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A mathematical model for a transmissible disease with two variants

Research Article

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Abstract: A mathematical model describing the propagation of an infectious disease with one strain and its two variants is considered. Model analysis is carried out to obtain and establish the stability of the five equilibrium points, using Lienard-Chipart criterion and Lyapunov functions. The existence of these equilibria are characterized using exact methods of algebraic geometry and computer algebra. The asymptotic or global stability of endemic equilibria is established and the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$. Model simulation is done with Python software to study the effects of health precautions and treatment and the results are analyzed. It is observed that the high treatment rate accompanied by a suitable rate of compliance with health precautions allows for the control the disease.

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Keywords: Computer algebra • Stability • Mathematical model • Two variants

1. Introduction

Mathematical models have become important tools for the analysis of propagation, control of infectious diseases and impacts of different interventions [1–3]

All viruses evolve and can produce variants. While in most cases the properties of the virus change little and the mutation hardly affects the population, in other cases there may be a greater impact. This is the case of Coronavirus disease 2019 (COVID-19) which is an infectious disease caused by the Sars-cov-2 virus.

The first case was registered in Wuhan, China, in December 2019. The initial strain of Sars-cov-2 spread around the world in 2020 and has already led to the appearance of variants like Delta and Omicron. The emergence of other variants are not excluded. It therefore becomes important to understand the dynamics of the evolution of such a type of disease with the emergence of variants from an initial strain that regularly mutates. Some models have been proposed to simulate the spread of COVID-19 with this type of variants. T. Li and Y. Guo, in [4], develop a mathematical model to simulate the possible impact of vaccination, isolation and nucleic acid testing measures to control the spread of the disease with Delta variant. G. Gilberto and A. Abraham propose in [5], a mathematical model based on ordinary differential equations to investigate potential consequences of the appearance of a new more transmissible Sars-cov-2 in a given region. In [6], a mathematical model to examine the impact of non pharmaceutical interventions, including the COVID-test, genome sequencing test capacity, contact tracing and quarantine strength, on the induced epidemic wave is developped.

A two-strain compartmental epidemic model is proposed in [7], to explore the impact of non-pharmaceutical interventions. A novel compartmental model which captures new strategies that promote self testing and adjust the

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eligibility for PCR tests, social behaviours, booster vaccines campaign and features of the newest variant Omicron is presented in [8]. Note that the majority of infected with COVID-19 have only mild or moderate symptoms and will recover without specific treatment. However, some will become seriously ill and require medical attention.

In this paper, we presented a compartmental model of a disease transmission with one virus and its variant with treatment and health precautions. The purpose of the current study is to assess the combined use of observation, treatment and health precautions strategies to an infectious epidemiological disease with two variants. The model presented has 5 equilibria.

The use of algebraic geometry and computer algebra approaches is of a valuable contribution for the characterization and study of the equilibria stability, particularly when the number of strains or variants increases.

We start with the presentation of the model in section 2. In the section 3, we determine and characterize the equilibria of the model algebraically using Gröebner base. In section 4, we study the stability of equilibria of the model by the methods of algebraic geometry and in particular for disease-free equilibrium, we calculate the basic reproduction number for a verification of algebraic characterizations. The global stability of disease-free equilibrium is studied in section 5. Section 6 is devoted to numerical simulation.

2. Mathematical model

A mathematic model to study the transmission dynamics of an infectious disease which initial strain have two new variants, in a constant population is formulated. The population is divided into six classes. The susceptible are in class S, the infected with initial strain in are in class I_1, those who are infected by the first and the second variant are respectively in class I_2 and I_3. Those under treatment are in class T and people in observation in class O. The state variable of each class also represents the proportion of its individuals. The transfer of individuals between the different classes of the model is carried out as follows: The transfer of individuals between the different classes of the model is carried out as follows: The size of all classes decreases due to the mortality rate. The classes of infected I_1, I_2 and I_3 receive the individuals of class S infected by the forces of infection $(1-e)\beta_1 I_1$, $(1-e)\beta_2 I_2$ and $(1-e)\beta_3 I_3$ respectively. Individuals of I_2 and I_3 are also derived from the mutation of those of I_1 due to the ν_1 and ν_2 rates respectively. Individuals from I_1, I_2, I_3 and O progress to T due to the treatment rate γ. Class S receives elements of the total population due to the birth rate, and of the classes O and T due to the rates $(1-\gamma)$ and r respectively. Individuals in S progress to T by the identification force $(1-e)\beta T$, and to I_1, I_2 and I_3 by the infection forces $(1-e)\beta_1 I_1$, $(1-e)\beta_2 I_2$ and $(1-e)\beta_3 I_3$ respectively. Class O receives from class S the elements which have been in contact with the individuals of T by the identification force $(1-e)\beta T$. Individuals from O progress to S and T due to the rates $(1-\mu)(1-\gamma)$ and $(1-\mu)\gamma$ respectively. Using parameters and their description in table 1 and the model presentation and formulation given in section 2, the model transfer diagram is given by fig. 1.

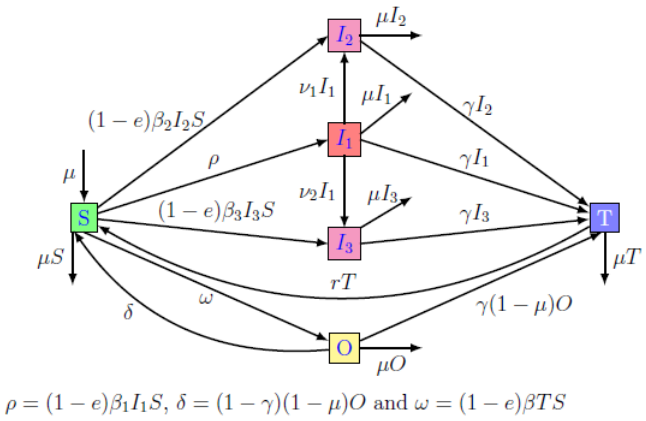

Fig. 1. Model Transfer Diagram

From the transfer diagram of the model in fig. 1, the dynamical system of the model is as follows: The model is

represented by the following system of ordinary differential equations

$$\begin{cases} \dot{S} = \mu + r\,T + (1-\gamma)(1-\mu)O - ((1-e)(\beta T + \beta_3 I_3 + \beta_2 I_2 + \beta_1 I_1) + \mu)S \\ \dot{I_3} = \nu_2 I_1 + (1-e)\beta_3 I_3 S - (\gamma+\mu) I_3 \\ \dot{I_2} = \nu_1 I_1 + (1-e)\beta_2 I_2 S - (\gamma+\mu) I_2 \\ \dot{I_1} = (1-e)\beta_1 I_1 S - (\nu_1 + \nu_2 + \gamma + \mu) I_1 \\ \dot{T} = \gamma(I_3 + I_2 + I_1 + (1-\mu)O) - (r+\mu)T \\ \dot{O} = (1-e)\beta T S - O \end{cases} \tag{1}$$

where $\beta_1 > \max\{\beta_2, \beta_3\}$ We easily verify that for

Table 1. Parameters and their Biological meaning.

