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MATHEMATICAL MODELLING OF TUBERCULOSIS AND DIABETES CO-INFECTION USING THE NON- STANDARD FINITE DIFFERENCE SCHEME

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ABSTRACT. One of the major health challenge facing Africa and in particular, Kenya is the risk of Tuberculosis and Diabetes. To understand the dynamics of this, a nine compartmental model for tuberculosis-diabetes co-infection is formulated. The Non-standard finite difference Scheme (NSFD) of the model is formulated from the first-order ordinary differential equations (ode) to avoid full implicit schemes that are computationally expensive. The overly small step sizes in NSFD give the user autonomy in controlling the accuracy of the results, making it suitable for disease control applications. Numerical simulations with different step sizes of the NSFD for the TB-Diabetes model are carried out to find the optimal step size, h. A comparison of the best resultant numerical simulation based on optimal h in NSDF indicates NSFD gives better results when compared with the corresponding first-order ode. The phase-plane analysis revealed that the NSFD formulated for tuberculosis and diabetes co-infection is generally asymptotically stable. Future studies should consider formulating the proposed model with varied control parameters such as medication to compare the results with those from first-order ode.

1. INTRODUCTION

Tuberculosis is an ailment that affects both human and animal population. It is caused by mycobacterium tuberculosis complex (MTBC) which includes seven TB causing mycobacterium [1]. It is an airborne disease and is transmitted through fluids particles called droplet nuclei of 1-5 microns in diameter generated from the respiratory system of TB infected individuals when they cough, sneeze, speak, sing or spit [2]. These droplet nuclei can be suspended in the air for several hours. The World health organization posits that TB is one of the top ten causes of death in the world [3]. 10.4 million People were exposed to TB in 2016 and 1.7 million died that year [4]. In 2019, it was estimated that 10 million people got infected while 1.4 million people died [5].

Kenya is a high TB burden country ranked 13^{th} amongst the 22 countries, contributing 80 percent of the global case load [6]. Diabetes Mellitus on the other hand, is a syndrome of disordered metabolism which occurs when the pancreas does not produce enough insulin or when the body does not effectively use the insulin produced. Insulin which is made by the beta cells of the pancreas regulates the blood sugar. If not

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well regulated, one develops hyperglycemia (high sugar levels) which leads to serious damage of various body systems especially the nerves, blood vessels, eyes and kidneys [7]. The global prevalence of diabetes over the past few decades has shown a trend of rapid increase and therefore raising a major concern. Two individuals develop diabetes every 10 seconds and two individuals die of diabetes every 10 seconds as per the International Diabetes Federation (IDF) statistics [8].

Diabetes is one of the risk factors of tuberculosis due to its immune-compromising effect. It is known to have an effect on the natural course of TB by making individuals to have a lifetime risk of getting TB infection activated from the latent stage of infection, getting more severe symptoms, treatment failure, more lapses as well as more prone to death [9]. There is also likelihood for misdiagnosis of patients with TB who have diabetes because these patients show typical imaging changes and lesion distribution in the lower lobe instead of the upper lobe which TB infected patient shows [10]. This has a serious clinical implication because lesion in the lower lobe is easily misdiagnosed as a tumor or community acquired pneumonia. The misdiagnosis may delay early treatment of TB and therefore increasing the spread and affecting the control of transmission [11]. It is therefore very crucial to study TB-diabetes co-infection in order to comply with the WHO's end TB blueprint, aiming at reducing TB incidences by 80 percent and the deaths by 90 percent by 2030.

The analysis of tuberculosis and diabetes co-infection through mathematical modeling has been done by many authors. For instance [11, 12] similarly analyzed the dynamics for the transmission of TB in people with diabetes using a SEIR model, and they found a need to look at intervention strategies. In their study, they proposed chemoprophylaxis treatment for latent TB individuals. They also stressed the need to treat active TB individuals and control glucose levels for diabetic individuals. Malik developed Moualeu's work by looking at the possibility of diabetes being transmitted vertically from newborns and included this aspect in their model. Pinto *et al.,* [13] studied Diabetes Mellitus and TB Co-existence and their Clinical implications from a fractional-order perspective.

Differential equations are commonly used to analyze the dynamics of biological systems [14]. These systems are usually so complex that their exact analytical solutions are usually unattainable. As a result, numerical methods such as Euler and Runge-Kutta schemes are used to analyze them. Approximation theory is used in constructing these methods, and in ways, finite representation of the functions is produced [15]. These methods are often prone to numerical instabilities [16]. For that reason, an idea to construct numerical schemes that do not deal on the approximation problem but rather than look at dynamical information emerged and was developed by Mickens in 1989 and he named the new scheme non-standard schemes to distinguish them from the classical ones [17]. These sets of numerical analysis methods offered numerical solutions to differential equations by constructing discrete models whose solutions are of the same qualitative properties as the corresponding differential equations for all step sizes. The scheme thereby gave reliable numerical results by preserving the significant properties of their continuous analogs [18]. Non-standard finite difference schemes have performed better than those that are more prone to numerical instabilities. In addition, the non-standard finite difference scheme performs better regarding positivity and boundedness of solutions compared to the standard finite difference schemes [19].

Non-standard difference schemes are popular numerical methods for solving differential equations involving non-local, non-linear, or non-integer operators [20]. The schemes are used when traditional numerical methods fail to provide accurate solutions or become computationally expensive. In epidemiology, non-standard finite difference schemes have been used to solve models of the spread of infectious diseases. These schemes are useful when traditional methods fail to capture the non-local and non-linear interactions between infected and susceptible individuals [21]. In modeling the spread of tuberculosis, non-standard

finite difference schemes have been used to capture the non-local and non-linear interactions between infected and susceptible individuals [22], these schemes have been used to model the effect of treatment and quarantine on the spread of the disease.

Furthermore, the construction and analysis of NSFDM to solve mathematical models was conducted by [23], which has been used by several researchers including [24] who constructed a nonstandard finite scheme of influenza and were able to show that the differential equation model shows the same properties as the continuous model. [25] solved the smoking prevalence model for Spain using a Nonstandard finite difference scheme to which they compared their results with the Euler, Runge-Kutta, and Trapezoidal methods and concluded that the scheme was a good numerical method in solving mathematical models.

[19] on their study on cholera dynamics showed that in the nonstandard finite difference model, the results obtained were similar to the case when the basic reproduction number was less than one, and the equilibrium points were both locally and globally stable regardless of the time step-size used. [26], in their study on modeling the dynamics of campylobacteriosis represented by a nonlinear system of ordinary differential equations [26]. The numerical results presented confirmed the applicability of the proposed NSFDM for biological systems. These methods preserved the solution's positivity and converged to stability properties to the correct equilibria for arbitrary step-sizes, which is not the case with solutions obtained by other numerical methods, since this is obtained with many difficulties.

In a recent study, by [27] used a fractional order model of tuberculosis and diabetes co-infection to develop a non-standard finite difference scheme to simulate the model. The results showed that the non-standard scheme provided a more accurate and efficient co-infection dynamics simulation than traditional schemes.

Contribution. The study is guided by the following contributions:

- The model is an extension of tuberculosis and diabetes co-infection using nonstandard finite difference scheme to avoid full implicit schemes. The extension is due to computational expensiveness of associated with continuous-time model due to overly small step sizes needed for accuracy.
- The proposed model extends [12] by including diabetes complications and tuberculosis complications due to diabetes. This gives the models a holistic understanding of both diseases, which have significant global challenges. The proposed model allows for a more comprehensive understanding of the disease's interactions, their influences on each other, and patient effects on outcomes.
- The proposed model extends the models by [11–13] by incorporating the non-standard finite difference method to carry out the numerical simulation of the model to compare it with the traditional simulation of the continuous-time model. This allows the researcher to establish an accurate approach feasible for real-life application studies.
- The study investigates the optimal value of h (step-size) for NSFD, which is essential for ensuring that the numerical solution converges to the true solution of the TB and diabetes co-infection problem presented in Figure 1.

The remainder of this paper is organized as follows: Section 2 outlines the formulated model in the paper, parameter estimation. Section 3 outlines both standard finite difference method and non-standard finite difference methods, section 4 outlines the numerical solutions and phase-plane analysis, and in Section 5 conclusion to the study is given.

2. METHODS

2.1. **Model formulation.**

S The model description that represents the dynamics of tuberculosis and diabetes co-infection is divided into compartments where the compartment S are healthy individuals, and is increased by Λ, the recruitment of individuals by birth. It is decreased by λS (individuals getting TB), $\alpha_1 S$ (Individuals acquiring Diabetes) and by μS (natural deaths).

