

An analytical approach to modeling conjunctival viral disease using fuzzy logic and time-delay dynamics

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ABSTRACT

Conjunctivitis, commonly known as pink eye, is the inflammation of the conjunctiva, often accompanied by redness, itchiness, and the discharge of thick white or greyish pus. Highly contagious in settings involving close contact, it poses significant public health and economic concerns. This study proposes a fuzzy mathematical modeling framework to investigate Conjunctival Viral Disease (CVD) transmission dynamics, with particular attention to the roles of asymptomatic carriers and environmental influences. Unlike conventional models that rely solely on deterministic parameters, the incorporation of fuzzy theory allows for representing uncertainties and variabilities inherent in real-world disease transmission. The model further incorporates time-delay terms to account for incubation periods and other latent effects, enhancing the accuracy of system dynamics. This fuzzy framework performs key analyses, including identifying equilibrium points, computation of the basic reproduction number, sensitivity analysis, and assessment of local and global stability. Numerical solutions are obtained using the Forward Euler and Nonstandard Finite Difference (NSFD) methods. The NSFD scheme is rigorously examined for convergence, non-negativity, boundedness, and consistency properties. Simulation results confirm that the NSFD approach maintains the qualitative features of the model even under larger time steps. Overall, the study underscores the importance of integrating fuzzy logic and time delays in epidemic modeling and presents a robust methodological approach for understanding and managing the spread of infectious diseases in uncertain and dynamic environments.

1. Introduction

This research aims to create a fuzzy mathematical model that studies conjunctival viral disease spread by incorporating asymptomatic carriers and environmental variables. The study pursues the integration of time delays to depict incubation timeframes and disease developmental stages that will strengthen the model's performance in handling genuine reality dynamics. Understanding equilibrium states forms one goal, while the other objectives consist of calculating basic reproduction numbers, performing sensitivity assessments, and evaluating fuzzy system stability. This research utilizes the Forward Euler method in

conjunction with NSFD to obtain numerical solutions for which the NSFD scheme receives special evaluation regarding its convergence properties while addressing non-negativity preservation, boundedness, and consistency. The research develops a comprehensive management system for infectious diseases spread under uncertain conditions.

The classical epidemic models require specific parameter values for their operation; however, these values do not adequately capture the elements of real-world disease transmission that are influenced by underlying uncertainty. The parameters in conjunctival viral disease models remain difficult to understand due to the available data showing variable contact rates and undetectable carriers. The researchers address

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the imprecision of parameters through fuzzy modeling, as this methodology effectively combines the evaluation of disease complexity with imprecise data. This motivation underpins the decision to frame the study within a fuzzy system, aiming for more realistic and applicable outcomes in uncertain environments.

Conjunctivitis, or pink eye, refers to the inflammation or infection of the conjunctiva, the transparent membrane that lines the sclera, which is the white part of the eye and covers the inner surface of the eyelids. It can be contagious, that is, caused by a virus or bacteria, and some people develop it due to an allergic reaction to pollen, smoke, dust, or toxins. With that in mind, this paper centers on viral conjunctivitis as the central subject of discussion. This is signified by redness of the conjunctiva, the white part of the eye area, itchiness of the eyes, blurred vision, and excessive tearing. Each has direct contact with an infected person, contact with the eyes, or with tears from an infected person or an object contaminated with tears, or contact with other objects contaminated with tears or flies that tend an infected eye. In recent years, viral conjunctivitis has become a significant public health concern, especially in densely populated regions like Pakistan. Despite being labeled as a "minor infection," conjunctivitis outbreaks can overwhelm healthcare systems and carry significant economic and social burdens. Nisar et al. [1] conducted a detailed analysis of a fractional-order model for eye infection with treatment dynamics. Their results show how early intervention and public awareness can reduce infection rates. Onruk and Naowarat [2] performed a sensitivity analysis to pinpoint critical parameters influencing conjunctivitis spread, highlighting the importance of model-driven policy. The 2023 surge in pink eye cases in Pakistan is well documented. Sajjad [3] called it a "silent epidemic," stressing the lack of preparedness. Ahmed et al. [4] focused on urban hotspots like Karachi and Lahore, noting behavioral and environmental factors in the outbreak. Raqib et al. [5] conducted a comprehensive review of viral strains and outbreak triggers, while Chansaenroj et al. [6] linked conjunctivitis outbreaks to enteroviruses like coxsackievirus A24, emphasizing the need for viral-specific modeling. These insights validate conjunctivitis as a timely and relevant case study to apply advanced epidemic modeling techniques, particularly fuzzy delay-based models.

Mathematical models are essential tools in epidemiology to explore disease transmission patterns and evaluate control measures for infectious diseases. During the previous few decades, mathematical modeling has become essential for epidemiology, while scientists have focused on implementing nonlinear dynamics alongside time delays and uncertainties. Fuzzy logic-based modeling has proven valuable for epidemic research because it effectively handles uncertainties, ambiguities, and imprecise data [7,8]. Since it involves crucial real-life applications, this research intends to improve epidemiological modeling techniques through fuzzy time-delayed transmission analytics for conjunctivitis. Deterministic and stochastic mathematical frameworks simplify reality through their ability to maintain disease transmission principles. The fundamental building blocks of epidemiological investigations are the Susceptible Exposed Infected Recovered (SEIR) model and its derivatives, the Susceptible Infected Recovered Cross-immune (SIRC) model, and their generalized versions. Khan et al. [9] developed a fractional-order SEIR framework through a generalized incidence rate that exhibits memory attributes while mimicking authentic epidemics. In their research, SIRC models received analysis through optimal control theory from Iacoviello and Stasio [10] to demonstrate mathematical methods for optimal vaccination and treatment strategies. Wei and Xue [11] studied extinction conditions and stability properties of generalized SEIR disease models with nonlinear incidence rates because these will determine long-term disease management outcomes. Their research demonstrates that model choice depends on specific epidemiological contexts. According to Champagne and Cazelles [12], dengue modeling requires stochastic approaches because selecting this method ensures better results when data quantities and signal quality are low. Zhang and Ma [13] presented a saturating contact SEIR model that prevents unrealistic infection number

increases. Mangal et al. [14] further conducted their research through the introduction of media awareness along with fractional stochastic components to understand disease-human behavior interactions. The analyzed works show that adaptable, flexible mathematical models should evolve alongside disease control systems and their dynamic settings.

Uncertainty remains an essential hindrance for modern mathematical models, which aim to model real-life disease patterns. The complete knowledge of transmission rates, recovery stages, and public behavior data is rarely achievable. Fuzzy logic introduces a disruptive methodology for this purpose. Zadeh initiated the fuzzy theory as a new concept in 1965 [15]. Alamin et al. [16] established fundamental principles for stability analysis of difference equations in fuzzy environments, which serve as the basis for discrete fuzzy models. Lefevre et al. [17] presented a comprehensive overview of fuzzy logic applications in biological systems, arguing that fuzzy techniques are particularly suited for systems with imprecise boundaries and stochasticity. Shatanawi et al. introduced fractional derivatives into fuzzy models, enabling memory and non-locality to coexist with fuzziness [18]. Mahato et al. [19] analyzed fuzzy epidemic models with vaccination, providing both analytical solutions and simulations that mirror real-world behavior. Abdy et al. [20] applied the fuzzy susceptible-infected-recovered (SIR) models to COVID-19, showing how fuzzy parameters better captured the variability in disease outcomes. In behavioral modeling, fuzzy logic has been applied to smoking cessation by Allaoui et al. [21] and general SIR dynamics by Verma et al. [22], who emphasized fuzzy membership functions to characterize susceptible and infected classes. In a pioneering study, Ortega et al. [23] introduced fuzzy dynamical systems into epidemic modeling as early as 2000, predicting the trajectory of this now-burgeoning field.

Using fuzzy theory specifically strengthens epidemic modeling because it best handles scarce, precise data and normal uncertainty. The integration process allows experts to include knowledge, linguistic elements, and imprecise variables to create a realistic model of disease spread. The modeling methodology succeeds best during conjunctival viral disease assessments because it handles difficult-to-quantify variables of individual differences, environmental effects, and symptomatic carriers. The fuzzy framework enables researchers to include variable parameter values because it addresses the natural vagaries of biological systems, while traditional models function with static, fixed parameters. When modeling traditional systems, the application becomes limited, and predictions become inaccurate due to minor changes in parameter values. The fuzzy approach permits researchers to determine possible parameter values for transmission and recovery rates and contact levels; thus, it can simulate various disease progressions instead of producing just a single certain outcome. This approach provides enhanced comprehension regarding how slight uncertainties transform disease patterns and stabilizing states and control strategy outcomes. The conduct of sensitivity tests inside a fuzzy environment allows researchers to precisely recognize important parameters for guiding intervention strategies. The fuzzy logic modeling process contains challenges since it requires a subjective design of fuzzy sets and higher computational needs. However, it still proves superior as a flexible model type for infectious disease analysis compared to traditional integer-order models.

Fuzzy modeling uses a mathematical method that effectively handles crucial ambiguous conditions appearing during underreported case reports or delayed reporting and variable environmental influences. Fuzzy systems that apply truth degrees and membership functions replace models based on crisp values, leading to improved model reliability. Fuzzy mathematics combined with time-delayed modeling draws significant attention, enabling researchers to develop multi-scale epidemic modeling for emerging diseases and re-emerging health threats, including conjunctivitis.

