



Research article

On a novel fuzzy fractional retarded delay epidemic model

Prasantha Bharathi Dhandapani^{1,*}, Jayakumar Thippan¹, Dumitru Baleanu^{2,3,4} and Vinoth Sivakumar¹

¹ Department of Mathematics, Sri Ramakrishna Mission Vidyalaya College of Arts and Science, Coimbatore-641 020, Tamilnadu, India

² Department of Mathematics, Cankaya University, Ankara, 06530, Turkey

³ Institute of Space Sciences, Magurele-Bucharest, Romania

⁴ Department of Medical Research, China Medical University Hospital, China Medical University, Taichung, Taiwan

* **Correspondence:** Email: d.prasanthabharathi@gmail.com.

Abstract: The traditional compartmental epidemic models such as SIR, SIRS, SEIR consider mortality rate as a parameter to evaluate the population changes in susceptible, infected, recovered, and exposed. We present a modern model where population changes in mortality are also considered as the parameter. The existing models in epidemiology always construct a system of the closed medium in which they assume that new birth, as well as new death, will not be possible. But in real life, such a concept will not be assumed to not exist. From our wide observation, we find that the changing rate in every population case is notably negligible, That's why we are preferring to calculate them fractionally using FFDE. Using Lofti's fuzzy concept we are picturing the models after that we are estimating their non-integer values using three distinct methodologies LADM-4, DTM-4 for arbitrary fractional-order α_i , and RKM-4. At $\alpha_i = 1$, comparison of the estimations will be done. In addition to the simulation, works of numerical estimations, the existence of steady states, equilibrium points, and stability analysis are all done.

Keywords: SIRD epidemic model; disease free and disease dependent steady states; stability analysis; LADM-Laplace Adomian decomposition method; DTM-differential transformation method; RKM-Runge-Kutta method

Mathematics Subject Classification: 26A33, 74H15, 34A07

1. Introduction

The life we are leading today in a civilized society is created in such a way that the living space of each and every species is occupied by our human race. Being concentrated on this well-sophisticated life or at least a better life, people are working all the time without rest to fulfill the needs of the family like bringing education to children, after that, their employment, etc., This is the normal family life of a middle-class people who are especially from India. Due to the commitment all the time for the family well being, there is a lack of care for their own personal health. This is one of the reasons behind losing good health. The cultivation lands now became corporate land. So good health is not easy to expect by means of properly farmed organic foods. Though medically many new innovative inventions are progressing every day there is also a vacuum in the answer when there is a question about non-eradicated diseases. Many new diseases are slowly occupying us and we are all victims to any kind of such transferring diseases. Even not having symptoms every one of us is a victim and a host of such ailments. We must prepare ourselves to be strong to fight against daily emerging new diseases. Till yesterday, the whole world was busy with the hot topic called COVID-19. But today the trend has changed to Omicron. Omicron is nothing but the new variant of the coronavirus.

In the literature of biomathematics modeling, population dynamics and the spread of epidemics are the most prominent topics since they have a wide historical background. In this paper, we study an epidemic model which is the pioneer of all studies about the transmission of ailments. The old models in epidemiology science are bound with the assumptions that new cases in the count of birth, death is not all allowed. And one more interesting concept in the epidemic case study is that they will not take death as a parameter subject to change as susceptible or infected. Recently many new mathematicians started to break that rule. We people are a step ahead in making death a parameter. We are hereby considering two cases of death. i.e., one due to the infection and the other as a natural death.

The model we are framing in this innovative manuscript is new, say SIRD-susceptible-Infected-recovered-death-population epidemic model. Few people may observe that they are not physically well and after that, they will go to the hospital and diagnose themselves to prevent any infections by taking the precautions and prescriptions with proper medical advice. But many people will not be aware of the symptoms they are having. Those people are always in the mindset that the symptoms they are having will be because of climate change or may cause a fever and will recover soon. But medically every time is valuable. Every hour will be treated as an hour of the diamond moment. Think, about what would happen if the infections were not observed or diagnosed on time. Definitely, that disease may have a chance to lead to a severe stage of infections which might put the patients' life to death. Simply we can say a time delay in the study of or diagnosis of infections may make the infections sustain severely. In order to research them mathematically, we are using the concept of time delay while implementing the rate of infection population change. Instead of the time-shift property of Laplace transform we are using a simple approach called linear operation technique in both (LADM) and in (DTM). So we can hereby declare that the model we are about to construct here is of non-integer order with infectious population study of delay due to time. As there will always arise randomness and fluctuations in modeling natural ailment dynamics we are converting our model into the fuzzy model.

The manuscript was solely prepared with dedication by the authors but it was not created all of the sudden. There are numerous fore-paper studies that have been carried out before proceeding with our analysis. They are summarized below so that the readers may get fruitful knowledge while passing

through them. They are Lofti Zadeh's theory of Fuzzy sets [1]. In 2000, the Buckley-Feuring proposal for FDE [2] created a great impact in the early 2000s. Abbasbandy's modified ADM in 2005 [3]. Allen's, mathematical biology [4]. Makinde [6], SIR-model for invariant vaccination procedure by ADM technique. Ongun's LADM for HIV infection of [7]. Arafa et al.'s fractional-order infant disease model of invariant vaccination procedure [8]. Atangana-Baleanu's novel fractional derivatives with nonlocal and non-singular kernel [9]. In [10], Aliyu provided an HIV-I cure model under ABC derivative. Farman presented the SEIR-measles model for the fractional-order derivatives using the technique known as LADM [13]. Authors in [14, 15] solved influenza models. Authors like [17, 18] presented a stochastic epidemic ODE model with perturbations. Authors like Prasantha Bharathi et al., frequently solved many types of FDEs [5, 12, 16]. Recently, Authors like P. Singh et al. contributed their innovative ideas about the dynamics of epidemic spread in [21, 22].

The paper highlights if death is used also as a variable, what can be the model? How to analyze such a model?. In this manuscript, the retarded delay is introduced and studied extensively with the necessary analysis in the upcoming sections.

Other than the introductory section, the paper was arranged as follows. Under the Sections 2–7, SIRD-with delay-model creation, the fore-studies, qualitative analysis, analytical solutions, numerical estimations, and the respective graphical illustrations are presented. Finally, in Section 8, the manuscript was concluded in detail.

2. SIRD delay epidemic model-formulation

We shall construct the fuzzy epidemic compartmental model under ABC fractional derivative ${}^{ABC}\Delta_{0,t}^\alpha$. Usually, $S(t)$, $I(t)$, $R(t)$ denote Susceptible, Infected and Recovered hosts respectively and $D(t)$ is given to the Death population where t here is taken in days (see Figure 1). The main assumption behind the model formulations is that there arises a new strange disease among the society. Due to this, there arises ' $S(t)$, $I(t)$, $R(t)$ and $D(t)$ ' here the death is not only because of the severity of infections but also due to some other health-related issues like heart attack, etc. Both are separately defined with the aid of separate parameters. Also, no additional birth or death exceeding the past count. The models that we are framing here are presented to bring the changes of approach to the classical Kermack-Mckendrick models [19, 20]. Mathematics definitions for both death and recovery are the same under which there is no chance of infection ratio can be observed. All these assumptions and strategies are put together to form the following model. Throughout the entire manuscript D is given for death population and Δ is given for d/dt .

The important thing we want to share to the readers is that the notations $S(t)$, $I(t)$, $R(t)$ and $D(t)$ are representing the number of susceptible, infected, recovered and death cases varying in time (in days) and they are dimensionless. $S(t)$, $I(t)$, $R(t)$ and $D(t)$ are initial populations in numbers at time $t = 0$. The rates β , δ , ψ , γ are representing the rates also. So our equations seem to be dimensionless. Also one can see when $D(t) = 0$, our entire study will match with the properties of SIR model [19, 20] as in SIR model [19, 20] assumes rates but dimensionless, i.e., $S(t)$, $I(t)$ and $R(t)$ are representing the numbers.

$$\begin{aligned}
\Delta S(t) &= (-\beta SI - \gamma S) \\
\Delta I(t) &= (\beta SI(t - \tau) - (\delta + \gamma)I(t)) \\
\Delta R(t) &= (2\delta - \psi)I(t) - \gamma R(t) \\
\Delta D(t) &= (\psi - \delta)I(t) - \gamma D(t)
\end{aligned} \tag{2.1}$$

SIRD with the initial function defined as $S(t_0) = m_1$, $I(t_0) = m_2$, $R(t_0) = m_3$ and $D(t_0) = m_4$ for all $-\tau \leq t_0 \leq 0$. Such that the initial functions are always constant as they are representing the population (numbers) at the time of study. All the symbols and parameters used in the above Eq (2.1) are completely described at the end of Section 3.

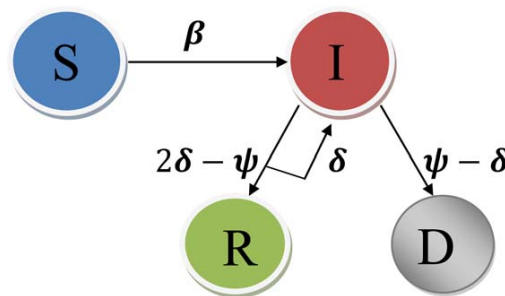


Figure 1. Model formulation.

