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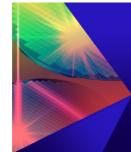
Evolutionary computational method for tuberculosis model with fuzziness

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ABSTRACT

This work investigates the computational study of a six-compartmental mathematical model of tuberculosis disease dynamics with the impact of vaccination. Traditional mathematical models presume that all variables are precise and can be measured or calculated precisely. However, in many real-world scenarios, variables may need to be more accurate or easier to quantify, resulting in model uncertainty. Considering this, fuzziness is introduced into the model by taking the contact, recovery, and death rates due to disease as fuzzy membership functions. Two numerical computational schemes, forward Euler and nonstandard finite difference (NSFD), are designed to solve the model. The positivity and convergence for the developed method are investigated, which are significant characteristics of these dynamical models, and it is revealed that these features are preserved in the extended scheme. Numerical computations are performed to support the analytical results. The numerical and computational results indicate that the proposed NSFD method adequately represents the dynamics of the disease despite the uncertainty and heterogeneity. Moreover, the obtained method generates plausible predictions that regulators can use to design and develop control strategies to support decision-making.

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I. INTRODUCTION

The bacterium Mycobacterium tuberculosis causes tuberculosis (TB). It is a significant public health problem worldwide, especially in developing countries. TB typically affects the lungs; however, it can also affect other parts of the body. TB's primary transmission mode is inhaling airborne droplets containing the bacteria. An infected person can spread bacteria into the air by coughing, sneezing, speaking, or singing. These droplets can be inhaled by other people nearby, and if they reach the lungs, the bacteria can

cause TB infection. Although tuberculosis can be fatal, it is frequently prevented and cured. TB affects one-quarter of the world's population, which means they have the germs but have not yet developed symptoms and cannot spread the disease. The bacterium that causes tuberculosis spreads when an infected person coughs or sneezes. Most people with the bacteria that causes tuberculosis do not exhibit any symptoms. When symptoms manifest, they frequently include fever, weight loss, nocturnal sweats, and a cough that can be bloody.¹⁻⁴ TB transmission is more likely in crowded and poorly ventilated settings, such as prisons, homeless shelters, and refugee camps. Close and prolonged contact with an infected person increases the risk of transmission. People with compromised immune systems, such as HIV infection or malnutrition, are also at higher risk of TB infection and disease. Despite having tuberculosis bacteria in their bodies, a person may not exhibit any symptoms. The immune systems of most humans can contain bacteria, stopping them from spreading and causing illness. In this situation, a person will have a TB infection but not an active disease. Latent TB is the name for this condition. It may be more challenging for the body to keep the TB bacteria under control. This is more likely to happen when the immune system is compromised by illness or the use of specific drugs. When this happens, the bacteria may grow and produce symptoms, which could result in active tuberculosis. Those who have active tuberculosis can infect others with the disease. People who do not exhibit any symptoms typically do not need treatment. A lengthy antibiotic treatment involving several drugs will be necessary for active symptoms. It continues to be a worldwide health concern due to its high mortality and is one of the top causes of death in most sub-Saharan African nations.

Mathematical models are frequently used to improve understanding infectious disease transmission and prevention. In epidemiology, mathematical modeling has become a valuable tool for understanding disease transmission dynamics, predicting the impact of interventions, and developing control strategies. Mathematical equations are used in mathematical models to describe how infectious diseases propagate through a population. Simple compartmental models to more complex network models that incorporate individual-level behavior and contact patterns are examples of these models. Compartmental models categorize the population as susceptible, infected, recovered, etc. The models track the movement of individuals between these compartments based on the rate of transmission, recovery, and other parameters. Mathematical modeling has been extensively used to study TB's transmission dynamics and develop disease control strategies. Yang et al. examined two TB models with insufficient care.⁵ After deriving the basic reproduction numbers for each, a study of the two models' intuitive epidemiological interpretations is done. There is also a discussion of certain methods to stop the spread of TB. Okuonghae proposed a mathematical model that distinguishes susceptibility in the population by classifying susceptible as having no, partial, or full natural resistance to TB and latently infected individuals as rapid, normal, or very slow (or no) progressors to active TB depending on the genes.⁶ Mishra and Srivastava presented a mathematical model to understand the spread of tuberculosis disease in both pulmonary and drug-resistant subjects.⁷ The mathematical model was developed to fit those data best and obtain the model's optimal parameter values. Preventive measures for tuberculosis control were also investigated.⁸ Ludji et al. proposed modifying deterministic mathematical models for tuberculosis with vaccination. The reproduction number is calculated, and a sensitivity analysis is carried out. The model's stability is also investigated.9 Nkamba et al. used a deterministic epidemic model to study the impact of vaccination on the spread of tuberculosis. There were also numerical simulations.¹⁰ Ullah et al. investigated the impact of effective contact rate, treatment rate, and incomplete treatment vs efficient treatment on a deterministic TB epidemic model. The asymptotic behavior, spread, and potential tuberculosis eradication are also discussed.¹¹ Liu and Zhang created a mathematical model to describe how vaccination and treatment affect the spread

