{S}{S^*_{s_2}}\right) - 1\right) + V^*_{s_2}\left(\frac{V}{V^*_{s_2}} - \ln\left(\frac{V}{V^*_{s_2}}\right) - 1\right) + I_1 + I^*_{2,s_2}\left(\frac{I_2}{I^*_{2,s_2}} - \ln\left(\frac{I_2}{I^*_{2,s_2}}\right) - 1\right).$$

The time derivative of L_2 is given by

$$\begin{aligned}\dot{L}_2(S,\,V,\,I_1,\,I_2) &= \left(1-\frac{S^*_{s_2}}{S}\right)\dot{S}+\left(1-\frac{V^*_{s_2}}{V}\right)\dot{V}+\dot{I}_1+\left(1-\frac{I^*_{s_2}}{I}\right)\dot{I}_2\\ &= \Lambda-\mu S-\mu V-dI_1-eI_2-\Lambda\frac{S^*_{s_2}}{S}+\alpha(1-u_1)S^*_{s_2}I_1+\beta(1-u_2)S^*_{s_2}I_2\\ &+(\sigma+\mu)S^*_{s_1}-\sigma S\frac{V^*_{s_2}}{V}+\eta V^*_{s_2}I_2+\mu V^*_{s_2}-\beta(1-u_2)SI^*_{2,s_2}-\eta I^*_{2,s_2}V+eI^*_{2,s_2},\end{aligned}$$

As E_2 is an equilibrium point of system (3.1),
we will have:

$$\begin{cases}\Lambda=\beta(1-u_2)S^*_{s_2}I^*_{2,s_2}+(\sigma+\mu)S^*_{s_2},\\ \mu V^*_{s_2}=\sigma S^*_{s_2}-\eta V^*_{s_2}I^*_{2,s_2},\\ \beta(1-u_2)S^*_{s_2}I^*_{2,s_2}+\eta V^*_{s_2}I^*_{2,s_2}=eI^*_{2,s_2}.\end{cases}$$

Therefore

$$\begin{aligned}\dot{L}_2 &= \sigma S^*_{s_2}\left(3-\frac{S^*_{s_2}}{S}-\frac{SV^*_{s_2}}{S^*_{s_2}V}-\frac{V}{V^*_{s_2}}\right)+(\mu S^*_{s_2}+\beta(1-u_2)S^*_{s_2}I^*_{2,s_2})\left(2-\frac{S^*_{s_2}}{S}-\frac{S}{S^*_{s_2}}\right)\\ &+I_2(\alpha(1-u_1)S^*_{s_2}-d)+I_2(\beta(1-u_2)S^*_{s_2}-e)\\ &\le \sigma S^*_{s_2}\left(3-\frac{S^*_{s_2}}{S}-\frac{SV^*_{s_2}}{S^*_{s_2}V}-\frac{V}{V^*_{s_2}}\right)+(\mu S^*_{s_2}+\beta(1-u_2)S^*_{s_2}I^*_{2,s_2})\left(2-\frac{S^*_{s_2}}{S}-\frac{S}{S^*_{s_2}}\right)\\ &+dI_1(R^1_0-1)+I_2(\beta(1-u_2)S^*_{s_2}-e).\end{aligned}$$

We replace $S^*_{s_2}$ we obtain

$$\beta(1-u_2)S^*_{s_2}-e\le 0.$$

As the arithmetic mean is greater than or equal to the geometric mean, we will have

$$2-\frac{S^*_{s_2}}{S}-\frac{S}{S^*_{s_2}}\le 0$$

and

$$3-\frac{S^*_{s_2}}{S}-\frac{SV^*_{s_2}}{S^*_{s_2}V}-\frac{V}{V^*_{s_2}}\le 0.$$

Then, when $R^1_0\le 1$, we will have $\dot{L}_2\le 0$. Therefore the strain 2 endemic equilibrium point E_2 is globally asymptotically stable. □

Theorem 3.4 *If $R^1_0>1$ and $R^2_0>1$, then total endemic equilibrium E_t is globally asymptotically stable.*