3. Fore-studies

The operators with non-integer order seem in such a way that the many mathematical-physical-chemical and biological theories can be turned into a simplified model. Different types of fractional operators are available to easily analyze those theories. singular kernels are notably inconvenient while considering Riemann-Liouville and Caputo-fractional operators since the solutions are not fine and smooth. To get rid of this, Atangana-Baleanu(AB) introduced a very novel advanced operator which consists of Mittag-Leffler kernel which is [9] non-local as well as non-singular. The merit of using this operator is that it can be very useful in modeling the biological dynamical systems since AB-derivative eliminates the difficulty to model any before said problems with singularity. Various types of problems on physical phenomena have been constructed and studied with proper analyses by using the required operator (ABC). The below section lists out of the set of necessary results in the FFDE.

Definition 3.1. [5] The fuzzy non-integral single retarded DDE can be described in ABC way as

$$\begin{cases}
{}^{ABC}\Delta_{0,t}^{\alpha} \tilde{y}(t) = \tilde{f}(t, \tilde{y}(t), \tilde{y}(t - \tau)), & t \geq t_0 \geq 0 \\
\tilde{y}(t) = \tilde{\phi}(t), & -\tau \leq t \leq 0 \\
\tilde{y}(t_0) = \tilde{y}_0 \in \tilde{\phi}(t)
\end{cases}$$

where $\tilde{f} : [0, \infty) \times R \times R \rightarrow E^n$ and $\tilde{\phi} \in R$ is a continuous fuzzy mapping and the initial condition $y_0 \in \phi$ then $y_0(s) = y(s) = \phi(s)$, $-\tau \leq s \leq 0$. Also y_0 is fuzzy valued with r -level intervals, $[y_0]^r = [y_0^r, \bar{y}_0^r]$, $0 \leq r \leq 1$.

Definition 3.2. [9] The Mittag-Leffler function can be defined as the result of the following fractional DE $\Delta^{\alpha} = ay$, $0 < \alpha < 1$ where generalized Mittag-Leffler function $E_{\alpha}(-t^{\alpha}) = \sum_{k=0}^{\infty} \frac{(-t)^{\alpha k}}{\Gamma(\alpha k + 1)}$ is considered as non local function.

Definition 3.3. [9] The fuzzy ABC derivative of f over $[t_0, t_n]$ is

$${}^{ABC}\Delta_{t_0, t_n}^\alpha \tilde{f}(t) = \frac{F(\alpha)}{(1-\alpha)} \int_0^t \tilde{f}'(\mu) E_\alpha[-\alpha \frac{(t-\mu)^\alpha}{1-\alpha}] d\mu,$$

where $F(\alpha)$ is a normalized function which obviously satisfying $F(0) = F(1) = 1$ and $F(\alpha)$ will satisfy the properties in ABC in a same way as that in Caputo and Fabrizio case [11].

Definition 3.4. [9] We can make fuzzy ABC derivative to undergo Laplace transform implying,

$$L\{{}^{ABC}\Delta_{0,t}^\alpha \tilde{f}(t)\}(s) = \frac{F(\alpha)}{1-\alpha} \frac{s^\alpha L\{\tilde{f}(t)\}(s) - s^{\alpha-1}}{s^\alpha + \frac{\alpha}{1-\alpha}} \text{ where } 0 < \alpha \leq 1.$$

Definition 3.5. [9] The AB integral of the function $\tilde{f}(t)$ having order $\alpha > 0$ is defined as

$${}_{AB}J_{0,t}^\alpha \tilde{f}(t) = 1 - \alpha F(\alpha) \tilde{f}(t) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t f(\mu)(t-\mu)^{\alpha-1} d\mu.$$

Also when $\alpha \rightarrow 1$ then the classical integral is obtained.

The DE referring (2.1) can be overwritten as the fractional DE which is given as

$$\begin{aligned} {}^{ABC}\Delta_{0,t}^{\alpha_1} S(t) &= (-\beta S(t)I(t) - \gamma S(t)), \\ {}^{ABC}\Delta_{0,t}^{\alpha_2} I(t) &= (\beta S(t)I(t-\tau) - (\delta + \gamma)I(t)), \\ {}^{ABC}\Delta_{0,t}^{\alpha_3} R(t) &= (2\delta - \psi)I(t) - \gamma R(t), \\ {}^{ABC}\Delta_{0,t}^{\alpha_4} D(t) &= (\psi - \delta)I(t) - \gamma D(t), \end{aligned} \quad (3.1)$$

which in turn implies fuzzy fractional DE (FFDE) by the concepts that are given in the fore-studies. The primary functions are the same as that of (2.1).

$$\begin{aligned} {}^{ABC}\Delta_{0,t}^{\alpha_1} \tilde{S}(t) &= (-\beta \tilde{S}(t)\tilde{I}(t) - \gamma \tilde{S}(t)), \\ {}^{ABC}\Delta_{0,t}^{\alpha_2} \tilde{I}(t) &= (\beta \tilde{S}(t)\tilde{I}(t-\tau) - (\delta + \gamma)\tilde{I}(t)), \\ {}^{ABC}\Delta_{0,t}^{\alpha_3} \tilde{R}(t) &= (2\delta - \psi)\tilde{I}(t) - \gamma \tilde{R}(t), \\ {}^{ABC}\Delta_{0,t}^{\alpha_4} \tilde{D}(t) &= (\psi - \delta)\tilde{I}(t) - \gamma \tilde{D}(t), \end{aligned} \quad (3.2)$$

where $\tilde{f}(t) = (0.75 + 0.25r, 1.125 - 0.125r)f(t)$ is the fuzzy function with $r \in [0, 1]$.

The primary conditions are satisfied and also implies that the total population is initially constant with size N . i.e., $S(t_0) + I(t_0) + R(t_0) + D(t_0) = N$.

Here we assume the following initial populations.

$$S(t_0) = S_0 = m_1 = 50, \quad t = t_0,$$

$$I(t_0) = I_0 = m_2 = 60, \quad -\tau \leq t \leq t_0,$$

$$R(t_0) = R_0 = m_3 = 40, \quad t = t_0$$

$$D(t_0) = D_0 = m_4 = 50, \quad t = t_0.$$

The parameters their descriptions and their respective assumed rates from the (2.1)–(3.2) are shown here under.

$\beta \rightarrow$ rate of susceptible becoming infectious=0.00012;

$\delta \rightarrow$ the rate of infectious becoming recovered=0.06;

$\psi \rightarrow$ rate of infectious becoming death due to severity of infections=0.04;

$\gamma \rightarrow$ rate of infectious becoming death due to age or any other health issues=0.02.

4. Steady states and stability analysis

This section is devoted to analyzing the stability of the system. We are finding the eigenvalues of the steady states to study the stability. Take (3.2), since the term $D(t)$ is not involved in ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{S}(t)$, ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{I}(t)$, ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{R}(t)$, ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{D}(t)$ considered for analyzing the stability. Also, ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{R}(t)$ is not considered because its population is not known since the total population is not known. It is now obvious to consider ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{S}(t)$ and ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{I}(t)$ for which the steady states and the stability analysis can be done.

$$\begin{aligned} {}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{S}(t) &= (-\beta\tilde{S}(t)\tilde{I}(t) - \gamma\tilde{S}(t)), \\ {}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{I}(t) &= (\beta\tilde{S}(t)\tilde{I}(t - \tau) - (\delta + \gamma)\tilde{I}(t)). \end{aligned} \quad (4.1)$$

Take

$$\lim_{t \rightarrow \infty} [S(t)] = \lim_{t \rightarrow \infty} [S(t - \tau)] = S^*$$

and

$$\lim_{t \rightarrow \infty} [I(t)] = \lim_{t \rightarrow \infty} [I(t - \tau)] = S^*.$$

Now set ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{I}(t) = 0$ to find disease free steady state E_1 along with basic reproduction number of the delayed $I(t)$ and put ${}^{ABC}\Delta_{0,t}^{\alpha_1}\tilde{S}(t) = 0$ to find disease depending steady state E_2 . In previous existing models, when $R_0 = 1$ or $R_0^{Delay} = 1$, $E_1 = E_2$. In this model, We obtain the steady states as $E_1 = (\frac{\delta+\gamma}{\beta}, 0)$ and $E_2 = (0, \frac{-\gamma}{\beta})$. These states infer that the growth of susceptible rate cannot turn out be infective whereas the decrease of infection can nullify the susceptible rate. If the inverse of these existing conditions prevail, these steady states remain to be steady always. In order to claim this, we shall prove the next theorem.