of tuberculosis.¹² Andrawus *et al.* developed a new mathematical model of TB transmission dynamics, including first- and secondline treatment. The control reproduction number and equilibrium points are calculated for the studied model, and their stability is examined. Some numerical simulation was performed to support the analytical results.¹³ Zadeh first proposed the fuzzy theory in 1965.¹⁴ Fuzzy theory plays an important role in mathematical modeling by providing a way to handle uncertain or ambiguous information in a mathematical framework. Traditional mathematical models assume that all variables are precise and can be accurately measured or calculated. However, in many real-world situations, variables may be imprecise or difficult to quantify, leading to uncertainty in the model. The fuzzy theory provides a mathematical framework for dealing with this uncertainty by allowing variables to take on values that are not precisely defined but are characterized by degrees of membership in a set. This allows for a more flexible and realistic representation of variables and enables mathematical models to reflect the complex and uncertain nature of real-world systems accurately. Fuzzy parameters are a type of parameter used in mathematical modeling that represents the degree of uncertainty or imprecision in the values of the parameters. Fuzzy parameters are instrumental when the relationships between variables are complex and precise data are unavailable. They allow for the representation of uncertainty and imprecision quantitatively, making it possible to incorporate subjective knowledge and expert opinion into mathematical models. The fuzzy theory has been applied in many areas of mathematical modeling. By incorporating fuzzy theory into mathematical models, researchers can build more accurate and robust models that can handle uncertainty and imprecision, leading to better predictions and decisions. Many researchers have applied fuzzy theory to epidemiology. The fuzzy theory has been applied in various ways to develop and enhance epidemic models. Barros et al.¹⁵ and Mondal et al.¹⁶ investigated epidemic models with fuzzy transmission coefficients. For the fuzzy transmission of worms in a computer network, Mishra and Pandey proposed a SIR'S model. The developed system of equations is solved and simulated using numerical methods.¹² The high-order extrapolated nonstandard finite difference schemes (NSFD) are designed to model infectious diseases, as discussed in Refs. 18 and 19. The NSFD theory proposed by Mickens²⁰ is extensively used in disease mathematical and numerical modeling, to mention a few. Allehiany et al. investigated a Covid-19 model with fuzziness and numerically solved it using the NSFD scheme.²⁴ Alhebshi et al. looked into a computer virus model that used fuzzy criteria.²⁶ The evolutionary computational method for tuberculosis model with fuzziness provides a significant contribution to the field of TB research and control. Its ability to incorporate fuzziness and evolutionary computation techniques enables more accurate predictions and simulations of TB transmission dynamics, aiding in developing effective control strategies and policies. Furthermore, this approach has the potential to be extended to other infectious diseases, where uncertainties and complex interactions play a critical role, providing a valuable tool for public health decision-making and intervention planning. The development, accomplishment, and numerical analysis of the first-order explicit numerical computational technique with NSFD in a fuzzy environment, particularly with fuzzy parameters, is the novelty of the current work. Using fuzzy theory in TB modeling can improve our understanding of TB transmission and inform the development of more effective control

(3)

(4)

strategies. To our knowledge, the model under study has never been studied in the literature in the NSFD and fuzzy senses, and this is the first study of this model in this sense. The remainder of this paper is designed as we begin with a TB model and a mathematical analysis of the model is presented. The following sections contain the forward Euler and NSFD mathematical techniques and their simulation results. This article is concluded in the final section.

II. TB MODEL WITH FUZZY PARAMETERS

We consider the TB model talked about by Mishra and Srivastava,⁷

$$\begin{cases} \frac{dS}{dt} = \Lambda + \varepsilon R + \rho V - \beta IS - (\sigma + d)S, \\ \frac{dE}{dt} = \beta IS - (\gamma + d)E, \\ \frac{dI}{dt} = -(\alpha + \phi + d + \delta)I + \gamma E, \\ \frac{dQ}{dt} = \alpha I - (\eta + d + \delta)Q, \\ \frac{dR}{dt} = \phi I + \eta Q - (\varepsilon + d)R, \\ \frac{dV}{dt} = \sigma S - (\rho + d)R. \end{cases}$$
(1)

