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Research Article

A Mathematical Model for a Transmissible Disease with a Variant

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The outbreak of the Coronavirus (COVID-19) pandemic around the world has caused many health and socioeconomic problems, and the identification of variants like Delta and Omicron with similar and often even more transmissible modes of transmission has motivated us to do this study. In this article, we have proposed and analyzed a mathematical model in order to study the effect of health precautions and treatment for a disease transmitted by contact in a constant population. We determined the four equilibria of the system of ordinary differential equations representing the model and characterized their existence using exact methods of algebraic geometry and computer algebra. The model is studied using the stability theory for systems of differential equations and the basic reproduction number R_0. The stability of the equilibria is analyzed using the Lienard-Chipart criterion and Lyapunov functions. 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 if the rate of treatment and compliance with health precautions are high, the number of infections decreases in the classes of infectious and is canceled out over time. It is concluded that the high treatment rate accompanied by a suitable rate of compliance with health precautions allows for the control the disease.

1. Introduction

The initial strain of SARS-CoV-2 that appeared at the end of 2019 and which spread around the world in 2020 has already led to the appearance of other variants. To the Delta variant was added a new Omicron variant in november 2021 which has become pratically dominant. The emergence of other variants is 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. Li and Guo, in [1], develop a mathematical model to simulate the possible impact of three control measures (vaccination, isolation, and nucleic acid testing) to control the spread of the disease with the Delta variant. The technique used is the weighted nonlinear least square estimation method. Gilberto and Abraham propose in [2] 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 [3], the authors develop a mathematical model to examine the impact of nonpharmaceutical interventions, including the COVID test, genome sequencing test capacity, contact tracing, and quarantine strength, on the induced epidemic wave. A novel compartmental model which captures new strategies that promote self-testing and adjust the eligibility for PCR tests, social behaviours, booster vaccine campaigns, and features of the newest variant Omicron is presented in [4]. However, to our knowledge, none of the above models incorporates the effects of potential SARS-CoV-2 variants together with the treatment and health precautions in the spread of COVID-19.

In this paper, we present 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

Table 1: Parameters and their biological meaning.

Parameters	Biological meaning
β	Contact rate
β_1	Transmission rate for the initial strain
β_2	Transmission rate for the variant ($\beta_2 < \beta_1$)
ν_1	Mutation rate for the variant
μ	Death rate
r	Cure rate
γ	Treatment rate
e	Health precaution rate

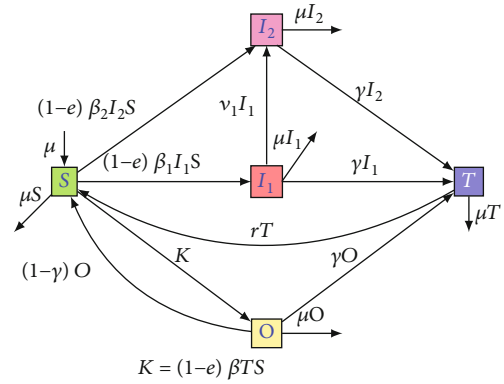

Figure 1: Model transfer diagram.

assess the combined use of observation, treatment, and health precaution strategies to an infectious epidemiological disease with one variant. The model presented has 4 equilibria. Note that the basic reproduction number denoted R_0 can be calculated using the Van den Drissche and Watmough method [5, 6]. The disease-free equilibrium is stable if $R_0 < 1$ and unstable if $R_0 > 1$. If there are only two equilibria, the second is endemic. When there are more than two equilibria, the basic reproduction number does not allow to control exactly the stability of endemic equilibria.

When the number of strains or variants increases, the number of equilibria can very drastically increase. In this case, the use of algebraic geometry and computer algebra approaches is of a valuable contribution for the characterization and study of the equilibrium stability.

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 the 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. Model Presentation

We present a model to study the spread of an infectious disease in a constant population required to respect sanitation precautions with a portion under treatment. The population is divided into five classes. The susceptible are in class S, and the infected are in class I_1 for the strain and in class I_2 for the variant. The individuals under treatment are in class T, and those under observation are 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 size of all classes decreases due to the mortality rate. The classes of infected I_1 and I_2 receive the individuals of class S infected by the forces of infection $(1-e)\beta_1 I_1$ and $(1-e)\beta_2 I_2$, respectively. Individuals of I_2 are also derived from the mutation of those of I_1 due to the ν_1 rate. Individuals from I_1, I_2, 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 and I_2 by the infection forces $(1-e)\beta_1 I_1$ and $(1-e)\beta_2 I_2$, 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 $1-\gamma$ and γ, 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 Figure 1.