L_f The compartment L_f (individuals with fast propagating TB) is increased by $p\lambda S$ (proportion of susceptible individuals developing a fast propagating TB) and πL_s (individuals with slow propagating TB developing fast propagating TB), and is decreased by μ (natural death), $\sigma_1 L_f$ (individuals with fast propagating TB becoming infectious).

L_s The compartment L_s(individuals with slow propagating TB) is increased by $(1-p)\lambda S$ (susceptible individuals developing slow propagating TB) and $\rho_1 I_T$ (those infectious and treated individual getting a slow propagation TB) and is decreased by μL_s (natural deaths), λL_f (individuals with slow propagating TB developing fast propagating TB), $\sigma_2 L_s$ (individuals with slow propagating TB becoming infectious), $\alpha_2 L_s$ (individuals with slow propagating TB acquiring diabetes) and $\delta_1 L_s$ (TB induced deaths).

I_T The compartment I_T (TB infectious individuals) is increased by $\sigma_2 L_s$ (individuals with slow propagating TB becoming infectious) and $\sigma_1 L_f$ (individuals with fast propagating TB becoming infectious) and is decreases by $\rho_1 I_T$ (those infectious and treated gets a slow propagation TB) , μI_T (natural deaths) and $\delta_2 I_T$ (TB induced deaths).

D_T The compartment D_T (individuals with TB and diabetes) and is increased by $\alpha_2 L_s$ (individuals with slow propagating TB acquiring diabetes), ηD (individuals with diabetes getting TB) and $\rho_2 I_{DT}$ individuals with diabetes but treated of TB becomes exposed to TB. The class is decreased by μD_T (natural death) and $\sigma_3 D_T$ (individuals becomes infectious of TB).

I_{DT} The compartment I_{DT} (individuals with diabetes and infectious of TB) is increased by $\sigma_3 D_T$ (individuals become infectious of TB) and $\omega_2 C_{DT}$ (proportion of individuals with diabetes complications due to TB). The class is decreased by $\rho_2 I_{DT}$ (individuals with diabetes but treated of TB becomes exposed to TB), μ (natural death rate), $\delta_2 I_{DT}$ (disease induced deaths) and $\theta_2 I_{DT}$ (proportion of individuals progressing to individual with diabetes complications due to TB).

C_{DT} The compartment C_{DT} (individuals with diabetes complications due to TB) and is increased by $\theta_2 I_{DT}$ (individuals infectious with TB and has Diabetes developing complications) and decreases by $\omega_2 C_{DT}$ (individuals receiving treatment of the complications), μC_{DT} (natural death) and $\delta_3 C_{DT}$ (TB induced deaths due to complications).

D The compartment D (individuals with diabetes) is increased by $\alpha_1 S$ (susceptible individuals acquiring diabetes) and $\omega_1 C$ (individuals with diabetes complications getting treatment of the complications) and is decreased by μD (natural deaths), $\theta_1 D$ (diabetic individuals getting diabetes complications) and λD (individuals with diabetes getting TB).

C The compartment C(diabetic individuals with complications) is increased by $\theta_1 D$ (diabetic individuals getting diabetes complications) and is decreased by μC (natural deaths), $\delta_3 C$ (induced deaths due to diabetes complications) and $\omega_1 C$ (individuals with diabetes complications getting treatment of the complications).

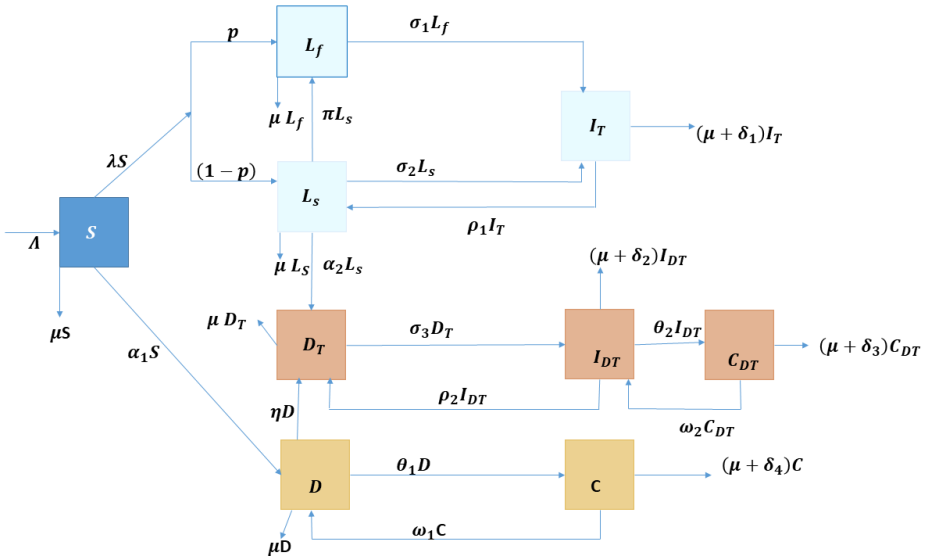


FIG. 1. TB-Diabetes model

Following the description given in the flow diagram, the model system is described by the first order differential equations given in (2.1).

$$
\left.
\begin{aligned}
\frac{dS}{dt} &= \overbrace{\Lambda}^{\text{recruitment}} - \overbrace{\beta IS}^{L_f L_s \text{ transmission}} - \overbrace{\alpha_1 S}^{D \text{ transmission}} - \overbrace{\mu S}^{\text{natural deaths}}, \\
\frac{dL_f}{dt} &= \overbrace{p\beta IS}^{S \text{ becomes } L_f} + \overbrace{\pi L_s}^{L_s \text{ becoming } L_f} - \overbrace{\sigma_1 L_f}^{L_f \text{ becoming } I_T} - \overbrace{\mu L_f}^{\text{natural deaths}}, \\
\frac{dL_s}{dt} &= \overbrace{(1-p)\beta IS}^{\text{S becoming } L_s} + \overbrace{\rho_1 I_T}^{I_T \text{ transforming to } L_s} - \overbrace{\pi L_s}^{L_s \text{ becoming } L_f} - \overbrace{\sigma_2 L_s}^{I_T \text{ becoming } L_s} - \overbrace{\alpha_2 L_s}^{L_s \text{ becoming } D_T} - \overbrace{\mu L_s}^{\text{natural deaths}}, \\
\frac{dI_T}{dt} &= \overbrace{\sigma_2 L_s}^{L_s \text{ becomes infectious}} + \overbrace{\sigma_1 L_f}^{L_f \text{ becomes infectious}} - \overbrace{\rho_1 I_T}^{I_T \text{ becomes } L_s} - \overbrace{\delta_1 I_T}^{\text{disease induced death}} - \overbrace{\mu I_T}^{\text{natural deaths}}, \\
\frac{dD_T}{dt} &= \overbrace{\alpha_2 L_s}^{L_s \text{ become diabetic}} + \overbrace{\eta D}^{D \text{ acquires TB}} + \overbrace{\rho_2 I_{DT}}^{I_{DT} \text{ exposed to TB}} - \overbrace{\sigma_3 D_T}^{D_T \text{ become infectious}} - \overbrace{\mu D_T}^{\text{natural deaths}}, \\
\frac{dI_{DT}}{dt} &= \overbrace{\sigma_3 D_T}^{D_T \text{ become infectious}} + \overbrace{\omega_2 C_{DT}}^{C_{DT} \text{ becomes } I_{DT} \text{ due to TB}} - \overbrace{\rho_2 I_{DT}}^{I_{DT} \text{ is exposed to TB}} - \\
&\quad \overbrace{\theta_2 I_{DT}}^{I_{DT} \text{ gets complication due to TB}} - \overbrace{\delta_2 I_{DT}}^{\text{disease induced death}} - \overbrace{\mu I_{DT}}^{\text{natural deaths}}, \\
\frac{dC_{DT}}{dt} &= \overbrace{\theta_2 I_{DT}}^{I_{DT} \text{ gets complication due to TB}} - \overbrace{\omega_2 C_{DT}}^{C_{DT} \text{ becomes } I_{DT} \text{ due to TB}} - \overbrace{\delta_3 C_{DT}}^{\text{disease induced death}} - \overbrace{\mu C_{DT}}^{\text{natural deaths}}, \\
\frac{dD}{dt} &= \overbrace{\alpha_1 S}^{S \text{ acquires diabetes}} + \overbrace{\omega_1 C}^{\text{treated } C} - \overbrace{\eta D}^{D \text{ gets TB}} - \overbrace{\theta_1 D}^{D \text{ getting complications}} - \overbrace{\mu D}^{\text{natural deaths}}, \\
\frac{dC}{dt} &= \overbrace{\theta_1 D}^{D \text{ getting complications}} - \overbrace{\omega_1 C}^{C \text{ getting treatment}} - \overbrace{\delta_4 C}^{\text{disease induced deaths}} - \overbrace{\mu C}^{\text{natural deaths}},
\end{aligned}
\right\} \tag{2.1}
$$

where $\lambda = \beta I$ and $I = I_T + I_{DT}$, subject to the initial conditions in (2.2).

$$\left.\begin{aligned} &S(0)=S_0,\ L_s(0)=L_{s0},\ I_T(0)=I_{T0},\ L_f(0)=L_{f0},\ I_{DT}=I_{0DT},\\ &C_{DT}=C_{0DT},\ D_T(0)=D_{T0},\ D(0)=D_0,\ C(0)=C_0. \end{aligned}\right\} \tag{2.2}$$

The model (2.1) is an extension of some earlier mentioned modelling studies such as:

(1) [12] by including diabetes complications and tuberculosis complications due to diabetes.
(2) [11–13] by incorporating the non-standard finite difference method to carry out the numerical simulation of the model.