Symbol	Biological meaning
β	contact rate
β_1	transmission rate for the initial strain
β_2	transmission rate for the first variant
β_3	transmission rate for the second variant
ν_1	mutation rate for the first variant
ν_2	mutation rate for the second variant
μ	death rate
r	cure rate

$$0 = \dot{S} + \dot{I_1} + \dot{I_2} + \dot{I_3} + \dot{T} + \dot{O} = \mu(1 - S - I_1 - I_2 - I_3 - T - O),$$

we have $S + I_1 + I_2 + I_3 + T + O = 1$, for any system solution (1).

3. Model equilibria

The system (1) can be written as $\dot{x} = f(x,u)$, where
$u = (\gamma, e, \beta, \beta_1, \beta_2, \beta_3, \nu_1, \nu_2, \mu, r)$ is the list of parameters and $x = (S, I_3, I_2, I_1, T, O)$ is the list of state variables. An important feature of this model is common to a large class of epidemiological models, see [9, 10], is that the components of the vector field f are polynomials as a function of u and x. Thus we can use the powerful tools of computer algebra such as Gröbner bases, see [11–13], to determine the equilibria of the model, which are the solutions of the algebraic equations system $f(u,x) = 0$:

$$\begin{cases} \mu + r\,T + (1-\gamma)(1-\mu)O - ((1-e)(\beta T + \beta_3 I_3 + \beta_2 I_2 + \beta_1 I_1) + \mu)S &= 0 \\ \nu_2 I_1 + (1-e)\beta_3 I_3 S - (\gamma+\mu) I_3 &= 0 \\ \nu_1 I_1 + (1-e)\beta_2 I_2 S - (\gamma+\mu) I_2 &= 0 \\ (1-e)\beta_1 I_1 S - (\nu_1 + \nu_2 + \gamma + \mu) I_1 &= 0 \\ \gamma(I_3 + I_2 + I_1 + (1-\mu)O) - (r+\mu)T &= 0 \\ (1-e)\beta T S - O &= 0 \end{cases} \tag{2}$$

The calculation of the Groebner base [13] of the system $f_1, f_2, f_3, f_4, f_5, f_6$ according to the lexicographical order $S \prec I_3 \prec I_2 \prec I_1 \prec T \prec O$ allows us to have a system (3) with a triangular form of six equations according to the given order of variables. The first element of the Groebner base calculated is a polynomial of degree 5 in S with roots

$$s_0 = 1, s_1 = \frac{\gamma+\mu}{\beta_2(1-e)}, s_2 = \frac{\gamma+\mu}{\beta_3(1-e)}, s_3 = \frac{(\mu+r)}{\gamma\beta(1-e)(1-\mu)}$$

and

$$s_4 = \frac{\gamma+\mu+\nu_1+\nu_2}{\beta_1(1-e)}.$$

Substituting S by s_0 into system (3), we obtain a single equilibrium, noted E_0, whose components are

$$(1,0,0,0,0,0)$$

This is the disease free equilibrium of the model, and it exists for all values of parameters.
Replacing S by s_1 into system (3), we obtain a single equilibrium, noted E_1, whose components are

$$\begin{aligned}
s_1 &= \frac{\gamma+\mu}{(1-e)\beta_2}\\
i_{31} &= 0\\
i_{21} &= -\frac{V_2V_5}{\beta_2(1-e)(\beta\gamma\mu(\gamma+\mu)+\beta_2(\gamma+\mu+r))}\\
i_{11} &= 0\\
t_1 &= \frac{\gamma V_2}{(1-e)(\beta\gamma\mu(\gamma+\mu)+\beta_2(\gamma+\mu+r))}\\
o_1 &= \frac{\gamma\beta(\gamma+\mu)V_2}{\beta_2(1-e)(\beta\gamma\mu(\gamma+\mu)+\beta_2(\gamma+\mu+r))}
\end{aligned}$$

where $V_2 = (1-e)\beta_2 - (\gamma+\mu)$ and $V_5 = \gamma\beta(\gamma+\mu)(1-\mu) - \beta_2(\mu+r)$.
This equilibrium corresponds to the non-existence of infectious cases linked to the strain and to the second variant. It is endemic and exist if and only if $V_2 \geq 0$ and $V_5 \leq 0$.
By replacing S by s_2 into system (3), we also obtain a single equilibrium, noted E_2, whose components are

$$\begin{aligned}
s_2 &= \frac{\gamma+\mu}{(1-e)\beta_3}\\
i_{32} &= -\frac{V_3V_6}{\beta_3(1-e)(\beta\gamma\mu(\gamma+\mu)+\beta_3(\gamma+\mu+r))}\\
i_{22} &= 0\\
i_{12} &= 0\\
t_2 &= \frac{\gamma V_3}{(1-e)(\beta\gamma\mu(\gamma+\mu)+\beta_3(\gamma+\mu+r))}\\
o_2 &= \frac{\gamma\beta(\gamma+\mu)V_3}{\beta_3(1-e)(\beta\gamma\mu(\gamma+\mu)+\beta_3(\gamma+\mu+r))}
\end{aligned}$$

where $V_3 = (1-e)\beta_3 - (\gamma+\mu)$ and $V_6 = \gamma\beta(\gamma+\mu)(1-\mu) - \beta_3(\mu+r)$.
This equilibrium which corresponds to the non-existence of infectious cases linked to the strain and to the first variant is endemic and exist if and only if $V_3 \geq 0$ and $V_6 \leq 0$.

By replacing S by s_3 in the system (3), we obtain an equilibrium, noted E_3, whose components are

$$\begin{aligned}
s_3 &= \frac{\mu+r}{(1-e)(1-\mu)\gamma\beta}\\
i_{33} &= 0\\
i_{23} &= 0\\
i_{13} &= 0\\
t_3 &= \frac{\gamma V_4}{\beta(1-e)(\gamma+\mu+r-\gamma\mu)}\\
o_3 &= \frac{(r+\mu)V_4}{\gamma\beta(1-e)(1-\mu)(\gamma+\mu+r-\gamma\mu)}
\end{aligned}$$

where $V_4 = \gamma\beta(1-e)(1-\mu) - \mu - r$.
This equilibrium corresponds to the non-existence of infectious cases in circulation and exist if and only if $V_4 \geq 0$.
Finally the substitution of S by s_4 in the system (3) gives a single equilibrium, noted E_4, whose components are

$$\begin{aligned}
s_4 &= \frac{\nu_1+\nu_2+\gamma+\mu}{(1-e)\beta_1}\\
i_{34} &= \frac{\nu_2V_1V_7V_9}{(\nu_1+\nu_2+\gamma+\mu)(1-e)(\beta\gamma\mu(\nu_1+\nu_2+\gamma+\mu)+\beta_1(\gamma+\mu+r))V_8}\\
i_{24} &= \frac{\nu_1V_1V_7V_{10}}{(\nu_1+\nu_2+\gamma+\mu)(1-e)(\beta\gamma\mu(\nu_1+\nu_2+\gamma+\mu)+\beta_1(\gamma+\mu+r))V_8}\\
i_{14} &= -\frac{V_1V_7V_9V_{10}}{\beta_1(\nu_1+\nu_2+\gamma+\mu)(1-e)(\beta\gamma\mu(\nu_1+\nu_2+\gamma+\mu)+\beta_1(\gamma+\mu+r))V_8}\\
t_4 &= \frac{\gamma V_1}{(1-e)(\gamma\beta\mu(\nu_1+\nu_2+\gamma+\mu)+\beta_1(\gamma+\mu+r))}\\
o_4 &= \frac{\gamma\beta(\nu_1+\nu_2+\gamma+\mu)V_1}{\beta_1(1-e)(\gamma\beta\mu(\nu_1+\nu_2+\gamma+\mu)+\beta_1(\gamma+\mu+r))}
\end{aligned}$$

where $V_1 = (1-e)\beta_1 - (\nu_1+\nu_2+\gamma+\mu)$, $V_7 = \gamma\beta(1-\mu)(\nu_1+\nu_2+\gamma+\mu) - \beta_1(\mu+r)$, $V_8 = \nu_1\beta_3(\beta_2-\beta_1) + (\beta_3-\beta_1)(\nu_2\beta_2 + (\gamma+\mu)(\beta_2-\beta_1)(\beta_3-\beta_1))$,
$V_9 = (\nu_1+\nu_2)\beta_2 + (\beta_2-\beta_1)(\gamma+\mu)$ and $V_{10} = (\nu_1+\nu_2)\beta_3 + (\beta_3-\beta_1)(\gamma+\mu)$.