From the transfer diagram of the model in Figure 1, the dynamical system of the model is as follows:

$$\begin{cases} \dot{S} = \mu + rT + (1-\gamma)O - ((1-e)(\beta T + \beta_2 I_2 + \beta_1 I_1) + \mu)S, \\ \dot{I}_2 = \nu_1 I_1 + (1-e)\beta_2 I_2 S - (\gamma + \mu)I_2, \\ \dot{I}_1 = (1-e)\beta_1 I_1 S - (\nu_1 + \gamma + \mu)I_1, \\ \dot{T} = \gamma(I_2 + I_1 + O) - (r + \mu)T, \\ \dot{O} = (1-e)\beta TS - (1+\mu)O, \end{cases} \tag{1}$$

where

We easily verify that for $0 = \dot{S} + \dot{I}_1 + \dot{I}_2 + \dot{T} + \dot{O} = \mu(1 - S - I_1 - I_2 - T - O)$, we have $S + I_1 + I_2 + T + O = 1$ for any solution of system (1).

3. Model Equilibria

System (1) can be written as $\dot{x} = f(x, u)$, where $u = (\gamma, e, \beta, \beta_1, \beta_2, \nu_1, \mu, r)$ is the list of parameters and $x = (S, I_2, I_1, T, O)$ is the list of state variables. An important feature of this model is common to a large class of epidemiological models, see [7, 8], and 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 the Gröebner base, see [9–11], to determine the equilibria of the model, which are the solutions of the algebraic equations system $f(u, x) = 0$:

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

The calculation of the Groebner base [9] of the system f_1, f_2, f_3, f_4, f_5 according to the lexicographical order $S \prec I_2 \prec I_1 \prec T \prec O$ allows us to have a system (3) with a triangular form of five equations according to the given order of variables. The first element of the Groebner base calculated is a polynomial of degree 4 in S with roots $s_0 = 1$, $s_1 = (\gamma + \mu)/\beta_2(1-e)$, $s_2 = (\gamma + \mu + \nu_1)/\beta_1(1-e)$ and $s_3 = (\mu + r)(\mu + 1)/\beta(1-e)$.

Replacing S by s_0 in system (3) and solving for the other variables, we obtain a single equilibrium, noted as E_0, whose components are

$$(1, 0, 0, 0, 0). \tag{3}$$

This is the disease-free equilibrium of the model, and it exists for all values of parameters.

By replacing S by s_1 in system (3) and solving for the other variables, we obtain a single equilibrium, noted as E_1, whose components are

$$\begin{aligned} s_1 &= \frac{\gamma + \mu}{(1-e)\beta_2}, \\ i_{21} &= -\frac{V_2 V_3}{\beta_2^2 (1+\mu)(1-e)(\gamma + \mu + r)}, \\ i_{11} &= 0, \\ t_1 &= \frac{\gamma V_2}{\beta_2 (1-e)(\gamma + \mu + r)}, \\ o_1 &= \frac{\gamma\beta(\gamma + \mu) V_2}{\beta_2^2 (1+\mu)(1-e)(\gamma + \mu + r)}, \end{aligned} \tag{4}$$

where $V_2 = (1-e)\beta_2 - (\gamma + \mu)$ and $V_3 = \gamma\beta(\gamma + \mu) - \beta_2(\mu + r)(\mu + 1)$.

This equilibrium which corresponds to the nonexistence of infectious cases linked to the strain is endemic and exists if and only if $V_2 \geq 0$ and $V_3 \leq 0$.

By replacing S by s_2 in system (3) and solving for the other variables, we obtain a single equilibrium, noted as E_2, whose components are

$$\begin{aligned} s_2 &= \frac{\gamma + \mu + \nu_1}{\beta_1 (1-e)}, \\ i_{22} &= \frac{-V_1 V_4}{\beta_1 (\beta_1 - \beta_2)(1+\mu)(1-e)(\gamma + \mu + r)(\nu_1 + \gamma + \mu)}, \\ i_{12} &= \frac{-V_1 V_4 V_6}{\beta_1^2 (\beta_1 - \beta_2)(1+\mu)(1-e)(\gamma + \mu + r)(\nu_1 + \gamma + \mu)}, \\ t_2 &= \frac{\gamma V_1}{\beta_1 (1-e)(\gamma + \mu + r)}, \\ o_2 &= \frac{\gamma\beta(\nu_1 + \gamma + \mu) V_1}{\beta_1^2 (1+\mu)(1-e)(\gamma + \mu + r)}, \end{aligned} \tag{5}$$

where $V_1 = (1-e)\beta_1 - (\nu_1 + \gamma + \mu)$, $V_4 = \beta\gamma(\nu_1 + \gamma + \mu) - \beta_1(\mu + r)(\mu + 1)$, and $V_6 = (\beta_1 - \beta_2)(\gamma + \mu) - \nu_1\beta_2$.

