

Analysis of a discrete mathematical COVID-19 model

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ABSTRACT

To describe the main propagation of the COVID-19 and has to find the control for the rapid spread of this viral disease in real life, in current manuscript a discrete form of the SEIR model is discussed. The main aim of this is to describe the viral disease in simplest way and the basic properties that are related with the nature of curves for susceptible and infected individuals are discussed here. The elementary numerical examples are given by using the real data of India and Algeria.

Introduction

As throughout this pandemic, we are hearing the increase of its frequency and other side all the health organizations, health ministry, and WHO are in struggle to control its rapid spread. It is clear that this is not a direct request to the population, but it translates into concrete actions that affect our lives: social isolation, use of masks, etc. The study of epidemics is old as old civilizations. The first medical document referring to infectious diseases is presented by Ebers Papyrus (approximately 1500 BC); more specifically, to malaria. Over the world, systematic studies on the different epidemics are discussed, while in 20th century the start of mathematical models occurred. Daniel Bernoulli [1] presented the famous SIR model for smallpox then discussed by some famous mathematicians such as d'Alembert one. But it was Ronald Ross, an Indian-born physician who, after years of researching precisely on malaria and named – also precisely – SIR in 1911, would highlight the importance of mathematical models as cited for example in [2,3]. In 1927, William Kermack and Anderson McKendrick [4] introduced the first complete mathematical model in epidemiology, with which they analyzed a plague epidemic in India in 1906 based on previous work by Ross and William Hamer [5,6]. After this, the duration of the disease is also taken into account, since someone who is infected for many days is potentially going to infect a greater number of individuals. These factors are sometimes offset: for

example, for a virus like HIV the probability of contagion is low, but an infected person can transmit it over several years.

The basic SIR model consists of three groups: susceptible, infected and recovered. The population is assumed to be constant, that is, there are no births or deaths. This may seem like an oversimplification, in the style of the famous spherical horses, especially when it comes to a disease that takes the lives of thousands of people every day. While some diseases may come over the exposed class and similarly with different aspects in literature different studies are found for infectious diseases in continues and discrete forms see [7–14]. However, what it means is that it is a system closed, in which individuals are not added or subtracted from the population.

Epidemic models are related to the variation of a disease over time and from one place to another, must be considered mathematically that how many variables are involved regarding the nature of diseases. In this sense, here we consider the dynamics of population in four classes as follows:

$$S \rightarrow E \rightarrow I \rightarrow R \rightarrow S.$$

The usual version of this model consists of a system of differential equations, although for simplicity we will deal here only with the discrete version, that is, with a system of difference equations. Ultimately, this

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simplification is reasonable considering that the data does not reach us in a continuous if not spaced: for example, the daily reports from the health ministry. On the other hand, the discrete version is enough to capture the essential properties of the model and will allow us to understand what that happy (at least when it is low) value called R . Finally, a detail that is not minor: to pose the equations in differences, only elementary mathematics is required.

Consequently, the discrete SEIR model can be an excellent motivation to work with students and better process all the information that comes to us on these topics. In what follows, to refer to the original model of differential equations, we will use the expression ‘continuous model’ and for more details see [15–27].

The article is organized in the following manner. Section “Mathematical models for epidemics” presents some generalities and a brief description of the mathematical model for the spread of COVID-19 in form of SEIR. Basics properties like existence of fixed point and equilibria of the model are discussed in Section “Basic properties”. Section “Bifurcation analysis” is related the bifurcation analysis of proposed model and in Section “Conclusion” the graphical results are presented related the data of India and Algeria. Finally, concluding remarks are given in last section.

Mathematical models for epidemics

In this section, we will present the assumptions according to which displacements occur from one class to the other and the difference equations that follow from them. This will finally allow us to study the dynamics of discrete time model for COVID-19.