Theorem 4.1. *The steady states remain to be steady always when the inverse of the product of steady-state matrices exists.*

Proof. First let us confirm from the list of parameters given above that $\beta \neq \gamma \neq \delta \neq \psi$. Let us consider E_1 and E_2 in the algebraic form as $E_1(S, I) = (\frac{\delta+\gamma}{\beta}S)$ and $E_2(S, I) = (\frac{-\gamma}{\beta}I)$. Now assume, the determinant of the product of the steady state matrices to be zero so that the inverse of the product of steady state matrices does not exist.

$$|E_1 E_2| = \begin{vmatrix} (\frac{\delta+\gamma}{\beta}) & 0 \\ 0 & \frac{-\gamma}{\beta} \end{vmatrix} = 0. \quad (4.2)$$

It was found from the above determinant, that $|E_1 E_2| = 0$ only when $\delta = \gamma$. But this is a contradiction to our parametric values. so the determinant of the product of steady-state matrices exists and obviously, the inverse of the product of steady-state matrices exists. This implies that these steady states always remain to be steady. \square

4.1. Basic reproduction number

The basic reproduction number is the calculation of the minimum susceptible rate when there is no change in the rate of infection. Though the model is the system of delay differential equations, the basic

reproduction number will be the same as that of the system of ordinary differential equations. Because for $-\tau \leq t_0 \leq 0$, we have $I(0) = I(-\tau)$. At $t=0$ the observations will be made on the rate at which the infectious cases progress and the estimation of this number will be found. When ${}^{ABC}\Delta_{0,t}^{\alpha_2} \tilde{I}(t_0) = 0$,

$$\beta \tilde{S}(t_0) \tilde{I}(t_0) - (\delta + \gamma) \tilde{I}(t_0) = 0, \beta S_0 I_0 - (\delta + \gamma) I_0 = 0, I_0 [\beta S_0 - (\delta + \gamma)] = 0,$$

$$\beta S_0 = \delta + \gamma, S_0 = \frac{\delta + \gamma}{\beta},$$

$R_0 = R_0^{delay} = S_0 \frac{\beta}{\delta + \gamma}$ is the Basic Reproduction number, Where $\frac{\gamma + \delta}{\beta} = S_c$.

When $S_0 < S_c$, the epidemic will come to an end but if $S_0 > S_c$, the disease may propagate again and again until it comes to an end. i.e., there is an epidemic.

4.2. Characteristic equations

Let us find the characteristic equations of the reduced system (4.1). The Jacobian matrix of the system is given by $J = [A_1 + A_2 \cdot e^{-\lambda\tau}]$. Where,

$$A_1 = \begin{bmatrix} -\beta I^* - \gamma & -\beta S^* \\ \beta I^* & -(\delta + \gamma) \end{bmatrix}, \quad (4.3)$$

$$A_2 = \begin{bmatrix} 0 & 0 \\ 0 & \beta S^* \end{bmatrix}. \quad (4.4)$$

Then the Jacobian matrix J is given by

$$J = \begin{bmatrix} -\beta I^* - \gamma & -\beta S^* \\ \beta I^* & -(\delta + \gamma) + \beta S^* e^{-\lambda\tau} \end{bmatrix}. \quad (4.5)$$

4.3. Jacobian matrix at disease free steady state

Now Substitute E_1 at J , We get JE_1 as

$$JE_1 = \begin{bmatrix} -\gamma & -(\delta + \gamma) \\ 0 & -(\delta + \gamma)(1 - e^{-\lambda\tau}) \end{bmatrix}. \quad (4.6)$$

The characteristic equation at the steady state E_1 is $CE_1 = |JE_1 - \lambda I|$ is given by

$$CE_1 = \begin{vmatrix} -(\gamma + \delta) & -(\delta + \gamma) \\ 0 & -(\delta + \gamma)(1 - e^{-\lambda\tau}) - \lambda \end{vmatrix} = 0. \quad (4.7)$$

i.e., $CE_1 = (-\lambda - \gamma)(\lambda\delta + \lambda\gamma - \lambda\delta e^{-\lambda\tau} - \lambda\gamma e^{-\lambda\tau})$

We can conclude that one of the eigenvalue, $\lambda_1(CE_1) = -\gamma$. If other eigenvalues also have negative real parts, then we can confirm that there is no Hopf bifurcation for E_1 .

Consider,

$$-\delta - \gamma + \delta e^{-\lambda\tau} + \gamma e^{-\lambda\tau} - \lambda = 0,$$

$$\lambda = -\delta - \gamma + \delta e^{-\lambda\tau} + \gamma e^{-\lambda\tau},$$

$$= -(\delta + \gamma) + e^{-\lambda\tau}(\lambda + \gamma)$$

$$\lambda = -(\delta + \gamma)[1 - e^{-\lambda\tau}],$$

i.e., $\lambda_2(CE_1)$ may lead to the infinite number of eigenvalues. Now take, $\frac{\lambda}{\delta+\gamma} = e^{-\lambda\tau} - 1$. Now we have to establish that $(e^{-\lambda\tau} - 1)$ lies in the left half of the complex plane. So that we can prove that E_1 is asymptotically stable. Now assume that $\lambda = a + ib$ with a as zero or a positive real number and b as a real number. Then the magnitude of $e^{-\lambda\tau}$ is

$$\begin{aligned} |e^{-\lambda\tau}| &= |e^{-a\tau - ib\tau}| = |e^{-a\tau} e^{-ib\tau}| \\ &= e^{-a\tau} |e^{-ib\tau}| \\ &= e^{-a\tau} |\cos b\tau - i \sin b\tau| \\ &= e^{-a\tau} \sqrt{(\cos^2 b\tau + \sin^2 b\tau)} \\ &= e^{-a\tau} \sqrt{1} \\ |e^{-\lambda\tau}| &= e^{-a\tau}. \end{aligned}$$

Since a is a zero or positive real number, and $\tau > 0$ the following cases will arrive.

- When a is zero, $|e^{-\lambda\tau}|$ is independent of τ will produce $|e^{-\lambda\tau}| = 1$.
- When a is positive, and since τ is always positive, $|e^{-\lambda\tau}| < 1$.

Hence $|e^{-\lambda\tau}| = e^{-a\tau} \leq 1$. Then definitely, $(e^{-\lambda\tau} - 1)$ is the complex number that lies in the left half of the complex plane. i.e., $\frac{(a+ib)}{\delta+\gamma} = e^{-\lambda\tau} - 1$. The right-hand side of the equation is already proved to lie in the left half of the complex plane irrespective of a is zero or positive real number. Suppose if a is the positive real number. i.e., if $a > 0$ then the left-hand side of the equation will be the complex number in the right half of the complex plane. This contradicts the right-hand side. So a cannot be the positive real number. So $\lambda_2(CE_1) = (\delta + \gamma)(e^{-\lambda\tau} - 1)$ leading to infinite eigenvalues are all negative which will never cross from left to right half of the complex plane. Then there is not a Hopf bifurcation. The disease-free steady state E_1 is asymptotically stable regardless of time delay τ .

4.4. Jacobian matrix at disease depending steady state

Now Substitute E_2 at J, We get JE_2 as

$$JE_2 = \begin{bmatrix} 0 & 0 \\ -\gamma & -(\delta + \gamma) \end{bmatrix} = 0. \quad (4.8)$$

The characteristic equation at the steady state E_2 is $CE_2 = |JE_2 - \lambda I|$ is given by

$$CE_2 = \begin{vmatrix} -\lambda & 0 \\ -\gamma & -(\lambda + \delta + \gamma) \end{vmatrix} = 0. \quad (4.9)$$

$CE_2 = \lambda^2 + \lambda\delta + \lambda\gamma = 0$, here also, one of the eigenvalues $\lambda_1(CE_2)$ is negative as $\lambda_1(CE_2) = -(\delta + \gamma)$. Since there is no $e^{-\lambda\tau}$ term $\lambda_2(CE_2)$ will not lead to the infinite eigenvalue. The other eigenvalue $\lambda_2(CE_2)$ is given by $\lambda_2(CE_2) = 0$. So the eigenvalues of disease depending on steady-state E_2 is negative semi-definite. This is called a stable line of equilibrium. In order to prove that the steady-state is stable, even if one of the eigenvalues is zero, we have to prove the following theorem.

Theorem 4.2. *The steady state is stable but not asymptotically stable when $\Delta > 0$, $\text{Det}(E_2) = 0$ and $\text{tr}(E_2) < 0$.*

Proof. From (4.8), It was found that $tr(E_2) = -(\delta + \gamma) < 0$, $Det(E_2) = 0$ and $\Delta \equiv [tr(E_2)]^2 - 4Det(E_2) \equiv [-(\delta + \gamma)]^2 \equiv (\delta + \gamma)^2 > 0$. Hence the steady state E_2 is stable but not asymptotically stable. Since one of the eigenvalues is zero and the other is negative the solutions on the eigenspace are time independent, so obviously independent of time delay. The steady state E_2 is thus having an attractive line of equilibria. Hence the system is thus stable at each steady state independent of time delay. \square

5. Analytical solution-SIRD-delay model

5.1. Application of LADM technique

The analytical estimations can be addressed through the application of LADM of order 4 as that of [13]. Though the system is delay-dependent in I we are not using the time shift property of the Laplace transform. Instead of that, we are treating the delay term $I(t - \tau)$ by using the concept of linear operations. We provide the entire study by taking $\tau = 1$. Because the initial function is unvaried constant for the period of delayed time from $-\tau$ to 0. This method is very direct. i.e., We can take $L[I(t - \tau)]$ with $I(t_0) = c_2$ for $-\tau \leq t_0 \leq 0$. as $L[I(t) + I(-\tau)]$ which becomes $L[I(t)] + L[I(-\tau)]$ which in turn gives $L[I(t)] + L[I(t_0)]$. i.e., $L[I(t)] + L[I_0]$. Suppose the initial functions are the functions of t then this direct linear operation method may not get worked out. Then we must go for the time-shifting property of Laplace transform or else we have to linearize the system and then we have to go for Laplace transform. All the cases can be solved by following procedures.