This equilibrium is endemic and exists if and only if $V_1 \geq 0$, $V_4 \leq 0$, and $V_6 \geq 0$.

By replacing S by s_3 in system (3) and solving for the other variables, we obtain a single equilibrium, noted as E_3, whose components are

$$\begin{aligned} s_3 &= \frac{(\mu + r)(\mu + 1)}{(1-e)\gamma\beta}, \\ i_{23} &= 0, \\ i_{13} &= 0, \\ t_3 &= \frac{-V_5}{\beta(1-e)(\gamma + \mu + r)}, \\ o_3 &= \frac{-(r + \mu) V_5}{\gamma\beta(1-e)(\gamma + \mu + r)}, \end{aligned} \tag{6}$$

where $V_5 = \gamma\beta(1-e) - (\mu + r)(\mu + 1)$.

This equilibrium corresponds to the nonexistence of infectious cases in circulation and exists if and only if $V_5 \leq 0$.

4. Equilibrium Stability

In this section, we studied the local stability of the model equilibria. We used a Lyapunov function or the classical linearization method and the Lienard-Chipart criterion (see [12]). In other words, we calculated the characteristic polynomial of the Jacobian of the system in each equilibrium and analyzed its roots. In addition, for the 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 the disease-free equilibrium.

4.1. Stability of the Disease-Free Equilibrium E_0. For disease-free equilibrium E_0, the characteristic polynomial of the Jacobian matrix,

$$\partial_x f(u, E_0) = \begin{bmatrix} -\mu & (e-1)\beta_2 & (e-1)\beta_1 & r + (e-1)\beta & 1 - \gamma \\ 0 & V_2 & \nu_1 & 0 & 0 \\ 0 & 0 & V_1 & 0 & 0 \\ 0 & \gamma & \gamma & -r - \mu & \gamma \\ 0 & 0 & 0 & (1-e)\beta & -1 - \mu \end{bmatrix}, \tag{7}$$

is factorized [13]:

$$\chi_0 = (Z+\mu)(Z-V_1)(Z-V_2)\left(Z^2+(2\mu+r+1)Z-V_5\right). \tag{8}$$

Using the Lienard-Chipart criterion for $Z^2+(2\mu+r+1)Z-V_5$, we can deduce that the equilibrium E_0 is hyperbolic and locally asymptotically stable if and only if $V_1<0$, $V_2<0$, and $V_5<0$.

4.1.1. Computation of the Basic Reproduction Number. From the variations of the infectious compartments,

$$\begin{aligned}\dot{I}_2 &= \nu_1 I_1 + (1-e)\beta_2 I_2 S - (\gamma+\mu)I_2,\\ \dot{I}_1 &= (1-e)\beta_1 I_1 S - (\nu_1+\gamma+\mu)I_1,\end{aligned} \tag{9}$$

and by posing $w=(I_2,I_1)$ and $\mathscr{F}(w)=\begin{bmatrix}(1-e)\beta_2 I_2 S\\ (1-e)\beta_1 I_1 S\end{bmatrix}$ which is the column matrix of the rates of occurrence of new infections by infectious compartment and $\mathscr{W}(w)=\begin{bmatrix}(\gamma+\mu)I_2-\nu_1 I_1\\ (\gamma+\mu+\nu_1)I_1\end{bmatrix}$ which is 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

$$\begin{aligned}F &= \partial_w \mathscr{F}(w) = \begin{bmatrix}(1-e)\beta_2 s_0 & 0\\ 0 & (1-e)\beta_1 s_0\end{bmatrix},\\ W &= \partial_w \mathscr{W}(w) = \begin{bmatrix}\gamma+\mu & -\nu_1\\ 0 & \gamma+\mu+\nu_1\end{bmatrix}.\end{aligned} \tag{10}$$

Then, we calculated the matrix $F\cdot W^{-1}$ whose spectral radius is the basic reproduction number.

$$\begin{aligned}W^{-1} &= \begin{bmatrix}\dfrac{1}{\gamma+\mu} & \dfrac{\nu_1}{(\gamma+\mu)(\gamma+\mu+\nu_1)}\\ 0 & \dfrac{1}{\gamma+\mu+\nu_1}\end{bmatrix},\\ F\cdot W^{-1} &= \begin{bmatrix}\dfrac{(1-e)\beta_2 s_0}{\gamma+\mu} & \dfrac{(1-e)\beta_2 s_0 \nu_1}{(\gamma+\mu)(\gamma+\mu+\nu_1)}\\ 0 & \dfrac{(1-e)\beta_1 s_0}{\gamma+\mu+\nu_1}\end{bmatrix}.\end{aligned} \tag{11}$$

The basic reproduction number R_0 of the model is the spectral radius of the matrix $F\cdot W^{-1}$; therefore $R_0=\max\{(1-e)\beta_2/(\gamma+\mu),(1-e)\beta_1/(\gamma+\mu+\nu_1)\}$. Thus, E_0 is locally asymptotically stable if and only if $R_0<1$ [5], which exactly reflects conditions $V_1<0$ and $V_2<0$.