The law of mass action

In this subsection, we will see how the model raises the passage from the first group to the second and third classes according to the scheme

$$S \xrightarrow[\beta_1(t)SE]{} E,$$

and

$$S \xrightarrow[\beta_2(t)SI]{} E.$$

Parameters $\beta_1(t) > 0$ and $\beta_2(t) > 0$, are known as the infection rates and is given by the relationship

$$\beta_1 = \frac{p_1 \kappa_1}{N}, \quad \beta_2 = \frac{p_2 \kappa_2}{N},$$

where κ_i for $i = 1, 2$ are the average numbers of contacts per capita (per unit of time), p_i for $i = 1, 2$ are the probabilities of contagion and N is the total population. So

$$p_1 \kappa_1 \frac{S}{N} = \text{contagions produced by exposed person per unit of time,}$$

and

$$p_2 \kappa_2 \frac{S}{N} = \text{contagions produced by an infected person per unit of time.}$$

Finally, by applying the total numbers of exposed and infected people to their related forces, we have respectively

$$p_1 \kappa_1 \frac{S}{N} E = \text{number of new exposed people per unit of time,}$$

and

$$p_2 \kappa_2 \frac{S}{N} I = \text{number of new infected per unit of time.}$$

Means that, the number of individuals passing from the class S to the classes E and I in the instant n are $\beta_1 S(n)E(n)$ and $\beta_2 S(n)I(n)$ respectively, and hence we have

$$S(n+1) = S(n) - \beta_1(t)E(n)S(n) - \beta_2(t)I(n)S(n). \quad (1)$$

Going from group E to group I

All this allows us to calculate how the exposed population is changing, losing a quantity per unit of time $\phi E(n)$ of individuals as

$$E \xrightarrow[\phi E]{} I,$$

and, in turn, receives a quantity $\beta_1 S(n)E(n)$ and $\beta_2 S(n)I(n)$ from the first group:

$$E(n+1) = E(n) + \beta_1 S(n)E(n) + \beta_2 S(n)I(n) - \phi E(n).$$

Going from group I to group R

As in the previous section, the displacement of individuals from the group I to the group R . It is also governed by a law, which in this case is simpler: the infected leave the second group as they ... cease to be.

$$I \xrightarrow[\gamma I]{} R.$$

In other words, there is a recovery time denoted with the letter γ , which is interpreted as the inverse of the average duration D of the disease and indicates the proportion of infected who pass (as always, per unit of time) to the third group:

$$\gamma = \frac{1}{D}.$$

Like β , we will assume that γ is constant and, furthermore, it is clear that it must be a number between 0 and 1, why γI must be less than I . In this way, the group R increases per unit time according to the rule

$$R(n+1) = R(n) + \gamma I(n). \quad (2)$$

Further, assume that the rate at which the recovered population form infection again join the susceptible class S is τ , is a constant and it must be a number between 0 and 1, why τR must be less than R . In this way, the group R decreases per unit time according to the rule and (2) leads new form as

$$R(n+1) = R(n) + \gamma I(n) - \tau R(n) = (1 - \tau)R(n) + \gamma I(n), \quad (3)$$

and (1) get the form of

$$S(n+1) = S(n) - \beta_1 S(n)E(n) - \beta_2 S(n)I(n) + \tau R(n). \quad (4)$$

All this allows us to calculate how the number of infected is changing, losing a quantity per unit of time $\gamma I(n)$ of individuals and, in turn, receives a quantity $\phi E(n)$ from the second group:

$$I(n+1) = I(n) + \phi E(n) - \gamma I(n).$$

This equation is what determines the behavior of the infected curve; However, this does not depend only on $I(n)$ but also of the other unknown variables. Therefore, what we must study is not a single equation but a system.

The game of 3 differences

$$S(n+1) - S(n) = -\beta_1 S(n)E(n) - \beta_2 S(n)I(n) + \tau R(n) \quad (5)$$

$$E(n+1) - E(n) = \beta_1 S(n)E(n) + \beta_2 S(n)I(n) - \phi E(n)$$

$$I(n+1) - I(n) = \phi E(n) - \gamma I(n)$$

$$R(n+1) - R(n) = \gamma I(n) - \tau R(n),$$

for which the initial conditions are assumed:

$$S(0) = S_0 > 0, E(0) = E_0 > 0, I(0) = I_0 > 0, R(0) = R_0.$$

This is quite clear: on the one hand, it is logical to assume that at the beginning of the disease there are still no recovered individuals; on the other hand, if the initial number of infected were zero, then we would continue going to class and sharing the mate with complete peace of

mind, since no one could be infected. It should be mentioned that precisely the solutions with $I(0) = 0$ correspond to the only balances of the system, that is, the constant solutions.