This equilibrium is endemic and exist if and only if $V_1 \geq 0$, $V_7 \leq 0$, $V_8 > 0$, $V_9 \leq 0$ and $V_{10} \leq 0$.

4. Equilibria stability

In this section we studied the local stability of the model equilibria. We used Lyapunov function or the classical linearization method and the Lienard-Chipart criterion, see [14] . In other words, we calculated the characteristic polynomial of the Jacobian of the system in each equilibrium and analyzed its roots. In addition, for disease-free equilibrium we calculated the basic reproduction number of the model. We will write the characteristic polynomial without the factors that are not involved in the stability analysis. We started with disease-free equilibrium.

4.1. Stability of the disease-free equilibrium E_0

We discuss the local stability of the disease-free equilibrium by examining the linearized form of system (3) at the equilibrium E_0. The Jacobian matrix evaluated at the disease-free equilibrium is given by

$$\partial_x f(u,E_0)=\begin{bmatrix} -\mu & (e-1)\beta_3 & (e-1)\beta_2 & (e-1)\beta_1 & r+(e-1)\beta & (1-\gamma)(1-\mu)\\ 0 & V_3 & 0 & \nu_2 & 0 & 0\\ 0 & 0 & V_2 & \nu_1 & 0 & 0\\ 0 & 0 & 0 & V_1 & 0 & 0\\ 0 & \gamma & \gamma & \gamma & -r-\mu & \gamma(1-\mu)\\ 0 & 0 & 0 & 0 & (1-e)\beta & -1 \end{bmatrix}$$

The characteristic polynomial of the Jacobian matrix is factorized [15]:

$$\chi_0=\left(Z+\mu\right)(Z-V_2)(Z-V_3)\left(Z^2+\left(2\mu+r+1\right)Z-V_4\right)(Z-V_1).$$

Using Lienard-Chipart criterion, we can deduce that the equilibrium E_0 is hyperbolic and locally asymptotically stable if and only if $V_1<0$,$V_2<0$, $V_3<0$ and $V_4<0$.

4.1.1. Computation of the basic reproduction number:

From the variations of the infectious compartments:
$\dot{I}_3=\nu_2 I_1+(1-e)\beta_3 I_3 S-(\gamma+\mu)I_3$
$\dot{I}_2=\nu_1 I_2+(1-e)\beta_2 I_2 S-(\gamma+\mu)I_2$
$\dot{I}_1=(1-e)\beta_1 I_1 S-(\nu_1+\nu_2+\gamma+\mu)I_2$
and by posing $w=(I_3,I_2,I_1)$ and $\mathscr{F}(w)=\begin{bmatrix}(1-e)\beta_3 I_3 S\\(1-e)\beta_2 I_2 S\\(1-e)\beta_1 I_1 S\end{bmatrix}$ which is the column matrix of the rates of occurrence of new infections by infectious compartment and $\mathscr{W}(w)=\begin{bmatrix}(\gamma+\mu)I_3-\nu_2 I_1\\(\gamma+\mu)I_2-\nu_1 I_1\\(\gamma+\mu+\nu_1+\nu_2)I_1\end{bmatrix}$ the column matrix of differences between the rate of individuals leaving per infectious compartment and the rate of those arriving in the same compartment, we determine the matrices
$F=\partial_w\mathscr{F}(w)=\begin{bmatrix}(1-e)\beta_3 s_0 & 0 & 0\\ 0 & (1-e)\beta_2 s_0 & 0\\ 0 & 0 & (1-e)\beta_1 s_0\end{bmatrix}$
and $W=\partial_w\mathscr{W}(w)=\begin{bmatrix}\gamma+\mu & 0 & -\nu_2\\ 0 & \gamma+\mu & -\nu_1\\ 0 & 0 & \gamma+\mu+\nu_1+\nu_2\end{bmatrix}$.
Then we calculated the matrix $F\cdot W^{-1}$ whose spectral radius is the basic reproduction number.

$$W^{-1}=\begin{bmatrix}\frac{1}{\gamma+\mu} & 0 & \frac{\nu_2}{(\gamma+\mu)(\gamma+\mu+\nu_1+\nu_2)}\\ 0 & \frac{1}{\gamma+\mu} & \frac{-\nu_1}{(\gamma+\mu)(\gamma+\mu+\nu_1+\nu_2)}\\ 0 & 0 & \frac{1}{\gamma+\mu+\nu_1+\nu_2}\end{bmatrix},$$

$$F\cdot W^{-1}=\begin{bmatrix}\frac{(1-e)\beta_3 s_0}{\gamma+\mu} & 0 & \frac{(1-e)\beta_3 s_0\nu_2}{(\gamma+\mu)(\gamma+\mu+\nu_1+\nu_2)}\\ 0 & \frac{(1-e)\beta_2 s_0}{\gamma+\mu} & \frac{(1-e)\beta_2 s_0\nu_1}{(\gamma+\mu)(\gamma+\mu+\nu_1+\nu_2)}\\ 0 & 0 & \frac{(1-e)\beta_1 s_0}{\gamma+\mu+\nu_1+\nu_2}\end{bmatrix}.$$

The basic reproduction number R_0 of the model is the spectral radius of the matrix $F\cdot W^{-1}$, therefore $R_0=\max\left\{\frac{(1-e)\beta_3}{\gamma+\mu},\frac{(1-e)\beta_2}{\gamma+\mu},\frac{(1-e)\beta_1}{\gamma+\mu+\nu_1+\nu_2}\right\}$. Thus E_0 is locally asymptotically stable if and only if $R_0<1$ [16].