Proof: We consider the Lyapunov function in $\mathbb{R}^4_+$

$$\begin{aligned}L_3(S,\,V,\,I_1,\,I_2) &= S^*_t\left(\frac{S}{S^*_t}-\ln\left(\frac{S}{S^*_t}\right)-1\right)+V^*_t\left(\frac{V}{V^*_t}-\ln\left(\frac{V}{V^*_t}\right)-1\right)\\ &+I_1\left(\frac{I_1}{I^*_{1,t}}-\ln\left(\frac{I_1}{I^*_{1,t}}\right)-1\right)+I^*_{2,t}\left(\frac{I_2}{I^*_{2,t}}-\ln\left(\frac{I_2}{I^*_{2,t}}\right)-1\right).\end{aligned}$$

The time derivative of L_2 is given by

$$\begin{aligned}\dot{L}_3(S,\,V,\,I_1,\,I_2) &= \left(1-\frac{S^*_t}{S}\right)\dot{S}+\left(1-\frac{V^*_t}{V}\right)\dot{V}+\left(1-\frac{I^*_{1,t}}{I}\right)\dot{I}_1+\left(1-\frac{I^*_{2,t}}{I}\right)\dot{I}_2\\ &= \Lambda-\mu S-\mu V-dI_1-eI_2-\Lambda\frac{S^*_t}{S}+\alpha(1-u_1)S^*_tI_1+\beta(1-u_2)S^*_tI_2+(\sigma+\mu)S^*_t\\ &-\sigma S\frac{V^*_t}{V}+\eta V^*_tI_2+\mu V^*_t-\alpha(1-u_1)SI^*_{1,t}+\beta(1-u_2)SI^*_{2,t}-\eta I^*_{2,t}V+eI^*_{2,t},\end{aligned}$$

as E_t is an equilibrium point of system (3.1),
we will have:

$$\begin{cases} \Lambda = \alpha(1-u_1)S_t^*I_{1,t}^* + \beta(1-u_2)S_t^*I_{2,t}^* + (\sigma+\mu)S_t^*, \\ \mu V_t^* = \sigma S_t^* - \eta V_t^* I_{2,t}^*, \\ \alpha(1-u_1)S_t^*I_{1,t}^* = dI_{1,t}^*, \beta(1-u_2)S_t^*I_{2,t}^* + \eta V_t^* I_{2,t}^* = eI_{2,t}^*. \end{cases}$$

Therefore

$$\dot{L_3} \leq \sigma S_t^* \left(3 - \frac{S_t^*}{S} - \frac{SV_t^*}{S_t^*V} - \frac{V}{V_t^*}\right) + (\mu S_t^* + \beta(1-u_2)S_t^*I_{2,t}^* + dI_{1,t}^*)\left(2 - \frac{S_t^*}{S} - \frac{S}{S_t^*}\right)$$
$$+ I_1(\alpha(1-u_1)S_t^* - d) + I_2(\beta(1-u_2)S_t^* - e).$$

We replace S_t^* we obtain

$$\alpha(1-u_1)S_t^* - d = 0,$$

and

$$\beta(1-u_2)S_t^* - e \leq 0.$$

As the arithmetic mean is greater than or equal to the geometric mean, we will have

$$2 - \frac{S_t^*}{S} - \frac{S}{S_t^*} \leq 0$$

and

$$3 - \frac{S_t^*}{S} - \frac{SV_t^*}{S_t^*V} - \frac{V}{V_t^*} \leq 0.$$

Then $\dot{L_3} \leq 0$. The total endemic equilibrium E_t is globally asymptotically stable. □

4. Numerical simulations

Numerical simulations will be given in this section to check to global stability of all equilibrium points and to confirm and value our theoretical results.

4.1. The equibria stability

In this subsection, we will study the stability of each equilibrium point. More precisely the following figures show the evolution of the disease for the given parameters (see Table 2).

Table 2: Parameters values of two-strain model.

Parameters	Fig. 2	Fig. 3	Fig. 4	Fig. 5
Λ	1	1	1	1
α	0.1	0.2	0.1	0.2
β	0.15	0.15	0.2	0.2
u_1	0.2	0.1	0.1	0.1
u_2	0.4	0.4	0.3	0.3
σ	0.3	0.3	0.3	0.3
μ	0.1	0.1	0.1	0.1
η	0.02	0.02	0.1	0.1
γ_1	0.3	0.2	0.3	0.2
γ_2	0.4	0.4	0.3	0.3