2.1.1. *Assumptions.* The following assumptions were made when developing the model:

(1) Recruitment is by birth only.
(2) Individuals in the infectious class are treated and develop latent slow TB to which they can also become infectious.
(3) No permanent immunity upon treatment.
(4) Individuals with TB complications due to diabetes even when treated are still infectious of TB.

2.2. **Parameter estimation.** The estimated birth rate in Kenya was reported at 27.67 births per year for 1000 births in 2022 according to the World Bank collection of development indicators [28]. Thus, $\Lambda = 100$ per year. The mortality rate in Kenya was estimated to 5.09 deaths per 1000 people. Thus, $\mu = 0.00746$ per year While the mortality rate due to TB estimated at 3.875 deaths per 1000 people per year according to World Health Organization [6]. $\delta_1 = 0.173$ Diabetes death rate is estimated at 30.43 per 100000 per year people by the World health organization [6]. Table 1 shows the description of the parameters used in the model.

TABLE 1. Parameter description and estimation

Description	Symbol	Value/yr	Range	Source
TB transmission coefficient	β	$\frac{233}{100{,}000}$	$\frac{188 \text{ to } 266}{100{,}000}$	[29]
Recruitment rate	Λ	$\frac{8.06}{1000} \times 53 \times 10^6$	-	Assumed
TB induced mortality	δ_1	$\frac{50}{100{,}000}$	$\frac{9 \text{ to } 97}{}$	[30]
Diabetic-TB induced mortality	δ_2	$\frac{426}{100{,}000}$	$\frac{89 \text{ to } 616}{100{,}000}$	[31]
Diabetic complication induced mortality for diabetic individuals	δ_3	$\frac{10.9}{100{,}000}$	$\frac{7.2 \text{ to } 82.6}{100{,}000}$	[32, 33]
Diabetes induced mortality	δ_4	$\frac{20.9}{100000}$	$\frac{7.2-82.6}{100000}$	[6]
Rate of Latent fast TB individuals becoming infectious	σ_1	$\frac{558}{100{,}000}$	$\frac{455 \text{ to } 662}{100{,}000}$	[29]
Rate of latent slow TB individuals becomes infectious	σ_2	3.33%	2% - 5%	[34]
Rate of diabetes individuals becoming infectious of TB	σ_3	14.8%	7.1% - 23.8%	[35]
Rate of acquiring diabetes	α_1	2.2%	1.4% - 3.1%	[36]
Rate of acquiring diabetes when exposed to TB	α_2	2.16	1.19 - 3.93	[37]
Rate of treated TB individuals exposed to TB	ρ_1	80.1%	$78\% - 85$	[38]
Rate of treated TB-diabetes individuals exposed to TB	ρ_2	31%	$12\% - 44\%$	[39]
Rate of treatment of complications due to diabetes	ω_1	38%	25% - 45%	[40]
Rate of treatment of TB complications in diabetic	ω_2	0.5%	0.1% - 45%	[41]
Rate of developing Latent fast TB	p	6%	5%–10%	[42]
Natural mortality rate	μ	$\frac{8.06}{1000}$	$\frac{7.9-8.1}{1000}$	[43]
Rate of acquiring complicated diabetes	θ_1	3.2%	2.8% - 4.4%	[44]
Rate of acquiring complicated diabetes due to TB	θ_2	$\frac{1}{20}$	$\frac{1 \text{ to } 6}{20}$	[45]
Rate of latent slow individuals becoming latent fast	π	$\frac{1220}{10^6} \times 0.08$	$\frac{48 \text{ to } 1299}{10^6}$	[46]
Rate of diabetic individuals acquiring TB	η	$\frac{112}{1134}$	$\frac{90 \text{ to } 130}{1134}$	[45]

3. MODEL ANALYSIS

In this section, a brief overview of the finite difference scheme as well as the non-finite difference scheme of the TB-Diabetes model is developed and analyzed.

3.1. **Finite Difference Method.** Finite difference methods are well known numerical methods that offer an alternative way of solving differential equations by approximating them with difference equations [47]. These methods work by discretizing the continuous domain into a finite number of grid points and approximating the derivatives at each grid point using finite differences. The basic idea behind finite difference methods is to replace the continuous derivative in a differential equation with a finite difference approximation that involves only function values at nearby points. The resulting differential equations can then be solved using standard numerical techniques.

Different types of finite difference methods depend on the order of the difference approximation used to approximate the derivatives [48]. For example, forward, backward, and central differences can approximate the first derivative, and second-order central differences can approximate the second derivative. Finite difference methods are widely used in many areas of science and engineering to solve partial differential equations (PDEs) that describe the behaviour of physical systems [49]. They are relatively simple to implement and can be used to solve problems that are too complex to solve analytically. However, they can be computationally expensive, especially for problems with large domains or high-dimensional spaces.

Finite difference formulas can be used at equally spaced grid points which are used to approximate the differential equations by transforming them into a system of algebraic equations to solve. Therefore in this method finite differences are used to approximate derivatives. In the transformation of a continuous time model to a discrete time model, discrete variable $k \in N$ replaces the continuous variable $t \in (o, \infty)$, while the continuous variable y is replaced by discrete value y_k and the resulting equation is a difference equation.

For instance, we are going to use the Taylor's theorem to introduce the standard finite difference method, we let h be the step size between the terms of the independent variable x and we also increase x by h to get the Taylor series expansion:

$$f(x+h) = f(x) + hf'(x) + \frac{h^2}{2!}f''(x) + ...$$

The Taylor series expansion solves to:

$$f'(x) = \frac{f(x+h) - f((x)}{h} \tag{3.1}$$

$$f'(x) = \frac{f(x) - f((x-h)}{h} \tag{3.2}$$

$$f'(x) = \frac{f(x+h) - f((x-h)}{2h} \tag{3.3}$$

equation (3.1), (3.2) and (3.3) above are referred to as the forward difference, backward difference and central difference approximation respectively. The central difference approximation is taken as the most accurate of the three because its truncation error is given by $O(h^2)$. When using the finite difference Methods,

the discretization error between the numerical solution and the exact solution is determined by the errors that arise when changing from a differential operator to a difference operator.

3.1.1. *Forward and Backward Euler method.* The Euler method is a basic algorithm for numerically solving first-order differential equations. Leonhard Euler Beethoven of mathematics introduced it in 1768 [50]. Forward and backward Euler methods are two common types of finite difference methods used to numerically approximate solutions to ordinary differential equations (ODEs). The forward Euler method is a first-order method that uses a forward difference approximation to the first derivative of the solution at a given time point [51]. The main difference between these methods is that the forward Euler method uses the derivative at the current time point to update the solution. In contrast, the backward Euler method uses the derivative at the next time point [52]. This makes the backward Euler method more stable, especially for stiff ODEs, but also more computationally expensive because it requires solving a nonlinear equation at each time step. Both methods have advantages and disadvantages, and the choice of method depends on the specific problem being solved and the desired level of accuracy and computational efficiency.

Euler idea changes a differential equation to an algebraic one and is used to solve initial valued problem of the form:

$$y'(t) = f(t, y(t)), y(t_0) = y_0$$

Truncating the Taylor series expansion forms the basis of the Euler method.i.e

$$\begin{aligned} y(t_n + h) = y_{n+1} &= y_n + h\frac{dy}{dt}\Big|_{t_n,y_n} + \frac{1}{2}h^2\frac{d^2y}{dt^2}\Big|_{t_n,y_n} + \frac{1}{3!}h^3\frac{d^3y}{dt^3}\Big|_{t_n,y_n} + ... \\ &= y_n + hf(t_n, y_n) + O(h^2). \end{aligned} \tag{3.4}$$

Equation (3.4) is the Euler forward equation which is explicit. A method can be explicit if one finds the y_{n+j}, $j = 0, 1, 2, 3, ..., k$ by recursively determining y_{n+j} iterate from the previous iterate. otherwise, it is implicit. The Euler backward method is an example of an implicit method. It is obtained by truncating the Taylor series expansion of the form:

$$\begin{aligned} y_n &= y(t_{n+1} - h) = y(t_{n+1}) - h\frac{dy}{dt}\Big|_{t_{n+1}} + \frac{1}{2}h^2\frac{d^2y}{dt^2}\Big|_{t_n,y_n} - ..... \\ y_{n+1} &= y_n + hf(t_{n+1}, y_{n+1}) + O(h^2). \end{aligned}$$

For the finite difference method to be useful, the convergence property is required as the minimum property. A numerical method is said to be convergent if the numerical solutions obtained approaches the exact solution as the step lengths approaches zero. For convergence property, the Euler forward method requires overly small step sizes. On the other hand, the Euler backward method is computational expensive since it gives an implicit equation for computation of y_{n+1} which is time consuming.