4.2. Stability of the endemic equilibrium E_1

For endemic equilibrium E_1 the characteristic polynomial specialized by replacing I_3 by i_{31}, I_1 by i_{11}, T by t_1 and O by o_1 of the jacobian matrix

$$\partial_x f(u,E_1)=\begin{bmatrix} m_{11} & m_{12} & (e-1)\beta_2 s_1 & (e-1)\beta_1 s_1 & r+(e-1)\beta s_1 & (1-\gamma)(1-\mu)\\ 0 & m_{22} & 0 & \nu_2 & 0 & 0\\ m_{31} & 0 & m_{33} & \nu_1 & 0 & 0\\ 0 & 0 & 0 & m_{44} & 0 & 0\\ 0 & \gamma & \gamma & \gamma & -r-\mu & \gamma(1-\mu)\\ (1-e)\beta t_1 & 0 & 0 & 0 & (1-e)\beta s_1 & -1 \end{bmatrix}$$

with

$$m_{11}=(e-1)(t_1\beta+\beta_2 i_{21})-\mu, m_{12}=(e-1)\beta_3 s_1$$

$$m_{31}=(1-e)\beta_2 i_{21}, m_{22}=(1-e)\beta_3 s_1-\gamma-\mu, m_{33}=(1-e)\beta_2 s_1-\gamma-\mu,$$

$$m_{44}=(1-e)\beta_1 s_1-\nu_1-\nu_2-\gamma-\mu$$

was not fully factorized, we then used another method. The not completely specialized characteristic polynomial is factorized [15] as

$$\chi_1=(Z+\mu)\left(Z+\frac{(\gamma+\mu)(\beta_2-\beta_3)}{\beta_3}\right)\left(Z+\frac{V_9}{\beta_2}\right)Q_1,$$

where $Q_1=u_3Z^3+u_2Z^2+u_1Z+u_0$ is a polynomial of degree 3. To study the stability of E_1 we applied Lienard-Chipart criterion [14] to the polynomial Q_1. we obtained the coefficients:
$u_3=1$
by replacing S by s_1 $u_2=(1-e)(t_1\beta+i_{21}\beta_2)+\mu+r+1$
$u_1=(1-e)\beta(\gamma+\mu+r-\gamma\mu)t_1+\frac{1}{\beta_2}((1-e)i_{21}\beta_2^2(\gamma+\mu+r+1)-V_5)$ and
$u_0=(1-e)(\beta\gamma\mu(\gamma+\mu)+\beta_2(\gamma+\mu+r))i_{21}$ which are all three strictly positive if i_{21} is strictly positive. The only subresultant we have to calculate is $sr=u_0(u_2u_1-u_0u_3)$. After the calculation, we have $u_2u_1-u_0u_3=q_2i_{21}^2+q_1i_{21}+q_0$ with

$$q_2=\beta_2^2(e-1)^2(\gamma+2\mu+r+1),$$

$$q_1=(1-e)[\beta\beta_2(1-e)(2\gamma+2\mu+2r+1-\gamma\mu)t_1+\beta_2(\mu+r+1)^2+\gamma(\beta_2(\mu+r)-\beta(\gamma+\mu))].$$

$q_1>0$ if $V_2\geq 0$

$$q_0=\frac{1}{\beta_2}((1-e)T\beta+\mu+r+1)((1-e)\beta\beta_2(\gamma+\mu+r-\gamma\mu)t_1-V_5).$$

q_0 is obviously and strictly positive if $V_2>0$ and $V_5\leq 0$. So u_3,u_2,u_1,u_0 and sr are all strictly positive. E_1 is therefore hyperbolic and locally asymptotically stable if and only if $\beta_2>\beta_3$, $V_2\geq 0$ and $V_5\leq 0$.

4.3. Stability of the endemic equilibrium E_2

For endemic equilibrium E_2 the characteristic polynomial specialized by replacing S by s_2, I_2 by i_{22}, I_1 by i_{12} and O by o_2 of the jacobian matrix

$$\partial_x f(u,E_2)=\begin{bmatrix} n_{11} & -\gamma-\mu & -\frac{\beta_2(\gamma+\mu)}{\beta_3} & -\frac{\beta_1(\gamma+\mu)}{\beta_3} & r-\frac{\beta(\gamma+\mu)}{\beta_3} & (1-\gamma)(1-\mu)\\ n_{21} & 0 & 0 & \nu_2 & 0 & 0\\ 0 & 0 & n_{33} & \nu_1 & 0 & 0\\ 0 & 0 & 0 & -\frac{V_{10}}{\beta_3} & 0 & 0\\ 0 & \gamma & \gamma & \gamma & -r-\mu & \gamma(1-\mu)\\ (1-e)\beta t_2 & 0 & 0 & 0 & \frac{\beta(\gamma+\mu)}{\beta_3} & -1 \end{bmatrix}$$

with

$$n_{11}=(e-1)(\beta t_2+\beta_3 i_{32})-\mu, n_{21}=(1-e)\beta_3 i_{32}, n_{33}=\frac{(\beta_2-\beta_3)(\gamma+\mu)}{\beta_3}$$

$$\chi_2 = (Z+\mu)(\beta_3 Z + (\gamma+\mu)(\beta_3-\beta_2))(\beta_3 Z + V_{10})Q_2$$

where

$$Q_2 = p_3 Z^3 + p_2 Z^2 + p_1 Z + p_0$$

with

$$p_3 = \beta_3, p_2 = \beta_3((1-e)(\beta t_2 + \beta_3 i_{32}) + \mu + r + 1),$$

$$p_1 = (1-e)\beta_3(t_2\beta(\gamma+\mu+r-\gamma\mu) + i_{32}\beta_3(\gamma+\mu+r+1)) - V_6,$$

$$p_0 = \beta_3(1-e)i_{32}(\beta_3(\gamma+\mu+r) + \beta\gamma\mu(\gamma+\mu)).$$

$$p_1 p_2 - p_0 p_3 = \beta_3^4(e-1)^2(\gamma+\mu+r+1)i_{32}^2 + \beta_3^2(1-e)[(1-e)(2\gamma+2\mu$$

$$+2r+1-\gamma\mu)t_2\beta\beta_3 - \gamma\beta(\gamma+\mu) + \beta_3((\mu+r)(\mu+r+2) + \gamma(\mu+r)+1)]i_{32}$$

$$+((1-e)\beta t_2 + \mu + r + 1)\beta_3((\gamma+\mu+r-\gamma\mu)(1-e)\beta\beta_3 t_2 - V_6).$$

E_2 is therefore hyperbolic and locally asymptotically stable if and only if $\beta_2 < \beta_3$, $V_3 \geq 0$, $V_{10} > 0$ and $V_6 \leq 0$.

4.4. Stability of the equilibrium E_3

For endemic equilibrium E_3 the characteristic polynomial specialized by replacing S by s_3, I_3 by i_{33} I_2 by i_{23} and I_1 by i_{13} of the jacobian matrix

$$\partial_x f(u, E_3) = \begin{bmatrix} (e-1)\beta t_3 - \mu & a_{12} & a_{13} & a_{14} & r - \frac{r+\mu}{(1-\mu)\gamma} & (1-\gamma)(1-\mu) \\ 0 & a_{22} & 0 & \nu_2 & 0 & 0 \\ 0 & 0 & a_{33} & \nu_1 & 0 & 0 \\ 0 & 0 & 0 & a_{44} & 0 & 0 \\ 0 & \gamma & \gamma & \gamma & -r-\mu & \gamma(1-\mu) \\ (1-e)\beta t_3 & 0 & 0 & 0 & \frac{(r+\mu)}{\gamma(1-\mu)} & -1 \end{bmatrix}$$

with

$$a_{12} = -\frac{\beta_3(r+\mu)}{(1-\mu)\gamma\beta},\ a_{13} = -\frac{\beta_2(r+\mu)}{(1-\mu)\gamma\beta},\ a_{14} = -\frac{\beta_1(r+\mu)}{(1-\mu)\gamma\beta},\ a_{22} = \frac{-V_6}{(1-\mu)\gamma\beta},$$

$$a_{33} = \frac{-V_5}{(1-\mu)\gamma\beta},\ \text{and}\ \mathrm{a}_{44} = \frac{-\mathrm{V}_7}{(1-\mu)\gamma\beta}.$$

$$\chi_3 = (Z+\mu)(\beta\gamma(\mu-1)Z - V_6)(\beta\gamma(\mu-1)Z - V_5)(\beta\gamma(\mu-1)Z - V_7)Q_3$$

where

$$Q_3 = Z^2 + ((1-e)\beta t_3 + \mu + r + 1)Z + (1-e)\beta t_3(\gamma+\mu+r-\gamma\mu).$$

E_3 is therefore hyperbolic and locally asymptotically stable if and only if $V_4 \geq 0$, $V_5 > 0$, $V_6 > 0$ and $V_7 > 0$.