4.2. Stability of the Endemic Equilibrium E_1. For endemic equilibrium E_1, the specialized characteristic polynomial of the Jacobian matrix was not fully factorized, but by substituting (S,I_1,O) by (s_1,i_{11},o_1), we obtain

Table 2: Parameter values used in numerical simulations.

Parameters	Biological meaning	Value
β	Contact rate	0.00006
β_1	Transmission rate for the initial strain	0.00005
β_2	Transmission rate for the variant	0.00003
ν_1	Mutation rate for the variant	0.001
μ	Death rate	0.0003
r	Cure rate	0.7

$$\partial_x f(u,E_1) = \begin{bmatrix} m_{11} & -\gamma-\mu & m_{13} & \dfrac{-\beta(\gamma+\mu)+r\beta_2}{\beta_2} & 1-\gamma\\ (1-e)\beta_2 i_{21} & 0 & \nu_1 & 0 & 0\\ 0 & 0 & \dfrac{V_6}{\beta_2} & 0 & 0\\ 0 & \gamma & \gamma & -\mu-r & \gamma\\ (1-e)\beta t_1 & 0 & 0 & \dfrac{\beta(\gamma+\mu)}{\beta_2} & -1-\mu \end{bmatrix}, \tag{12}$$

with $m_{11}=-((t_1\beta+i_{21}\beta_2)(1-e)+\mu)$ and $m_{13}=-\beta_1(\gamma+\mu)/\beta_2$, and the characteristic polynomial is factorized [13] as

$$\chi_1 = (Z+\mu)(\beta_2 Z - V_6)Q_1, \tag{13}$$

where $Q_1=a_3Z^3+a_2Z^2+a_1Z+a_0$ is a polynomial of degree 3. To study the stability of E_1, we applied the Lienard-Chipart criterion [12] to the polynomial Q_1. We obtained the following coefficients:

$$\begin{aligned}a_3 &= \beta_2\\ a_2 &= \beta_2((t_1\beta+i_{21}\beta_2)(1-e)+2\mu+r+1),\\ a_1 &= -V_3+\beta_2(1-e)(t_1\beta\gamma+t_1\beta\mu+t_1\beta r\\ &\quad +i_{21}\gamma\beta_2+2i_{21}\mu\beta_2+i_{21}r\beta_2+i_{21}\beta_2),\\ a_0 &= (1-e)\beta_2^2(1+\mu)(\gamma+\mu+r)i_{21},\end{aligned} \tag{14}$$

which are all strictly positive if i_{21} is strictly positive and $V_2>0$. The only subresultant we have to calculate is $sr=a_0(a_2a_1-a_0a_3)$. After the calculation, we have

$$a_2a_1-a_0a_3 = q_2i_{21}^2+q_1i_{21}+q_0, \tag{15}$$

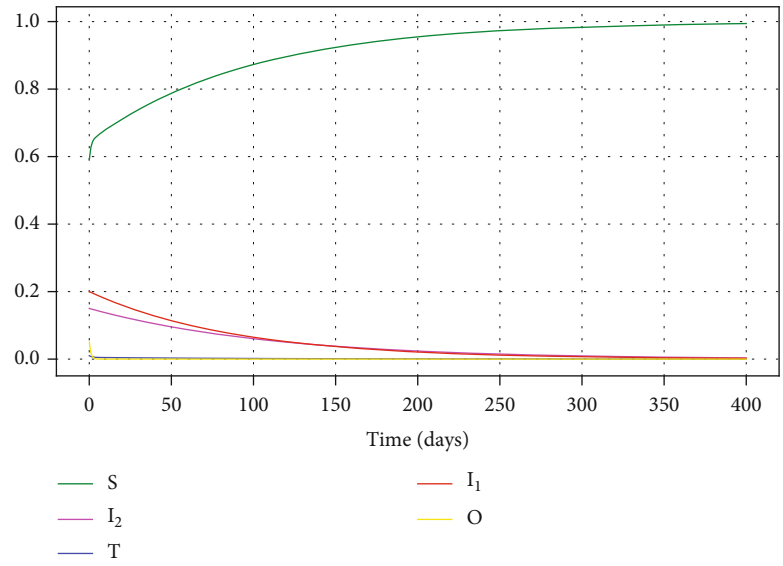

Figure 2: Disease spread at $\gamma = 0.01$ and $e = 0.1$.