Time delays are essential when analyzing infectious disease models for interpreting disease incubation times, treatment delays, and

detection intervals. According to Pathak and Kota, time-delayed epidemic models offer better disease progression simulation because they include the natural delays that occur during transmission and disease control processes [24]. The incidence rates became general in Allali's work [25] as he extended the previous research by creating a two-strain model to describe intricate disease connections. Guglielmi et al. [26] showed how the delay differential equations apply to the COVID-19 epidemic spreading through spatial areas while demonstrating improved predictive capabilities. Their research confirms that spatially resolved delay models work effectively in practical decision-making. The research by Niu et al. [27] proves that integrating infectious disease modeling with public health policies generates superior response outcomes, particularly if time-based factors are considered. Ivorra et al. [28] added unreported infections and time delays when modeling COVID-19 transmission in China to demonstrate the need for accurate assumptions in disease modeling. The SIRC model for influenza A by Casagrandi et al. [29] included a recovered and contagious class, as described by their work. Suryaningrat et al. [30] expanded the SIR framework using fuzzy parameters and time delays to enable the modeling of COVID-19 dynamics in Indonesia, as shown in their research. Time delays and fuzzy parameters influenced disease dynamics and changed stability and bifurcation patterns. Ben Lahbib and Azrar [31] explored stochastic SIRC models with time delay and nonlinear incidence, highlighting delay-driven fluctuations. Similarly, Mezouaghi et al. [32] studied partial protection delays, showing their significant effect on global stability. Yan and Jia [33] analyzed Hopf bifurcation under delayed medical resource constraints, while Beretta and Breda [34] focused on constant latency and infectious periods. Liu [35] investigated general nonlinear incidence in a delayed SIR model, emphasizing the role of functional response forms. Rihan [36], in his seminal work, further unified delay differential equations with biological systems, establishing a foundational framework. Recent works by Meziane et al. [37], Ghosh et al. [38], and Almuqati and Allehiany [39] have continued to refine these models to incorporate logistic growth, eco-epidemiological interactions, and disease duration-based delays. Hussien and Naji [40] contributed a delay-based model with nonlinear eco-epidemiological feedback, stressing the ecosystem's effect on disease dynamics. These studies collectively establish time delay as a crucial structural feature in epidemic modeling, necessary for aligning theoretical predictions with empirical data.

This study presents a novel fuzzy time-delayed epidemic model of CVD, incorporating asymptomatic carriers and environmental variability, which are often overlooked in traditional models. The model integrates fuzzy theory to address uncertainties in transmission parameters and incorporates time delays to better reflect disease dynamics. Additionally, a comparative numerical analysis of the Forward Euler and NSFD methods highlights the advantages of the NSFD method in capturing accurate and stable results under fuzzy conditions.

The research utilized the Susceptible Exposed Infected Vaccinated Recovered and Treated (SEIVRT) model because it effectively models the intricate transmission dynamics of the conjunctivitis virus, which include both incubation durations and treatment responses. This model accounts for the multiple infection and treatment phases as a crucial method to understand virus spread and control strategies. The paper's organization is as follows: Section 2 presents the development of a fuzzy-parameterized delayed mathematical model for CVD dynamics that is established and analyzed; it defines the endemic equilibrium, and considers the Reproduction number along with sensitivity analysis for the stabilization of the model. Section 3 focuses on the discretization of the parameters using the Euler and NSFD enumeration methods. While evaluating the study at a specific class level, Section 4 quantitatively compares the outcomes, and Section 5 encompasses the synthesis of all the conclusions.

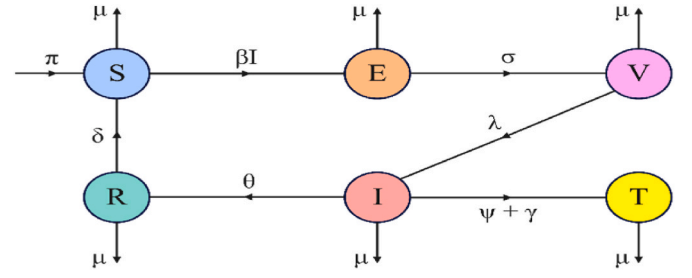


Fig. 1. Flowchart of CVD infection transmission dynamics.

2. Model formulation

We considered the SEIVRT model as presented by Kulachi et al. [41]. First, we will mention some useful definitions for this study.

2.1. Fuzzy subset

A fuzzy subset (or fuzzy set) of T is defined as a function $\mu_{\tilde{C}}$ where $\mu_{\tilde{C}} : T \rightarrow [0, 1]$ is represented as $\tilde{C} = \{x \mid \mu_{\tilde{C}} : x \in T\}$, and $\mu_{\tilde{H}}$ is the membership function linked to the fuzzy set.

2.2. Fuzzy number

The Fuzzy subset $\tilde{C}$ of T qualifies as a fuzzy number if all its α -levels are closed and non-empty intervals within T , and the Support of $\mu_{\tilde{C}}$ is bounded where $\text{Support} = \{x : \mu_{\tilde{C}}(x) > 0\} \subset \mathbb{R}$.

2.3. Fuzzy triangular number (FTN)

A fuzzy triangular number, defined in Ref. [42], has the membership function $\mu_{\tilde{F}}(\rho)$, which is expressed as

$$\mu_{\tilde{F}}(\rho) = \begin{cases} 0, & \rho \leq l \\ \frac{\rho - l}{v - l}, & l \leq \rho \leq m \\ \frac{n - \rho}{n - m}, & m < \rho \leq n \\ 0, & n < \rho \end{cases} \quad (1)$$

Where $l \leq m \leq n$.

2.4. Fuzzy triangular number (FTN) value

The Central Value of an FTN $\tilde{F}$ is presented by [43]

$$E(\tilde{F}) = \frac{l + 2m + n}{4}. \quad (2)$$

2.5. Fuzzified parametric model

A disease progression model is considered to have parameters that are better expressed as fuzzy sets rather than numbers. With this approach, each parameter is classified as a fuzzy set in which membership characterizes all possible values [44]. This method efficiently quantifies ambiguity, vagueness, or irregularity within the framework, providing additional assistance when empirical data is conflicting or intrinsic random errors disturb the system. In various conditions and contexts, fuzzy parameters are usually more applicable in probability models than fixed parameters. The corresponding differential equations originated from the flowchart of the model, as shown in Fig. 1.

In this compartmental model presented in Fig. 1, π denotes the constant recruitment rate of individuals into the susceptible population

(S) for the transmission dynamics of conjunctival disease. The parameter β represents the effective contact rate through which susceptible individuals become exposed (E) due to interaction with infectious individuals (I). The exposed individuals may progress to the vaccinated class (V) at a rate σ , indicating preventive medical intervention. Despite vaccination, individuals may still become infected at a rate λ , moving them into the infected class. Infected individuals may then begin treatment at a combined rate of $\psi + \gamma$, where ψ represents the natural progression toward treatment and γ captures the active intervention rate, moving them to the treated class (T).

Additionally, infected individuals may recover and move to the recovered class (R) at a rate θ . In contrast, some recovered individuals may lose immunity and return to the susceptible class at a rate δ . The natural death rate μ affects all compartments equally. This model captures the critical pathways of disease transmission prevention through vaccination, treatment, and recovery within a population affected by conjunctival disease.

$$\begin{cases} \frac{dS}{dt} = \pi - \beta SI - \mu S + \delta R, \\ \frac{dE}{dt} = \beta SI - (\sigma + \mu)E, \\ \frac{dV}{dt} = \sigma E - (\lambda + \mu)V, \\ \frac{dI}{dt} = \lambda V - (\psi + \gamma + \theta + \mu)I, \\ \frac{dR}{dt} = \theta I - (\delta + \mu)R, \\ \frac{dT}{dt} = (\psi + \gamma)I - \mu T. \end{cases} \quad (3)$$

A fuzzy framework extends the classical model by incorporating real-world data imprecision that affects transmission and treatment processes in conjunctival disease spread. The model represents transmission rate β as fuzzy parameter $\beta(\rho)$ and treatment rate ψ as $\psi(\rho)$, through which ρ indicates the uncertainty level. This transformation enables a model structure that represents disease dynamic behavior by showing varied patterns while delivering secure analytical possibilities and interpretation results.

The fuzzified form of the preceding model presented in (3) is defined as:

$$\begin{cases} \frac{dS}{dt} = \pi - \beta(\rho)SI - \mu S + \delta R, \\ \frac{dE}{dt} = \beta(\rho)SI - (\sigma + \mu)E, \\ \frac{dV}{dt} = \sigma E - (\lambda + \mu)V, \\ \frac{dI}{dt} = \lambda V - (\psi(\rho) + \gamma + \theta + \mu)I, \\ \frac{dR}{dt} = \theta I - (\delta + \mu)R, \\ \frac{dT}{dt} = (\psi(\rho) + \gamma)I - \mu T. \end{cases} \quad (4)$$

These parameters are denoted as $\beta(\rho)$ and $\psi(\rho)$, respectively, and are designed as:

$$\beta(\rho) = \begin{cases} 0, & \rho \leq \rho_{\min} \\ \frac{\rho - \rho_{\min}}{\rho_{\max} - \rho_{\min}}, & \rho_{\min} < \rho \leq \rho_{\max} \\ 1, & \rho_{\max} \leq \rho. \end{cases} \quad (5)$$

Where $\rho_{\min}$ and $\rho_{\max}$ are the minimum and maximum values of the variable ρ beyond which the function $\beta(\rho)$ becomes constant. and

$$\psi(\rho) = \frac{(\psi_0 - 1)}{\rho_{\max}} \rho + 1, \quad 0 \leq \rho \leq \rho_{\min} \quad (6)$$

Where ψ_0 is a constant parameter in $\psi(\rho)$ that determines the slope of the linear function in the interval $0 \leq \rho \leq \rho_{\min}$.

2.6. Fuzzified time-delay model

Infectious disease differential models are characterized by a significant period shift between the infection and the transmission phases, since incorporating time delays is crucial for accurately analyzing and predicting the progression of such infectious diseases. This is the time taken by an individual of the population to get infected by the virus from the source to the point when such an individual turns into an infective agent and can infect other people and is denoted by τ . The inclusion of the $e^{-\mu\tau}$ is given based on what for some periods it is believed to reduce linearly, as given by the mortality or recovery rate at τ . Therefore, the standard fuzzy epidemic model is extended to the time-lagged fuzzy epidemic model by introducing the factor $(t - \tau)e^{-\mu\tau}$ among the compartments of susceptible and exposed populations.