4.5. Stability of the endemic equilibrium E_4

4.5.1. Invariant domain

Given a differentiable vector field $f : \mathbb{R}^n \to \mathbb{R}^n$, we recall that $\mathbb{R}^n_+$ is positively invariant under f if and only if for all $i \in [1, n]$ and $x \in \mathbb{R}^n_+$ such that $x_i = 0$ we have $f_i(x) \geq 0$, see [17]. The application of this property makes it easy to verify that $\mathbb{R}^n_+$ is positively invariant under the vector field associated with the system (1). Let's recall that an domain D is positively invariant for $\dot{x} = f(x(t), u)$, if the trajectory of any solution of $\dot{x} = f(x(t), u)$ that starts in D remains in D for any positive value of t.

4.5.2. Global stability of E_4

For the equilibrium E_4, we use a Lyapunov function to study his stability.

Note that $\dot{S}+\dot{I}_3+\dot{I}_2+\dot{I}_1+\dot{T}+\dot{O}=\mu(1-(S+I_3+I_2+I_1+T+O)=0$, so the domain $\Omega=\{(S,I_3,I_2,I_1,T,O)\in\mathbb{R}^6_+ : S+I_3+I_2+I_1+T+O=1\}$ is positively invariant under $\dot{x}=f(x(t),u)$.

Let $L=(I_1-i_{14})^2$,

$\frac{dL}{dt}=2(I_1-i_{14})\dot{I}_1=2I_1(I_1-i_{14})((1-e)\beta_1 S-(\nu_1+\nu_2+\mu+\gamma))$,

then $\frac{\mathrm{dL}}{\mathrm{dt}}\leq 2\mathrm{I}_1(\mathrm{I}_1-\mathrm{i}_{14})\mathrm{V}_1\leq 0$.

Indeed E_4 exists if $V_1\geq 0$, then in this case $(1-e)\beta_1 S-(\nu_1+\nu_2+\mu+\gamma)\geq 0$. so, I_1 increase towards i_{14}, then $I_1-i_{14}\leq 0$. So L is a Lyapunov function.

E_4 is therefore hyperbolic and globally asymptotically stable if and only in Ω if $V_1\geq 0$, $V_7\leq 0$, $V_8>0$, $V_9\leq 0$ and $V_{10}\leq 0$.

Thus, we have the following result.

Theorem 4.1.
The model represented by the system (1) has five equilibria:

1. *a disease free equilibrium E_0 which is hyperbolic and locally asymptotically stable if and only if $V_1<0$,$V_2<0$, $V_3<0$ and $V_4<0$;*
2. *an equilibrium E_1 which is hyperbolic and locally asymptotically stable if and only if $\beta_2>\beta_3$, $V_2\geq 0$ and $V_5\leq 0$;*
3. *an equilibrium E_2 which is hyperbolic and locally asymptotically stable if and only if $\beta_2<\beta_3$, $V_3\geq 0$, $V_6\leq 0$ and $V_{10}>0$;*
4. *an equilibrium E_3 which is hyperbolic and locally asymptotically stable if and only if $V_4\geq 0$, $V_5>0$, $V_6>0$ and $V_7>0$;*
5. *an equilibria E_4 which is hyperbolic and globally asymptotically stable in Ω if and only if $V_1\geq 0$, $V_7\leq 0$, $V_8>0$, $V_9\leq 0$ and $V_{10}\leq 0$.*

Remark: Note that if $\beta_2>\beta_3$, E_2 is unstable and if $\beta_2<\beta_3$ E_1 is unstable.

5. Global stability of disease free-equilibrium

Theorem 5.1.
If $R_0<1$ then the disease free equilibrium $E_0=(1,0,0,0,0,0)$ is globaly asymptotically stable.

Consider the function $\begin{array}{rl} L: & R_+^6\to R_+ \\ & x\mapsto I_1 \end{array}$.

Its derivative with respect to time following the solutions of system (E) is

$$\frac{dL(x(t))}{dt}=((1-e)\beta_1 S-(\nu_1+\nu_2+\gamma+\mu))I_1\leq((1-e)\beta_1-(\nu_1+\nu_2+\gamma+\mu))I_1=V_1 I_1.$$

Let's recall that E_0 is locally asymptotically stable if $V_1<0$. So L is a lyapunov function.

Note that if $R_0<1$, we have $(1-e)\beta_3-(\gamma+\mu)<0$, $(1-e)\beta_2-(\gamma+\mu)<0$ and $(1-e)\beta_1-(\nu_1+\nu_2+\gamma+\mu)<0$, therefore $V_1<0$.

Finally, if $R_0<1$ then E_0 is globally asymptotically stable in Ω.

6. Numerical Simulation

Numerical simulations are done using Python computer software program. Parameter values are given in table 2:

6.1. Effect of varying treatment rate on different epidemiological classes

In this part, the health precaution rate e is fixed to $0,1$. The effect of treatment on the dynamics of the model is studied for the following values of treatment rates $\gamma=0,01;0,1;0,4$ and $0,7$. It is observed that there is a drastic decrease of infectious classes when the treatment rate increases as shown in figs. 2 to 5.

Table 2. Parameter values used in numerical simulations.

Symbol	Biological meaning	Value
β	contact rate	0,00006
β_1	transmission rate for the initial strain	0,00001
β_2	transmission rate for the first variant	0,00005
β_3	transmission rate for the second variant	0,00003
ν_1	mutation rate for the first variant	0,001
ν_2	mutation rate for the second variant	0,002
μ	death rate	0,0003
r	cure rate	0,7