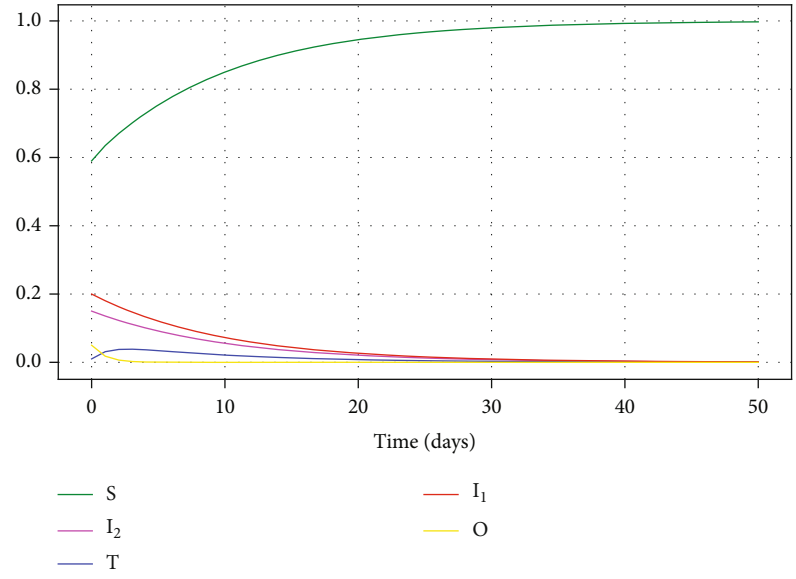

Figure 3: Disease spread at $\gamma = 0.1$ and $e = 0.1$.

with

$$\begin{aligned} q_2 &= \beta_2^4(e-1)^2(\gamma + 2\mu + r + 1),\\ q_1 &= \beta_2^2(1-e)[t_1\beta\beta_2(1-e)(2\gamma + 3\mu + 2r + 1) - V_3\\ &\quad + \beta_2(\gamma\mu + \gamma r + 3\mu^2 + 3\mu r + r^2 + 3\mu + r + 1)],\\ q_0 &= \beta_2(t_1\beta(1-e) + 2\mu + r + 1)\\ &\quad \cdot (t_1\beta\beta_2(1-e)(r + \mu + \gamma) - V_3), \end{aligned} \tag{16}$$

which is obviously and strictly positive if $V_3 < 0$ and $V_2 > 0$. So a_3, a_2, a_1, a_0, and sr are all strictly positive. E_1 is therefore hyperbolic and locally asymptotically stable if and only if $V_3 < 0$, $V_2 > 0$ and $V_6 < 0$.

4.3. Stability of the Endemic Equilibrium E_2

4.3.1. Invariant Domain. Given a differentiable vector field $f : \mathbb{R}^n \longrightarrow \mathbb{R}^n$, 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 [14]). The application of this property makes it easy to verify that $\mathbb{R}_+^n$ is positively invariant under the vector field associated with system (1). Let us recall that a 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.

4.3.2. Global Stability of E_2. For the equilibrium E_2, we can use a Lyapunov function to study this stability. Note that $\dot{S} + \dot{I}_2 + \dot{I}_1 + \dot{T} + \dot{O} = \mu(1 - (S + I_2 + I_1 + T + O)) = 0$, so the

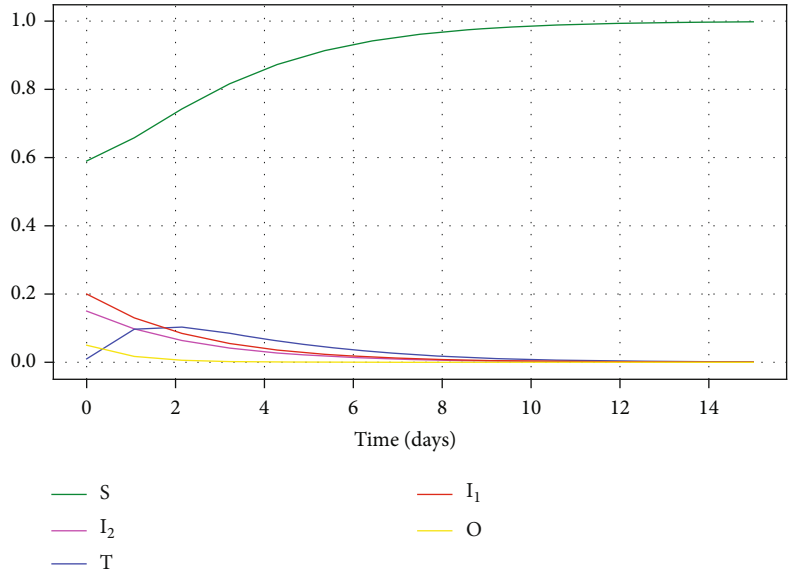

Figure 4: Disease spread at $\gamma = 0.4$ and $e = 0.1$.