By including the recruitment rate in the first group as λ and natural plus disease death rate in each group represents by μ , so the above system leads to

$$S(n+1) - S(n) = \lambda - \beta_1 S(n)E(n) - \beta_2 S(n)I(n) - \mu S(n) + \tau R(n) \quad (6)$$

$$E(n+1) - E(n) = \beta_1 S(n)E(n) + \beta_2 S(n)I(n) - (\phi + \mu)E(n)$$

$$I(n+1) - I(n) = \phi E(n) - (\gamma + \mu)I(n)$$

$$R(n+1) - R(n) = \gamma I(n) - (\tau + \mu)R(n),$$

Basic properties

Existence of the fixed points and method summary

According to $N = \frac{\lambda}{\mu}$ the model (6) can be reduced to the following model

$$\begin{cases} S(n+1) = \left(\lambda + \frac{\lambda\tau}{\mu}\right) + (1 - \mu - \tau)S(n) \\ \quad - \beta S(n)E(n) - \beta S(n)I(n) - \tau I(n) - \tau E(n), \\ E(n+1) = (1 - \phi - \mu)E(n) + \beta S(n)E(n) + \beta S(n)I(n), \\ I(n+1) = (1 - \gamma - \mu)I(n) + \phi E(n), \end{cases} \quad (7)$$

where $\beta_1 = \beta_2 = \beta$.

To obtain the fixed points of model (7), we must solve the following equations

$$\begin{cases} \left(\lambda + \frac{\lambda\tau}{\mu}\right) + (1 - \mu - \tau)S - \beta SE - \beta SI - \tau I - \tau E = S, \\ (1 - \phi - \mu)E + \beta SE + \beta SI = E, \\ (1 - \gamma - \mu)I + \phi E = I. \end{cases} \quad (8)$$

The solutions of (8) are:

$$F_0 = \left(\frac{\lambda}{\mu}, 0, 0\right), \quad F_1 = (S_*, E_*, I_*),$$

where

$$S_* = \frac{(\gamma + \mu)(\mu + \phi)}{\beta(\gamma + \mu + \phi)},$$

$$E_* = \frac{\gamma + \mu}{\phi} I_*,$$

$$\begin{aligned} I_* &= \frac{\lambda - \frac{\mu(\phi + \mu)(\gamma + \mu)}{\beta(\phi + \mu + \gamma)}}{(\phi + \mu)(\phi + \gamma)(\tau + \mu) - \tau\gamma\phi} \\ &= \frac{\mu(\phi + \mu)(\gamma + \mu)}{\beta(\phi + \mu + \gamma)} \left(\frac{\frac{\lambda\beta(\phi + \mu + \gamma)}{\mu(\phi + \mu)(\gamma + \mu)} - 1}{(\phi + \mu)(\phi + \gamma)(\tau + \mu) - \tau\gamma\phi} \right), \\ &= \frac{\mu(\phi + \mu)(\gamma + \mu)}{\beta(\phi + \mu + \gamma)} \left(\frac{R_0 - 1}{(\phi + \mu)(\phi + \gamma)(\tau + \mu) - \tau\gamma\phi} \right), \\ &> 0, \text{ for } R_0 > 1. \end{aligned}$$

where $R_0 = \frac{\lambda\beta(\phi + \mu + \gamma)}{\mu(\phi + \mu)(\gamma + \mu)}$ is the reproductive number. The model (7) can be considered as the map:

$$\begin{pmatrix} S \\ E \\ I \end{pmatrix} \mapsto M^{SEI}(S, E, I) = \begin{pmatrix} \left(\lambda + \frac{\lambda\tau}{\mu}\right) + (1 - \mu - \tau)S - \beta SE - \beta SI - \tau I - \tau E \\ (1 - \phi - \mu)E + \beta SE + \beta SI \\ (1 - \gamma - \mu)I + \phi E \end{pmatrix}. \quad (9)$$

The Jacobian matrix of the map (9) is as follows:

$$A^{SEI} = \begin{pmatrix} (-E - I)\beta - \mu - \tau + 1 & -\beta S - \tau & -\beta S - \tau \\ \beta E + \beta I & \beta S - \mu - \phi + 1 & \beta S \\ 0 & \phi & 1 - \gamma - \mu \end{pmatrix}.$$

The multi-line forms corresponding to the map (9) are defined as follows:

$$B_i(\Gamma, \Sigma) = \sum_{j,k=1}^3 \frac{\partial^2 M_i^{SEI}(S, E, I)}{\partial \xi_j \partial \xi_k} \gamma_j \sigma_k,$$

$$C_i(\Gamma, \Sigma, Y) = \sum_{j,k,l=1}^3 \frac{\partial^3 M_i^{SEI}(S, E, I)}{\partial \xi_j \partial \xi_k \partial \xi_l} \gamma_j \sigma_k v_l,$$

where

$$\xi = (S, E, I), \quad \Gamma = (\gamma_1, \gamma_2, \gamma_3)^T, \quad \Sigma = (\sigma_1, \sigma_2, \sigma_3)^T, \quad Y = (v_1, v_2, v_3)^T.$$

For more detail see [8–10].