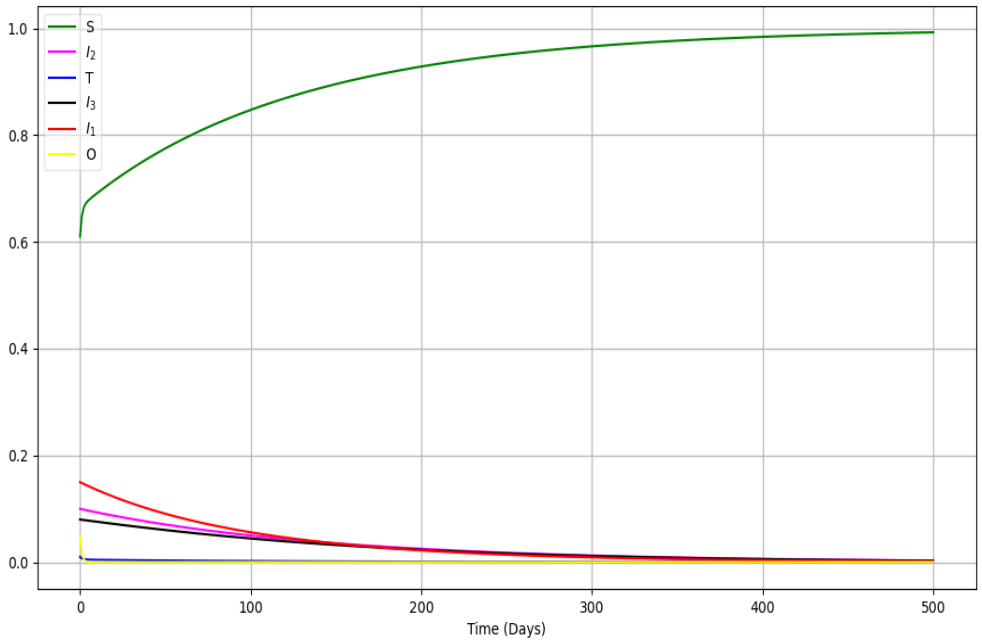

Fig. 2. Disease spreed at $\gamma = 0,01$ and $e = 0,1$

6.2. Effect of varying health precaution rate on different epidemiological classes

In this part the treatment rate γ is fixed to $0,1$. The effect of health precaution rate on the dynamics of the model is studied for the following values of health precaution rates $e = 0,01; 0,1; 0,4$ and $0,7$. It is observed that there is a no significant decrease of infectious classes when the health precaution rate increases in figs. 6 to 9. As the simulation shows, increasing the rate of health precautions stops disease transmission over time but does not eliminate it. The epidemic disappears after 50 days for all the given values of e, for $\gamma = 0,1$ even if the variation is less noticeable with the rate increase in the of health precautions.

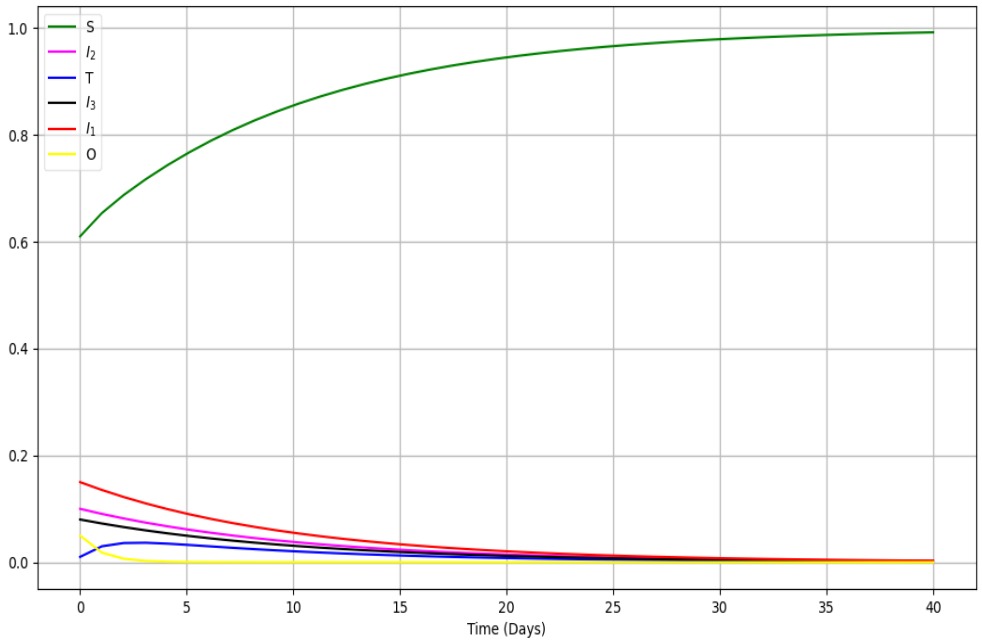

Fig. 3. Disease spread at $\gamma = 0,1$ and $e = 0,1$

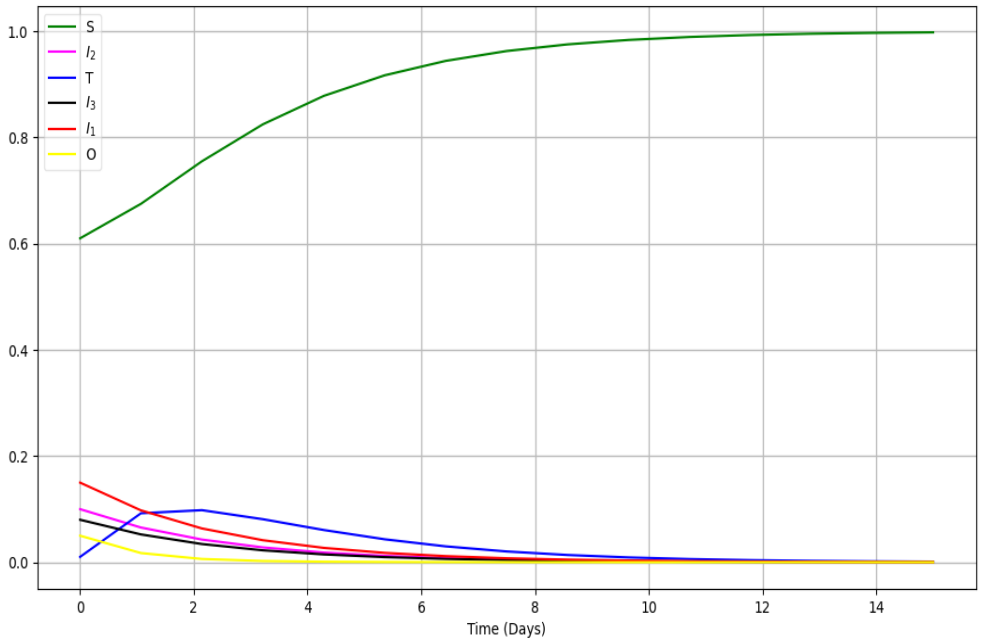

Fig. 4. Disease spread at $\gamma = 0,4$ and $e = 0,1$

7. Conclusion:

In this paper, an epidemic model with two variants is proposed to study the effect of treatment and health preautions on the transmission dynamics of Covid-19. The model was analyzed for equilibrium points and their stability. The basic reproduction number, R_0 that describes the dynamics of the disease was obtained. It was established that