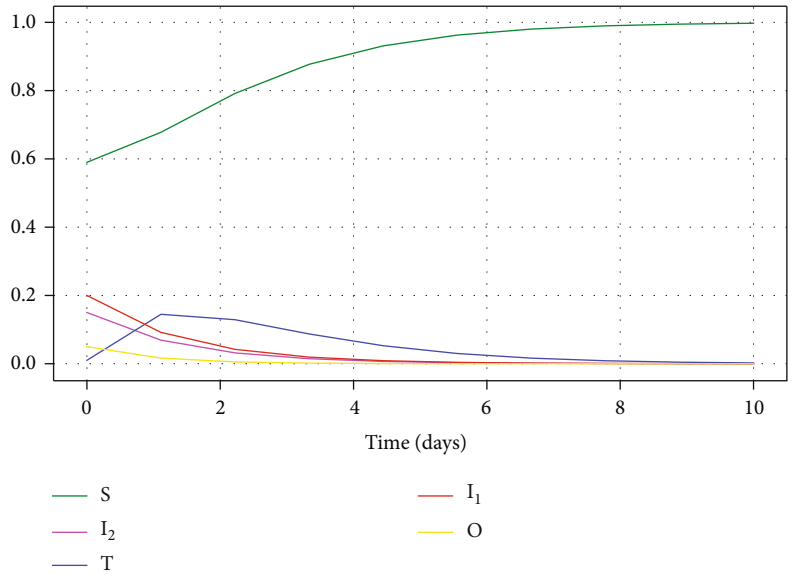

Figure 5: Disease spread at $\gamma = 0.7$ and $e = 0.1$.

domain $\Omega = \{(S, I_2, I_1, T, O) \in \mathbb{R}_+^5 : S + I_2 + I_1 + T + O = 1\}$ is positively invariant under $\dot{x} = f(x(t), u)$.

Let $L = (I_1 - i_{12})^2$,

$$\frac{dL}{dt} = 2(I_1 - i_{12})\dot{I}_1 = 2I_1(I_1 - i_{12})((1-e)\beta_1 S - (\nu_1 + \mu + \gamma)) \leq 2I_1(I_1 - i_{12})V_1 \leq 0. \tag{17}$$

Indeed, $V_1 > 0$ and $(1-e)\beta_1 S - (\nu_1 + \mu + \gamma) \geq 0$ if $S \geq s_2$, so I_1 increases to i_{12}; then, $I_1 - i_{12} \leq 0$.

Therefore, E_2 is hyperbolic and globally asymptotically stable in Ω if and only if $V_1 \geq 0$, $V_4 \leq 0$, and $V_6 \geq 0$.

4.4. Stability of the Equilibrium E_3. For endemic equilibrium E_3, the specialized characteristic polynomial of the Jacobian matrix was not fully factorized, but by substituting (S, I_2, I_1) by (s_3, i_{23}, i_{13}), we obtain

$$\partial_x f(u, E_3) = \begin{bmatrix} (e-1)\beta t_3 - \mu & a_{12} & a_{13} & a_{14} & 1-\gamma \\ 0 & -\dfrac{V_3}{\gamma\beta} & \nu_1 & 0 & 0 \\ 0 & 0 & -\dfrac{V_4}{\gamma\beta} & 0 & 0 \\ 0 & \gamma & \gamma & -r-\mu & \gamma \\ (1-e)\beta t_3 & 0 & 0 & \dfrac{(r+\mu)(1+u)}{\gamma} & -1-\mu \end{bmatrix}, \tag{18}$$