Theorem 1. (i) For $R_0 = \frac{\lambda\beta(\phi + \mu + \gamma)}{\mu(\phi + \mu)(\gamma + \mu)} < 1$, the model (7) has only one disease free equilibrium point F_0 . (ii) For system (7) there exists a positive equilibrium point F_1 if $R_0 = \frac{\lambda\beta(\phi + \mu + \gamma)}{\mu(\phi + \mu)(\gamma + \mu)} > 1$.

More basic properties:

1. The total population $N = S + E + I + R$ remains constant.
2. For $n > 0$, all values of $S(n)$, $E(n)$, $I(n)$ and $R(n)$ are positive and less than N . Indeed, as an inductive hypothesis that $I(n)$, $E(n)$, $S(n) > 0$ and $R(n) \geq 0$, which is obviously true for $n = 0$. From there it is clear that $R(n+1) = (1 - (\tau + \mu))R(n) + \gamma I(n) > R(n) \geq 0$. On the other hand, $\beta_1 E(n) < \beta_1 N < 1$, and $\beta_2 I(n) < \beta_2 N < 1$, from where

$$S(n+1) = (1 - \beta_1 E(n) - \beta_2 I(n) - \mu)S(n) + \lambda + \tau R(n) > 0,$$

while

$$I(n+1) = (1 - \gamma - \mu)I(n) + \phi E(n) > 0.$$

3. The sequences $S(n)_{n \in \mathbb{N}}$ and $R(n)_{n \in \mathbb{N}}$ are strictly monotonous and coupled; therefore, they converge. Indeed, as $I(n)$ it's positive, we already saw in (2) what $R(n)$ it is growing; on the other hand,

$$S(n+1) - S(n) = \lambda - \beta_1 E(n)S(n) - \beta_2 I(n)S(n) - \mu S(n) + \tau R(n) < 0,$$

so that $S(n)$ is decreasing.

The behavior of $I(n)$ it is the one that interests us the most and deserves that we analyze it separately as following.

As in discrete for we cannot define real curves. However, for practical purposes it can always be assumed that we interpolate the points reasonably and graph them as continuous functions for $t \geq 0$. Here, we study directly the growth or decrease of the succession $I(n)_{n \in \mathbb{N}}$, as from the formula we have:

$$I(n+1) - I(n) = -(\gamma + \mu)I(n) + \phi E(n) > 0.$$

Since it is a recurrence, it is useful to first see what happens at the beginning, when $n = 0$. This leads us to consider two cases:

case 1. If $\phi E(n) - (\gamma + \mu)I(n) < 0$ for all $n > 0$. In turn, this implies that $I(n+1) - I(n) < 0$, that is to say: $I(n)$ it is always decreasing because the recovery rate become more than infected rate ϕ . This leads a good news that there is no epidemic. But, unfortunately, epidemics do happen, so it is better to also take a look at the other possible situation:

case 2. $\phi E_0 - (\gamma + \mu)I_0 > 0$. This means that, at the beginning, $I(n)$ it grows. Let us see what does it up to a certain value n_{\max} and then it decreases. If $\phi E(n) - (\gamma + \mu)I(n) \leq 0$ for certain value of n , thereafter the song number of infected is decreasing; then it is enough to prove that $I(n)$ cannot be growing for everything n . But this can be seen as an immediate consequence of a fundamental fact, which deserves to be demonstrated separately for $\beta_1 = \beta_2 = \beta$:

Theorem 2. If $\beta \in (0, \frac{1}{N})$, $\gamma \in (0, 1)$, $S_0, E_0, I_0 > 0$, then:

$$\lim_{n \rightarrow \infty} I(n) = 0.$$

Proof. We already saw that $0 < I(n), R(n) < N$ for all $n > 0$. Moreover, given the equality

$$R(n+1) - R(n) = \gamma I(n) - \tau R(n),$$

we can add telescopically up to Σ certain value m , from where (remembering what $R(0) = 0$) result:

$$R(m+1) = \gamma \sum_{n=0}^m I(n).$$

It only remains to observe which series Σ as $R(m)$ remains limited and also $I(n) > 0$, the $\sum_{n=0}^{\infty} I(n)$ converges and consequently $I(n) \rightarrow 0$.