The corresponding model with fuzzy parameters can be written as

$$\begin{cases} \frac{dS}{dt} = \Lambda + \varepsilon R + \rho V - \beta(\xi)IS - (\sigma + d)S, \\ \frac{dE}{dt} = \beta(\xi)IS - (\gamma + d)E, \\ \frac{dI}{dt} = -(\alpha + \phi + d + \delta(\xi))I + \gamma E, \\ \frac{dQ}{dt} = \alpha I - (\eta(\xi) + d + \delta(\xi))Q, \\ \frac{dR}{dt} = \phi I + \eta(\xi)Q - (\varepsilon + d)R, \\ \frac{dV}{dt} = \sigma S - (\rho + d)R. \end{cases}$$
(2)

Here, *S*, *E*, *I*, *Q*, *R*, and *V* denote susceptible, exposed, TB infected, quarantined, recovered, and vaccinated classes, respectively. The birth rate is represented by Λ , the natural death rate by *d*, *y* is the rate of transmission from exposed to infected type, the rates of transmission from infected to quarantined class and infected to recovered class are denoted by α and ϕ respectively, and the rates of transfer from recovered compartment to susceptible compartment and vaccinated to susceptible compartment are represented by ε and ρ . At the same time, σ is the vaccination rate coefficient for the susceptible population. Moreover, the TB contact rate β , recovery rate η , and TB-induced death rate δ are considered fuzzy numbers due to their uncertain natures and are defined as follows:

and

$$\delta(\xi) = \begin{cases} \frac{(1-\xi) - \psi_0}{\xi_{\min}} \xi + \psi_0, & 0 \le \xi \le \xi_{\min}, \\ 1-\xi, & \xi_{\min} < \xi. \end{cases}$$
(5)

 $\beta(\xi) = \begin{cases} 0, & \zeta \ge \zeta_{\min} \\ \frac{\xi - \xi_{\min}}{\xi_M - \xi_{\min}}, & \xi_{\min} < \xi \le \xi_M \\ 1, & \xi_M < \xi, \end{cases}$

 $\eta(\xi) = \frac{\eta_0 - 1}{\xi_M} \xi + 1, 0 \le \xi \le \xi_{\min},$

III. NUMERICAL MODELING

Numerical modeling is a cornerstone of modern scientific investigation, empowering researchers to unlock the natural world's mysteries and address pressing global challenges. As computational techniques advance, the impact of numerical modeling on our understanding of the universe and our ability to shape a better tomorrow will only continue to expand, making it an indispensable tool in pursuing knowledge and progress. We could use Euler and nonstandard finite difference methods in this section for a given fuzzy epidemic model (2).

A. Forward Euler scheme

The method is based on discretizing the continuous-time domain into a series of discrete time steps, where the solution to the differential equation is approximated at each time step. The forward Euler method for model (2) is as follows:

$$\begin{cases} S^{n+1} = S^{n} + h \Big[\Lambda + \varepsilon R + \rho V - \beta(\xi) I^{n} S^{n} - (\sigma + d) S^{n} \Big], \\ E^{n+1} = E^{n} + h \Big[\beta(\xi) I^{n} S^{n} - (\gamma + d) E^{n} \Big], \\ I^{n+1} = I^{n} + h \Big[\gamma E^{n} - (\alpha + \phi + d + \delta(\xi)) I^{n} \Big], \\ Q^{n+1} = Q^{n} + h \Big[\alpha I^{n} - (\eta(\xi) + d + \delta(\xi)) Q^{n} \Big], \\ R^{n+1} = R^{n} + h \Big[\phi I^{n} + \eta(\xi) Q^{n} - (\varepsilon + d) R^{n} \Big], \\ V^{n+1} = V^{n} + h \Big[\sigma S^{n} - (\rho + d) R^{n} \Big]. \end{cases}$$
(6)

Here, "*h*" is the time step size and $n \ge 0$. $S^{0}(0), E^{0}(0), I^{0}(0), Q^{0}(0), R^{0}(0)$, and $V^{0}(0) \ge 0$.

B. NSFD scheme

The Nonstandard Finite Difference (NSFD) method is a numerical technique for solving differential equations. Unlike traditional finite difference methods that utilize standard central or forward/backward difference formulas, NSFD methods employ nonstandard stencils. The Nonstandard Finite Difference (NSFD) method for model (2) is as follows:

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$$\begin{cases}
S^{n+1} = \frac{S^{n} + h(\Lambda + \varepsilon R^{n} + \rho V^{n})}{1 + h(\beta(\xi)I^{n} + \sigma + d)}, \\
E^{n+1} = \frac{E^{n} + h\beta(\xi)I^{n}S^{n}}{1 + h(\gamma + d)}, \\
I^{n+1} = \frac{I^{n} + h\gamma E^{n}}{1 + h(\alpha + \phi + d + \delta(\xi))}, \\
Q^{n+1} = \frac{Q^{n} + h\alpha I^{n}}{1 + h(\eta(\xi) + d + \delta(\xi))}, \\
R^{n+1} = \frac{R^{n} + h(\phi I^{n} + \eta(\xi)Q^{n})}{1 + h(\varepsilon + d)}, \\
V^{n+1} = \frac{V^{n} + h\sigma S^{n}}{1 + h(\rho + d)}.
\end{cases}$$
(7)

Here, "h" is the time step size and $n \ge 0$. $S^{0}(0), E^{0}(0), I^{0}(0), Q^{0}(0), R^{0}(0)$, and $V^{0}(0) \ge 0$.