$$\begin{aligned} S(k+1) &= L^{-1}(-\beta/s^{\alpha_1} \times L(A_k) - \gamma/s^{\alpha_1} \times L(S_k)), \\ I(k+1) &= L^{-1}(\beta/s^{\alpha_2} \times L(A_k + (S_{k+1} \times I_0)) - (\delta + \gamma)/s^{\alpha_2} \times L(I_k)), \\ R(k+1) &= L^{-1}((2\delta - \psi)/s^{\alpha_3} \times L(I_k) - (\delta)/s^{\alpha_3} \times L(R_k)), \\ D(k+1) &= L^{-1}((\psi - \delta)/s^{\alpha_4} \times L(I_k) - (\gamma)/s^{\alpha_4} \times L(D_k)). \end{aligned} \quad (5.1)$$

Where (A_k) is an Adomian polynomial defined by $A_k = \frac{1}{k!} \frac{d^k}{d\lambda^k} (\sum_{l=0}^k (\lambda^l \cdot S_l \lambda^l \cdot I_l))|_{\lambda=0}$, i.e.,

$$A_0 = S_0 I_0,$$

$$A_1 = S_0 I_1 + S_1 I_0,$$

$$A_2 = S_0 I_2 + S_1 I_1 + S_2 I_0 \text{ and so on.}$$

$$S(t) = \sum_{k=0}^{\infty} (S(k)),$$

$$I(t) = \sum_{k=0}^{\infty} (I(k)),$$

$$R(t) = \sum_{k=0}^{\infty} (R(k)),$$

$$D(t) = \sum_{k=0}^{\infty} (D(k)).$$

As it involves, For the model (3.2), the LADM-4 solutions for $(\alpha_1, \alpha_2, \alpha_3, \text{ and } \alpha_4)=1$ are obtained by neglecting the terms t^5 and above.

$$\begin{aligned} \tilde{S}(t) &= (50 - 1.36t + 0.031816t^2 - 0.000838977t^3 + 0.0000213707t^4 + 2.02011 \times 10^{-8}t^5 \\ &\quad + 2.60432 \times 10^{-12}t^6 + 2.1755 \times 10^{-17}t^7), \\ \tilde{I}(t) &= (60 - 4.44t + 0.154488t^2 - 0.00341645t^3 + 0.000049643t^4 + 9.18547 \times 10^{-8}t^5 \\ &\quad + 3.59767 \times 10^{-11}t^6 + 2.94704 \times 10^{-15}t^7 + 1.95795 \times 10^{-20}t^8), \\ \tilde{R}(t) &= (40 + 4.t - 0.2176t^2 + 0.00557035t^3 - 0.0000961808t^4 - 9.74151 \times 10^{-8}t^5 \\ &\quad - 1.52092 \times 10^{-11}t^6 - 2.90067 \times 10^{-16}t^7), \\ \tilde{D}(t) &= (50 - 2.2t + 0.0664t^2 - 0.00147259t^3 + 0.0000244452t^4 + 2.43538 \times 10^{-8}t^5 \\ &\quad + 3.80229 \times 10^{-12}t^6 + 7.25168 \times 10^{-17}t^7), \end{aligned} \quad (5.2)$$

where $0 \leq r \leq 1$. Note that the powers of t do not mean the order of the system. Since we are using (LADM of order 4) we had computed the system up to S_4, I_4, R_4 and D_4 . Their expansion were given in (5.2).

5.2. Application of DTM

The alternate method called DTM-4 is also taken in reference to [13]. The DTM was manifested with the aid of Taylor's expansion for the series. The complete DTM-4 solutions series is about $t = 0$. For $I(t - \tau)$, The same idea of linear operation as applied in (LADM-4) is applied here which was given in detail below. The differential transformation of the function $f(x)$ when $k/\alpha \in Z^+$ can be defined as $D_t(f(x)) = 1/(k/\alpha)! [\frac{d^{k/\alpha} f(x)}{dx^{k/\alpha}}]_{x=0}$. Also If $f(x) = g(x)h(x)$, $F(k) = \sum_{n=0}^k G(n)H(k-n)$. Now for $(\alpha_1, \alpha_2, \alpha_3, \text{ and } \alpha_4)=1$, the system of system of Eq (3.2) is defined as

$$\begin{aligned}\tilde{S}(k+1) &= \frac{1}{k+1}(-\beta \sum_{n=0}^k \tilde{S}(n)\tilde{I}(k-n) - \gamma\tilde{S}(k)), \\ \tilde{I}(k+1) &= \frac{1}{k+1}(\beta \sum_{n=0}^k \tilde{S}(n)\tilde{I}(k-n) + S(k+1)I(t_0) - (\delta + \gamma)\tilde{I}(k)), \\ \tilde{R}(k+1) &= \frac{1}{k+1}(2\delta - \psi)\tilde{I}(k) - \gamma\tilde{R}(k), \\ \tilde{D}(k+1) &= \frac{1}{k+1}(\psi - \delta)\tilde{I}(k) - \gamma\tilde{D}(k),\end{aligned}\tag{5.3}$$

When the initial time is considered as zero, i.e., $t_0 = 0$. The inverse DT of $S(k), I(k), R(k)$ and $D(k)$ are obtained as $S(t) = \sum_{k=0}^{\infty} S(k)t^k$, $I(t) = \sum_{k=0}^{\infty} I(k)t^k$, $R(t) = \sum_{k=0}^{\infty} R(k)t^k$ and $D(t) = \sum_{k=0}^{\infty} D(k)t^k$. For the model (3.2) the DTM-4 solutions are given by

$$\begin{aligned}S(t) &= (50 - 1.36t + 0.0318454t^2 - 0.000850522t^3 + 0.0000219972t^4), \\ I(t) &= (60 - 4.44979t + 0.159861t^2 - 0.00362678t^3 + 0.0000548306t^4), \\ R(t) &= (40 + 4.t - 0.217992t^2 + 0.00571624t^3 - 0.000101117t^4), \\ D(t) &= (50 - 2.2t + 0.0664979t^2 - 0.00150906t^3 + 0.0000256792t^4),\end{aligned}\tag{5.4}$$

where $0 \leq r \leq 1$. Here also we had computed the system up to S_4, I_4, R_4 and D_4 and had gotten the above expansion. But the highest power of t here is 4 because of expansion of (5.3). In general, $S(t), I(t), \tilde{R}(t), D(t) = (0.75 + 0.25r, 1.125 - 0.125r)(S(t), I(t), R(t), D(t))$.

6. Numerical solution of SIRD-Delay model

6.1. Fourth order Runge-Kutta method

In this section the RKM-4 for $(\alpha_1, \alpha_2, \alpha_3, \text{ and } \alpha_4)=1$ is applied to get the desired solution. Estimating *SIRD* with delay at $h = 0.1$ is considered to get the better result over $0 \leq r \leq 1$.

Evaluation:

$$\begin{aligned}\tilde{S}(t+1) &= (\tilde{S}(t) + (1/6(K_1 + 2K_2 + 2K_3 + K_4))), \\ \tilde{I}(t+1) &= (\tilde{I}(t) + (1/6(L_1 + 2L_2 + 2L_3 + L_4))), \\ \tilde{R}(t+1) &= (\tilde{R}(t) + (1/6(M_1 + 2M_2 + 2M_3 + M_4))), \\ \tilde{D}(t+1) &= (\tilde{D}(t) + (1/6(N_1 + 2N_2 + 2N_3 + N_4))).\end{aligned}\tag{6.1}$$

To estimate (6.1), consider the following.