In short, the behavior of the infected curve depends essentially on a value that allows us to know in advance whether or not there will be an epidemic:

$$\mathcal{R}_0 = \left(\frac{\beta_1}{\phi + \mu} + \frac{\beta_2 \phi}{(\phi + \mu)(\gamma + \mu)} \right) S_0,$$

for $\beta = \beta_1 + \beta_2$, we have

$$\mathcal{R}_0 = \frac{\lambda \beta (\phi + \mu + \gamma)}{\mu (\phi + \mu)(\gamma + \mu)}.$$

Here, \mathcal{R}_0 determines the situation of case 1 for $\mathcal{R}_0 \leq 1$, while case 2 occurs when $\mathcal{R}_0 > 1$. In short, it is a fundamental parameter and that is why it is so important. Knowing it, as it indicates what would happen to the epidemic if no action is taken to contain it. Of course, it is not a directly observable value but it is estimated; for this, there are various techniques such as those mentioned for example in (Martcheva, 2015). In general, it is not clear how many are infected at the beginning of the process; for this reason, the so-called basic play number, defined as $\mathcal{R}_b = \left(\frac{\beta_1}{\phi + \mu} + \frac{\beta_2 \phi}{(\phi + \mu)(\gamma + \mu)} \right) N$, which is essentially the same as the previous one while the proportion of infected is small. Once the epidemic is advanced, the amount, being an important indicator, since it allows to know if the infection curve $\left(\frac{\beta_1}{\phi + \mu} + \frac{\beta_2 \phi}{(\phi + \mu)(\gamma + \mu)} \right) S(n)$ follow all grows or decreases, depending on whether its value is greater or less than one. That is precisely the so-called \mathcal{R} ; public health policies are aimed at keeping it as low as possible. From here, we conclude that the possible paths are:

- Reduce D , by finding and producing effective antiviral.
- Reduce p (transmissibility): hygiene measures, chinstraps.
- Reduce κ : isolation measures.

However, the discrete case requires a bit more care and we will see that in the next output.

Theorem 3. If $\beta \in (0, \frac{1}{N})$, $\gamma \in (0, 1)$, $S_0, E_0, I_0 > 0$, then:

$$\lim_{n \rightarrow \infty} S(n) > 0.$$

Proof. From As from the last result, we have that the limit exists, well $S(n)_{n \in \mathbb{N}} \subset \mathbb{R} > 0$ and is decreasing. On the other hand, let us see that, as in addition, it makes sense to propose a “telescopic product”. Indeed, since

$$\frac{S(n+1)}{S(n)} = 1 - \beta_1 E(n) - \beta_2 I(n) + \tau \frac{R(n)}{S(n)},$$

then for $m > n$ we have:

$$\frac{S(m+1)}{S(m)} \frac{S(m)}{S(m-1)} \dots \frac{S(n+1)}{S(n)} = \prod_{j=n}^m (1 - \beta_1 E(j) - \beta_2 I(j) + \tau \frac{R(j)}{S(j)}),$$

that is

$$\frac{S(m+1)}{S(n)} = \prod_{j=n}^m (1 - \beta_1 E(j) - \beta_2 I(j) + \tau \frac{R(j)}{S(j)}).$$

Now a common idea in the study of infinite products come in our help. Let us start by rewriting the Σ previous equality in form

$$\ln \left(\frac{S(m+1)}{S(n)} \right) = \sum_{j=n}^m \ln(1 - \beta_1 E(j) - \beta_2 I(j) + \tau R(j)),$$

and note that, for any $x > 0$ small enough, ok for example what $\ln(1-x) > -2x$. How $E(j) \rightarrow 0$, $I(j) \rightarrow 0$ and $R(j) \rightarrow 0$, we can choose a large value of n such that the above inequality holds for $E(j)$, $I(j)$ and $R(j)$ such that $j \geq n$

$$\ln \left(\frac{S(m+1)}{S(n)} \right) \geq \sum_{j=n}^m [-2(\beta_1 E(j) + \beta_2 I(j))] = -2 \left[\beta_1 \sum_{j=n}^m E(j) + \beta_2 \sum_{j=n}^m I(j) \right].$$