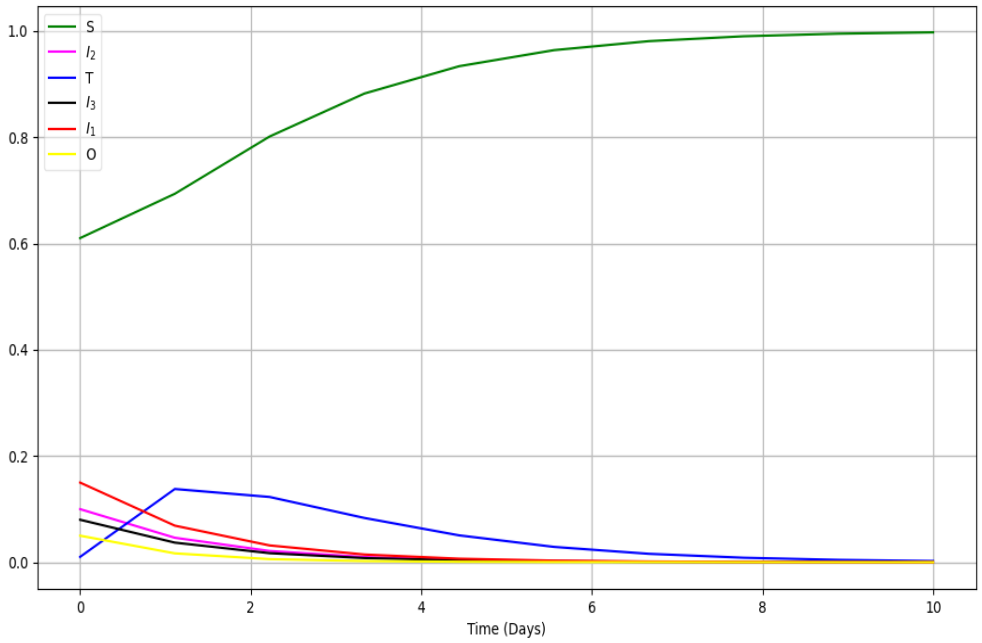

Fig. 5. Disease spread at $\gamma = 0,7$ and $e = 0,1$

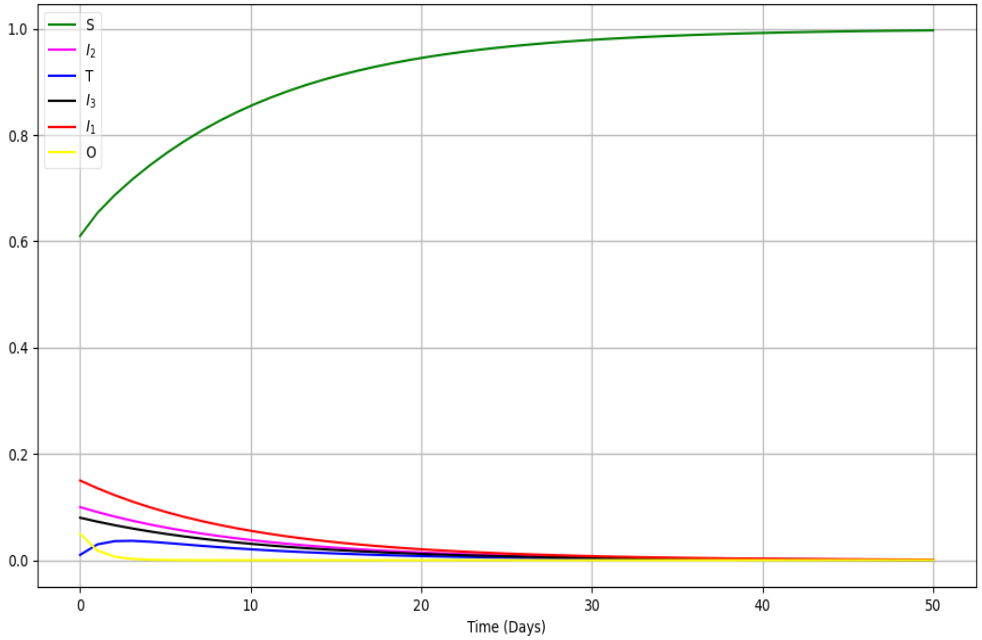

Fig. 6. Disease spread at $\gamma = 0,1$ and $e = 0,01$

for $R_0 < 1$, the disease-free equilibrium E_0 is locally asymptotically stable and the disease dies out.

For the model presented, all the five equilibria and their stability are exactly characterized by the use of algebraic geometry and computer algebra methods. The disease-free equilibrium is globally asymptotically stable, i.e. a disease-free environment can be achieved if treatment and health precautions are respected. Numerical simulations of the model show that the singular use of a health precaution/treatment strategy may lead to the effective disease control