Outlined below is the fuzzy model incorporating time delay for CVD

$$\begin{cases} \frac{dS}{dt} = \pi - \beta(\rho)S(t - \tau)I(t - \tau)e^{-\mu\tau} - \mu S + \delta R, \\ \frac{dE}{dt} = \beta(\rho)S(t - \tau)I(t - \tau)e^{-\mu\tau} - (\sigma + \mu)E, \\ \frac{dV}{dt} = \sigma E - (\lambda + \mu)V, \\ \frac{dI}{dt} = \lambda V - (\psi(\rho) + \gamma + \theta + \mu)I, \\ \frac{dR}{dt} = \theta I - (\delta + \mu)R, \\ \frac{dT}{dt} = (\psi(\rho) + \gamma)I - \mu T. \end{cases} \quad (7)$$

2.7. The fuzzy basic reproductive number BRN ($\mathcal{R}_0^f$)

The basic reproduction number $\mathcal{R}_0$ is a crucial tool for describing the transmission dynamics of infections in the population. To compute the basic reproduction number, we apply the next-generation matrix method. Now, from system (7), we can obtain the vectors for the new infection and transition terms, respectively, as

$$f = \begin{bmatrix} \beta(\rho)SIe^{-\mu\tau} \\ 0 \\ 0 \end{bmatrix}, \quad v = \begin{bmatrix} (\sigma + \mu)E \\ -\sigma E + (\lambda + \mu)V \\ -\lambda V + (\psi(\rho) + \gamma + \theta + \mu)I \end{bmatrix},$$

where $\mathcal{F}$ and $\mathcal{V}$ are the Jacobians of f and v , respectively.

$$\mathcal{F} = \begin{bmatrix} 0 & 0 & \beta(\rho)\pi e^{-\mu\tau} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\sigma + \mu) & 0 & 0 \\ -\sigma & (\lambda + \mu) & 0 \\ 0 & -\lambda & (\psi(\rho) + \gamma + \theta + \mu) \end{bmatrix}.$$

So, the regeneration matrix is given by

$$\mathcal{F}\mathcal{V}^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta(\rho)\sigma\lambda\pi e^{-\mu\tau}}{\mu(\sigma + \mu)(\lambda + \mu)(\psi(\rho) + \gamma + \theta + \mu)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

Since the basic reproduction number $\mathcal{R}_0$ is the spectral radius of the next-generation matrix, the spectral radius of $\mathcal{F}\mathcal{V}^{-1}$ is

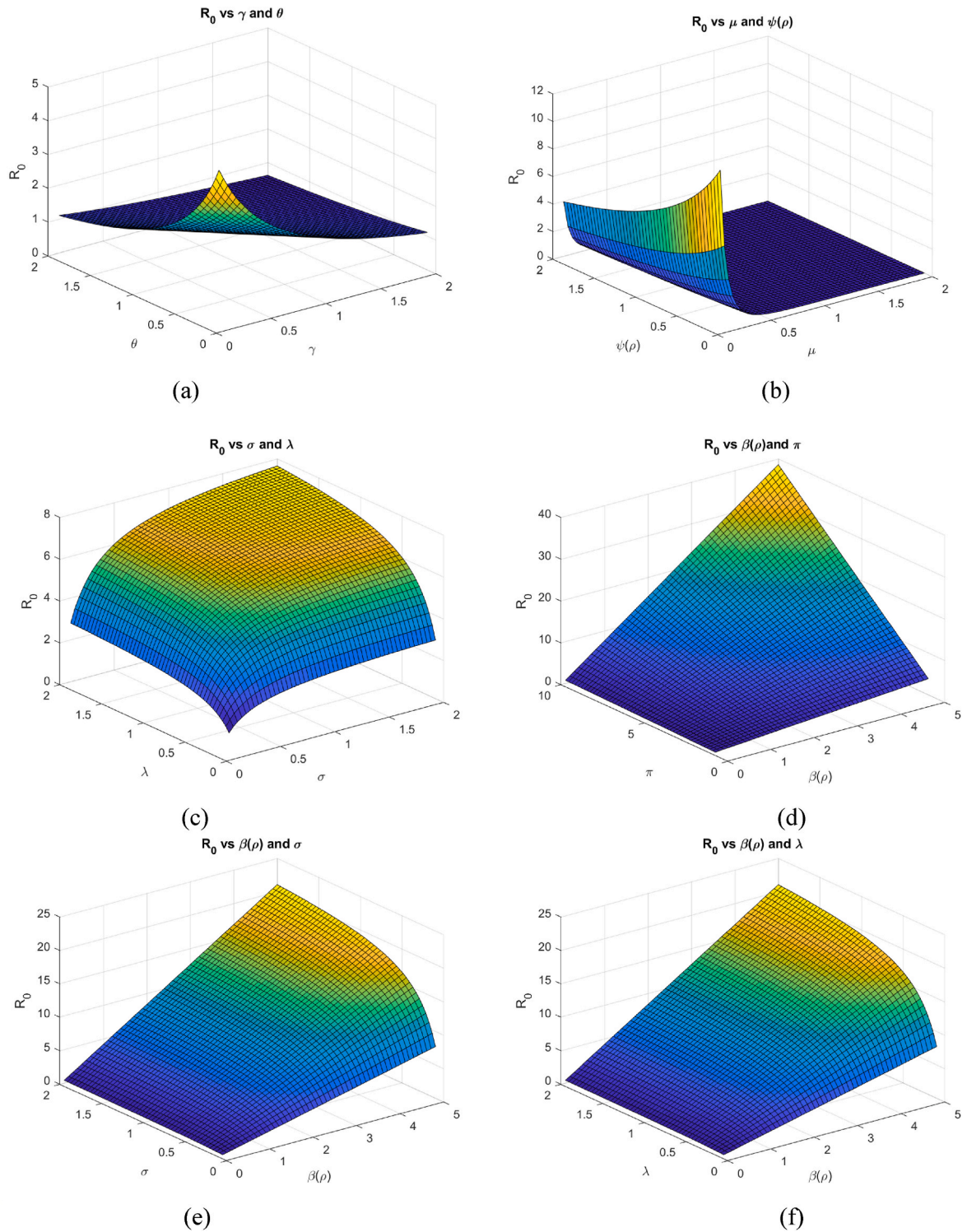


Fig. 2. Different parameters influence reproductive number behavior in the developed system.

$$\mathcal{R}_0 = \frac{\beta(\rho)\sigma\lambda\pi e^{-\mu\tau}}{\mu(\sigma + \mu)(\lambda + \mu)(\psi(\rho) + \gamma + \theta + \mu)}.$$

The BRN $\mathcal{R}_0$ by inserting the DFE point is given by

$$\mathcal{R}_0 = \frac{\beta(\rho)\sigma\lambda\pi e^{-\mu\tau}}{\mu k_1 k_2 k_3}. \quad (8)$$

Where $k_1 = (\sigma + \mu)$, $k_2 = (\lambda + \mu)$, $k_3(\psi(\rho) + \gamma + \theta + \mu)$, $k_4 = (\delta + \mu)$ and $k_5 = (\psi(\rho) + \gamma)$.

The value of the FBRN number $\mathcal{R}_0$ is determined as shown below:

Case 1. If $\rho \leq \rho_{min}$, then $\beta(\rho) = 0$ and we obtain

$$\mathcal{R}_0(\rho) = 0.$$

Case 2. If $\rho_{min} < \rho \leq \rho_{max}$, we have $\beta(\rho) = \frac{\rho - \rho_{min}}{\rho_{max} - \rho_{min}}$, and we obtain

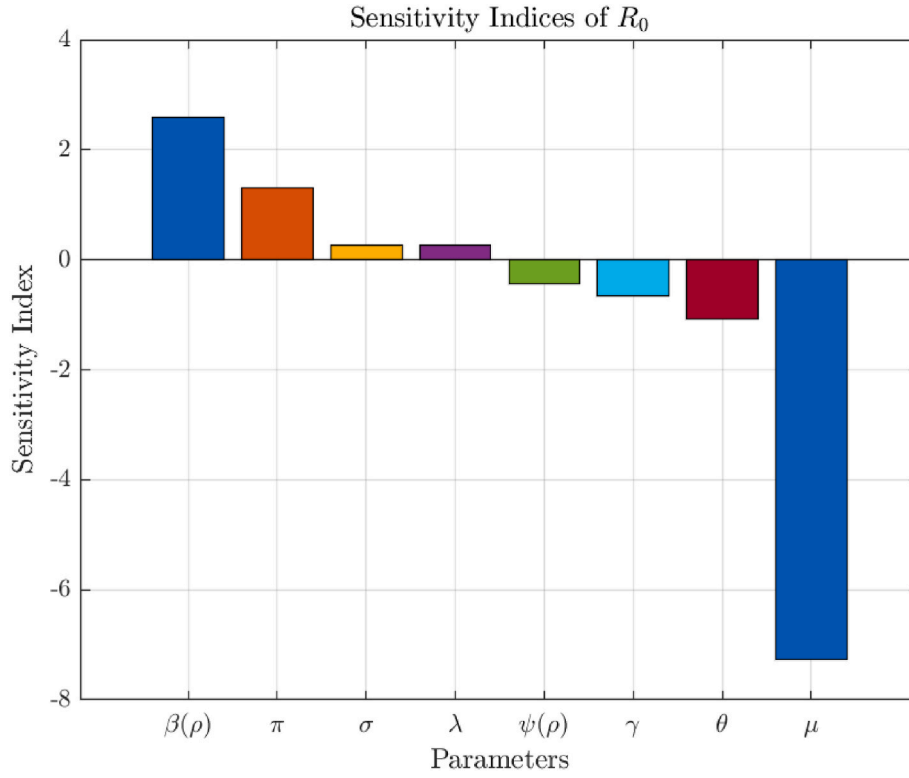


Fig. 3. Sensitivity of the basic reproduction number $\mathcal{R}_0$ to changes in the model parameters using sensitivity indices.

$$\mathcal{R}_0(\rho) = \frac{\beta(\rho)\sigma\lambda\pi e^{-\mu\tau}}{\mu k_1 k_2 k_3}.$$