C. Positivity of the scheme

Theorem. Let the state variables *S*, *E*, *I*, *Q*, *R*, and *V* involved in the scheme be positive at t = 0; moreover, if all the parameters are also positive, then $S^{n+1} \ge 0, E^{n+1} \ge 0, I^{n+1} \ge 0, Q^{n+1} \ge 0, R^{n+1} \ge 0$.

Proof. Taking into account the state variables S, E, I, Q, R, and V of the NSFD scheme (6) and by combining all the equations in the system above with n = 0, we arrive at the following expression:

$$\begin{cases} S^{1} = \frac{S^{0} + h(\Lambda + \varepsilon R^{0} + \rho V^{0})}{1 + h(\beta(\xi)I^{0} + \sigma + d)} \ge 0, \\ E^{1} = \frac{E^{0} + h\beta(\xi)I^{0}S^{0}}{1 + h(\gamma + d)} \ge 0, \\ I^{1} = \frac{I^{0} + h\gamma E^{0}}{1 + h(\alpha + \phi + d + \delta(\xi))} \ge 0, \\ Q^{1} = \frac{Q^{0} + h\alpha I^{0}}{1 + h(\eta(\xi) + d + \delta(\xi))} \ge 0, \\ R^{1} = \frac{R^{0} + h(\phi I^{0} + \eta(\xi)Q^{0})}{1 + h(\varepsilon + d)} \ge 0, \\ V^{1} = \frac{V^{0} + h\sigma S^{0}}{1 + h(\rho + d)} \ge 0. \end{cases}$$

By substituting n = 1 in, we can proceed to the next step.

$$\begin{cases} S^{2} = \frac{S^{1} + h(\Lambda + \varepsilon R^{1} + \rho V^{1})}{1 + h(\beta(\xi)I^{1} + \sigma + d)} \ge 0, \\ E^{2} = \frac{E^{1} + h\beta(\xi)I^{1}S^{1}}{1 + h(\gamma + d)} \ge 0, \\ I^{2} = \frac{I^{1} + h\gamma E^{1}}{1 + h(\alpha + \phi + d + \delta(\xi))} \ge 0, \\ Q^{2} = \frac{Q^{1} + h\alpha I^{1}}{1 + h(\eta(\xi) + d + \delta(\xi))} \ge 0, \\ R^{2} = \frac{R^{1} + h(\phi I^{1} + \eta(\xi)Q^{1})}{1 + h(\varepsilon + d)} \ge 0, \\ V^{2} = \frac{V^{1} + h\sigma S^{1}}{1 + h(\rho + d)} \ge 0. \end{cases}$$

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Next, assume that the above system of equations ensures that the value of variables has the attribute of positivity for n = 2, 3, 4, ..., n - 1, i.e., $S^{n+1} \ge 0, E^{n+1} \ge 0, I^{n+1} \ge 0, Q^{n+1} \ge 0, R^{n+1} \ge 0, V^{n+1} \ge 0$, for n = 2, 3, 4, ..., n - 1.

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The positivity will now be examined for a random positivity integer $n \in \mathbb{Z}$, and we observe that

$$\begin{cases} S^{n+1} = \frac{S^n + h(\Lambda + \varepsilon R^n + \rho V^n)}{1 + h(\beta(\xi)I^n + \sigma + d)} \ge 0, \\ E^{n+1} = \frac{E^n + h\beta(\xi)I^n S^n}{1 + h(\gamma + d)} \ge 0, \\ I^{n+1} = \frac{I^n + h\gamma E^n}{1 + h(\alpha + \phi + d + \delta(\xi))} \ge 0, \\ Q^{n+1} = \frac{Q^n + h\alpha I^n}{1 + h(\eta(\xi) + d + \delta(\xi))} \ge 0, \\ R^{n+1} = \frac{R^n + h(\phi I^n + \eta(\xi)Q^n)}{1 + h(\varepsilon + d)} \ge 0, \\ V^{n+1} = \frac{V^n + h\sigma S^n}{1 + h(\rho + d)} \ge 0. \end{cases}$$

As a result, the proposed scheme guarantees the positivity of the state variables for all positive integer values of *n*.