3.1.2. *Runge-Kutta method.* For the numerical solution of a differential equation, the Runge Kutta method of various orders could be adopted.The Runge Kutta first order are often referred to as the Euler forward method, where the derivatives of y at the given time step are used to do the extrapolations of the solution at the next time step [53]. On the other hand, the Runge-Kutta methods extrapolates the solution to the future time step using the information on the 'slope' at more than one point. To obtain the Runge-Kutta second order method, we truncate the Taylor series expansion by including one more term. such as

$$y_{n+1} = y_n + hf(t_n, y_n) + \frac{1}{2}h^2 f'(t_n, y_n) + O(h^3)$$

Runge-Kutta simplified the second order method by writing it as:

$$y_{n+1} = y_n + h(a_1k_1 + a_2K_2)$$

where, $k_1 = f(t_n, y_n)$ and $k_2 = f(t_n + p_I h, y_n + q_{II}k_1 h)$. The simplified form is easier to use since one is not needed to calculate $f'(t_n, y_n)$. The constants a_1, a_2, p and q have to be calculated so as the outcome is a method with local truncation error of $O(h^3)$ Runge-Kutta method of higher order can also be developed in the same way. Commonly used is the Runge Kutta fourth order method. Runge-Kutta fourth order method is given by (3.5).

$$\left.\begin{aligned} k_1 &= hf(y_n, t_n) \\ k_2 &= hf(y_n + k_1/2, t_n + h/2) \\ k_3 &= hf(y_n + k_2/2, t_n + h/2) \\ k_4 &= h(y_n + k_3, t_n + h) \\ y_{n+1} &= y_n + (k_1 + 2k_2 + 2k_3 + k_4)/6 \end{aligned}\right\} \tag{3.5}$$

The Runge- Kutta methods are explicit techniques therefore, they are conditionally stable.

3.1.3. *Euler method applied to the TB-Diabetes Model.* A forward Euler method can be constructed for our equation model (2.1) by replacing the derivative part with the forward difference approximation (3.4) and the non-derivative part approximated at base time level. This yields:

$$\left.\begin{aligned} \frac{S_1^{n+1} - S^n}{l} &= \Lambda - (\alpha_1 + \beta I^n + \mu)S_1^n, \\ \frac{L_f^{n+1} - L_f^n}{l} &= p\beta I^n S^n + \pi L_s^n - (\sigma_1 + \mu)L_f^n, \\ \frac{L_s^{n+1} - L_s^n}{l} &= (1-p_1)\beta I^n S_1^n + \rho_1 I_T^n - \pi L_s^n - (\alpha_2 + \sigma_2 + \mu)L_s^n, \\ \frac{I_T^{n+1} - I_T^n}{l} &= \sigma_2 L_s^n + \sigma_1 L_f^n - (\rho_1 + \delta_1 + \mu)I_T^n, \\ \frac{D_T^{n+1} - D_T^n}{l} &= \alpha_2 L_s^n + \eta D^n + \rho_2 I_{DT}^n - (\sigma_3 + \mu)D_T^n, \\ \frac{I_{DT}^{n+1} - I_{DT}^n}{l} &= \sigma_3 D_T^n + \omega_2 C_{DT}^n - (\rho_2 + \delta_2 + \mu)I_{DT}^n, \\ \frac{C_{DT}^{n+1} - C_{DT}^n}{l} &= \theta_2 I_{DT}^n - (\omega_2 + \mu + \delta_3)C_{DT}^n, \\ \frac{D^{n+1} - D^n}{l} &= \alpha_1 S^n + \omega_1 C^n - \eta D^n - (\theta_1 + \mu)D^n, \\ \frac{C^{n+1} - C^n}{l} &= \theta_1 D^n - (\delta_4 + \omega_1 + \mu)C^n. \end{aligned}\right\} \tag{3.6}$$

where $l > 0$ is an increment in time and $t \geq 0$ at the points $t_n = nl(n = o, 1, 2, 3, ...)$ is discretized in the standard way. Expression in (3.6) can be rewritten as given in (3.7).

$$
\left.
\begin{aligned}
S_1^{n+1} &= S^n + (\Lambda - (\alpha_1 + \beta I^n + \mu)S_1^n)l, \\
L_f^{n+1} &= L_f^n + (p\beta I^n S^n + \pi L_s^n - (\sigma_1 + \mu)L_f^n)l, \\
L_s^{n+1} &= L_s^n + ((1-p_1)\beta I^n S_1^n + \rho_1 I_T^n - \pi L_s^n - (\alpha_2 + \sigma_2 + \mu)L_s^n)l, \\
I_T^{n+1} &= I_T^n + (\sigma_2 L_s^n + \sigma_1 L_f^n - (\rho_1 + \delta_1 + \mu)I_T^n)l, \\
D_T^{n+1} &= D_T^n + (\alpha_2 L_s^n + \eta D^n + \rho_2 I_{DT}^n - (\sigma_3 + \mu)D_T^n)l, \\
I_{DT}^{n+1} &= I_{DT}^n + (\sigma_3 D_T^n + \omega_2 C_{DT}^n - (\rho_2 + \delta_2 + \mu)I_{DT}^n)l, \\
C_{DT}^{n+1} &= C_{DT}^n + (\theta_2 I_{DT}^n - (\omega_2 + \mu + \delta_3)C_{DT}^n)l, \\
D^{n+1} &= D^n + (\alpha_1 S^n + \omega_1 C^n - \eta D^n - (\theta_1 + \mu)D^n)l, \\
C^{n+1} &= C^n + (\theta_1 D^n - (\delta_4 + \omega_1 + \mu)C^n)l.
\end{aligned}
\right\} \tag{3.7}
$$

3.2. **Nonstandard Finite Difference Method.** This scheme is a set of numerical analysis method developed by [17] to offer numerical solutions to differential equations by constructing discrete model. However in 2003, he Constructed and analyzed a NSFDM to solve mathematical models using a set of rules [23]. These rules are mentioned below:

- Rule 1: The discrete and the corresponding derivative should be of the same order, if it occurs otherwise then there will be instabilities in the solution.
- Rule 2: For the discrete derivatives, the denominator functions should be expressed as a function of the step-sizes compared with those conventionally used. Considering the equation $\frac{du}{dt} = f(t, u, \lambda)$, the convection denominator is given by

$$\frac{du}{dt} \to \frac{u_{k+1} - u_k}{\Delta t}$$

which is replaced by a non-negative function

$$\phi(h) = h + O(h^2), \text{ where } h = \Delta t,\ t \to t_k = hk,\ u(t) \to u_k,$$

and k is an integer. The exact discrete first derivative is of the form

$$\frac{du}{dt} \to \frac{u_{k+1} - \psi_k}{\phi(h)}, \text{ where } \phi(h) = 1 + O(h^2)$$

- Rule 3: Non-linear terms should be replaced by nonlocal discrete representations. For example u^2 is replaced by $u_{k+1}u_k$.
- Rule 4: If there is any special condition holding for the solutions of the differential equation, then the same condition should hold for the finite difference scheme. Otherwise, numerical instabilities may arise. A good example is the condition of positivity that must be satisfied in modeling infectious diseases. If the discrete equations allows any negative solutions, then there will be numerical instabilities.