Case 3. If $\rho_{max} \leq \rho$, we have $\beta(\rho) = 1$ and we obtain

$$\mathcal{R}_0(\rho) = \frac{\sigma\lambda\pi e^{-\mu\tau}}{\mu k_1 k_2 k_3}.$$

$\mathcal{R}_0(\rho)$ can be written as

$$\mathcal{R}_0(\rho) = \left(0, \frac{\beta(\rho)\sigma\lambda\pi e^{-\mu\tau}}{\mu k_1 k_2 k_3}, \frac{\sigma\lambda\pi e^{-\mu\tau}}{\mu k_1 k_2 k_3}\right).$$

As per [43], the fuzzy reproductive number is expressed as:

$$\mathcal{R}_0^f = E[\mathcal{R}_0(\rho)],$$

$$\mathcal{R}_0^f = \frac{\sigma\lambda\pi e^{-\mu\tau}(2\beta(\rho) + 1)}{4\mu k_1 k_2 k_3}. \quad (9)$$

2.8. Sensitivity analysis of $\mathcal{R}_0$

This study investigates the proposed CVD model through sensitivity analysis to evaluate how various factors influence the dynamics of the disease. This work explores the role of each parameter in the spread of conjunctivitis. Such insights are crucial for developing strategic public health responses, as they enable us to identify key factors to manipulate without significantly disrupting the model's behavior. Moreover, understanding sensitivity aids in enhancing public health interventions, particularly by refining isolation strategies and boosting recovery rates for more effective outbreak management. The sensitivity index of a parameter is defined as follows [45]:

$$\zeta(v) = \frac{q}{R_0} \times \frac{\partial}{\partial q}(\mathcal{R}_0),$$

Where ζ denote sensitivity of the parameter q . We calculate the sensitivity of $\beta(v)$ as

$$\zeta(\beta(\rho)) = \frac{\beta(\rho)}{\mathcal{R}_0} \times \frac{\partial}{\partial \beta}(\mathcal{R}_0) = 1,$$

Similarly,

$$\zeta(\pi) = \frac{\pi}{\mathcal{R}_0} \times \frac{\partial}{\partial \pi}(\mathcal{R}_0) = 1,$$

$$\zeta(\sigma) = \frac{\sigma}{\mathcal{R}_0} \times \frac{\partial}{\partial \sigma}(\mathcal{R}_0) = \frac{\sigma\mu}{k_1},$$

$$\zeta(\lambda) = \frac{\lambda}{\mathcal{R}_0} \times \frac{\partial}{\partial \lambda}(\mathcal{R}_0) = \frac{\lambda\mu}{k_2},$$

$$\zeta(\psi(\rho)) = \frac{\psi(\rho)}{\mathcal{R}_0} \times \frac{\partial}{\partial \psi(\rho)}(\mathcal{R}_0) = -\frac{\psi(\rho)}{k_3},$$

$$\zeta(\gamma) = \frac{\gamma}{\mathcal{R}_0} \times \frac{\partial}{\partial \gamma}(\mathcal{R}_0) = -\frac{\gamma}{k_1},$$

$$\zeta(\theta) = \frac{\theta}{\mathcal{R}_0} \times \frac{\partial}{\partial \theta}(\mathcal{R}_0) = -\frac{\theta}{k_1},$$

$$\zeta(\mu) = \frac{\mu}{\mathcal{R}_0} \times \frac{\partial}{\partial \mu}(\mathcal{R}_0) = -\frac{\mu(\tau + k_2 k_3 + k_1 k_3 + k_1 k_2) + 1}{k_1 k_2 k_3}.$$

The sensitivity analysis of the basic reproduction number for conjunctivitis disease is depicted in Fig. 2, illustrating how various parameters affect disease dynamics. Plot (a) shows the relationship between $\mathcal{R}_0$, the vaccination rate θ , and the recovery rate without treatment γ . $\mathcal{R}_0$ is most sensitive when both rates are low, indicating a higher potential for disease spread. Plot (b) highlights the impact of the death rate μ and the recovery rate with treatment $\psi(\rho)$. As μ increases, $\mathcal{R}_0$ typically decreases, suggesting that higher mortality lowers

transmission potential. Conversely, plot (c) demonstrates that as the vaccination rate σ increases, $\mathcal{R}_0$ declines, while higher incubation rates λ elevate $\mathcal{R}_0$. Plots (d) and (e) focus on the fuzzy transmission rate $\beta(\rho)$ and inclusion rate π , indicating that increases in either lead to significant rises in $\mathcal{R}_0$. Finally, plot (f) illustrates that while $\mathcal{R}_0$ is sensitive to changes in both the transmission and incubation rates, it is more influenced by variations in the transmission rate. This analysis underscores the importance of vaccination and treatment strategies in controlling CVD outbreaks.

The bar chart presented in Fig. 3 represents the sensitivity indices of the reproductive number $\mathcal{R}_0$ for conjunctivitis disease concerning various parameters, showing the relative influence of each on the spread of the disease. The transmission rate $\beta(\rho)$ and the inclusion rate π have positive sensitivity indices, meaning an increase in these values leads to a higher $\mathcal{R}_0$, contributing to a greater spread of the disease. The vaccination rate σ and incubation rate λ have smaller positive indices, suggesting a relatively lower but still positive impact on $\mathcal{R}_0$. Conversely, the death rate μ has the most significant negative sensitivity, indicating that higher death rates strongly reduce $\mathcal{R}_0$, limiting the spread. Recovery rates with treatment $\psi(\rho)$ without medication γ and with medication θ also have negative indices, signifying that increased recovery lowers $\mathcal{R}_0$ and controls the disease spread. Overall, the Plot highlights that transmission and inclusion rates are the most influential in increasing $\mathcal{R}_0$, while death and recovery rates are key in reducing it.

2.9. Equilibrium analysis

Case 1. If $\rho \leq \rho_{\min}$, then $\beta(\rho) = 0$ and we obtain

$$E_p^0(S^0, E^0, V^0, I^0, R^0, T^0) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0\right),$$

Case 2. If $\rho_{\min} < \rho \leq \rho_{\max}$, we have $\beta(\rho) = \frac{\rho - \rho_{\min}}{\rho_{\max} - \rho_{\min}}$, and we obtain

and $E_p^*(S^*, E^*, V^*, I^*, R^*, T^*)$ we obtain

$$S^* = \frac{k_1 k_2 k_3}{\sigma \lambda \beta(\rho) e^{-\mu\tau}},$$

$$E^* = \frac{k_2 k_3 I^*}{\sigma \lambda},$$

$$V^* = \frac{k_3 I^*}{\lambda},$$

$$I^* = \frac{\frac{\mu M}{\beta e^{-\mu\tau}} - \pi}{\left(\frac{\partial \theta}{k_1} - M\right)},$$

$$R^* = \frac{\theta I^*}{k_4},$$

$$T^* = \frac{k_5 I^*}{\mu}.$$

where $M = \frac{k_1 k_2 k_3}{\sigma \theta}$.

Case 3. If $\rho_{\max} \leq \rho$, we have $\beta(\rho) = 1$ and we obtain $E_p^{**}(S^{**}, E^{**}, V^{**}, I^{**}, R^{**}, T^{**})$ as

$$S^{**} = \frac{k_1 k_2 k_3}{\sigma \lambda \beta(\rho) e^{-\mu\tau}},$$

$$E^{**} = \frac{k_2 k_3 I^{**}}{\sigma \lambda},$$

$$V^{**} = \frac{k_3 I^{**}}{\lambda},$$

$$I^{**} = \frac{\frac{\mu M}{\beta e^{-\mu\tau}} - \pi}{\left(\frac{\partial \theta}{k_1} - M\right)},$$

$$R^{**} = \frac{\theta I^{**}}{k_4},$$

$$T^{**} = \frac{k_5 I^{**}}{\mu}.$$

2.10. Stability behavior analysis

At the Disease-Free Equilibrium (DFE), the Jacobian matrix of system (7) is expressed as:

$$J(E_p^0) = \begin{bmatrix} -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & -k_1 & 0 & 0 & 0 & 0 \\ 0 & \sigma & -k_2 & 0 & 0 & 0 \\ 0 & 0 & \lambda & -k_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_4 & 0 \\ 0 & 0 & 0 & -k_5 & 0 & -\mu \end{bmatrix},$$

The matrix has the following eigenvalues $\lambda_1 = -\mu < 0$, $\lambda_2 = -k_1 < 0$, $\lambda_3 = -k_2 < 0$, $\lambda_4 = -k_3 < 0$, $\lambda_5 = -k_4 < 0$, $\lambda_6 = -k_5 < 0$. As all eigenvalues are negative, we deduce that the system of differential equations in the SEVIRT model is locally asymptotically stable at E_p^0 .

3. Numerical simulation

By integrating mathematical models with numerical algorithms, computational simulations enable scientists to explore complex phenomena, yielding insights that are often beyond the reach of direct experimentation. These simulations transform continuous mathematical systems into discrete forms, facilitating the study of system behavior under various conditions. To solve the discretized governing equations, we employ a range of numerical techniques, including both the Runge-Kutta method and the method of lines. This study specifically illustrates the application of the Runge-Kutta 5th order scheme and the NSFD approach to solve the proposed model.