D. Convergence analysis

Convergence analysis is crucial for assessing the reliability and efficiency of numerical methods, guiding the selection of appropriate parameters, and ensuring that numerical solutions provide accurate approximations of the underlying mathematical problems. In this section, we check the convergence analysis of the NSFD model given in (7). Let

$$\begin{cases} C_1 = \frac{S + h(\Lambda + \varepsilon R + \rho V)}{1 + h(\beta(\xi)I + \sigma + d)}, \\ C_2 = \frac{E + h\beta(\xi)IS}{1 + h(\gamma + d)}, \\ C_3 = \frac{I + h\gamma E}{1 + h(\alpha + \phi + d + \delta(\xi))} \\ C_4 = \frac{Q + h\alpha I}{1 + h(\eta(\xi) + d + \delta(\xi))}, \\ C_5 = \frac{R + h(\phi I + \eta(\xi)Q)}{1 + h(\varepsilon + d)}, \\ C_6 = \frac{V + h\sigma S}{1 + h(\rho + d)}. \end{cases}$$
(8)

The Jacobian matrix of the NSFD scheme at Disease Free Equilibrium (DFE) point is

$$J = \begin{bmatrix} \frac{1}{1+h(\sigma+d)} & 0 & 0 & 0 & \frac{h\varepsilon}{1+h(\sigma+d)} & \frac{h\rho}{1+h(\sigma+d)} \\ 0 & \frac{1}{1+h(\gamma+d)} & 0 & 0 & 0 & 0 \\ 0 & \frac{h\gamma}{1+h(\alpha+\phi+d+\delta(\xi))} & \frac{1}{1+h(\alpha+\phi+d+\delta(\xi))} & 0 & 0 & 0 \\ 0 & 0 & \frac{h\alpha}{1+h(\gamma(\xi)+d+\delta(\xi))} & \frac{1}{1+h(\eta(\xi)+d+\delta(\xi))} & 0 & 0 \\ 0 & 0 & \frac{h\phi}{1+h(\varepsilon+d)} & \frac{h\eta}{1+h(\varepsilon+d)} & \frac{1}{1+h(\varepsilon+d)} & 0 \\ \frac{h\sigma}{1+h(\rho+d)} & 0 & 0 & 0 & 0 & \frac{1}{1+h(\rho+d)} \end{bmatrix}.$$

Eigenvalues of the above matrix are $\lambda_1 = \frac{1}{1+h(\alpha+d)} < 1$, $\lambda_2 = \frac{1}{1+h(\gamma+d)} < 1$, $\lambda_3 = \frac{1}{1+h(\alpha+\phi+d+\delta(\xi))} < 1$, and $\lambda_4 = \frac{1}{1+h(\eta(\xi)+d+\delta(\xi))} < 1$. $\lambda_5 = \frac{1}{1+h(\varepsilon+d)} < 1$, and $\lambda_6 = \frac{1}{1+h(\rho+d)} < 1$. Since all eigenvalues are less than one, which proves the desired result.

E. Consistency analysis

We apply Taylor's series to check the proposed scheme's consistency. From the first equation of system (6), we have

$$S^{n+1}\left[1+h(\beta(\xi)I^n+\sigma+d)\right]=S^n+h(\Lambda+\varepsilon R^n+\rho V^n).$$
 (9)

Taylor's series expansion for S^{n+1} is

$$S^{n+1} = S^n + h\frac{dS}{dt} + \frac{h^2}{2!}\frac{d^2S}{dt^2} + \frac{h^3}{3!}\frac{d^3S}{dt^3} + \frac{h^4}{4!}\frac{d^4S}{dt^4} + \cdots$$

Equation (9) becomes

$$\left(S^n + h\frac{dS}{dt} + \frac{h^2}{2!}\frac{d^2S}{dt^2} + \frac{h^3}{3!}\frac{d^3S}{dt^3} + \frac{h^4}{4!}\frac{d^4S}{dt^4} + \cdots\right),$$

$$\left[1 + h(\beta(\xi)I^n + \sigma + d)\right] = S^n + h(\Lambda + \varepsilon R^n + \rho V^n),$$

$$h\frac{dS}{dt} + h(\beta(\xi)I^n + \sigma + d)S^n = h(\Lambda + \varepsilon R^n + \rho V^n).$$

We get the following by some simplification and applying $h \rightarrow 0$:

$$\frac{dS}{dt} = \Lambda + \varepsilon R^{n} + \rho V^{n} - \beta(\xi) I^{n} S^{n} - (\sigma + d) S^{n},$$
$$\frac{dS}{dt} = \Lambda + \varepsilon R + \rho V - \beta(\xi) IS - (\sigma + d) S,$$

which is consistent with the first equation of system (2). In a similar way,

$$E^{n+1} = E^n + h\frac{dE}{dt} + \frac{h^2}{2!}\frac{d^2E}{dt^2} + \frac{h^3}{3!}\frac{d^3E}{dt^3} + \frac{h^4}{4!}\frac{d^4E}{dt^4} + \cdots$$