3.2.1. *Non-standard finite Difference Scheme for TB-Diabetes Model.* We consider a non-standard finite difference scheme for the first order system of differential equations in (2.1) for the TB-diabetes model.

$$\left.\begin{aligned}
\frac{S^{n+1}-S^n}{\phi_1(h)} &= \Lambda-(\alpha_1+\beta I^n+\mu)S^{n+1},\\
\frac{L_f^{n+1}-L_f^n}{\phi_2(h)} &= p\beta I^n S^{n+1}+\pi L_s^{n+1}-(\sigma_1+\mu)L_f^{n+1},\\
\frac{L_s^{n+1}-L_s^n}{\phi_3(h)} &= (1-p)\beta I^n S^{n+1}+\rho_1 I_T^{n+1}-(\alpha_2+\sigma_2+\pi+\mu)L_s^{n+1},\\
\frac{I_T^{n+1}-I_T^n}{\phi_4(h)} &= \sigma_2 L_s^{n+1}+\sigma_1 L_f^{n+1}-(\rho_1+\delta_1+\mu)I_T^{n+1},\\
\frac{D_T^{n+1}-D_T^n}{\phi_5(h)} &= \alpha_2 L_s^{n+1}+\eta D^{n+1}+\rho_2 I_{DT}^{n+1}-(\sigma_3+\mu)D_T^{n+1},\\
\frac{I_{DT}^{n+1}-I_{DT}^n}{\phi_6(h)} &= \sigma_3 D_T^{n+1}+\omega_2 C_{DT}^{n+1}-(\rho_2+\delta_2+\mu)I_{DT}^{n+1},\\
\frac{C_{DT}^{n+1}-C_{DT}^n}{\phi_7(h)} &= \theta_2 I_{DT}^{n+1}-(\omega_2+\mu+\delta_3)C_{DT}^{n+1},\\
\frac{D^{n+1}-D^n}{\phi_8(h)} &= \alpha_1 S^{n+1}+\omega_1 C^{n+1}-\eta D^{n+1}-(\theta_1+\mu)D^{n+1},\\
\frac{C^{n+1}-C^n}{\phi_9(h)} &= \theta_1 D^{n+1}-(\delta_4+\omega_1+\mu)C^{n+1}.
\end{aligned}\right\}$$

where the denominator function is given by:

$$\phi_j(h,k_j^*)=\frac{1-e^{-k_j^*h}}{k_j^*},\ \text{and}\ k_j^*=\max\{|\gamma_i|\},\ \gamma_i=\left.\frac{\partial f}{\partial x}\right|_{x=x_i}$$

with $f(\bar{x})=0,\ i=1,2,3,4,\ldots\ldots,\ j=1,2,3,4,\ldots\ldots$ Therefore, we have

$$\left.\begin{aligned}
&\phi_1(h)=\frac{1-e^{-(\alpha_1+\mu)h}}{\alpha_1+\mu},\ \phi_2(h)=\frac{1-e^{-(\sigma_1+\mu)h}}{\sigma_1+\mu},\ \phi_3(h)=\frac{1-e^{-(\alpha_2+\sigma_2+\pi+\mu)h}}{(\alpha_2+\sigma_2+\pi+\mu)},\\
&\phi_4(h)=\frac{1-e^{-(\rho_1+\delta_1+\mu h}}{\rho_1+\delta_1+\mu},\ \phi_5(h)=\frac{1-e^{-(\sigma_3+\mu)h}}{\sigma_3+\mu},\ \phi_6(h)=\frac{1-e^{-(\rho_2+\delta_2+\mu)h}}{\rho_2+\delta_2+\mu},\\
&\phi_7(h)=\frac{1-e^{-(\omega_2+\delta_3+\mu)h}}{(\omega_2+\delta_3+\mu)},\ \phi_8(h)=\frac{1-e^{-(\theta_1+\mu)h}}{(\theta_1+\mu)},\ \phi_9(h)=\frac{1-e^{-(\delta_4+\omega_1+\mu)h}}{\delta_4+\omega_1+\mu}.
\end{aligned}\right\}$$

In its explicit form, the system becomes

$$\left.\begin{aligned}
S^{n+1}&=\frac{\Lambda\phi_1(h)+S^n}{1+(\lambda+\mu+\alpha_1)\phi_1(h)},\ L_f^{n+1}=\frac{p\beta I^n S^{n+1}\phi_2(h)+\pi L_s^{n+1}\phi_2(h)+L_f^n}{1+[(\sigma_1+\mu)]\phi_2(h)},\\
L_s^{n+1}&=\frac{[(1-p)\beta I^n S^{n+1}+\rho_1 I_T^{n+1}]\phi_3(h)}{1+(\sigma_2+\mu+\alpha_2+\pi)\phi_3(h)},\ I_T^{n+1}=\frac{[\sigma_1 L_f^{n+1}+\sigma_2 L_s^{n+1}]\phi_4(h)+I_T^n}{1+(\rho_1+\delta_1+\mu)\phi_4(h)},\\
D_T^{n+1}&=\frac{[\alpha_2 L_s^{n+1}+\eta D^{n+1}+\rho_2 I_{DT}^{n+1}]\phi_5(h)+D^n}{1+(\mu+\sigma_3)\phi_5(h)},\ I_{DT}^{n+1}=\frac{[\sigma_3 D_T^{n+1}+\omega_2 C_{DT}^{n+1}]\phi_6(h)+I_{DT}^n}{1+(\theta_2+\mu+\rho_2+\delta_2)\phi_6(h)},\\
C_{DT}^{n+1}&=\frac{[\theta_2 I_{DT}^{n+1}]\phi_7+C_{DT}^n}{1+(\omega_2+\mu+\delta_3)\phi_7(h)},\ D^{n+1}=\frac{[\alpha_1 S^{n+1}+\omega_1 C^{n+1}]\phi_8(h)+D^n}{1+(\eta+\theta_1+\mu)\phi_8(h)},\ C^{n+1}=\frac{\theta_1 D^{n+1}\phi_9(h)+C^n}{1+(\omega_1+\delta_4+\mu)\phi_9(h)}.
\end{aligned}\right\}$$

The following theorems shows positivity and boundedness of the system.

Theorem 3.1. If all the initial value as well as parameter values of the discrete system are positive, then the numerical solution of the system will also be positive for all $n\geq 0$.

Proof. Since the terms in the right-hand side of (3.2.1) are all positive, it follows that this method preserves the positivity property of our model. □

Theorem 3.2. The NSFD scheme defines the discrete dynamical system on the biologically feasible region

$$D = \left\{ (S^n, L_s^n, L_f^n, I_{DT}^n, D_T^n, I_T^n, D^n, C^n) \in R_+^8 : 0 \leq S^n + L_s^n + L_f^n + I_{DT}^n + D_T^n + I_T^n + D^n + C^n < \frac{\Lambda}{\mu} \right\}.$$

Proof. The denominator of the terms in the right-hand side of (3.2.1) are greater than one, and $S^n + L_s^n + L_f^n + I_{DT}^n + D_T^n + I_T^n + D^n + C^n < \frac{\Lambda}{\mu}$ in D, it follows that the numerical solutions given out by positivity-preserving method (3.2.1) are bounded for all t. □

4. Numerical solutions

Numerical solutions are often implemented for approximating mathematical problems using numerical methods, such as finite difference methods [54]. There are used when an analytical solution is either unavailable or not feasible to compute. The approach involves discretizing the problem into a finite number of points or elements and then approximating the values of the unknowns at these points or elements. The approximations are typically obtained by solving a system of linear or nonlinear equations, which can be constructed using various numerical techniques, such as matrix algebra, interpolation, or optimization. The accuracy of a numerical solution in this paper depends on the size of the discretization, the properties of the problem, and the precision of the numerical algorithms used to solve the problem.

4.1. **Continuous-time Model.** To simulate a first-order ordinary differential equation (ODE), we use parameter values in Table 1 to compute and simulate the state variable in (2.1) via MATLAB's built-in *ode45* solver. This is achieved via the fourth-order Runge-Kutta method. The results presented in Figure 2 are based on the initial condition of $S = L_F = L_s = I_T = D_T = I_{DT} = C_{DT} = D = C = 0$.