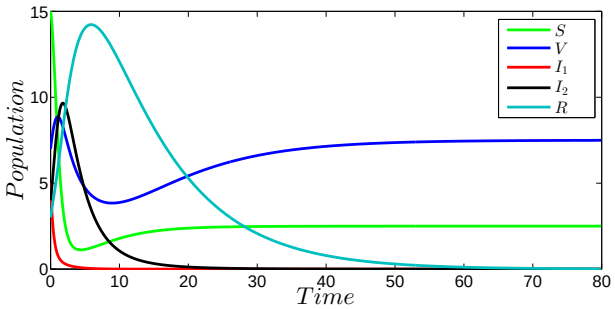

Figure 2: Stability of the disease-free equilibrium with $R_0^1 = 0.50$ and $R_0^2 = 0.75$.

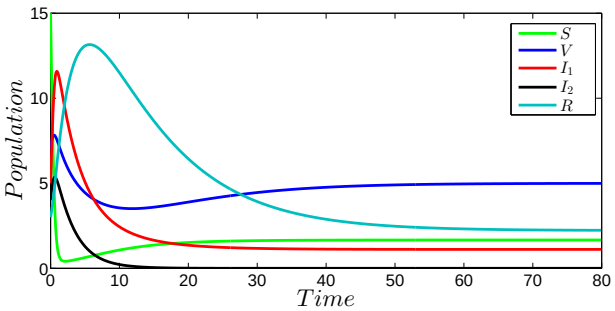

Figure 3: Stability of the strain-1 endemic equilibrium with $R_0^1 = 1.50$ and $R_0^2 = 0.75$.

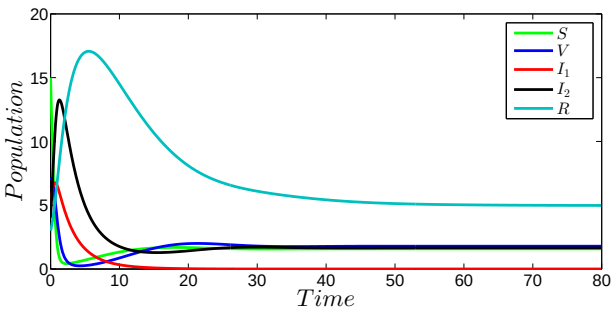

Figure 4: Stability of the strain-2 endemic equilibrium with $R_0^1 = 0.50$ and $R_0^2 = 2.75$.

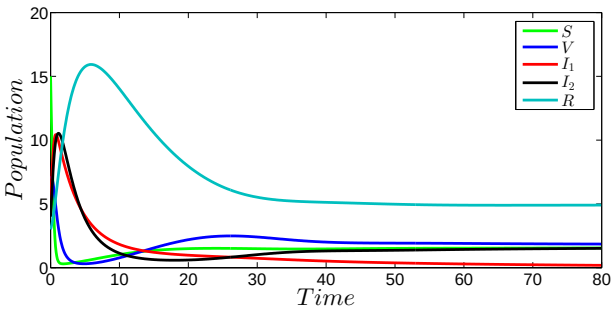

Figure 5: Stability of the total endemic equilibrium with $R_0^1 = 1.50$ and $R_0^2 = 2.75$.

Fig. 2 shows the evolution of all two-strain variables. We can observe that all curves converge to 0 except the curves of susceptible and vaccinated individuals. The two basic reproduction numbers is less than 1 ($R_0^1 = 0.50$ and $R_0^2 = 0.75$), which coincides with our result given in Theorem 3.1. Fig. 3 shows that the strain-1 persists while the other strain-2 dies out. The strain-1 reproduction number is greater than 1 while the other strain-2 reproduction number is less than 1 ($R_0^1 = 1.50$ and $R_0^2 = 0.75$), which coincides with our result given in Theorem 3.2. In Fig. 4 we can observe that the strain-2 persists while the other strain-1 dies out. The strain-2 reproduction number is greater than 1 while the other strain-1 reproduction number is less than 1 ($R_0^1 = 0.50$ and $R_0^2 = 2.75$), which coincides with our result given in Theorem 3.3. Finally, Fig. 5 we can see that both strains persist and both reproduction numbers are greater than 1 ($R_0^1 = 1.50$ and $R_0^2 = 2.75$), which coincides with our result given in Theorem 3.4. We can observe from this last figure that the strains-2 dominate the other strain-1, therefore the strain with the large reproduction number will dominate the other. All previous numerical results confirm our theoretical results.