3.1. Euler's forward method

The forward Euler technique is a frequently recognized explicit first-order numeric technique for resolving ordinary differential equations. Acclaimed for its computing power, it effectively furnishes timely estimates of solution trajectories over time. We implement a forward Euler scheme for the system mentioned above (7).

$$S^{n+1} = S^n + h(\pi + h\beta(\rho)I^n S^n e^{-\mu\tau} - \mu S^n + \delta R^n), \quad (10)$$

$$E^{n+1} = E^n + h(\beta(\rho)I^n S^n e^{-\mu\tau} - k_1 E^n), \quad (11)$$

$$V^{n+1} = V^n + h(\sigma E^n - k_2 V^n), \quad (12)$$

$$I^{n+1} = I^n + h(\lambda V^n - k_3 I^n), \quad (13)$$

$$R^{n+1} = R^n + h(\theta I^n - k_4 R^n), \quad (14)$$

$$T^{n+1} = T^n + h(k_5 I^n - \mu T^n). \quad (15)$$

$$\text{Where } k_4 = (\delta + \mu), k_5 = (\psi(\rho) + \gamma). \quad (16)$$

3.2. NSFD scheme

The NSFD technique is an advanced and efficient way to solve epidemic models involving fuzzy delayed differential equations. It is categorized as a computational methodology that preserves the characteristics of a continuous model. These strategies maintain important properties, such as convergent numerical solutions, positivity, and stability, making them more appealing and suitable for studying epidemic models. Compared to standard methods like the forward Euler scheme, NSFD schemes show enhanced performance in computational efficiency and provide a better alignment with continuous models regarding dynamical consistency. This paper presents a proposed framework that incorporates an NSFD approach along with a delay factor.

$$\left\{ \begin{array}{l} S^{n+1} = \frac{S^n + h(\pi + \delta R^n)}{1 + h(\beta(\rho)I^n e^{-\mu\tau} + \mu)}, \\ E^{n+1} = \frac{E^n + h\beta(\rho)S^n I^n e^{-\mu\tau}}{1 + hk_1}, \\ V^{n+1} = \frac{V^n + h\sigma E^n}{1 + hk_2}, \\ I^{n+1} = \frac{I^n + h\lambda V^n}{1 + hk_3}, \\ R^{n+1} = \frac{R^n + h\theta I^n}{1 + hk_4}, \\ T^{n+1} = \frac{T^n + hk_5 I^n}{1 + h\mu}. \end{array} \right. \quad (17)$$

3.3. Positivity of NSFD

To ensure applicability and validity, it is crucial to maintain positivity in the numerical solutions of epidemic models. In compartmental epidemic models, at least one state variable representing population segments must be positive, while all other values should remain non-negative. Preserving the positivity of these variables is vital. This fundamental property is rigorously guaranteed through mathematical induction within an implicit numerical integration scheme. The theorem [46] outlined below offers important insights.

Theorem. Let S, E, V, I, R and T are all positive at $t = 0$; furthermore, if $\psi(\rho) \geq 0, \pi \geq 0, \mu \geq 0, \gamma \geq 0, \delta \geq 0, \sigma \geq 0, \tau \geq 0, \lambda \geq 0, \theta \geq 0$ and $\beta(\rho) \geq 0$ then

$$\left\{ \begin{array}{l} S^{n+1} = \frac{S^n + h(\pi + \delta R^n)}{1 + h(\beta(\rho)I^n e^{-\mu\tau} + \mu)} \geq 0, \\ E^{n+1} = \frac{E^n + h\beta(\rho)S^n I^n e^{-\mu\tau}}{1 + hk_1} \geq 0, \\ V^{n+1} = \frac{V^n + h\sigma E^n}{1 + hk_2} \geq 0, \\ I^{n+1} = \frac{I^n + h\lambda V^n}{1 + hk_3} \geq 0, \\ R^{n+1} = \frac{R^n + h\theta I^n}{1 + hk_4} \geq 0, \\ T^{n+1} = \frac{T^n + hk_5 I^n}{1 + h\mu} \geq 0. \end{array} \right. \quad (18)$$

for all $n \in \mathbb{Z}^+$.

Proof. Now, for $n = 0$, system (18) becomes

$$\left\{ \begin{array}{l} S^1 = \frac{S^0 + h(\pi + \delta R^0)}{1 + h(\beta(\rho)I^0 e^{-\mu\tau} + \mu)} \geq 0, \\ E^1 = \frac{E^0 + h\beta(\rho)S^0 I^0 e^{-\mu\tau}}{1 + hk_1} \geq 0, \\ V^1 = \frac{V^0 + h\sigma E^0}{1 + hk_2} \geq 0, \\ I^1 = \frac{I^0 + h\lambda V^0}{1 + hk_3} \geq 0, \\ R^1 = \frac{R^0 + h\theta I^0}{1 + hk_4} \geq 0, \\ T^1 = \frac{T^0 + hk_5 I^0}{1 + h\mu} \geq 0. \end{array} \right. \quad (19)$$

Now, for $n = 1$, system (18) becomes

$$\left\{ \begin{array}{l} S^2 = \frac{S^1 + h(\pi + \delta R^1)}{1 + h(\beta(\rho)I^1 e^{-\mu\tau} + \mu)} \geq 0, \\ E^2 = \frac{E^1 + h\beta(\rho)S^1 I^1 e^{-\mu\tau}}{1 + hk_1} \geq 0, \\ V^2 = \frac{V^1 + h\sigma E^1}{1 + hk_2} \geq 0, \\ I^2 = \frac{I^1 + h\lambda V^1}{1 + hk_3} \geq 0, \\ R^2 = \frac{R^1 + h\theta I^1}{1 + hk_4} \geq 0, \\ T^2 = \frac{T^1 + hk_5 I^1}{1 + h\mu} \geq 0. \end{array} \right. \quad (20)$$

Please assume that the preceding set of equations guarantees that S, E, V, I, R and T values have the positivity property for $n = 2, 3, 4, \dots, n-1$. In other words, for $n = 2, 3, 4, \dots, n-1$, $S^{n+1} \geq 0, E^{n+1} \geq 0, V^{n+1} \geq 0, I^{n+1} \geq 0, R^{n+1} \geq 0$ and $T^{n+1} \geq 0$. The positivity will now be investigated for a random positive integer n , and we find that

$$\left\{ \begin{array}{l} S^{n+1} = \frac{S^n + h(\pi + \delta R^n)}{1 + h(\beta(\rho)I^n e^{-\mu\tau} + \mu)} \geq 0, \\ E^{n+1} = \frac{E^n + h\beta(\rho)S^n I^n e^{-\mu\tau}}{1 + hk_1} \geq 0, \\ V^{n+1} = \frac{V^n + h\sigma E^n}{1 + hk_2} \geq 0, \\ I^{n+1} = \frac{I^n + h\lambda V^n}{1 + hk_3} \geq 0, \\ R^{n+1} = \frac{R^n + h\theta I^n}{1 + hk_4} \geq 0, \\ T^{n+1} = \frac{T^n + hk_5 I^n}{1 + h\mu} \geq 0. \end{array} \right. \quad (21)$$

Thus, for any positive integer values of n , the suggested approach ensures positivity for the state variables S, E, V, I, R , and T .

3.4. Boundedness of NSFD scheme

Given that the model is about the human population, it is critical to ensure that at any given time 't,' the sum of populations in all compartments does not exceed the overall population. The following theorem [47] addresses this condition effectively.

Theorem. Let S, E, V, I, R and $T \geq 0$ are finite $S + E + V + I + R + T \leq N$; furthermore, $\psi(\rho) \geq 0, \pi \geq 0, \mu \geq 0, \gamma \geq 0, \delta \geq 0, \sigma \geq 0, \tau \geq 0, \lambda \geq 0, \theta \geq 0$ and $\beta(\rho) \geq 0$ then there is a constant such that $S^{n+1} + E^{n+1} + V^{n+1} +$

Table 1
Values of parameters.

Parameters	Values	Parameters	Values
π	3	τ	≥ 0
θ	0.5	γ	0.3
λ	0.2	μ	0.2
δ	0.2	$\beta(\rho)$	Variable
σ	0.8	$\psi(\rho)$	Variable

$$I^{n+1} + R^{n+1} + T^{n+1} \leq N_n \text{ for all } n \in \mathbb{Z}^+.$$

Proof. To establish the boundedness of the proposed NSFD scheme, we refer to system (38) above

$$\begin{aligned} & S^{n+1}(1 + h(\beta(\rho)I^n e^{-\mu\tau} + \mu)) + E^{n+1}(1 + h(\sigma + \mu)) + V^{n+1}(1 + h(\lambda + \mu)) \\ & + I^{n+1}(1 + h(\psi(\rho) + \gamma + \theta + \mu)) + R^{n+1}(1 + h(\delta + \mu)) \\ & + T^{n+1}(1 + h\mu) = S^n + h(\pi + \delta R^n) + E^n + h\beta(\rho)S^n I^n e^{-\mu\tau} + V^n + h\sigma E^n \\ & + I^n + h\lambda V^n + R^n + h\theta I^n + T^n + h(\psi(\rho) + \gamma)I^n, \end{aligned} \quad (22)$$

$$\begin{aligned} & (S^{n+1} + E^{n+1} + V^{n+1} + I^{n+1} + R^{n+1} + T^{n+1})(1 + h\mu) + h(\beta(\rho)I^n e^{-\mu\tau}) \\ & + h(\sigma + \lambda + \psi(\rho) + \gamma + \theta + \delta) = (S^n + E^n + V^n + I^n + R^n + T^n) \\ & + h(\pi + \delta R^n + \beta(\rho)S^n I^n e^{-\mu\tau} + \sigma E^n + \lambda V^n + \theta I^n + (\psi(\rho) + \gamma)I^n), \end{aligned} \quad (23)$$

$$\begin{aligned} & (S^{n+1} + E^{n+1} + V^{n+1} + I^{n+1} + R^{n+1} + T^{n+1}) \\ & + C = \frac{(S^n + E^n + V^n + I^n + R^n + T^n) + h(\pi + \delta R^n + \beta(\rho)S^n I^n e^{-\mu\tau} + \sigma E^n + \lambda V^n + \theta I^n + (\psi(\rho) + \gamma)I^n)}{(1 + h\mu)}, \end{aligned} \quad (24)$$

Where $C = h(\sigma + \lambda + \psi(\rho) + \gamma + \theta + \delta)$,

Now for $n = 0$ system (24) becomes

$$(S^1 + E^1 + V^1 + I^1 + R^1 + T^1) + C = \frac{(S^0 + E^0 + V^0 + I^0 + R^0 + T^0) + h(\pi + \delta R^0 + \beta(\rho)S^0 I^0 e^{-\mu\tau} + \sigma E^0 + \lambda V^0 + \theta I^0 + (\psi(\rho) + \gamma)I^0)}{(1 + h\mu)},$$

Since $N_1 = S^0 + E^0 + V^0 + I^0 + R^0 + T^0$,

$$S^1 + E^1 + V^1 + I^1 + R^1 + T^1 \leq N_1,$$

Similarly, for $n \in \mathbb{Z}^+$ system (24) becomes

$$(S^{n+1} + E^{n+1} + V^{n+1} + I^{n+1} + R^{n+1} + T^{n+1}) + C = \frac{(S^n + E^n + V^n + I^n + R^n + T^n) + h(\pi + \delta R^n + \beta(\rho)S^n I^n e^{-\mu\tau} + \sigma E^n + \lambda V^n + \theta I^n + (\psi(\rho) + \gamma)I^n)}{(1 + h\mu)}. \quad (25)$$

Consequently, $S^{n+1} \leq N_n$, $E^{n+1} \leq N_n$, $V^{n+1} \leq N_n$, $I^{n+1} \leq N_n$, $R^{n+1} \leq N_n$ and $T^{n+1} \leq N_n$, hence the proof.