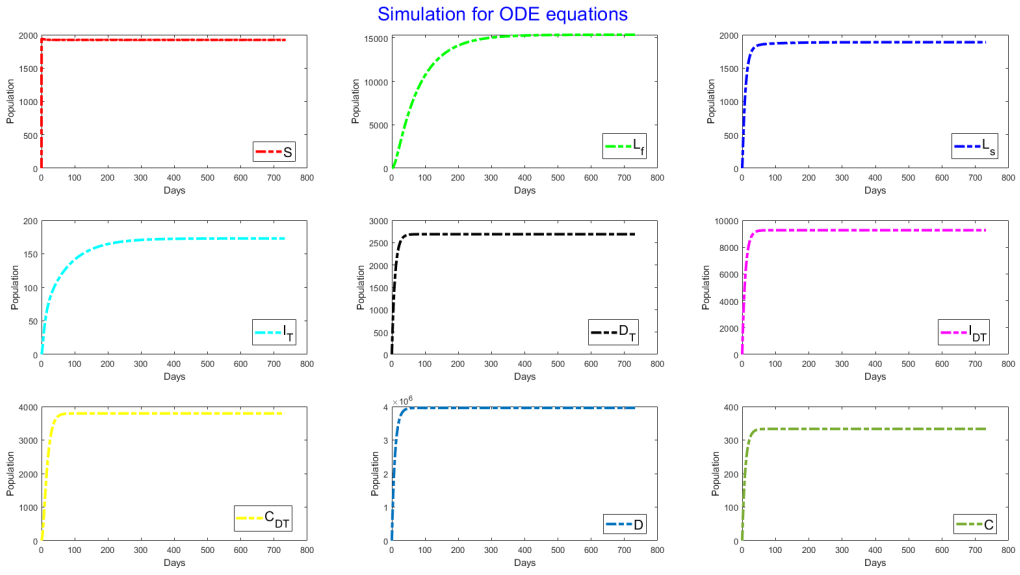


FIG. 2. The graphical simulation of all compartment in the model diagram in Figure L_s, I_{DT}, D, and C

Figure 2 indicated that S (healthy individuals) increases rapidly until an equilibrium is reached around 2000. The graphical simulation of $L_f, Ls, I_T, D_T, I_{DT}, C_{DT}, D$ and C increases gradually to around 15000, 1900, 170, 2700, 9261, 3800, 4×10^6 and 330, respectively.

4.2. **NSFD for TB-Diabetes Model.** To simulate NSFD, we use parameter values in Table 1 to compute and simulate the state variable in (3.2.1) via MATLAB's built-in *ode45* solver. This is achieved via the fourth-order Runge-Kutta method. The results presented in Figure 3 are based on the initial conditions being zeor, that is $S = L_F = L_s = I_T = D_T = I_{DT} = C_{DT} = D = C = 0$ and $h = [0 : 50]$ and $N = 99$.

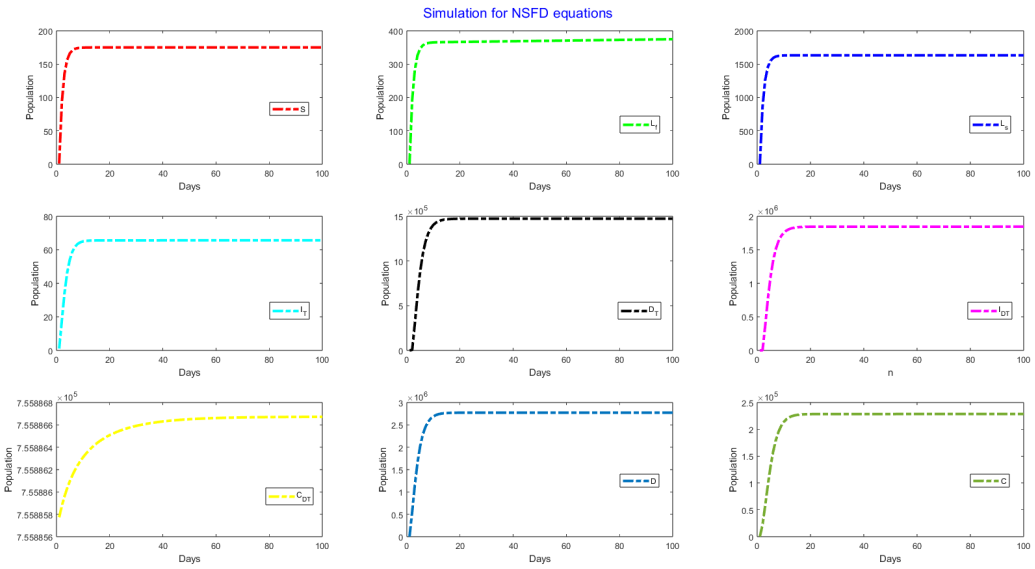


FIG. 3. Graphical simulation of NSFD for $S, L_s, I_{DT}, I_{DT}, C_{DT}, D$ and C

Figure 3 show that the simulation of the compartments reaches equilibrium points. Therefore, comparing case-by-case simulations for NSFD and order is inevitable to understand why NSFD was applied in this work. A comparison of the simulation results of NSFD and continuous-time model is presented in Figure 4.

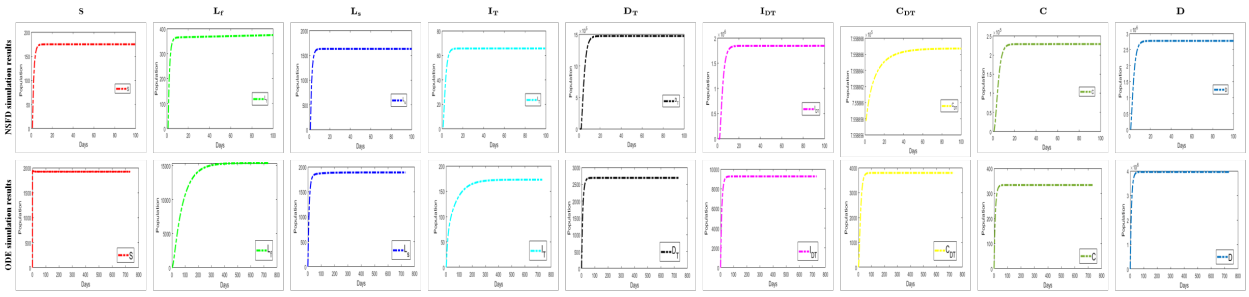


FIG. 4. Comparison of the graphical simulation of population of compartment for the system in (2.1) and (3.2.1).

Figure 4 suggests that for S under the continuous-time model and nsfd exhibit almost similar simulation results. However, the NSFD results tend to have a similar growth pattern to the continuous-time model,

which is inconsistent in cases such as S. The equilibrium points in NSFD are also attained at higher values when compared to their corresponding continuous-time model, except for those of S, L_f and I_T. In both methods, L_s and D have almost similar equilibrium points. A summary of the ablation comparison of equilibrium points attained in both cases is presented in Table 2.

TABLE 2. Comparison of numerical simulation results obtained based on continuous-time model and nsfd

Compartment	Higher Equilibrium	
	continuous-time	nsfd
S	✓	
L_f	✓	
L_s	✓	
I_T	✓	
D_T		✓
I_{DT}		✓
C_{DT}		✓
C	✓	
D		✓

Table 2 indicates that in many simulations, the continuous-time model simulation gave a higher approximated equilibrium when compared to nsfd results. The observations suggest that NSFD could be more accurate compared to continuous-time model. The accuracy could be due to overly small step sizes. For instance, an increase h, give results better results compared to lower have of h as illustrated in Figure 5

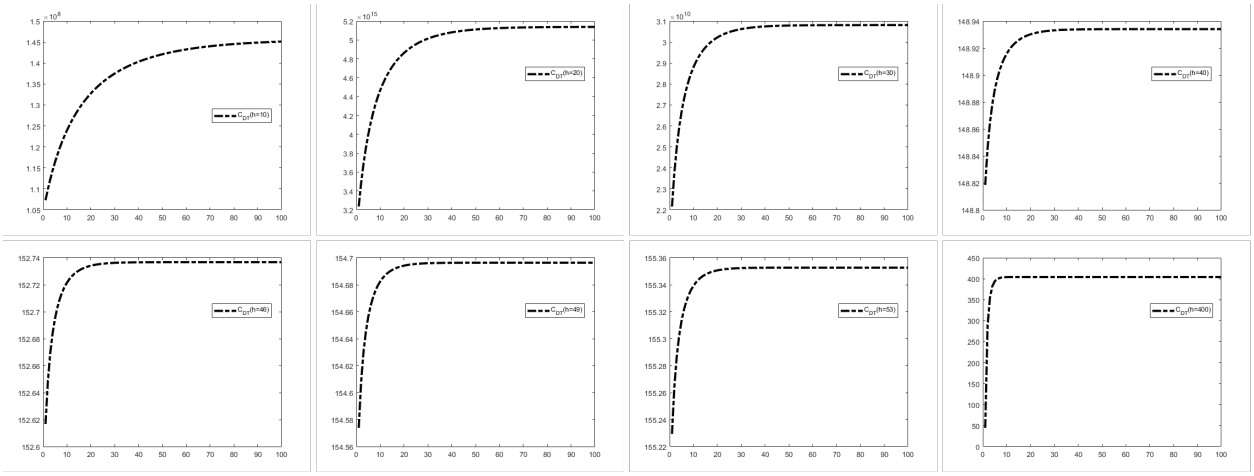
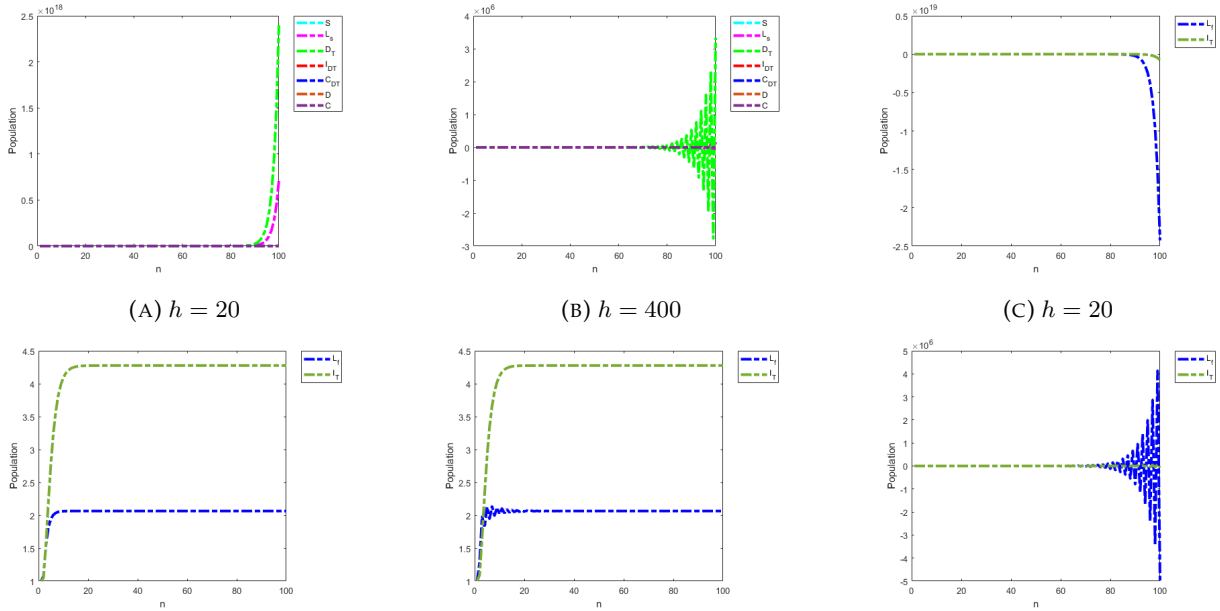