4.2. The quarantine effect

In this subsection, we show the quarantine effect to reduce the infection of COVID-19.

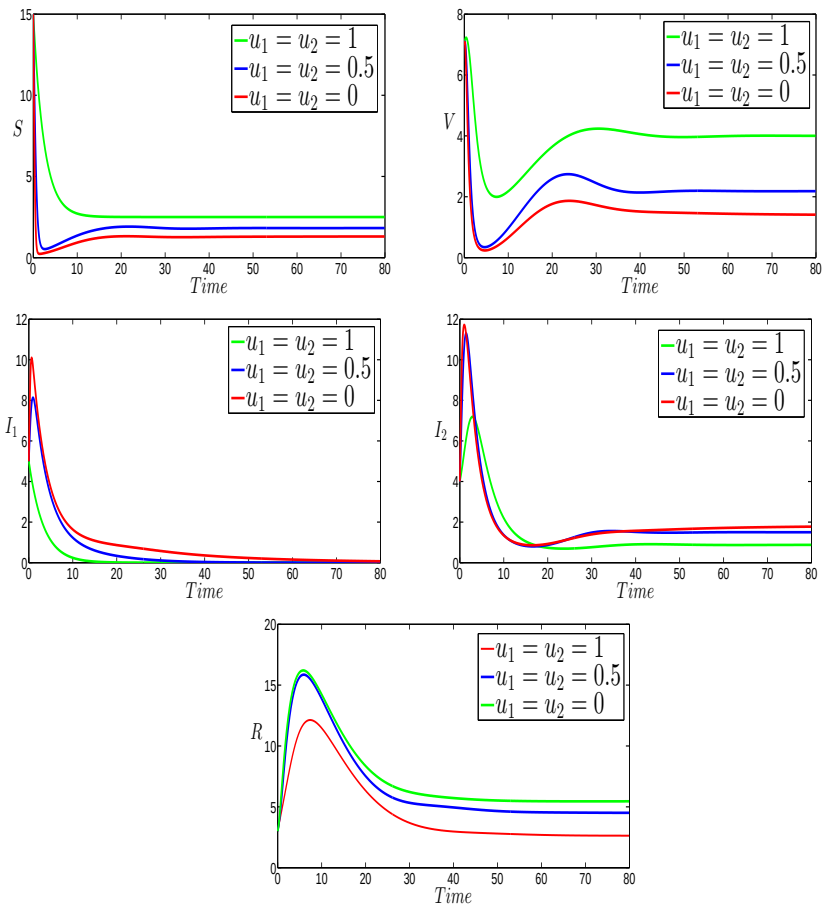

Figure 6: The dynamics of the population for different values of quarantine.

Fig. 6 illustrates the quarantine effect to reduce the infection, we can observe that the number of infected individuals I_1 and I_2 decreases in the existence of quarantine. The number of removed individuals also decreases, this is due to the fact that the number of infected individuals diminishes.

4.3. Model comparison with COVID-19 clinical data

In this section we will compare our model with the clinical data of COVID-19.

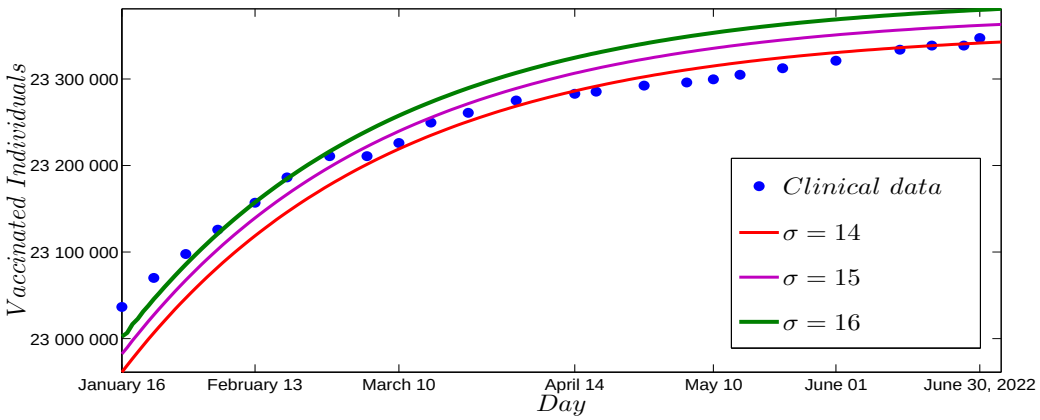

Figure 7: Fitting of the two-strain model to COVID-19 clinical data for different σ values.