But let us remember that

$$R(j+1) - R(j) = \gamma I(j),$$

so that (again the sum telescopic) ok, for everything $m > n$,

$$\ln \left(\frac{S(m+1)}{S(n)} \right) \geq -\frac{2\beta}{\gamma} \sum_{j=n}^m \gamma I(j) = -2\beta [R(m+1) - R(m)],$$

and, in short:

$$S(m+1) \geq S(n) e^{-2\beta [R(m+1) - R(m)]} \geq S(n) e^{-2\beta [N - R(m)]},$$

which is a positive constant.

Bifurcation analysis

Codimension one bifurcation of F_0

Theorem 4. F_0 undergo a flip bifurcation at

$$\begin{aligned} (1) \quad \lambda &= \frac{\mu (\gamma \mu + \gamma \phi + \mu^2 + \mu \phi - 2\gamma - 4\mu - 2\phi + 4)}{\beta (\gamma + \mu + \phi - 2)}, \\ (2) \quad \beta &= \frac{\mu (\gamma \mu + \gamma \phi + \mu^2 + \mu \phi - 2\gamma - 4\mu - 2\phi + 4)}{\lambda (\gamma + \mu + \phi - 2)}, \\ (3) \quad \phi &= \frac{\beta \gamma \lambda + \beta \lambda \mu - \gamma \mu^2 - \mu^3 - 2\beta \lambda + 2\gamma \mu + 4\mu^2 - 4\mu}{-\beta \lambda + \gamma \mu + \mu^2 - 2\mu} \end{aligned}$$

Proof. We only proof the first item.

It is clear that the Jacobian matrix A at F_0 and for $\lambda = \frac{\mu (\gamma \mu + \gamma \phi + \mu^2 + \mu \phi - 2\gamma - 4\mu - 2\phi + 4)}{\beta (\gamma + \mu + \phi - 2)}$ has a simple eigenvalue -1 and it does not have any eigenvalues on the unit circle. Therefore the map (9) at $\lambda = \frac{\mu (\gamma \mu + \gamma \phi + \mu^2 + \mu \phi - 2\gamma - 4\mu - 2\phi + 4)}{\beta (\gamma + \mu + \phi - 2)}$ can be considered as

$$\omega \mapsto -\omega + b_{PD} \omega^3 + \mathcal{O}(\omega^4),$$

where

$$Av = -v, \quad A^T w = -w, \quad \langle w, v \rangle = 1.$$

In this case we have

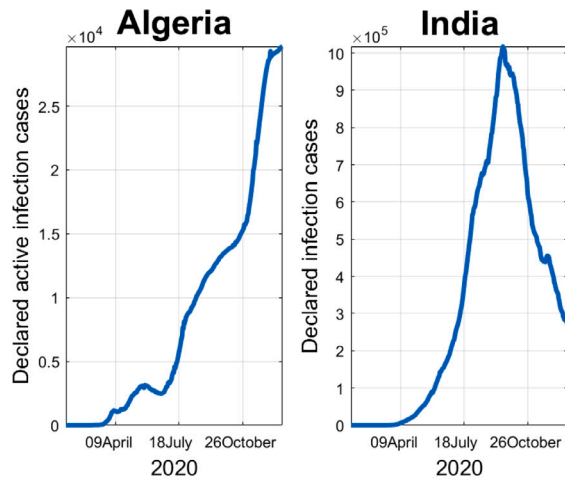
$$\begin{aligned} v &= \begin{pmatrix} \frac{-\mu^2 + (-\phi - \tau - \gamma + 4)\mu + (-\tau - \gamma + 2)\phi - (\gamma - 2)(\tau - 2)}{(\mu + \tau - 2)\phi} \\ \frac{\gamma + \mu - 2}{\phi} \\ 1 \end{pmatrix}, \\ w &= \begin{pmatrix} 0 \\ \frac{\gamma + \mu + \phi - 2}{\mu + \phi - 2} \left(1 + \frac{(\gamma + \mu + \phi - 2)(\gamma + \mu - 2)}{(\mu + \phi - 2)\phi} \right)^{-1} \\ \left(1 + \frac{(\gamma + \mu + \phi - 2)(\gamma + \mu - 2)}{(\mu + \phi - 2)\phi} \right)^{-1} \end{pmatrix}. \end{aligned}$$