FIG. 5. Graphical simulation of population of compartment C_{DT}. h (step size) increases from left to right.

Figure 5 shows comparison of simulated result for compartment C_{DT} with varied step size h. Lower h indicate unstable results compared to higher h. However, the increase is limited to optimal results posted by other compartments. Figure 6 illustrates that optimal value of h is feasible since very higher values yields results that are uninterpreted.



(A) $h = 20$ (B) $h = 400$ (C) $h = 20$

(D) $h = 50$ optimal (E) $h = 80$ (F) $h = 400$

FIG. 6. Graphical simulation showing the role of h (step size) in determining the accuracy in the simulation. h increases from left to right.

Figure 6a and Figure 6c shows that at $h = 20$, the simulation is not presentable; hence no results are attained. At higher $h = 400$ (see Figures 6b and 6f), the simulation, like that of L_f, becomes distorted. Thus, determining h that gives optimal values for all the compartments was the first stage during the simulation. Unlike the traditional first-order continuous-time model, NSFD offers an alternative to establishing accurate results by determining optimal h. Thus, scholars could rely on it to provide insightful recommendations for problems such as controlling tuberculosis and diabetes co-infection.

4.3. **Phase planes.** The phase plane is a graphical representation of the behavior of a dynamic system over time [55]. It is a two-dimensional plot that shows the relationship between the system's state variables and their rates of change. In a phase plane plot, the horizontal axis represents the value of one state variable, while the vertical axis represents the value of another state variable. The state of the system at any given time can be represented as a point in the phase plane. The trajectories in the phase plane represent the behavior of the system over time. The direction and shape of the trajectories can provide important information about the system's stability [56]. The slope of the trajectory at any given point represents the rate of change of the state variables at that point. Different types of behavior can be observed in the phase plane, such as stable and unstable equilibria. The phase plane is a powerful tool for understanding the behavior of complex dynamic systems, such as the relationship between tuberculosis and diabetes co-infection.

The model presented in Figure 1 suggests that there is a possible 11 phase plane plots ($S-D$, $S-L_f$, $S-L_s$, L_f-L_s, L_f-I_T, L_s-I_T, L_s-D_T, D_T-D, D_T-I_{DT}, $D-C$, $I_{DT}-C_{DT}$). Figures 7 - 16 present a summary of the phase plane plots.