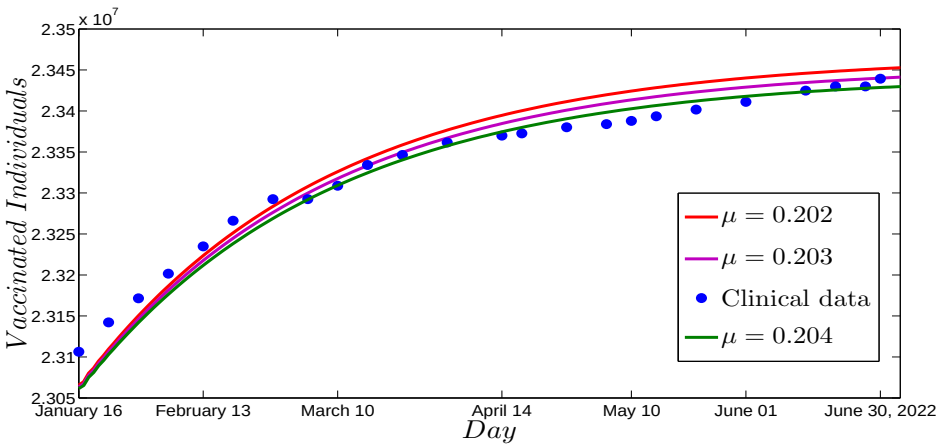

Figure 8: Fitting of the two-strain model to COVID-19 clinical data for different μ values.

Fig. 7 and Fig. 8 show the dynamics of vaccinated individuals between January 16 and June 30, 2022, the value of the following parameters: $\Lambda = 4756892$, $\alpha = 0.2$, $\beta = 0.2$, $u_1 = 0.1$, $u_2 = 0.3$, $\gamma_1 = 0.2$, $\gamma_2 = 0.3$, $\rho = 0.3$, $m_1 = 0.001$, $m_2 = 0.002$, $\phi_1 = 0.4$, $\phi_2 = 0.3$, $\psi_1 = 0.4$ and $\psi_2 = 0.3$. The solid curves show the dynamic of the two-strain model for different values of θ and δ, and the points show the clinical COVID-19 infected individuals reported in Morocco [42]. We can observe that our model results are very close to clinical data, so the model can fit COVID-19 clinical data especially for a short period.

5. Conclusion

In this work, we have studied a two-strain COVID-19 epidemic model with quarantine and vaccination strategy. We have proved the existence, positivity and boundedness of solutions, we have shown that the model has for equilibrium points, the first namely the disease-free equilibrium, the strain-1 endemic equilibrium, the strain-2 endemic equilibrium and the total endemic equilibrium. We have used the next

generation matrix method to find the both reproduction numbers R_0^1 and R_0^2 and by using some Lyapunov functions we have given the conditions of the global stability of all the equilibria: when both reproduction numbers are less than 1, the disease-free equilibrium is globally asymptotically stable which means that both strains die out. When R_0^1 is greater than 1 and R_0^2 is less than 1 which means that the first strain persists and the second strain dies out, the strain-1 endemic equilibrium is globally asymptotically stable, and when R_0^2 is greater than 1 and R_0^1 is less than 1 which means that the second strain persists and the first strain dies out, the strain-2 is globally asymptotically stable. The last equilibrium point is globally asymptotically stable when both reproduction numbers are greater than 1, we have shown also that the strain-1 with the greater reproduction number will dominate the other strain. Numerical simulations are given in order to support our theoretical results concerning the stability of equilibria and to show the quarantine efficiency to reduce the infection of COVID-19, and finally, we have compared our model with real vaccination COVID-19 data to value our analytical results. As the future direction of this present work, one can consider the same problem with generalized incidence rates.