From the second equation of the NSFD scheme, we have

$$\begin{split} E^{n+1}[1+h(\gamma+d)] &= E^n + h\beta(\xi)I^nS^n, \\ \left(E^n + h\frac{dE}{dt} + \frac{h^2}{2!}\frac{d^2E}{dt^2} + \frac{h^3}{3!}\frac{d^3E}{dt^3} + \frac{h^4}{4!}\frac{d^4E}{dt^4} + \cdots\right), \\ [1+h(\gamma+d)] &= E^n + h\beta(\xi)I^nS^n, \\ h(\gamma+d)E^n + h\frac{dE}{dt} = h\beta(\xi)I^nS^n, \\ \frac{dE}{dt} &= \beta(\xi)I^nS^n - (\gamma+d)E^n, \\ \frac{dE}{dt} &= \beta(\xi)IS - (\gamma+d)E, \\ I^{n+1} &= I^n + h\frac{dI}{dt} + \frac{h^2}{2!}\frac{d^2I}{dt^2} + \frac{h^3}{3!}\frac{d^3I}{dt^3} + \frac{h^4}{4!}\frac{d^4I}{dt^4} + \cdots, \\ I^{n+1}[1+h(\alpha+\phi+d+\delta(\xi))] &= I^n + h\gamma E^n, \\ \left(I^n + h\frac{dI}{dt} + \frac{h^2}{2!}\frac{d^2I}{dt^2} + \frac{h^3}{3!}\frac{d^3I}{dt^3} + \frac{h^4}{4!}\frac{d^4I}{dt^4} + \cdots\right), \end{split}$$

$$h(\alpha + \phi + d + \delta(\xi))I^{n} + h\frac{dI}{dt} = h\gamma E^{n},$$

$$\frac{dI}{dt} = -(\alpha + \phi + d + \delta(\xi))I^{n} + \gamma E^{n},$$

$$\frac{dI}{dt} = -(\alpha + \phi + d + \delta(\xi))I + \gamma E.$$

 $[1+h(\alpha+\phi+d+\delta(\xi))]=I^n+h\gamma E^n,$

Similarly,

$$Q^{n+1} = Q^n + h\frac{dQ}{dt} + \frac{h^2}{2!}\frac{d^2Q}{dt^2} + \frac{h^3}{3!}\frac{d^3Q}{dt^3} + \frac{h^4}{4!}\frac{d^4Q}{dt^4} + \cdots,$$

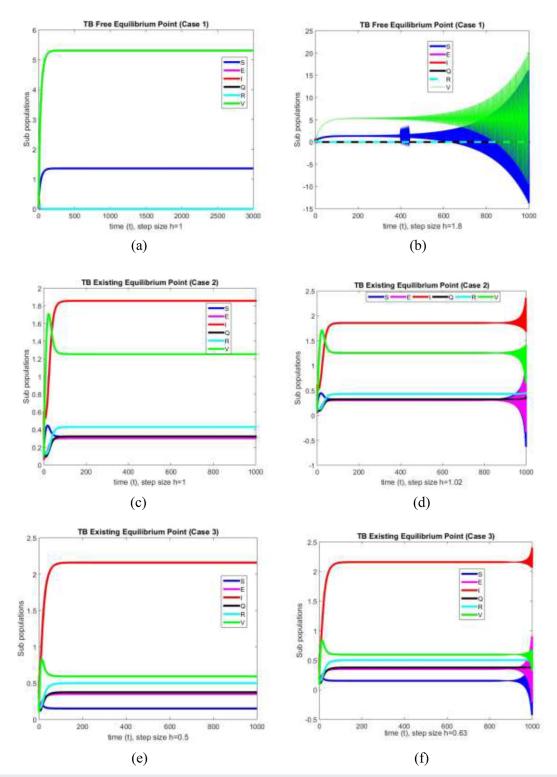


FIG. 1. Dynamics of subpopulations using forward Euler method for case 1, case 2, and case 3. (a) Graphical behavior of each subpopulation at TB-free equilibrium for case 1 at h = 1. (b) Graphical behavior of each subpopulation at TB-free equilibrium for case 2 at h = 1. (c) Graphical behavior of each subpopulation at TB-existing equilibrium for case 2 at h = 1. (d) Graphical behavior of each subpopulation at TB-existing equilibrium for case 2 at h = 1.0. (e) Graphical behavior of each subpopulation at TB-existing equilibrium for case 2 at h = 1.02. (e) Graphical behavior of each subpopulation at TB-existing equilibrium for case 3 at h = 0.5. (f) Graphical behavior of each subpopulation at TB-existing equilibrium for case 3 at h = 0.63.