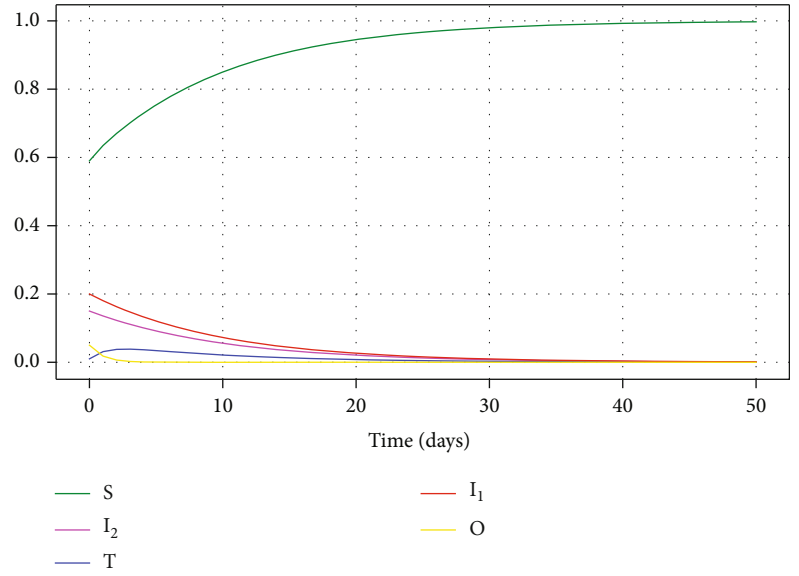

Figure 6: Disease spread at $\gamma = 0.1$ and $e = 0.01$.

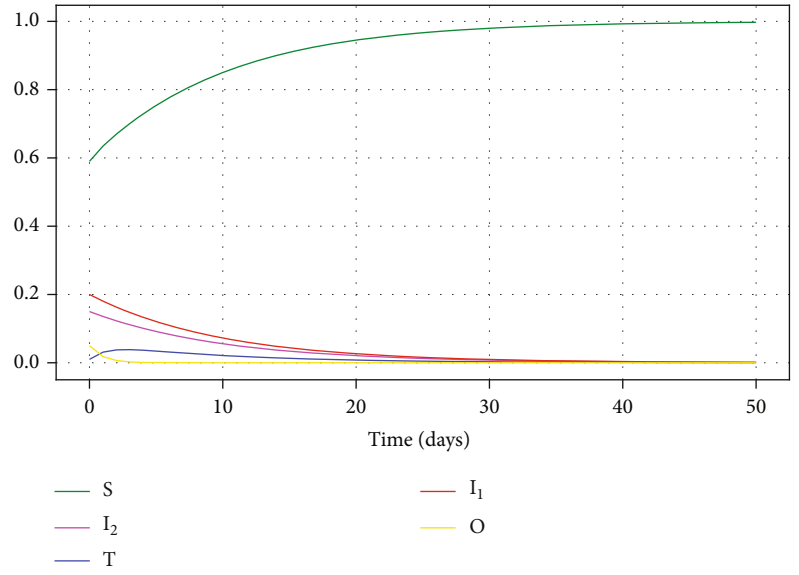

Figure 7: Disease spread at $\gamma = 0.1$ and $e = 0.1$.

with $a_{12} = -(\beta_2(r+\mu)(1+u)/\gamma\beta)$, $a_{13} = -(\beta_1(r+\mu)(1+u)/\gamma\beta)$, and $a_{14} = -((\gamma r - (r+\mu)(1+u))/\gamma)$, and the characteristic polynomial is factorized [13] as

$$\chi_3 = (Z+\mu)(\beta\gamma Z + V_3)(\beta\gamma Z + V_4)Q_3, \tag{19}$$

where $Q_3 = Z^2 + ((1-e)\beta t_3 + 2\mu + r + 1)Z + (1-e)\beta t_3(\gamma + \mu + r)$. To study the stability of E_3, we applied the Lienard-Chipart criterion [12] to the polynomial Q_3.

Therefore, E_3 is hyperbolic and locally asymptotically stable if and only if $V_3 > 0$, $V_4 > 0$, and $V_5 \leq 0$.

We have thus the following result.

Theorem 1. *The model represented by system (1) has four equilibria:*

(1) A disease-free equilibrium E_0 which exists for all values of the parameters. It is hyperbolic and locally asymptotically stable if and only if $V_1 < 0$, $V_2 < 0$, and $V_5 < 0$

(2) An equilibrium E_1 which exists if and only if $V_2 \geq 0$ and $V_3 \leq 0$ and is hyperbolic and locally asymptotically stable if and only if $V_3 < 0$, $V_2 > 0$, and $V_6 < 0$

(3) An equilibrium E_2 which exists and is hyperbolic and globally asymptotically stable in Ω if and only if $V_1 \geq 0$, $V_4 \leq 0$, and $V_6 \geq 0$