References

1. Nikolaou, Michael. "Revisiting the standard for modeling the spread of infectious diseases." Scientific reports 12.1 (2022): 1-16.
2. Kermark, M., and A. Mckendrick. "Contributions to the mathematical theory of epidemics. Part I." Proc. r. soc. a 115.5 (1927): 700-721.
3. Puri, Neha, et al. "Social media and vaccine hesitancy: new updates for the era of COVID-19 and globalized infectious diseases." Human vaccines & immunotherapeutics 16.11 (2020): 2586-2593.
4. Djilali, Salih, and Soufiane Bentout. "Global dynamics of SVIR epidemic model with distributed delay and imperfect vaccine." Results in Physics 25 (2021): 104245.
5. Zhang, Xinhong, and Qing Yang. "Threshold behavior in a stochastic SVIR model with general incidence rates." Applied Mathematics Letters 121 (2021): 107403.
6. Gao, Jianguo, Chao Zhang, and Jinliang Wang. "Analysis of a reaction–diffusion SVIR model with a fixed latent period and non-local infections." Applicable Analysis 101.2 (2022): 497-518.
7. Liu, Xinyu, and Yuting Ding. "Stability and Numerical Simulations of a New SVIR Model with Two Delays on COVID-19 Booster Vaccination." Mathematics 10.10 (2022): 1772.
8. Liu, Qun, and Daqing Jiang. "Global dynamical behavior of a multigroup SVIR epidemic model with Markovian switching." International Journal of Biomathematics 15.01 (2022): 2150080.
9. Seydou, Moussa, and Moussa Tessa. "Approximations of Quasi-Stationary Distributions of the Stochastic SVIR Model for the Measles." Journal of Applied Mathematics and Physics 9.9 (2021): 2277-2289.
10. Oke, M. O., et al. "On the application of optimal control strategies to a generalized SVIR model." Journal of Physics: Conference Series. Vol. 1734. No. 1. IOP Publishing, 2021.
11. Ogunmiloro, Oluwatayo Michael, Fatima Ohunene Abedo, and Hammed Kareem. "Numerical and stability analysis of the transmission dynamics of SVIR epidemic model with standard incidence rate." MALAYSIAN JOURNAL OF COMPUTING 4.2 (2019): 349-361.
12. Ouakka, Abdellah, Abdelhai El Azzouzi, and Zakia Hammouch. "Global dynamic behavior of a vaccination–age SVIR model with treatment and general nonlinear incidence rate." Journal of Computational and Applied Mathematics 422 (2023): 114848.
13. Marchiori, Massimo. "COVID-19 and the social distancing paradox: Dangers and solutions." arXiv preprint arXiv:2005.12446 (2020).
14. World Health Organization. https://covid19.who.int/
15. Hossain, Md Kamal, Majid Hassanzadeganroudsari, and Vasso Apostolopoulos. "The emergence of new strains of SARS-CoV-2. What does it mean for COVID-19 vaccines?." Expert review of vaccines 20.6 (2021): 635-638.
16. Walker, A. Sarah, et al. "Tracking the emergence of SARS-CoV-2 alpha variant in the United Kingdom." New England Journal of Medicine 385.27 (2021): 2582-2585.
17. Shiehzadegan, Shayan, et al. "Analysis of the delta variant B. 1.617. 2 COVID-19." Clinics and Practice 11.4 (2021): 778-784.
18. Collie, Shirley, et al. "Effectiveness of BNT162b2 vaccine against omicron variant in South Africa." New England Journal of Medicine 386.5 (2022): 494-496.
19. Singh, Pushpendra, and Anubha Gupta. "Generalized SIR (GSIR) epidemic model: An improved framework for the predictive monitoring of COVID-19 pandemic." ISA transactions 124 (2022): 31-40.

20. Nguyen, Tri K., et al. "Enhancing Covid-19 virus spread modelling using an activity travel model." Transportation Research Part A: Policy and Practice (2022).

21. Ghosh, Kalpita, and Asim Kumar Ghosh. "Study of COVID-19 epidemiological evolution in India with a multi-wave SIR model." Nonlinear Dynamics (2022): 1-9.

22. Ghosh, Kalpita, and Asim Kumar Ghosh. "Study of COVID-19 epidemiological evolution in India with a multi-wave SIR model." Nonlinear Dynamics (2022): 1-9.

23. Vega, Roberto, Leonardo Flores, and Russell Greiner. "SIMLR: Machine Learning inside the SIR model for COVID-19 Forecasting." Forecasting 4.1 (2022): 72-94.

24. Cooper, Ian, et al. "Dynamical analysis of the infection status in diverse communities due to COVID-19 using a modified SIR model." Nonlinear Dynamics (2022): 1-14.