$L_m = L_{m-1}$ for $m = 1$ and $I(t) = I(t - 1)$ for $t = 0$.

$$\begin{aligned}
 \tilde{K}(m)_1 &= h(-\beta(\tilde{S}(t))(\tilde{I}(t)) - \gamma(\tilde{S}(t))) \\
 \tilde{L}(m)_1 &= h(\beta(\tilde{S}(t))(\tilde{I}(t-1)) - (\delta + \gamma)(\tilde{I}(t))) \\
 \tilde{M}(m)_1 &= h((2\delta - \psi)(\tilde{I}(t)) - (\gamma)\tilde{R}(t)) \\
 \tilde{N}(m)_1 &= h((\psi - \delta)(\tilde{I}(t)) - (\gamma)\tilde{D}(t)) \\
 \tilde{K}(m)_2 &= h(-\beta(\tilde{S}(t) + (\tilde{K}(m)_1/2))(\tilde{I}(t) + (\tilde{L}(m)_1/2)) \\
 &\quad - (\gamma(\tilde{S}(t) + (\tilde{K}(m)_1/2)))) \\
 \tilde{L}(m)_2 &= h(\beta(\tilde{S}(t) + (\tilde{K}(m)_1/2))(I(t-1) \\
 &\quad + (\tilde{L}(m-1)_1/2)) - ((\delta + \gamma)(\tilde{I}(t) + (\tilde{L}(m)_1/2)))) \\
 \tilde{M}(m)_2 &= h((2\delta - \psi)(\tilde{I}(t) + (\tilde{L}(m-1)_1/2)) \\
 &\quad - ((\gamma)(\tilde{R}(t) + (\tilde{M}(m)_1/2)))) \\
 \tilde{N}(m)_2 &= h((\psi - \delta)(\tilde{I}(t) + (\tilde{L}(m)_1/2)) - \\
 &\quad ((\gamma)(\tilde{D}(t) + (\tilde{N}(m)_1/2)))) \\
 \tilde{K}(m)_3 &= h(-\beta(\tilde{S}(t) + (\tilde{K}(m)_2/2))(\tilde{I}(t) + (\tilde{L}(m)_2/2)) \\
 &\quad - (\gamma(\tilde{S}(t) + (\tilde{K}(m)_2/2)))) \\
 \tilde{L}(m)_3 &= h(\beta \times (\tilde{S}(t) + (\tilde{K}(m)_2/2))(\tilde{I}(t-1) \\
 &\quad + (\tilde{L}(m-1)_2/2)) - ((\delta + \gamma)(\tilde{I}(t) + (\tilde{L}(m)_2/2)))) \\
 \tilde{M}(m)_3 &= h((2\delta - \psi)(\tilde{I}(t) + (\tilde{L}(m)_2/2)) \\
 &\quad - ((\gamma)(\tilde{R}(t) + (\tilde{M}(m)_2/2)))) \\
 \tilde{N}(m)_3 &= h((\psi - \delta)(\tilde{I}(t) + (\tilde{L}(m)_2/2)) - \\
 &\quad ((\gamma)(\tilde{D}(t) + (\tilde{N}(m)_2/2)))) \\
 \tilde{K}(m)_4 &= h(-\beta(\tilde{S}(t) + (\tilde{K}(m)_3))(\tilde{I}(t) + (\tilde{L}(m)_3)) \\
 &\quad - (\gamma(\tilde{S}(t) + \tilde{K}(m)_3))) \\
 \tilde{L}(m)_4 &= h(\beta(\tilde{S}(t) + (\tilde{K}(m)_3))(\tilde{I}(t-1) \\
 &\quad + (\tilde{L}(m-1)_3)) - ((\delta + \gamma)(\tilde{I}(t) + \tilde{L}(m)_3))) \\
 \tilde{M}(m)_4 &= h((2\delta - \psi)(\tilde{I}(t) + (\tilde{L}(m)_3)) \\
 &\quad - (\gamma)((\tilde{R}(t) + \tilde{M}(m)_3))) \\
 \tilde{N}(m)_4 &= h((\psi - \delta)(\tilde{I}(t) + (\tilde{L}(m)_3)) - \\
 &\quad (\gamma)((\tilde{D}(t) + \tilde{N}(m)_3))).
 \end{aligned} \tag{6.2}$$

For $p \in [1, 4]$ and $r \in [0, 4]$,

$$\tilde{K}_p = \tilde{K}_p(t; r) = [\underline{K}_p(t; r), \overline{K}_p(t; r)],$$

$$\tilde{L}_p = \tilde{L}_p(t; r) = [\underline{L}_p(t; r), \overline{L}_p(t; r)],$$

$$\tilde{M}_p = \tilde{M}_p(t; r) = [\underline{M}_p(t; r), \overline{M}_p(t; r)],$$

$$\tilde{N}_p = \tilde{N}_p(t; r) = [\underline{N}_p(t; r), \overline{N}_p(t; r)].$$

For $t \in [0, n]$, $n = 1, 2, 3, \dots$,

and for $q = t, \bar{q} = t + 1, t = 0, 1, 2, 3, \dots$,

$$\tilde{S}(q) = \tilde{S}(q)(t; r) = [\underline{S}_q(t; r), \overline{S}_q(t; r)],$$

$$\tilde{I}(q) = \tilde{I}(q)(t; r) = [\underline{I}_q(t; r), \overline{I}_q(t; r)],$$

$$\tilde{R}(q) = \tilde{R}(q)(t; r) = [\underline{R}_q(t; r), \overline{R}_q(t; r)],$$

$$\tilde{D}(q) = \tilde{D}(q)(t; r) = [\underline{D}_q(t; r), \overline{D}_q(t; r)].$$

Where $[\underline{f}(t; r), \overline{f}(t; r)] = [0.75 + 0.25r, 1.125 - 0.125r]f(t)$.

7. Graphical representations

The associationship among $SIRD$ -delay at $\alpha_i = 1, i = 1, 2, 3, 4$ for $t \in [0, 1000]$ for the fuzzy valued model (3.2) is given in figure.

For the various values of $t, \alpha_1, \alpha_2, \alpha_3, \alpha_4$ and $r \in [0, 1]$ the following tables are given. In Tables 1–4 the susceptible, infected, recovered and dead population solutions are calculated using two various methods, say, LADM-4, DTM-4 and after that by RKM-4, all these methods are compared. Figure 2 is plotted by taking $S(t), I(t), R(t)$ and $D(t)$ for $\alpha_i = 1, i = 1, 2, 3, 4, t \in [0, 300]$ and $r = 1$. Figure 3 is plotted by taking $S(t), I(t), R(t)$ and $D(t)$ for $\alpha_i = 1, i = 1, 2, 3, 4, t \in [0, 3]$ and $r = 1$. In Tables 5 and 6, susceptible population values for $\alpha \in [0, 1]$ and $t \in [0, 1]$ are given and the respective plot is given in Figure 4. In Tables 7 and 8, infected population values for $\alpha \in [0, 1]$ and $t \in [0, 1]$ are given and the respective plot is given in Figure 5. In Tables 9 and 10, recovered population values for $\alpha \in [0, 1]$ and $t \in [0, 1]$ are given and the respective plot is given in Figure 6. In Tables 11 and 12, dead population values for $\alpha \in [0, 1]$ and $t \in [0, 1]$ are given and their respective plot is given in Figure 7. Figure 8 is given for $S(t), I(t), R(t), D(t)$ for $t \in [0, 1], \alpha \in [0, 1]$ and $r = 1$. After solving (3.1) for $\alpha \in [0, 1]$ the values are calculated for $t \in [0, 1]$ and $r \in [0, 1]$ in (3.2). As a sample, fixing $t = 1, \alpha_i = 1, i = 1, 2, 3, 4$ and considering $r \in [0, 1]$ fuzzy valued solutions of susceptible, infected, recovered and dead populations are given in Table 13. Similarly we found the remaining values in $t \in [0, 1]$ with $\alpha_i, i = 1, 2, 3, 4$ for $r \in [0, 1]$ and other plots are obtained. Figures 9–12 are given for $S(t), I(t), R(t), D(t)$ for $t \in [0, 1], \alpha \in [0, 1]$, and $r \in [0, 1]$ which are the fuzzy valued plots.

Table 1. The susceptible cases.

t	LADM-4	DTM-4	RKM-4
0	50	50	50
0.1	49.8643173	49.8643176	49.8643173
0.2	49.7292659	49.7292670	49.7289045
0.3	49.5948409	49.594843	49.5941225
0.4	49.4610374	49.4610413	49.4599662
0.5	49.3278504	49.327856	49.3264308
0.6	49.1952753	49.1952834	49.1935116
0.7	49.0633072	49.0633177	49.0612030
0.8	48.9319414	48.9319545	48.9295007
0.9	48.8011733	48.8011891	48.7983997
1.0	48.6709984	48.6710168	48.6678956

Table 2. The infected cases.

t	LADM-4	DTM-4	RKM-4
0	60	60	60
0.1	59.5575414	59.5566157	59.5575902
0.2	59.1181522	59.1164071	59.1187057
0.3	58.6818120	58.6793524	58.6825032
0.4	58.2485007	58.2454302	58.2503295
0.5	57.8181980	57.8146193	57.8207953
0.6	57.3908841	57.3868984	57.3943356
0.7	56.9665392	56.9622466	56.9709296
0.8	56.5451434	56.5406429	56.5505565
0.9	56.1266773	56.1220666	55.1331958
1	55.71112128	55.70649	55.7188268

Table 3. The recovered cases.

t	LADM	DTM	RKM-4
0	40	40	40
0.1	40.3978295	40.3978257	40.3978296
0.2	40.7913404	40.791325	40.7913470
0.3	41.1805656	41.1805342	41.1805831
0.4	41.5655380	41.5654845	41.5655715
0.5	41.9462902	41.9462102	41.9474980
0.6	42.3228547	42.3227446	42.3240883
0.7	42.6952635	42.6951204	42.6965300
0.8	43.0635485	43.0633706	43.0648559
0.9	43.4277416	43.4275275	43.4290983
1	43.7878740	43.7876234	43.7892893

Table 4. The death cases.

t	LADM-4	DTM-4	RKM-4
0	50	50	50
0.1	49.7806625	49.7806634	49.7806624
0.2	49.5626442	49.5626478	49.5626425
0.3	49.3459364	49.3459442	49.3459320
0.4	49.1305303	49.1305437	49.1305220
0.5	48.9164174	48.9164374	48.9161155
0.6	48.7035890	48.7036166	48.7032806
0.7	48.4920367	48.4920725	48.4917201
0.8	48.2817520	48.2817965	48.2814252
0.9	48.0727265	48.0727800	48.0723873
1	47.8649518	47.8650145	47.8645980

Table 5. The susceptible cases.