3.5. Convergence investigation

Analysis of convergence in epidemic models looks at the reliability and accuracy of the numerical techniques employed to resolve differential equations about the spread of diseases. The numerical solution method is assessed to determine its approximation to the accurate solution as the computational parameters decrease, ensuring both the reliability and precise outcomes of simulations and preserving the qualities of the continuous model, the proposed model and simulation uses the following Values summarised in Table 1.

$$G_1 = \frac{S + h(\pi + \delta R)}{1 + h(\beta(\rho)Ie^{-\mu\tau} + \mu)},$$

$$G_2 = \frac{E + h\beta(\rho)S I e^{-\mu\tau}}{1 + h k_1},$$

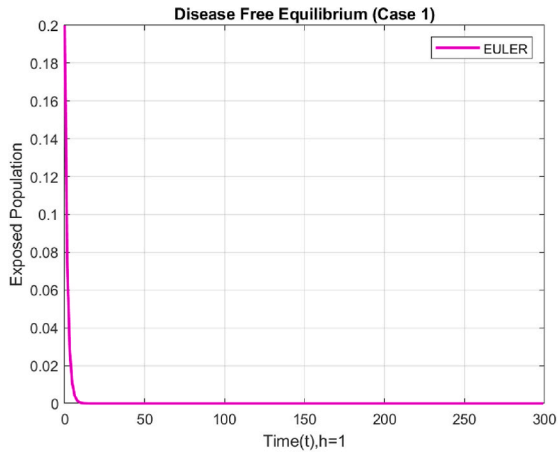
$$G_3 = \frac{V + h_\sigma E}{1 + h k_2},$$

$$G_4 = \frac{I + h\lambda V}{1 + h k_3},$$

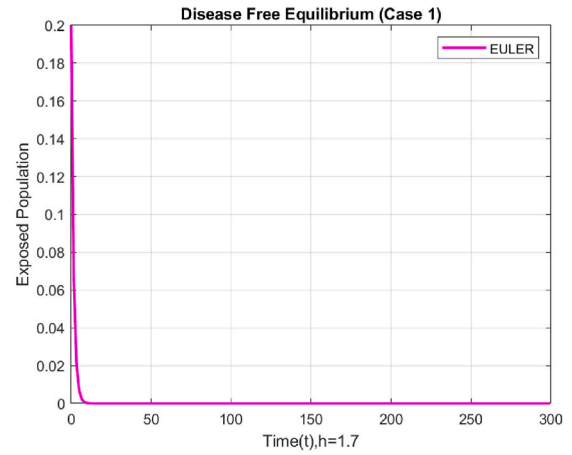
$$G_5 = \frac{R + h\theta I}{1 + h k_4}.$$

$$G_6 = \frac{T + h k_5 I}{1 + h\mu}.$$

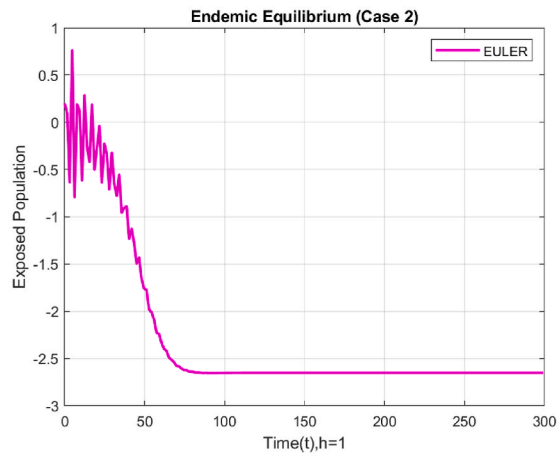
Jacobian matrix at the DFE point



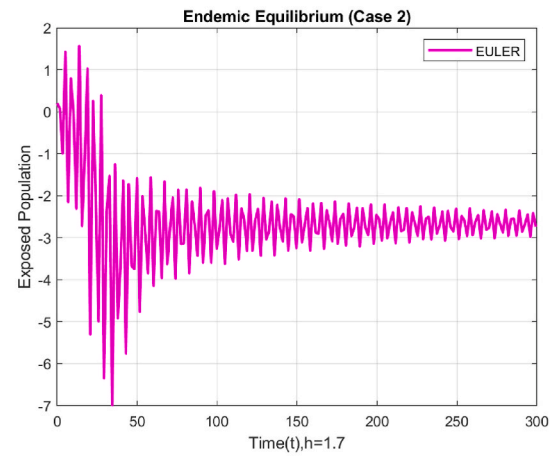
(a)



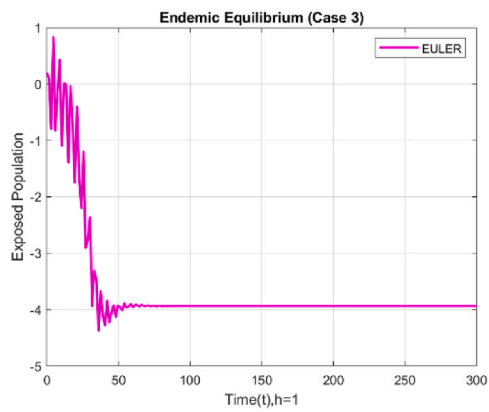
(b)



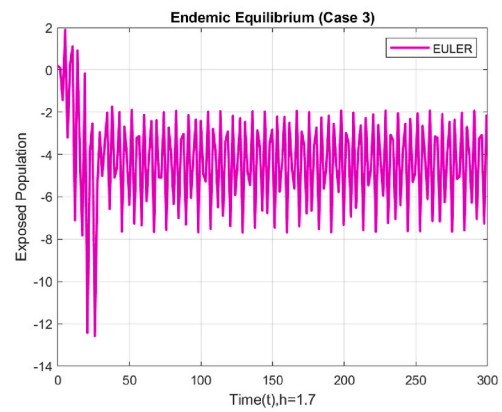
(c)



(d)



(e)



(f)

Fig. 4. Variation in exposed population with step size using the forward Euler technique.

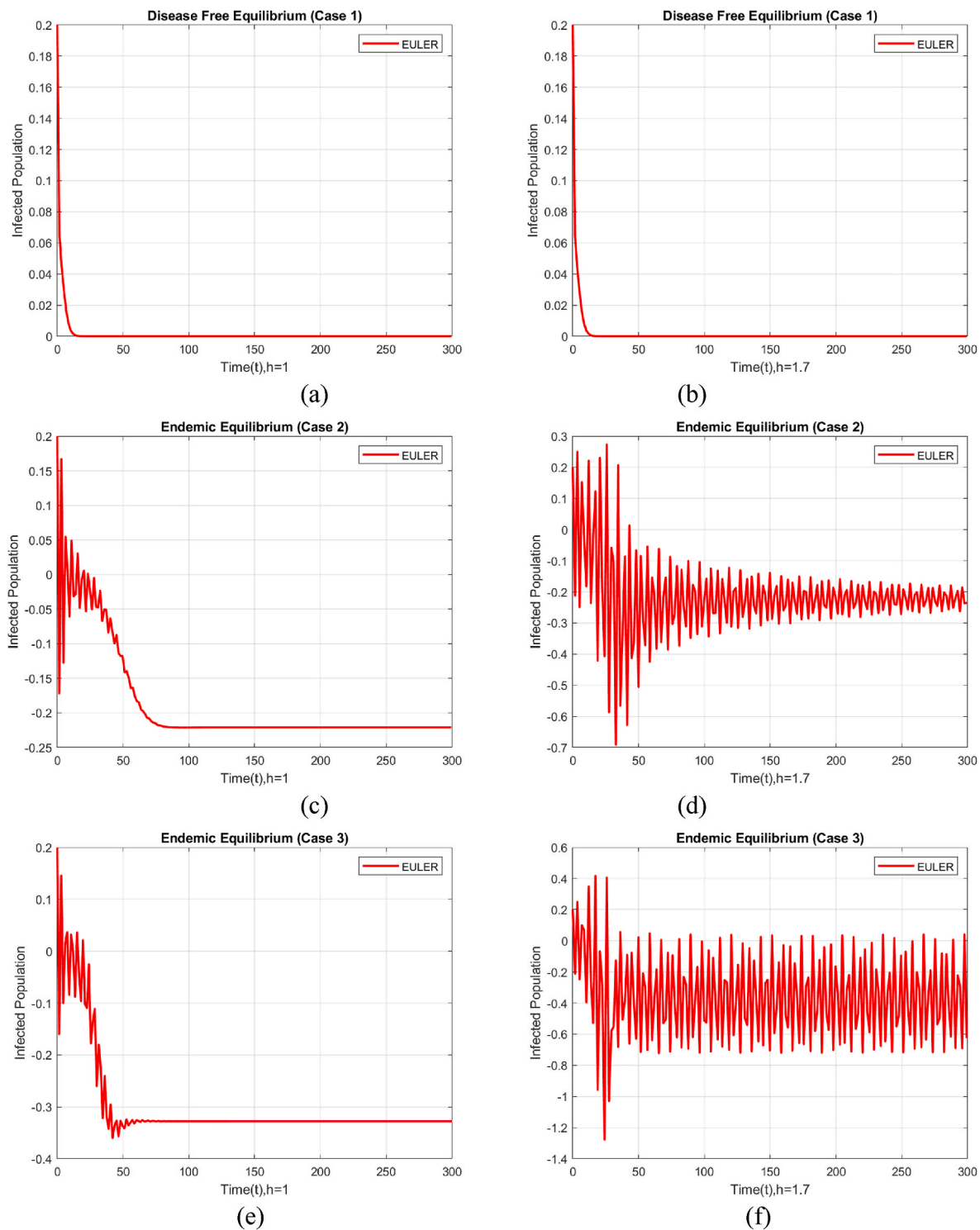


Fig. 5. Infected populations at various step sizes using the forward Euler approach.