We can find b_{PD} as follows:

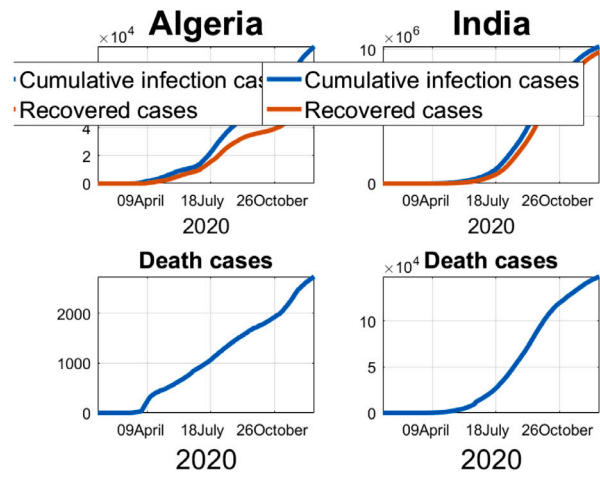
$$b_{PD} = \frac{b_{PD}^1}{b_{PD}^2},$$

where

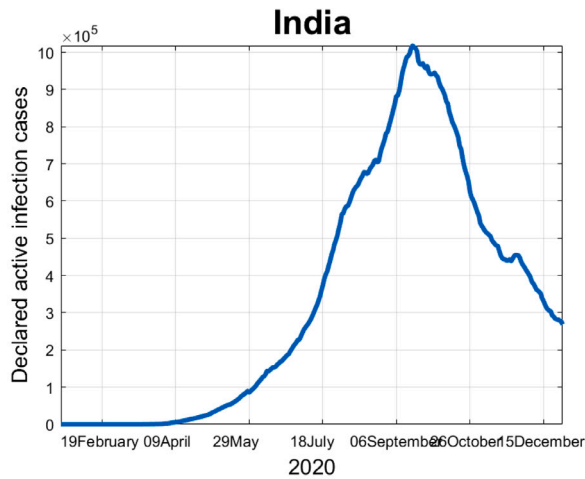
$$\begin{aligned} b_{PD}^1 &= (\gamma \mu + \gamma \phi + \gamma \tau + \mu^2 + \mu \phi + \mu \tau + \phi \tau - 2\gamma - 4\mu - 2\phi - 2\tau + 4) \\ &\quad \times (\gamma + \mu + \phi - 2)^3 \beta^2 (\gamma^2 \mu^2 + \gamma^2 \mu \phi \end{aligned}$$



(a) The Susceptible population by COVID-19



(b) The Infected population by COVID-19



(c) The Quarantined population by COVID-19

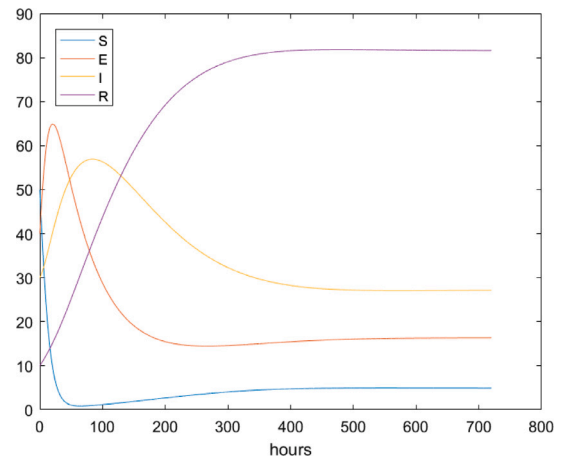


Fig. 1. Shows the numerical simulation proposed system. (a) Graph of susceptible population. (b) Graph of infected people. (c) Graph of quarantined people. Initial values are $S_0 = 50$, $E_0 = 40$, $I_0 = 30$, $R_0 = 10$, $R_0 = 0$, and parameters are $d = 0.001$, $\beta = 0.003$, $\delta = 0.003$, $\gamma = 0.002$, $b = 10$, $\eta = 0.05$, $\rho = 0.003$, $\sigma_1 = 0.003$, $\sigma_2 = 0.002$.