$\alpha \setminus t$	0	0.1	0.2	0.3	0.4	0.5
0	48.6997	48.6997	48.6997	48.6997	48.6997	48.6997
0.1	50	48.9059	48.8304	48.784	48.7499	48.7229
0.2	50	49.0928	48.9624	48.8778	48.8139	48.7619
0.3	50	49.2578	49.0911	48.9771	48.888	48.8138
0.4	50	49.4004	49.2131	49.0782	48.9691	48.8759
0.5	50	49.521	49.3261	49.178	49.0541	48.9455
0.6	50	49.6213	49.4287	49.2742	9.1404	49.0202
0.7	50	49.7034	49.5202	49.365	49.2258	49.0976
0.8	50	49.7697	49.6004	49.4491	49.3086	49.1757
0.9	50	49.8226	49.6699	49.5258	49.3873	49.2529
1	50	49.8643	49.7293	49.5948	49.461	49.3279

Table 6. The susceptible cases.

$\alpha \setminus t$	0.6	0.7	0.8	0.9	1
0	48.6997	48.6997	48.6997	48.6997	48.6997
0.1	48.7004	48.6811	48.6641	48.649	48.6353
0.2	48.7178	48.6793	48.6451	48.6142	48.586
0.3	48.7496	48.6928	48.6416	48.5948	48.5516
0.4	48.7938	48.7199	48.6523	48.5899	48.5317
0.5	48.848	48.7587	48.676	48.5987	48.5258
0.6	48.91	48.8074	48.7111	48.6199	48.5331
0.7	48.9776	48.8641	48.7559	48.6522	48.5524
0.8	49.0488	48.9268	48.8088	48.6942	48.5828
0.9	49.1219	48.9937	48.868	48.7444	48.6228
1	49.1953	49.0633	48.9319	48.8012	48.671

Table 7. The infected cases.

$\alpha \backslash t$	0	0.1	0.2	0.3	0.4	0.5
0	55.8492	55.8492	55.8492	55.8492	55.8492	55.8492
0.1	60	56.494	56.2569	56.1114	56.0049	55.9205
0.2	60	57.0818	56.6695	56.4033	56.2024	56.0394
0.3	60	57.6047	57.0741	56.7133	56.4321	56.1985
0.4	60	58.0592	57.4601	57.0307	56.6846	56.3901
0.5	60	58.4461	57.8199	57.3461	56.9511	56.6065
0.6	60	58.7693	58.1481	57.6517	57.2234	56.8402
0.7	60	59.0349	58.442	57.9417	57.4946	57.0841
0.8	60	59.2499	58.7008	58.2116	57.7588	57.332
0.9	60	59.4217	58.9256	58.4588	58.0112	57.5782
1	60	59.5575	59.1182	58.6818	58.2485	57.8182

Table 8. The infected cases.

$\alpha \backslash t$	0.6	0.7	0.8	0.9	1
0	55.8492	55.8492	55.8492	55.8492	55.8492
0.1	55.8502	55.79	55.7371	55.69	55.6474
0.2	55.9014	55.7812	55.6744	55.5781	55.4903
0.3	55.997	55.8189	55.6586	55.5124	55.3779
0.4	56.1312	55.8987	55.6867	55.4912	55.3094
0.5	56.2975	56.0157	55.7552	55.5122	55.2838
0.6	56.4897	56.1646	55.86	55.5724	55.2992
0.7	56.7011	56.3398	55.9964	55.6682	55.3531
0.8	56.9255	56.5355	56.1595	55.7956	55.4422
0.9	57.1572	56.7462	56.3441	55.9499	55.5629
1	57.3909	56.9665	56.5451	56.1267	55.7111

Table 9. The recovered cases.

$\alpha \backslash t$	0	0.1	0.2	0.3	0.4	0.5
0	43.5958	43.5958	43.5958	43.5958	43.5958	43.5958
0.1	40	43.0583	43.2575	43.3794	43.4682	43.5386
0.2	40	42.5625	42.9134	43.1384	43.3074	43.444
0.3	40	42.1157	42.5725	42.8806	43.1192	43.3165
0.4	40	41.7226	42.2435	42.6138	42.9101	43.1609
0.5	40	41.3846	41.9337	42.3456	42.6867	42.9825
0.6	40	41.0999	41.6482	42.083	42.4556	42.7872
0.7	40	40.8645	41.3906	41.8315	42.223	42.5806
0.8	40	40.6731	41.1622	41.5955	41.9943	42.3683
0.9	40	40.5195	40.9628	41.3779	41.774	42.1554
1.0	40	40.3978	40.7913	41.1806	41.5655	41.9463

Table 10. The recovered cases.

$\alpha \setminus t$	0.6	0.7	0.8	0.9	1
0	43.5958	43.5958	43.5958	43.5958	43.5958
0.1	43.597	43.647	43.6908	43.7299	43.7651
0.2	43.5594	43.6596	43.7483	43.8282	43.9009
0.3	43.486	43.6353	43.7691	43.8908	44.0025
0.4	43.3803	43.5764	43.7545	43.918	44.0697
0.5	43.2463	43.4858	43.7061	43.9107	44.1023
0.6	43.0887	43.3669	43.6264	43.8703	44.1009
0.7	42.9125	43.2239	43.5185	43.7987	44.0665
0.8	42.7228	43.0612	43.386	43.6988	44.0012
0.9	42.5247	42.8834	43.2329	43.5739	43.9073
1.0	42.3229	42.6953	43.0635	43.4277	43.7879

Table 11. The dead cases.

$\alpha \setminus t$	0	0.1	0.2	0.3	0.4	0.5
0	47.9246	47.9246	47.9246	47.9246	47.9246	47.9246
0.1	50	48.2497	48.1304	48.0571	48.0034	47.9608
0.2	50	48.5454	48.3384	48.2046	48.1035	48.0214
0.3	50	48.8076	48.54	48.361	48.2197	48.1023
0.4	50	49.035	48.7357	48.5207	48.3473	48.1994
0.5	50	49.2281	48.9158	48.6791	48.4815	48.3088
0.6	50	49.389	49.0798	48.8322	48.6183	48.4266
0.7	50	49.5211	49.2263	48.9771	48.7542	48.5492
0.8	50	49.628	49.3552	49.1118	48.8863	48.6734
0.9	50	49.7133	49.467	49.235	49.0123	48.7966
1	50	49.7807	49.5626	49.3459	49.1305	48.9164

Table 12. The dead cases.

$\alpha \setminus t$	0.6	0.7	0.8	0.9	1
0	47.9246	47.9246	47.9246	47.9246	47.9246
0.1	47.9254	47.8949	47.8683	47.8445	47.823
0.2	47.9518	47.8912	47.8373	47.7887	47.7444
0.3	48.0008	47.9111	47.8303	47.7566	47.6887
0.4	48.0693	47.9523	47.8456	47.7471	47.6555
0.5	48.1538	48.0123	47.8813	47.759	47.644
0.6	48.251	48.088	47.9352	47.7907	47.6533
0.7	48.3576	48.1768	48.0047	47.8401	47.6818
0.8	48.4705	48.2756	48.0874	47.9051	47.7279
0.9	48.5867	48.3815	48.1806	47.9834	47.7897
1	48.7036	48.492	48.2818	48.0727	47.865

Table 13. Fuzzy fractional epidemic model SIRD-delay.

r	S		I		R		D	
	mini	maxi	mini	maxi	mini	maxi	mini	maxi
0	36.5247	54.7871	41.8869	62.8303	32.6969	49.0453	35.9434	53.9151
0.1	37.6924	54.1068	43.1268	61.9078	33.918	48.6887	37.0628	53.2031
0.2	38.8688	53.4446	44.3923	61.0394	35.1207	48.2909	38.1955	52.5188
0.3	40.0551	52.7999	45.6867	60.2234	36.3021	47.8527	39.3431	51.8614
0.4	41.252	52.1716	47.013	59.4576	37.4592	47.3749	40.5071	51.2296
0.5	42.4601	51.5587	48.3734	58.7391	38.5895	46.8587	41.6885	50.6218
0.6	43.6798	50.9597	49.7693	58.0642	39.6908	46.3059	42.8879	50.0359
0.7	44.911	50.3732	51.2016	57.4288	40.7616	45.719	44.1057	49.4699
0.8	46.1536	49.7973	52.6701	56.8283	41.8011	45.1012	45.3415	48.9211
0.9	47.4072	49.2305	54.1739	56.2575	42.8096	44.4561	46.5949	48.3871
1.0	48.671	48.671	55.7111	55.7111	43.7879	43.7879	47.865	47.865

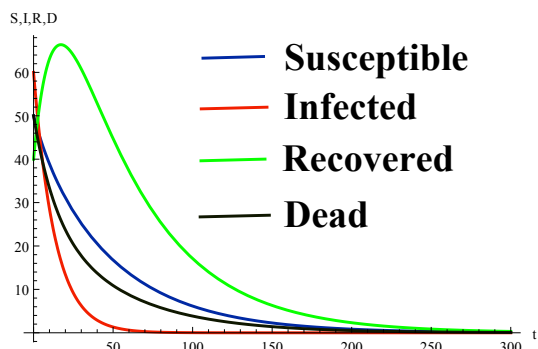


Figure 2. Fuzzy-fractional model for $t \in [0, 300]$.