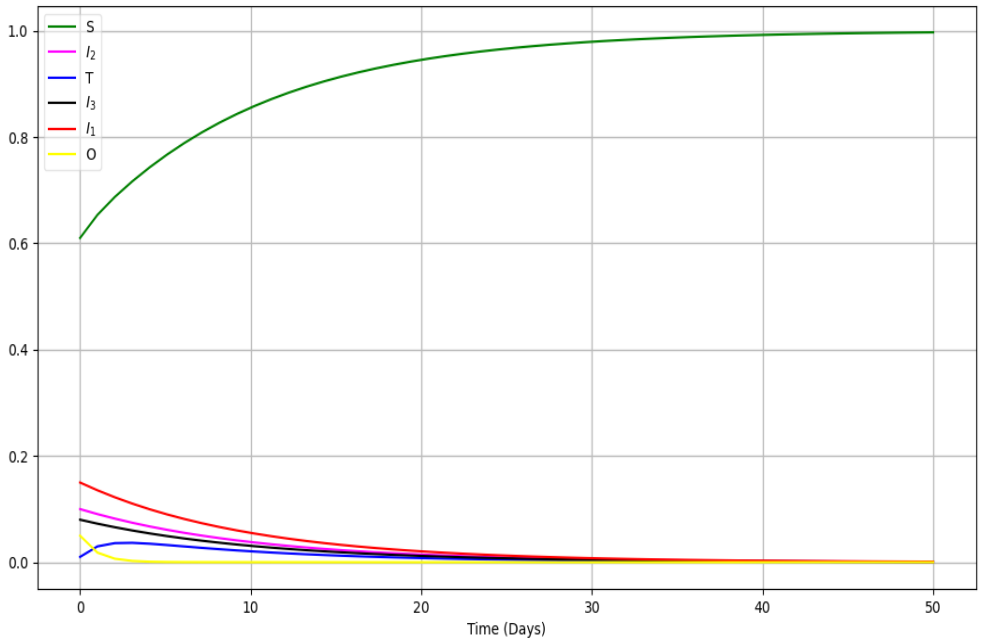

Fig. 7. Disease spread at $\gamma = 0,1$ and $e = 0,1$

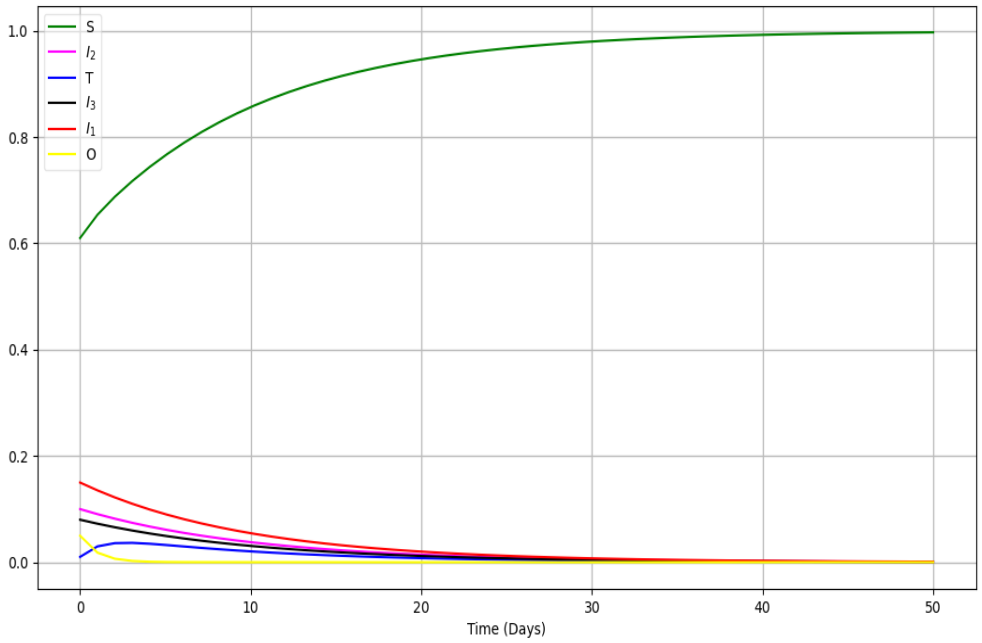

Fig. 8. Disease spread at $\gamma = 0,1$ and $e = 0,4$

(or elimination) if its effectiveness level is at least moderately high enough. Compliance with health precaution for instance can significantly reduce the cost of treatment. The epidemic tends to disappear quickly when the treatment rate increases. We note for example that for the given values of the parameters, the disease spread decreases from 500 days to 10 days when γ goes from $0,01$ to $0,7$. As the simulation shows, increasing the rate of health precautions stops disease transmission over time but does not eliminate it. But the combined effects of treatment measures and

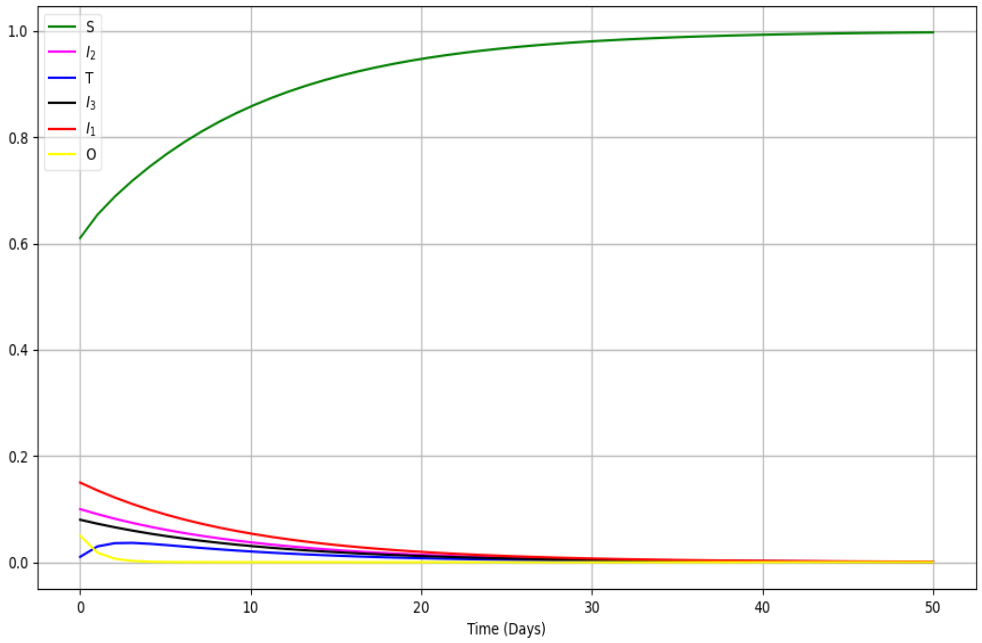

Fig. 9. Disease spreed at $\gamma = 0,1$ and $e = 0,7$

health precautions have a considerable effect on the spread of the disease. The disease tends to disappear with the increase in these rates. The model presented is such that the initial strain have only one variant. The use of algebraic geometry and computer algebra approaches is of a valuable contribution for the characterization of equilibria and their stability. Another perspective of this work is to extend this model to the case of disease with several variants.

References

[1] D. Biswasa, S. Pal. Stability analysis of a non-linear HIV/AIDS epidemic model with vaccination and antiretroviral therapy. Int. J. Adv. Appl. Math. and Mech. 5(2)(2017) 41-50.
[2] A. Radid, K. Rhofir. Partitioning differential transformation method to a SIR epidemic model under vaccination strategy Int. J. Adv. Appl.Math. and Mech. 7(1)(2019) 9-19.
[3] S. C, Mpeshea, N. Nyerereb. Modeling approach to assess the transmission dynamics of Hepatitis B infection in Africa. Int. J. Adv. Appl. Math. and Mech. 6(3)(2019) 51-61.
[4] T. Li, Y. Guo. Modeling and optimal control of mutated COVID-19 (Delta strain) with imperfect vaccination, Chaos, Solitons and Fractals 156 (2022) 111825.
[5] G. Gilberto, A. Abraham, L.Qualitative analysis of a mathematical model with presymptomatic individuals and two SARS-CoV-2 variants, Comput. Appl. Math. 40, No. 6, Paper No. 199, 25 p. (2021).
[6] M. Bushra, T. Marco, W. Jianhong, Variant specific interventions to slow down replacement and prevent outbreaks. Math. Biosci. 343(2022)108703.
[7] S. Zhao, et al. The non-pharmaceutical interventions may affect the advantage in transmission of mutated variants during epidemics: a conceptual model for COVID-19, J. Theor. Biol, 542 (2022).
[8] P. Yuan, E. Aruffo,Y. T, L. Yang, N. Ogden, A. Fazil, H. Zhu, Projections of the transmission of the Omicron variant for Toronto, Ontario, and Canada using surveillance data following recent changes in testing policies, Infectious Disease Modelling 7 (2022) 83-93.
[9] N. Ferguson, R. Anderson and S. Gupta. The effect of antibody-dependant enhancement on the transmission dynamics and persistence of multiple strain pathogens. Proc. Natl. Acad. Sci. USA, (1999), 96: 790-794.
[10] L. Billings, A. Fiorillo, and I. B. Schwartz. Vaccinations in disease models with antibody-dependent enhancement. Math. Biosci., (2008), 211(2): 265-281.
[11] T. Becker and V. Weispfenning In cooperation with Heinz Kredel. Gröbner bases. A computational approach to commutative algebra , volume 141 of Graduate Texts in Mathematics. Springer-Verlag, New York, 1993, 581 pages.
[12] D. Cox, J. Little, and D. O'Shea. Ideals, varieties, and algorithms. Undergraduate Texts in Mathematics. Springer, New York, third edition, 2007, 565 pages.

[13] W. W. Adams and P. Loustaunau. An introduction to Gröbner bases, volume 3 of Graduate Studies in Mathematics. American Mathematical Society, Providence, RI, 1994, 301 pages.
[14] S. Basu, R. Pollack, and M.-F. Roy. Algorithms in real algebraic geometry, volume 10 of Algorithms and Computation in Mathematics. Springer-Verlag, Berlin, second edition, 2006, 662 pages.
[15] C. W. Brown, M. El Kahoui, D. Novotni, and A. Weber. Algorithmic methods for investigating equilibria in epidemic modeling. J. Symbolic Comput.,(2006), 41(11): 1157-1173.
[16] P van den. Driessche and J. Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Math. Biosci., (2002), 180: 29-48.
[17] S. Wiggins. Introduction to applied nonlinear dynamical systems and chaos, volume 2 of Texts in Applied Mathematics. Springer-Verlag, New York second edition, 2003.