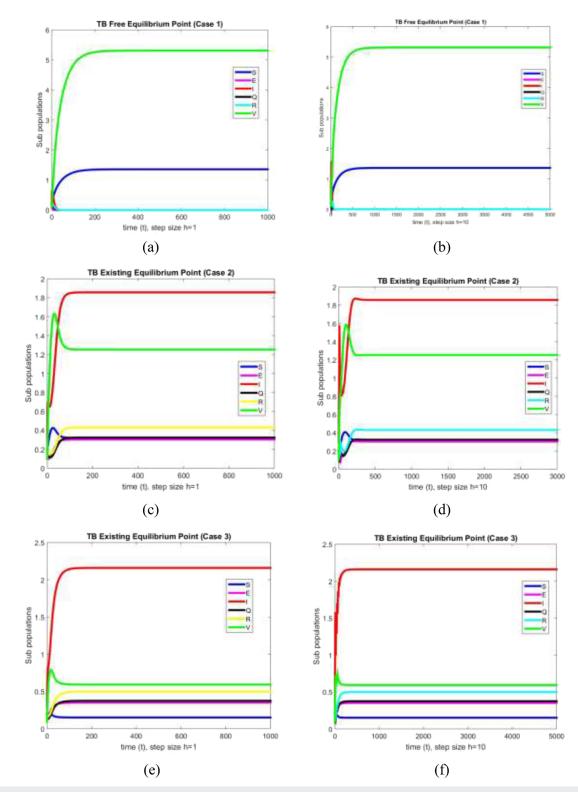


FIG. 2. Dynamics of subpopulations for case 1, case 2, and case 3 at h = 1 and h = 10. (a) TB-free equilibrium representation for case 1 at h = 1. (b) TB-free equilibrium for case 1 at h = 10. (c) TB-existing equilibrium for case 2 at h = 1. (d) TB-existing equilibrium for case 2 at h = 10. (e) TB-existing equilibrium for case 3 at h = 1. (f) TB-existing equilibrium for case 3 at h = 10. (e) TB-existing equilibrium for case 3 at h = 1. (f) TB-existing equilibrium for case 3 at h = 10.

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$$Q^{n+1}[1 + h(\eta(\xi) + d + \delta(\xi))] = Q^n + h\alpha I^n,$$
$$h\frac{dQ}{dt} + h(\eta(\xi) + d + \delta(\xi))Q^n = h\alpha I^n,$$
$$\frac{dQ}{dt} = \alpha I^n - (\eta(\xi) + d + \delta(\xi))Q^n,$$
$$\frac{dQ}{dt} = \alpha I - (\eta(\xi) + d + \delta(\xi))Q.$$

Similarly, we can get

$$\frac{dR}{dt} = \phi I + \eta(\xi)Q - (\varepsilon + d)R$$

and

$$\frac{dV}{dt} = \sigma S - (\rho + d)R$$

by applying the TSE of the last two equations of the NSFD scheme. As a result, system (2) and our discretized implicit numerical integration scheme is consistent.

IV. NUMERICAL SIMULATIONS

Here, we will use numerical simulation to demonstrate the behavior of the forward Euler method and the proposed NSFD scheme.

The dynamics of subpopulations using the forward Euler method are depicted in Figs. 1(a)-1(f). The way remains positive

and convergent for a small step size value and starts oscillating and gives negative values with a slight increase in the step size values. For case 1, the method behaves well at h = 1 and produces negative solutions at h = 1.8. For case 2, the method gives positive solutions at h = 1, making nonphysical oscillations and non-positive solutions at h = 1.02. Similarly, the technique works at h = 0.5 for the case, providing negative solutions and fluctuations at h = 0.63. It can be concluded from these graphs that the Euler method is not a reliable tool for studying TB disease dynamics in fuzzy conditions. The dynamics of subpopulations are shown in Figs. 2(a)-2(f) for all three cases at two different values of the time step sizes. It can be seen that all compartments are converging smoothly, showing positive behavior, and increasing step size does not affect its convergence and positivity. Positivity is an essential feature in epidemic models as these models consist of populations that cannot be negative. Many numerical schemes do not hold this feature.

The infected population at the TB-free equilibrium point is shown in Figs. 3(a) and 3(b) at two different step size values. The method shows positive, stable, and converging behavior in both cases, which are the main features of these types of models. The technique can reflect the disease dynamics for case 1. The behavior of the infected population for case 2 and case 3 is represented in Figs. 4(a), 4(b), 5(a), and 5(b), respectively. The proposed technique converges in both cases and is positive for different step-size values. This is an exciting feature of the developed method, which many other classical ways, such as Euler–Maruyama, Euler's Stochastics, and RK-4, do not keep it at increasing step sizes, as Raza *et al.*²⁷ pointed out. This makes it a valuable tool for disease modeling. The NSFD scheme is an invaluable tool for disease modeling that can provide accurate and efficient approximations of nonstandard differential equations.