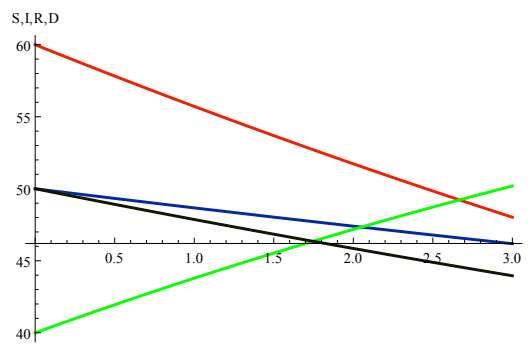


Figure 3. Fuzzy-fractional model for $t \in [0, 3]$.

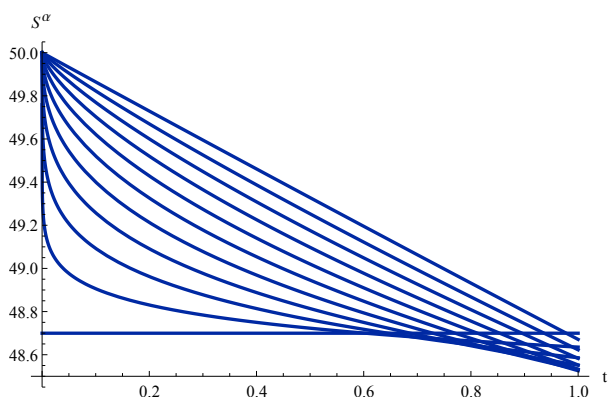


Figure 4. Fractional $S(t)$.

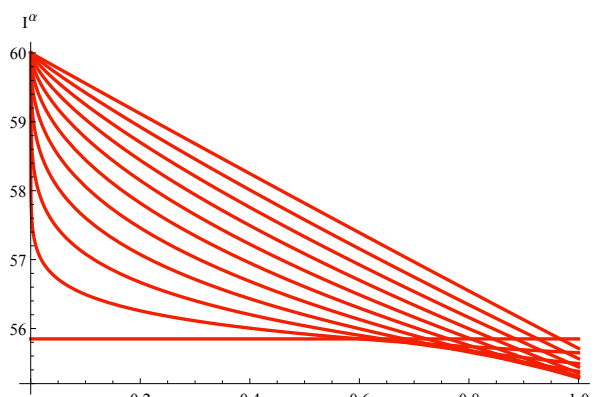


Figure 5. Fractional $I(t)$.

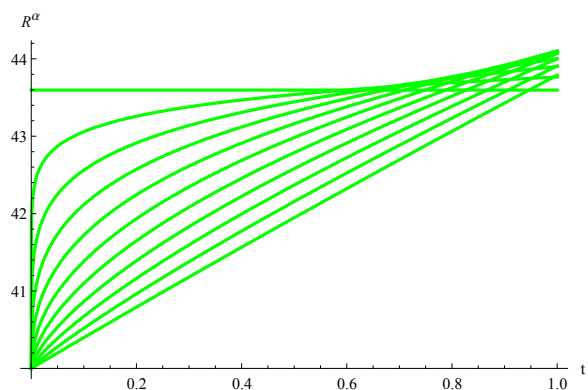


Figure 6. Fractional $R(t)$.

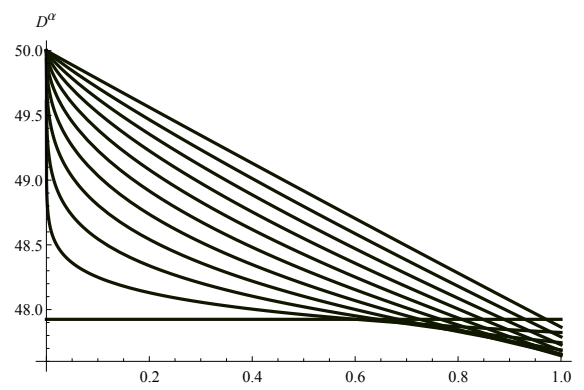


Figure 7. Fractional $D(t)$.

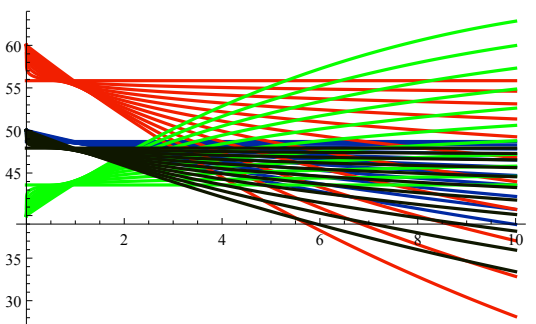


Figure 8. Fractional SIRD for $t \in [0, 1]$.

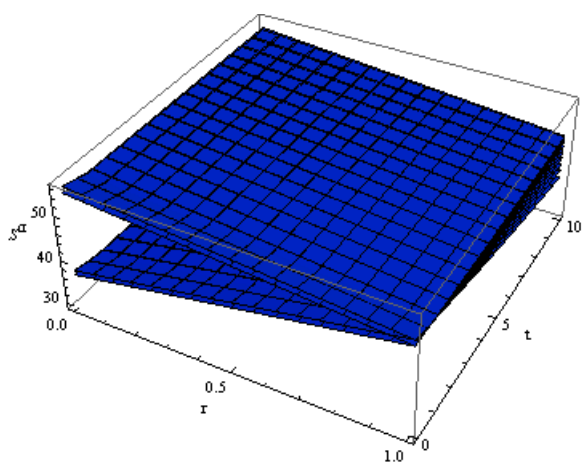


Figure 9. Fuzzy Fractional $S(t)$.

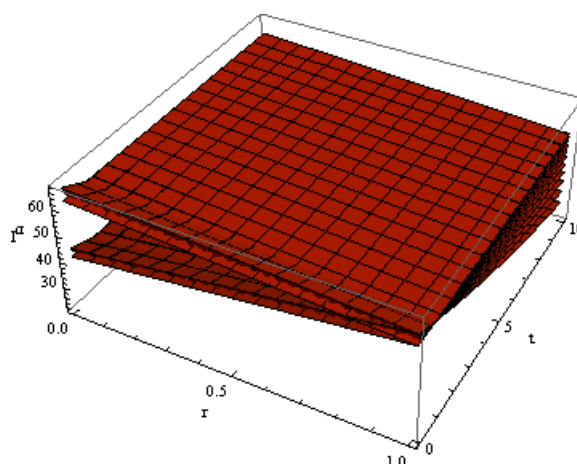


Figure 10. Fuzzy fractional $I(t)$.

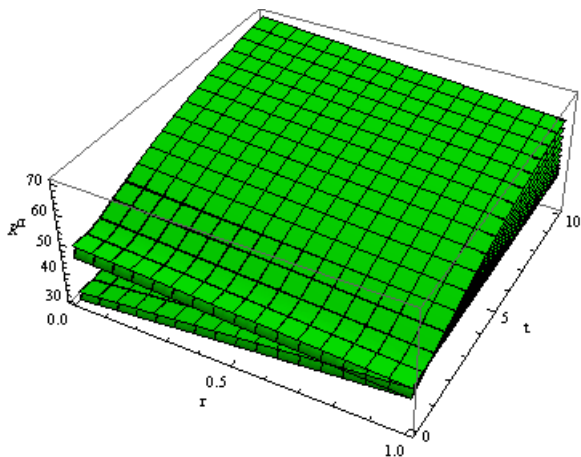


Figure 11. Fuzzy fractional $R(t)$.

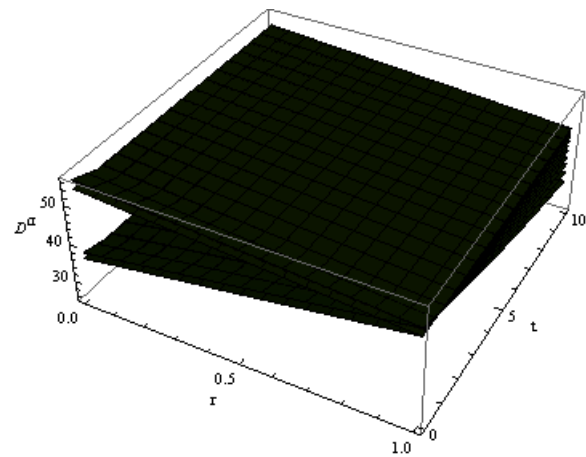


Figure 12. Fuzzy fractional $D(t)$.

From Tables 1–4, we can confirm the solutions are correct at least to one decimal place of accuracy by comparison of LADM-4, DTM-4, and RKM-4 methods.