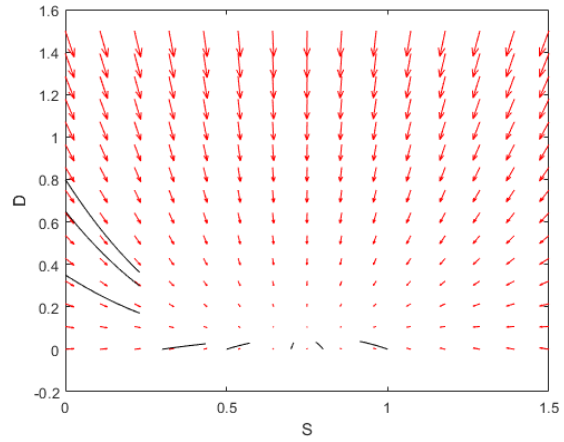


FIG. 7. S-D phase plane

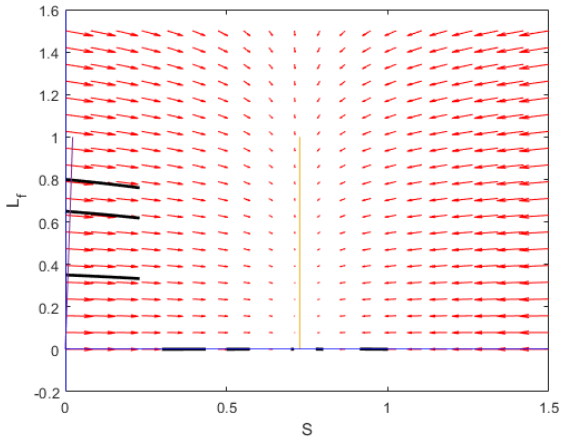


FIG. 8. S-L_f phase plane

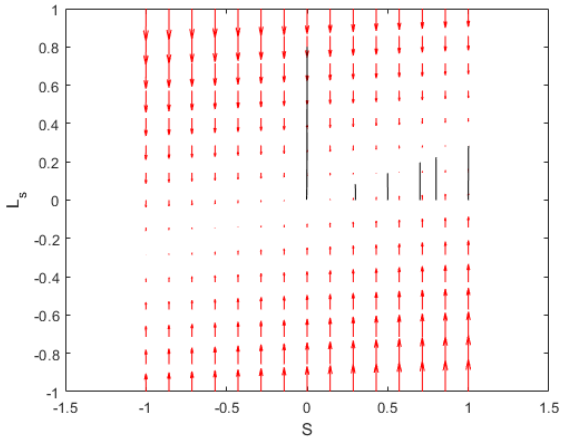


FIG. 9. S-L_s phase plane

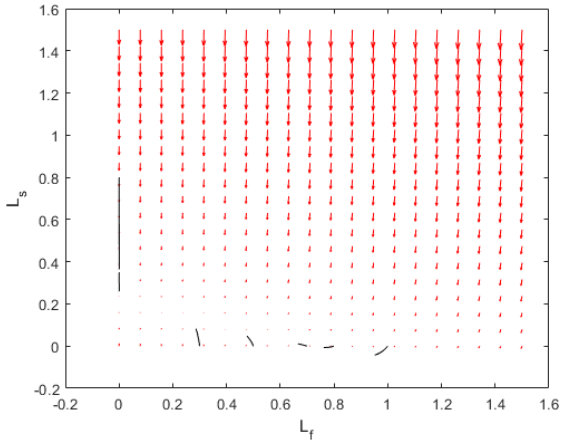


FIG. 10. L_f-L_s phase plane

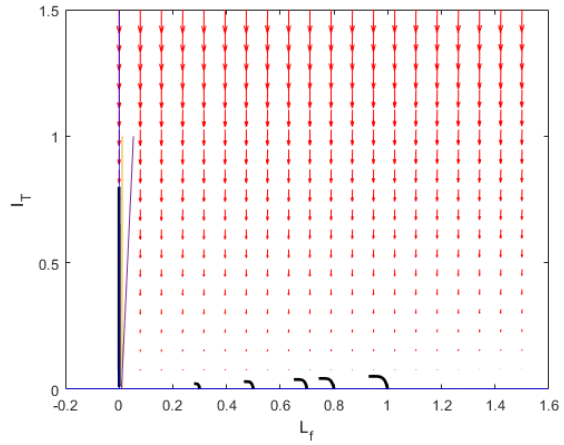


FIG. 11. L_f-I_T phase plane

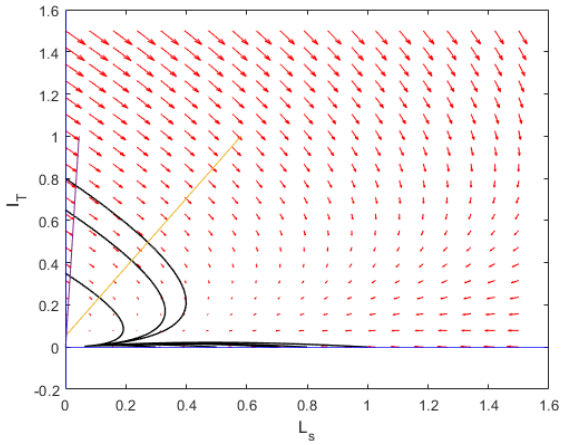


FIG. 12. L_s-I_T phase plane

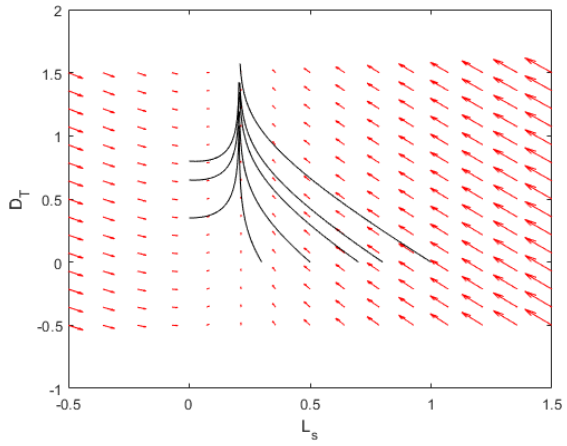


FIG. 13. L_S-D_T phase plane

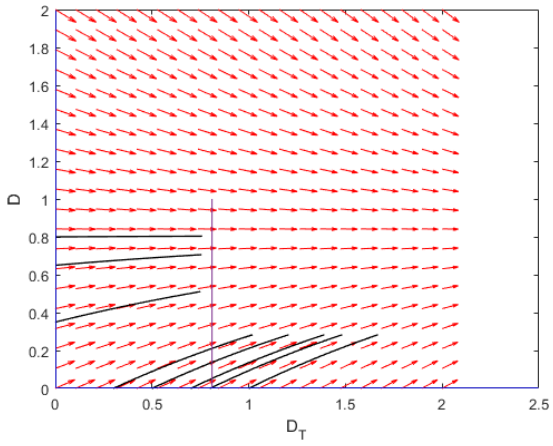


FIG. 14. D_T-D phase plane

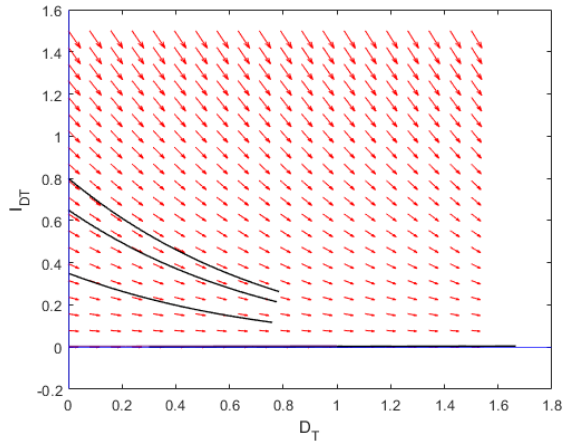


FIG. 15. D_T-I_{DT} phase plane

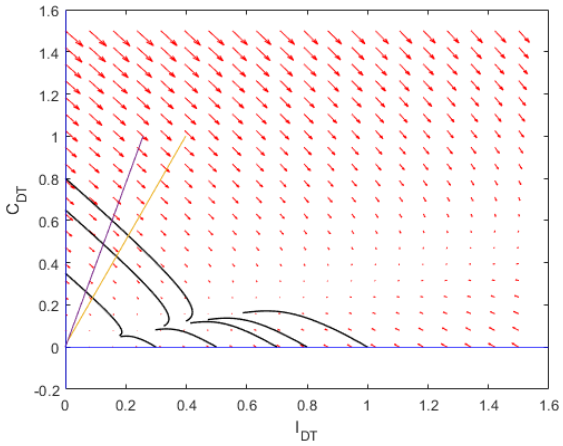


FIG. 16. C_{DT}-I_{DT} phase plane

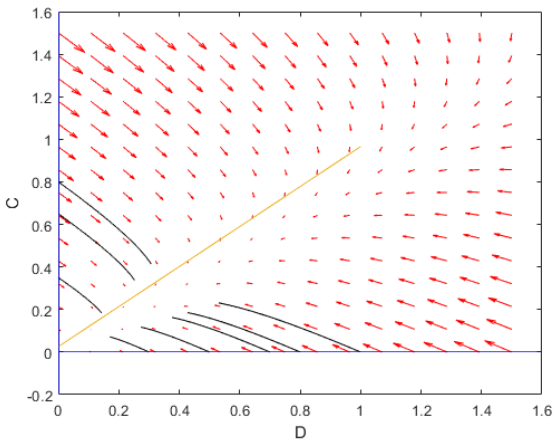


FIG. 17. D-C phase plane

Figure 7 plots a phase plane of S against D representing the state of the system. The system shows the equilibrium is at $\simeq 0.1$. All the arrows point to equilibrium, suggesting a stable system. The arrows slope towards the positive direction, suggesting S and D are increasing. The arrows converge to a stable equilibrium, suggesting asymptotically stable $S-D$. A similar observation in noted in the phase plane plot between S-L_f in Figure 8, S-L_s phase plane in Figure 9, L_f-I_T phase plane in Figure 11, L_s-I_T phase plane in Figure 12, L_S-D_T phase plane in Figure 13, D_T-D phase plane in Figure 14, D_T-I_{DT} phase plane in Figure 15, C_{DT}-I_{DT} phase plane in Figure 16 and D-C phase plane in Figure 17. However, in D-C phase plane in Figure 17, the direction of arrows at the equilibrium is points to the negative, hence D and C is decreasing. The phase plane between L_f-L_s in Figure 10 is invisible, a a sign that the system is unpredictable.

5. Conclusion

Existing studies have formulated many mathematical models for TB-Diabetes co-infection. However, the discrete TB-Diabetes models still have research value. In the current study, a NSFD scheme for TB-Diabetes co-infection is formulated. Numerical simulation is presented and compared with the corresponding first-order ode. The results indicated that the first-order ode gives a higher approximated equilibrium when compared to NSFD results. The observations suggest that NSFD could be more accurate compared to first-order ode. The accuracy could be due to overly small step sizes needed to obtain stable equilibrium. For instance, an increase h gives better results than lower h in the NSFD (see Figure 5). Although larger step sizes can save computational time and memory, they could not give meaningful results to disease control recommendations. Therefore, NSFD offers an alternative to establishing accurate results by determining optimal h. Thus, scholars could rely on it to provide insightful recommendations for problems such as controlling tuberculosis and diabetes co-infection. The phase plane (see Figures 7-17) stability conditions for different compartments were presented. The analysis is based on the convergence or divergence of the arrows from the equilibrium. The results indicated that other than that of and D-C, whose arrows point to a decreasing equilibrium, the rest are asymptotically stable, and their equilibrium is increasing. Future studies should consider formulating the proposed model with varied control parameters, such as medication, to compare the results with those from first-order ode.

Existing studies have formulated many mathematical models for TB-Diabetes co-infection. However, the discrete TB-Diabetes models still have research value. In the current study, a NSFD scheme for TB-Diabetes co-infection is formulated. Numerical simulations are presented and compared with the corresponding first-order ode. The results indicated that the first-order ode gives a higher approximated equilibrium when compared to nsfd results. The observations suggest that nsfd could be more accurate compared to first-order ode. The accuracy could be due to overly small step sizes needed to obtain stable equilibrium. For instance, increasing h gives better results than lower h in the NSFD (see Figure 5). Although larger step sizes can save computational time and memory, they could not give results prone to disease control recommendations. Therefore, NSFD offers an alternative to establishing accurate results by determining optimal h. Thus, scholars could rely on it to provide insightful recommendations for problems such as controlling tuberculosis and diabetes co-infection. The phase plane (see Figures 7-17) stability conditions different compartments. The analysis is based on the convergence or divergence of the arrows from the equilibrium. The results indicated that other than that of L_f-L_s, which is oscillatory, and D-C, whose arrows point to a decreasing equilibrium, the rest are asymptotically stable, and their equilibrium is increasing. Future studies should consider formulating the proposed model with varied control parameters, such as medication, to compare the results with those from first-order ode.

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