25. Fu, Yuting, et al. "Optimal lockdown policy for vaccination during COVID-19 pandemic." Finance research letters 45 (2022): 102123.

26. Bodini, Antonella, et al. "Underdetection in a stochastic SIR model for the analysis of the COVID-19 Italian epidemic." Stochastic Environmental Research and Risk Assessment 36.1 (2022): 137-155.

27. Bandekar, Shraddha Ramdas, and Mini Ghosh. "Mathematical modeling of COVID-19 in India and its states with optimal control." Modeling Earth Systems and Environment 8.2 (2022): 2019-2034.

28. Deniz, U. Ç. A. R., and Elcin Celik. "Analysis of Covid 19 disease with SIR model and Taylor matrix method." AIMS Mathematics 7.6 (2022): 11188-11200.

29. Majee, Suvankar, et al. "Complex dynamics of a fractional-order SIR system in the context of COVID-19." Journal of Applied Mathematics and Computing (2022): 1-24.

30. Wang, Jiale, et al. "A modified SIR model for the COVID-19 epidemic in China." Journal of Physics: Conference Series. Vol. 2148. No. 1. IOP Publishing, 2022.

31. Tchoumi, S. Y., H. Rwezaura, and J. M. Tchuenche. "Dynamic of a two-strain COVID-19 model with vaccination." Results in Physics (2022): 105777.

32. Martsenyuk, Vasyl, Marcin Bernas, and Aleksandra Klos-Witkowska. "Two-strain COVID-19 model using delayed dynamic system and big data." Ieee Access 9 (2021): 113866-113878.

33. de Leon, Ugo Avila-Ponce, Eric Avila-Vales, and Kuan-lin Huang. "Modeling COVID-19 dynamic using a two-strain model with vaccination." Chaos, Solitons & Fractals 157 (2022): 111927.

34. Yuliani, Endang, et al. "On the Modeling of COVID-19 Transmission Dynamics with Two Strains: Insight through Caputo Fractional Derivative." Fractal and Fractional 6.7 (2022): 346.

35. Amine, Saida, and Karam Allali. "Dynamics of a time-delayed two-strain epidemic model with general incidence rates." Chaos, Solitons & Fractals 153 (2021): 111527.

36. Yaagoub, Zakaria, Jaouad Danane, and Karam Allali. "Global Stability Analysis of Two-Strain SEIR Epidemic Model with Quarantine Strategy." Nonlinear Dynamics and Complexity: Mathematical Modelling of Real-World Problems. Cham: Springer International Publishing, 2022. 469-493.

37. Yaagoub, Zakaria, and Karam Allali. "Global Stability of Multi-Strain SEIR Epidemic Model with Vaccination Strategy." Mathematical and Computational Applications 28.1 (2023): 9.

38. Baba, Isa Abdullahi, and Evren Hincal. "Global stability analysis of two-strain epidemic model with bilinear and non-monotone incidence rates." The European Physical Journal Plus 132.5 (2017): 1-10.

39. Isa Abdullahi Baba, Evren Hincal and Sultan Hamed Khalifa Alsaadi, Global stability analysis of a two-strain model with awareness, Advances in Di erential Equations and Control Processes, 19(2)(2018), 83-100.

40. Dounia Bentaleb, Saida Amine, Lyapunov function and global stability for a two-strain SEIR model with bilinear and nonmonotone incidence, International Journal of Biomathematics 2019; 12(2): 1950021.

41. Meskaf, Adil, et al. "Global stability analysis of a two-strain epidemic model with non-monotone incidence rates." Chaos, Solitons & Fractals 133 (2020): 109647.

42. Statistics of Moroccan Health Ministry on COVID-19. https://www.sante.gov.ma/

43. Yaagoub, Zakaria, and Karam Allali. "Fractional HBV infection model with both cell-to-cell and virus-to-cell transmissions and adaptive immunity." Chaos, Solitons & Fractals 165 (2022): 112855.

Zakaria Yaagoub and Karam Allali,

Laboratory of Mathematics, Computer Science and Applications,

Faculty of Sciences and Technologies,

University Hassan II of Casablanca, PO Box 146, Mohammedia 20650, Morocco.

E-mail address: zakaria.yaag@gmail.com and allali@hotmail.com