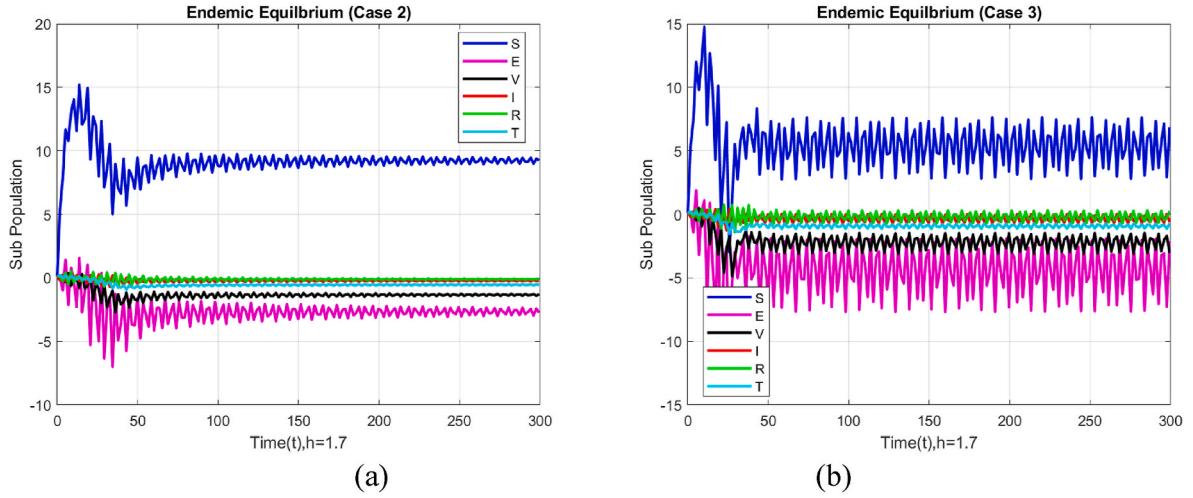


Fig. 6. Compartmental comparison of SEVIRT using the forward Euler approach.

$$\mathcal{J}(E_p^0) = \begin{bmatrix} \frac{1}{1+h\mu} & 0 & 0 & 0 & \frac{h\delta}{1+h\mu} & 0 \\ 0 & \frac{1}{1+hk_1} & 0 & 0 & 0 & 0 \\ 0 & \frac{h\sigma}{1+hk_3} & \frac{1}{1+hk_2} & 0 & 0 & 0 \\ 0 & 0 & \frac{h\lambda}{1+hk_3} & \frac{1}{1+hk_3} & 0 & 0 \\ 0 & 0 & 0 & \frac{h\theta}{1+hk_4} & \frac{1}{1+hk_4} & 0 \\ 0 & 0 & 0 & \frac{hk_5}{1+h\mu} & 0 & \frac{1}{1+h\mu} \end{bmatrix},$$

Eigenvalues of the above Jacobian matrix $\lambda_1 = 0.9804 < 1$, $\lambda_2 = 0.9434 < 1$, $\lambda_3 = 0.9615 < 1$, $\lambda_4 = 0.8621 < 1$, $\lambda_5 = 0.9091 < 1$ and $\lambda_6 = 0.9804 < 1$. Convergence of the proposed NSFD scheme at the DFE point is ensured, as all eigenvalues of the Jacobian matrix reside within the unit circle at E_p^0 .

3.6. Consistency properties of the NSFD scheme

Consistency pertains to the numerical method's capacity to accurately represent the underlying continuous model as computational factors, such as step size, are reduced. The analysis results highlight the importance of maintaining consistency in NSFD schemes to ensure the reliability and accuracy of numerical solutions.

Beginning with the first equation in system (17), we get

$$S^{n+1}(1 + h(\beta(\rho)I^n e^{-\mu\tau} + \mu)) = S^n + h(\pi + \delta R^n). \quad (26)$$

Taylor's series expansion of the S^{n+1} is as follows

$$S^{n+1} = \left(S^n + h \frac{dS}{dt} + \frac{h^2}{2!} \frac{d^2S}{dt^2} + \frac{h^3}{3!} \frac{d^3S}{dt^3} + \dots \right). \quad (27)$$

From equation (26), we obtain

$$\left(S^n + h \frac{dS}{dt} + \frac{h^2}{2!} \frac{d^2S}{dt^2} + \frac{h^3}{3!} \frac{d^3S}{dt^3} + \dots \right) (1 + h(\beta(\rho)I^n e^{-\mu\tau} + \mu)) = S^n + h(\pi + \delta R^n).$$

Simplifying and applying the limit $h \rightarrow 0$ yields:

$$S^n((\beta(\rho)I^n e^{-\mu\tau} + \mu)) + \frac{dS}{dt} = \pi + \delta R^n,$$

$$\frac{dS}{dt} = \pi + \delta R_{-\beta}^n(\rho) S^n I^n e^{-\mu\tau} - \mu S^n,$$

$$\Rightarrow \frac{dS}{dt} = \pi + \delta R_{-\beta(\rho)} S I e^{-\mu\tau} - \mu S.$$

Utilizing the second expression in the NSFD scheme, it follows that:

$$E^{n+1}(1 + hk_1) = E^n + h\beta(\rho) S^n I^n e^{-\mu\tau}, \quad (28)$$

By applying the Taylor series, E^{n+1} is as follows

$$E^{n+1} = \left(E^n + h \frac{dE}{dt} + \frac{h^2}{2!} \frac{d^2E}{dt^2} + \frac{h^3}{3!} \frac{d^3E}{dt^3} + \dots \right). \quad (29)$$

With the substitution of E^{n+1} in (28), we obtain

$$\left(E^n + h \frac{dE}{dt} + \frac{h^2}{2!} \frac{d^2E}{dt^2} + \frac{h^3}{3!} \frac{d^3E}{dt^3} + \dots \right) (1 + hk_1) = E^n + h\beta(\rho) S^n I^n e^{-\mu\tau}.$$

By applying $h \rightarrow 0$, we get

$$k_1 E^n + \frac{dE}{dt} = \beta(\rho) S^n I^n e^{-\mu\tau},$$

$$\frac{dE}{dt} = \beta(\rho) S^n I^n e^{-\mu\tau} - k_1 E^n,$$

$$\Rightarrow \frac{dE}{dt} = \beta(\rho) S I e^{-\mu\tau} - k_1 E.$$

Utilizing the third expression in the NSFD scheme, it follows that:

$$V^{n+1}(1 + hk_2) = V^n + h\sigma E^n, \quad (30)$$

Taylor series of V^{n+1} is as follows

$$V^{n+1} = \left(V^n + h \frac{dV}{dt} + \frac{h^2}{2!} \frac{d^2V}{dt^2} + \frac{h^3}{3!} \frac{d^3V}{dt^3} + \dots \right), \quad (31)$$

With the substitution of V^{n+1} in (28), we obtain

$$V^{n+1} = \left(V^n + h \frac{dV}{dt} + \frac{h^2}{2!} \frac{d^2V}{dt^2} + \frac{h^3}{3!} \frac{d^3V}{dt^3} + \dots \right) (1 + hk_2) = V^n + h\sigma E^n.$$

Apply $h \rightarrow 0$, we get

$$k_2 V^n + \frac{dV}{dt} = \sigma E^n,$$

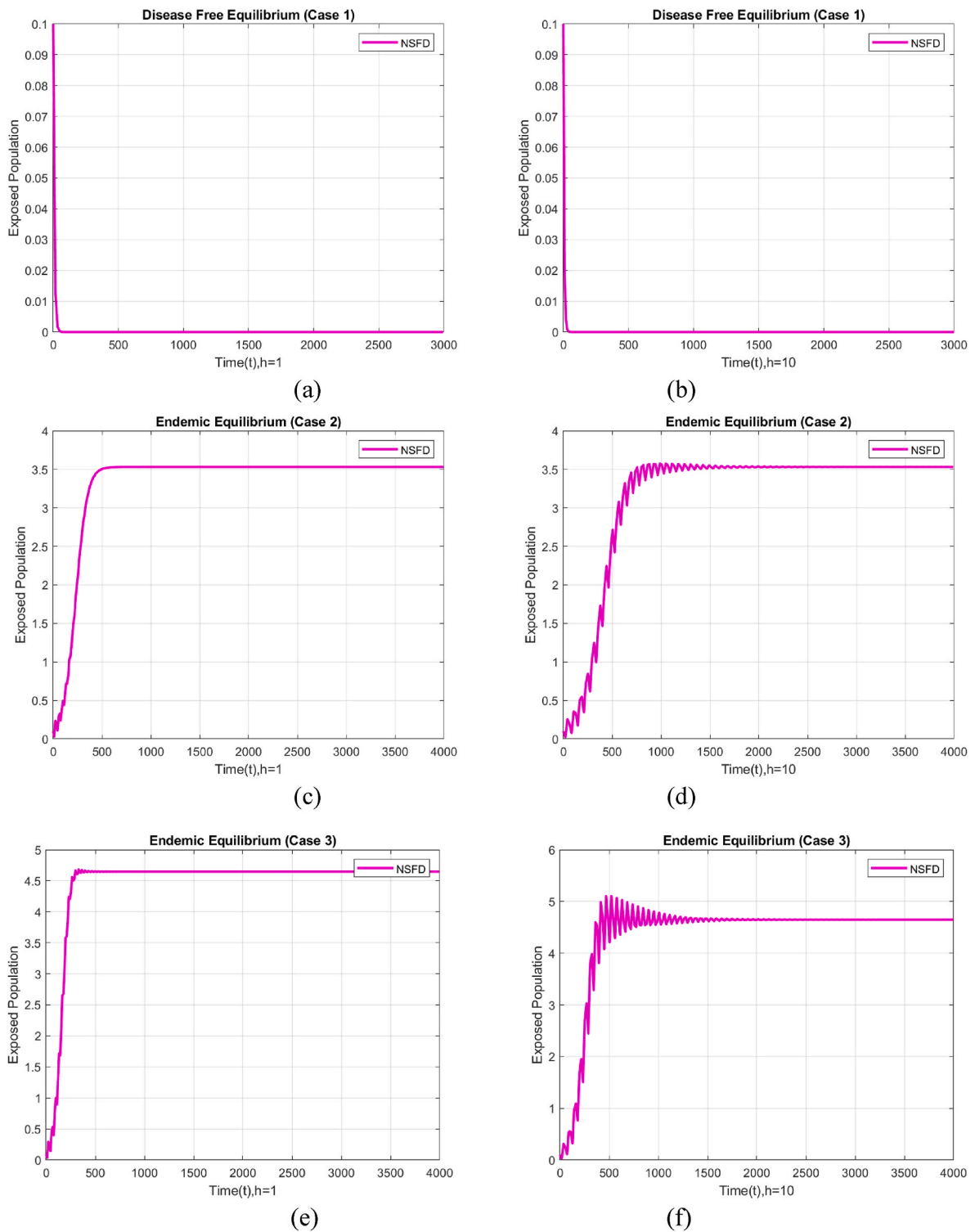


Fig. 7. Exposed population at various step sizes using the NSFD approach.