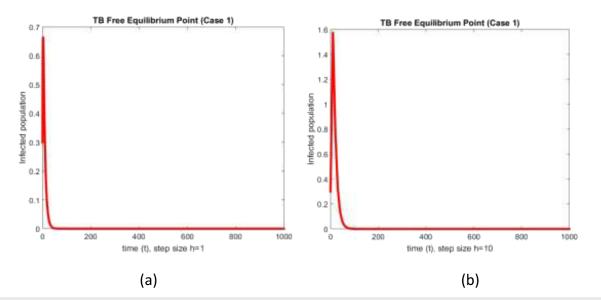


FIG. 3. (a) Graphical behavior of infected populations for case 1 at h = 1 (TB-free equilibrium). (b) Graphical behavior of infected populations for case 1 at h = 10 (TB-free equilibrium).

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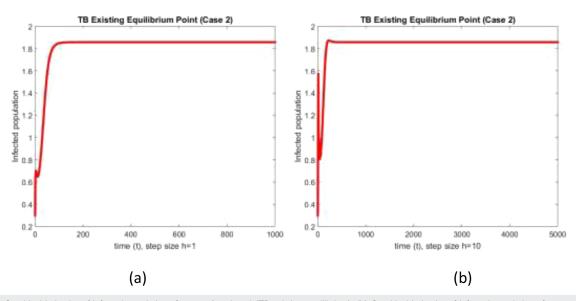


FIG. 4. (a) Graphical behavior of infected populations for case 2 at h = 1 (TB-existing equilibrium). (b) Graphical behavior of infected populations for case 2 at h = 10 (TB-existing equilibrium).

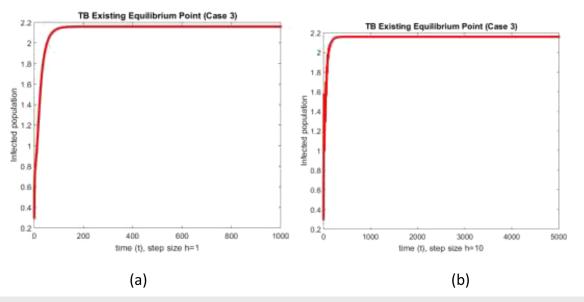


FIG. 5. (a) Graphical behavior of infected populations for case 3 at h = 1 (TB-existing equilibrium). (b) Graphical behavior of infected populations for case 3 at h = 10 (TB-existing equilibrium).

V. CONCLUSIONS

Data may not be precise in many real-world situations, making it challenging to build a traditional mathematical model. The fuzzy theory can handle such uncertain data. A disease may not be effective with a few viruses due to humanity's natural immune power. If the virus quantity is high, the system will become endemic. As a result, intensive treatment is not required for a small amount of virus. This phenomenon can only be observed in the fuzzy model and cannot be sustained in the crisp model. As a result, fuzzy models are more adaptable than classical models. This paper examines a mathematical model for TB transmission. In this study, the parameters $\beta(\xi)$, $\eta(\xi)$, and $\delta(\xi)$ are treated as fuzzy numbers. The reproduction number and fuzzy equilibrium analysis are investigated. An NSFD scheme is implemented in fuzzy environments to solve the studied model numerically, and its convergence and positivity are investigated. The proposed method's consistency is also investigated. The proposed method preserves the convergence and positive behavior of the numerical solutions at each time step, which are the main characteristics of this type of model. Delayed, stochastic, and fractional models with fuzziness and many more directions can be considered future directions. Furthermore, stochastic modeling and implementation of nonstandard finite difference methods may be extended in fuzzy epidemic models, as discussed in Refs. 28–30.

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AUTHOR DECLARATIONS

Conflict of Interest

The authors have no conflicts to disclose.

Author Contributions

Ateq Alsaadi: Data curation (equal); Formal analysis (equal); Methodology (equal). Fazal Dayan: Conceptualization (equal); Visualization (equal); Writing – original draft (equal); Writing – review & editing (equal). Nauman Ahmed: Conceptualization (equal); Data curation (equal); Project administration (equal). Dumitru Baleanu: Funding acquisition (equal); Investigation (equal); Project administration (equal). Muhammad Rafiq: Conceptualization (equal); Methodology (equal); Project administration (equal). Ali Raza: Investigation (equal); Methodology (equal); Project administration (equal); Resources (equal).

DATA AVAILABILITY

The data that support the findings of this study are available within the article.

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