Implications of Figures 2, 3 and 7

- (1) The model we have shown here is not the cumulative case study. It is the daily change in the SIRD cases due to the epidemic's spread.
- (2) If it is cumulative the graph of death will be fixed at a particular count and will not change, especially decreased with time.
- (3) If it is cumulative, the curve of D will not decrease since rebirth is physically impossible and it will increase or otherwise it is stable at a particular count if there is no new death.
- (4) The model we considered cannot be taken as closed since there is a change in death.
- (5) The model does not imply the death of people who died naturally due to age, accident, disaster, etc., it is only implying the death due to the epidemic spread. So decrease of curve D implies only that when time increases the daily new death count decreases.
- (6) The curve of S, I, D decreases but R increases for some t , and after that it also gets reduced. The curve I is not hiking and continuously reducing because the model we considered is a delay model having $I(t - \tau)$. At initial time $I(t)$ is the same as the $I(t - \tau)$. When there is a decrease in daily new susceptible, it not only means the existing susceptible went to the compartment I , being susceptible the people might be gone out of S due to recovering from the symptoms which is also the reason for the decrease of I , since S is decreasing and by which I is also decreasing, initially there is a hike in the curve of R . After a certain time, R is also decreased since there is a decrease in I .
- (7) In addition, the curves of D are more dominated by ψ than γ .
- (8) All the curves decrease as time increases because it is a daily case study and not a cumulative case study. After the time $t \geq 300$ S, I, R, D become zero means at time $t \geq 300^{th} day$, there is no new

susceptible, new infected, new recovered and new dead since we are studying only daily case study and not cumulative study.

- (9) Since the solutions are derived using the LADM technique they are not cumulative. Because in cumulative only the increase of time will increase at least one of the compartment cases mostly, R . But here we are addressing only a daily case study in which an increase of time need not imply an increase of at least a compartment case.
- (10) Due to the above implications, the reason for the decrease of D curves as shown in Figure 6 is also justified.

8. Conclusions

The system of the single retarded delay fuzzy fractional epidemic model under ABC derivative was presented. To study its stability the system was reduced to two steady states such as disease-free and disease dependent since if the steady states are stable then obviously the complete system is stable. The basic reproduction number was found and stability analysis is done for both the steady states. We proved that the steady states always remain to be steady, by a theorem. For a negative semi-definite eigenvalue, we proved the theorem to claim that the system is stable. From our study steady-state 1 is asymptotically stable and steady-state 2 is stable and both are not affected by the delay. so no Hopf bifurcation will occur. For $\alpha_i \in [0, 1], i = 1, 2, 3, 4$, Fuzzy valued $S(t), I(t), R(t)$ and $D(t)$ at $t \in [0, 1], r \in [0, 1]$ are shown in the tables and plots. Traditionally two numerical solutions will be compared with one analytical solution but we differently compared two analytical solutions with that of one numerical solution. Because the analytical solutions seem to coincide by only one decimal place of accuracy. In order to confirm the accuracy of the solution, we considered RKM-4 which is very direct. All these three methods were compared at $\alpha_i = 1$. The solutions by LADM, DTM and RKM are equally well-matched in accuracy up to one decimal place. But RKM-4 is considered as a quick as well as a direct solution in case of the delayed epidemic model. We are solving the delay term by means of linear operation instead of the time-shift property of Laplace transform in both LADM and in DTM. So one can get a doubt about the correctness of the solutions. That's why we are comparing LADM and DTM solutions with that of RKM-4 solutions which is very direct involving neither a transformation nor a linearization to confirm the correctness of the solutions. Also, we have to keep in mind that to solve the fuzzy ordinary epidemic model with a single retarded delay one can use RKM-4, and to solve the fuzzy fractional-order epidemic model with a single retarded delay one can use LADM-4 as well as DTM-4. The limitation of the model is the value of the delay term that could not be extended beyond the value used in the initial condition. In the future the authors would like to do the research on this retard delay epidemic model to predict the daily new cases.

Conflict of interest

The authors declare no conflict of interest.

References

1. L. A. Zadeh, Fuzzy sets, *Inf. Contr.*, **8** (1965), 338–353. [https://doi.org/10.1016/S0019-9958\(65\)90241-X](https://doi.org/10.1016/S0019-9958(65)90241-X)
2. J. J. Buckley, T. Feuring, Fuzzy differential equations, *Fuzzy Set. Syst.*, **110** (2000), 43–54. [https://doi.org/10.1016/S0019-9958\(65\)90241-X](https://doi.org/10.1016/S0019-9958(65)90241-X)
3. S. Abbasbandy, Extended Newton's method for a system of nonlinear equations by modified Adomian decomposition method, *Appl. Math. Comput.*, **170** (2005), 648–656. <https://doi.org/10.1016/j.amc.2004.12.048>
4. L. G. S. Allen, *Introduction to mathematical biology*, Prentice Hall, 2007.
5. P. B. Dhandapani, D. Baleanu, J. Thippan, V. Sivakumar, Fuzzy type RK4 solutions to fuzzy hybrid retarded delay differential equations, *Front. Phys.*, **7** (2019), 1–6. <https://doi.org/10.3389/fphy.2019.00168>
6. O. D. Makinde, Adomian decomposition approach to a SIR epidemic model with constant vaccination strategy, *Appl. Math. Comput.*, **184** (2007), 842–848. <https://doi.org/10.1016/j.amc.2006.06.074>
7. M. Y. Ongun, The Laplace Adomian decomposition method for solving a model for HIV infection of $CD4^+T$ cells, *Math. Comput. Model.*, **53** (2007), 597–603.
8. A. A. M. Arafa, S. J. Rida, M. Khalil, Solutions of the fractional order model of childhood disease with constant vaccination strategy, *Math. Sci. Lett.*, **1** (2012), 17–23. <https://doi.org/10.12785/msl/010103>
9. A. Antangana, D. Baleanu, New fractional derivative with non-local and non-singular kernel theory and application to heat transfer model, *Therm. Sci.*, **20** (2016), 763–769. <https://doi.org/10.2298/TSCI160111018A>
10. A. L. Aliyu, A. S. Alshomrani, Y. Li, Existence theory and numerical simulation of HIV-1 cure model with new fractional derivative possessing a non-singular kernel, *Adv. Diff. Equ.*, 2019, 1–17. <https://doi.org/10.1186/s13662-019-2336-5>
11. M. Caputo, M. Fabrizio, A new definition of fractional derivative without singular kernel, *Prog. Fract. Differ. Appl.*, **1** (2015), 73–85.
12. P. B. Dhandapani, D. Baleanu, J. Thippan, V. Sivakumar, New fuzzy fractional epidemic model involving death population, *Comput. Syst. Sci. Eng.*, **37** (2021), 331–346. <https://doi.org/10.32604/csse.2021.015619>
13. M. Farman, M. U. Saleem, A. Ahmed, M. O. Ahamed, Analysis and numerical solution of SEIR epidemic model of measles with non-integer time fractional derivatives by using Laplace Adomian decomposition method, *Ain Shams Eng. J.*, 2018, 3391–3397. <https://doi.org/10.1016/j.asej.2017.11.010>
14. M. El-Shahed, A. Alsacdi, The fractional SIRC model and influenza A, *Math. Prob. Eng.*, 2011, 1–9. <https://doi.org/10.1155/2011/480378>
15. P. Palese, J. F. Young, Variation of influenza A, B, and C, *Science*, **215** (1982), 1468–1474. <https://doi.org/10.1126/science.7038875>

16. P. B. Dhandapani, D. Baleanu, J. Thippan, V. Sivakumar, On stiff fuzzy IRD-14 day average transmission model of COVID-19 pandemic disease, *AIMS Bioeng.*, **7** (2020), 208–223. <https://doi.org/10.3934/bioeng.2020018>
17. X. Zhang, D. Jiang, T. Hayat, B. Ahmad, Dynamics of a stochastic SIS model with double epidemic diseases driven by Levy jumps, *Physica A*, **471** (2017), 767–777. <https://doi.org/10.1016/j.physa.2016.12.074>
18. G. Zaman, Y. Kang, I. H. Jung, Stability analysis and optimal vaccination of an SIR epidemic model, *Biosystems*, **93** (2017), 240–249. <https://doi.org/10.1016/j.physa.2016.12.074>
19. W. O. Kermack, A. G. Mckendrick, Contribution to the mathematical theory of epidemics, *Proc. Roy. Soc. Lond. A*, **115** (1927), 700–721. <https://doi.org/10.1098/rspa.1927.0118>
20. W. O. Kermack, A. G. Mckendrick, Contribution to the mathematical theory of epidemic—II. The problem of endemicity, *Proc. Roy. Soc. Lond. A*, **138** (1932), 55–83. <https://doi.org/10.1098/rspa.1932.0171>
21. P. Singh, A. Gupta, Generalized SIR (GSIR) epidemic model: An improved framework for the predictive monitoring of COVID-19 pandemic, *ISA T.*, 2021, In press.
22. P. Singh, A. Singhal, B. Fatimah, A. Gupta, An improved data driven dynamic SIRD model for predictive monitoring of COVID-19, *IEEE ICASSP*, 2021, 8158–8162. <https://doi.org/10.1109/ICASSP39728.2021.9414762>



AIMS Press

©2022 the Author(s), licensee AIMS Press. This is an open access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>)