$$\begin{aligned}
 & + 2\gamma^2\mu\tau + \gamma^2\phi\tau + \gamma^2\tau^2 + 2\gamma\mu^3 + 3\gamma\mu^2\phi + 4\gamma\mu^2\tau + \gamma\mu\phi^2 \\
 & + 5\gamma\mu\phi\tau + 2\gamma\mu\tau^2 + \gamma\phi^2\tau + 2\gamma\phi\tau^2 + \mu^4 \\
 & + 2\mu^3\phi + 2\mu^3\tau + \mu^2\phi^2 + 4\mu^2\phi\tau + \mu^2\tau^2 + 2\mu\phi^2\tau \\
 & + 2\mu\phi\tau^2 + \phi^2\tau^2 - 2\gamma^2\mu - \gamma^2\phi - 2\gamma^2\tau - 6\gamma\mu^2 \\
 & - 6\gamma\mu\phi - 8\gamma\mu\tau - \gamma\phi^2 - 5\gamma\phi\tau - 2\gamma\tau^2 - 4\mu^3 \\
 & - 6\mu^2\phi - 6\mu^2\tau - 2\mu\phi^2 - 8\mu\phi\tau - 2\mu\tau^2 \\
 & - 2\phi^2\tau - 2\phi\tau^2 + 4\gamma\mu + 2\gamma\phi + 4\gamma\tau + 4\mu^2 + 4\mu\phi + 4\mu\tau + 4\phi\tau, \\
 b_{PD}^2 &= \phi^2 (\gamma^2 + 2\gamma\mu + \gamma\phi + \mu^2 + 2\mu\phi + \phi^2 - 4\gamma - 4\mu - 4\phi + 4) \\
 & \times (\mu + \tau - 2)^2 (\gamma^2 + 2\gamma\mu + \gamma\phi + \mu^2 \\
 & + 2\mu\phi + \phi^2 - 2\gamma - 2\mu - 2\phi) (\mu + \tau).
 \end{aligned}$$

If $b_{PD} \neq 0$ the flip bifurcation is generic. When $b_{PD} > 0$ ($b_{PD} < 0$) the flip bifurcation is supercritical (subcritical)

Theorem 5. F_0 undergo a Neimark-Sacker bifurcation at

$$\begin{aligned}
 (1) \quad \lambda &= \frac{\mu(\gamma\mu + \gamma\phi + \mu^2 + \mu\phi - \gamma - 2\mu - \phi)}{\beta(\gamma + \mu + \phi - 1)}, \\
 (2) \quad \beta &= \frac{\mu(\gamma\mu + \gamma\phi + \mu^2 + \mu\phi - \gamma - 2\mu - \phi)}{\lambda(\gamma + \mu + \phi - 1)}, \\
 (3) \quad \phi &= \frac{\beta\gamma\lambda + \beta\lambda\mu - \gamma\mu^2 - \mu^3 - \beta\lambda\gamma\mu + 2\mu^2}{-\beta\lambda\gamma + \gamma\mu + \mu^2 - \mu}.
 \end{aligned}$$

Proof. The proof is similar to proof Theorem 4.

Conclusion

The conclusions are based the new discrete method that is generated in this article because of there is no appropriate model or method in the literature is found to control somehow the COVID-19. Further, no experimental work till now done for the control of this infectious disease. So for this purpose, to describe the main propagation of the COVID-19 and has to find the control for the rapid spread of this viral disease in real life, in current manuscript a discrete form of the SEIR model is discussed. Here, we discussed the nature of the disease related the reproductive number, if the reproductive number less than one extinction of the diseases happened while persistence occurred at reproductive number value greater than one. It is observed that discrete model has more plentiful dynamical behavior than the continuous models for COVID-19. The elementary numerical examples are given by using the real data of India and Algeria (see Fig. 1).

CRediT authorship contribution statement

Thanin Sitthiwiratham: Formal analysis, Funding acquisition, Resources, Validation, Visualization, Writing – review & editing. **Anwar Zeb:** Model investigated and drafted, Data curation, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review &

editing. **Saowaluck Chasreechai**: Formal analysis, Funding acquisition, Methodology, Validation, Visualization, Formally analyzed and viewed with revision, Writing – review & editing. **Zohreh Eskandari**: Formal analysis, Software, Validation, Visualization, Formally analyzed and viewed with revision, Writing – review & editing. **Mouhcine Tilioua**: Formal analysis, Investigation, Software, Validation, Visualization, Writing – review & editing. **Salih Djilali**: Model investigated and drafted, Data curation, Investigation, Methodology, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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