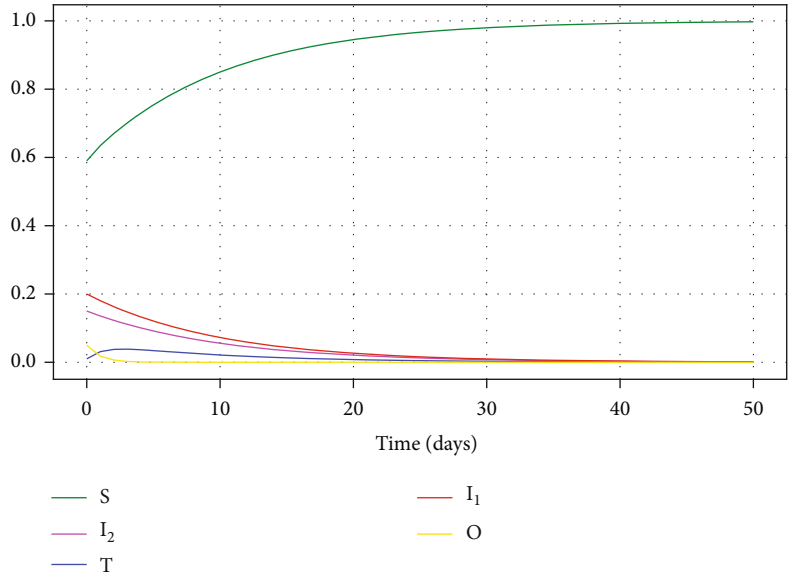

Figure 8: Disease spread at $\gamma = 0.1$ and $e = 0.4$.

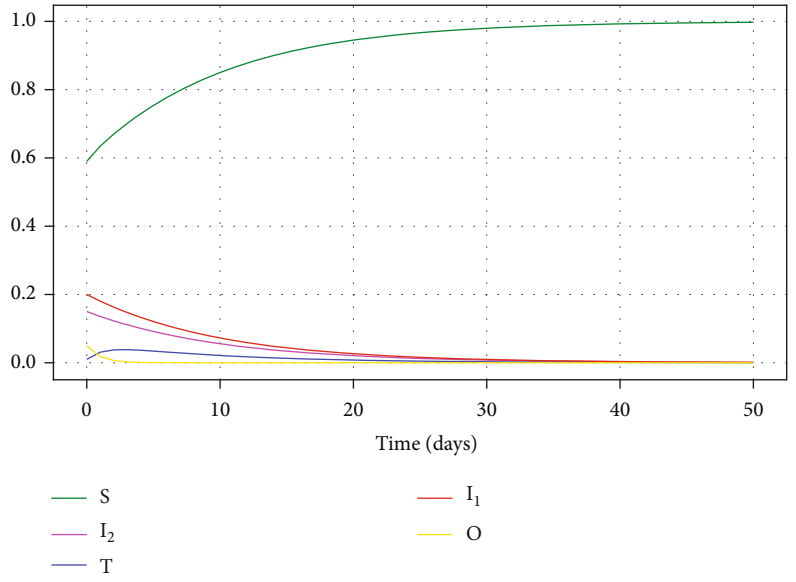

Figure 9: Disease spread at $\gamma = 0.1$ and $e = 0.7$.

(4) *An equilibrium E_3 which exists if $V_5 \leq 0$ and is hyperbolic and locally asymptotically stable if and only if $V_3 > 0$, $V_4 > 0$, and $V_5 \leq 0$*

5. Global Stability of Disease-Free Equilibrium

Theorem 2. *If $R_0 < 1$, then the disease-free equilibrium $E_0 = (1, 0, 0, 0, 0)$ is globally asymptotically stable.*

Consider the function

$$L: \begin{array}{c} R_+^5 \longrightarrow R_+ \\ x \mapsto I_1 \end{array}. \tag{20}$$

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

$$\begin{aligned} \frac{dL(x(t))}{dt} &= ((1-e)\beta_1 S - (\nu_1 + \gamma + \mu))I_1 \\ &\leq ((1-e)\beta_1 - (\nu_1 + \gamma + \mu))I_1 = V_1 I_1. \end{aligned} \tag{21}$$

This shows that it is a Lyapunov function if $V_1 < 0$. Thus, E_0 is globally asymptotically stable in Ω if $R_0 < 1$.

6. Numerical Simulation

Numerical simulations are done using the Python computer software program. Parameter values are given in Table 2.

6.1. Effect of Varying Treatment Rates 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 Figures 2, 3, 4, and 5.

6.2. Effect of Varying Health Precaution Rates on Different Epidemiological Classes. In this part, the treatment rate γ is fixed to 0.1. The effect of the 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 Figures 6, 7, 8, and 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.

7. Conclusion

For the model presented, all 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 (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 400 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 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 has 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.

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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