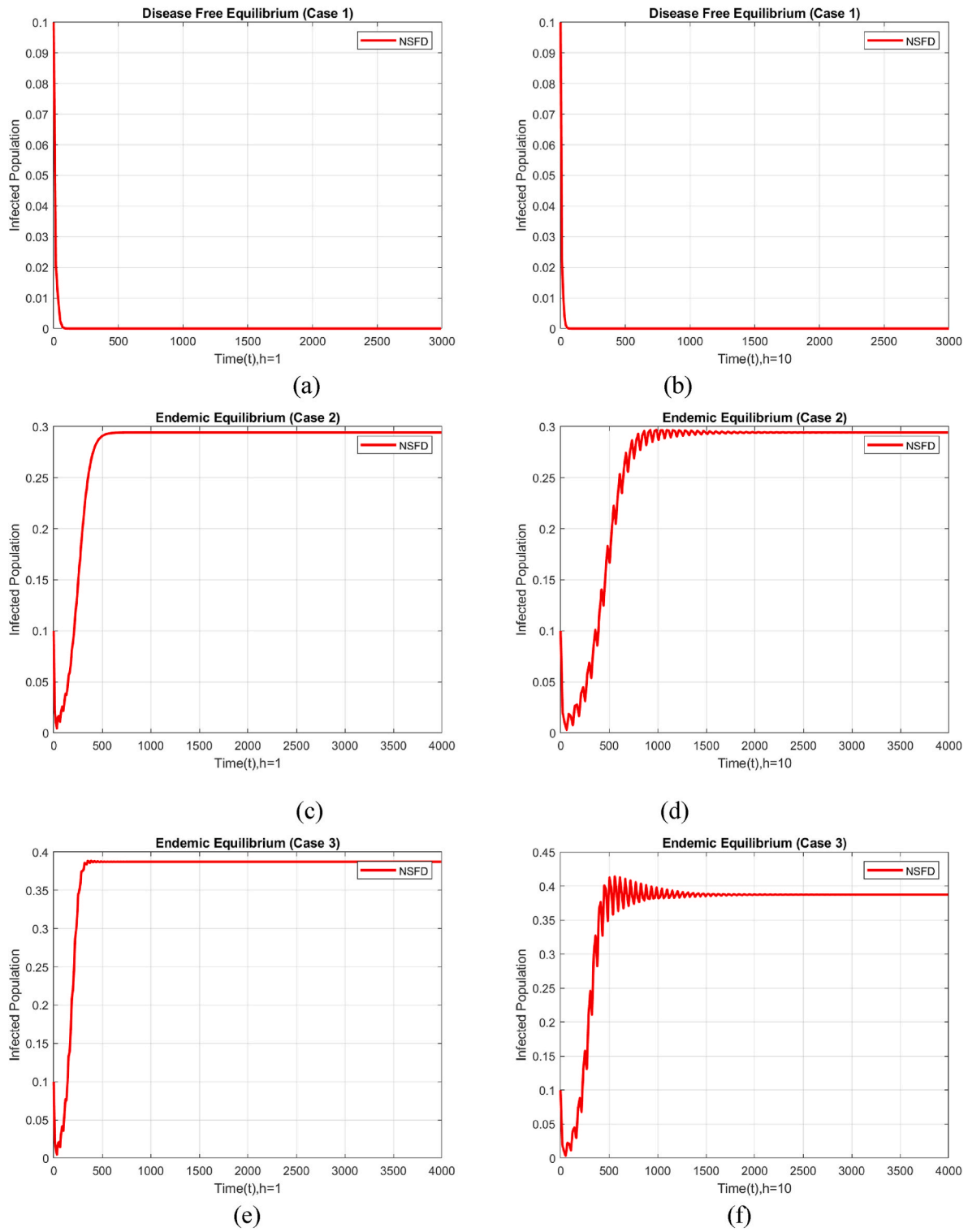


Fig. 8. Infected populations at various step sizes using the NSFD approach.

$$\frac{dI}{dt} = \sigma E^n - k_2 V^n,$$

$$\Rightarrow \frac{dI}{dt} = \sigma E - k_2 V.$$

Utilizing the fourth expression in the NSFD scheme, it follows that:

$$I^{n+1}(1 + hk_3) = I^n + h\lambda V^n. \quad (32)$$

Taylor series expansion to the I^{n+1} is as follows

$$I^{n+1} = \left(I^n + h \frac{dI}{dt} + \frac{h^2}{2!} \frac{d^2I}{dt^2} + \frac{h^3}{3!} \frac{d^3I}{dt^3} + \dots \right). \quad (33)$$

From equation (32), we obtain

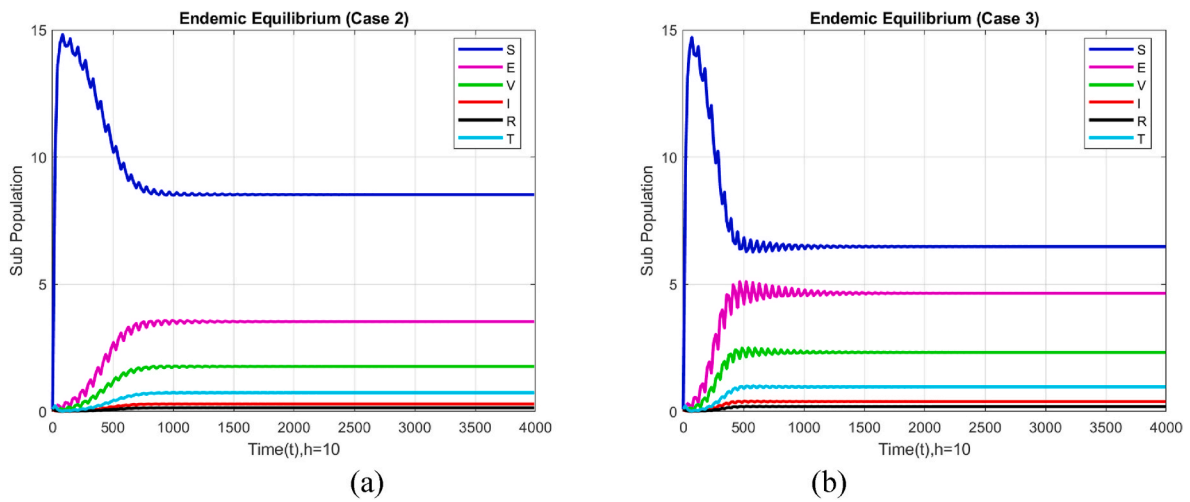


Fig. 9. Compartmental comparison of SEVIRT using the NSFD approach.

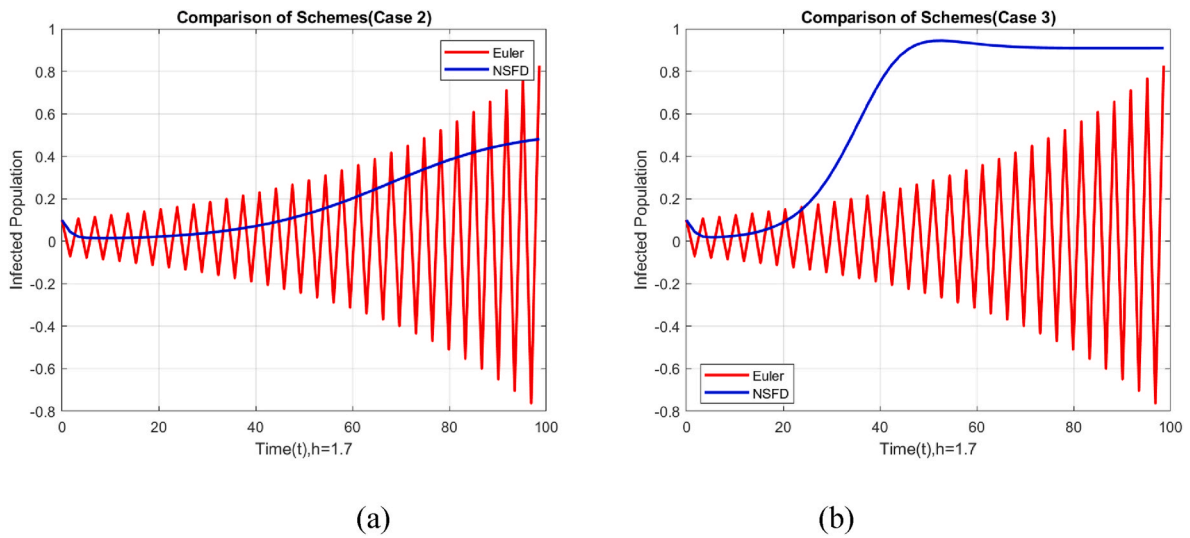


Fig. 10. Comparison of the SEIVRT Model using the Euler method and the NSFD approach.

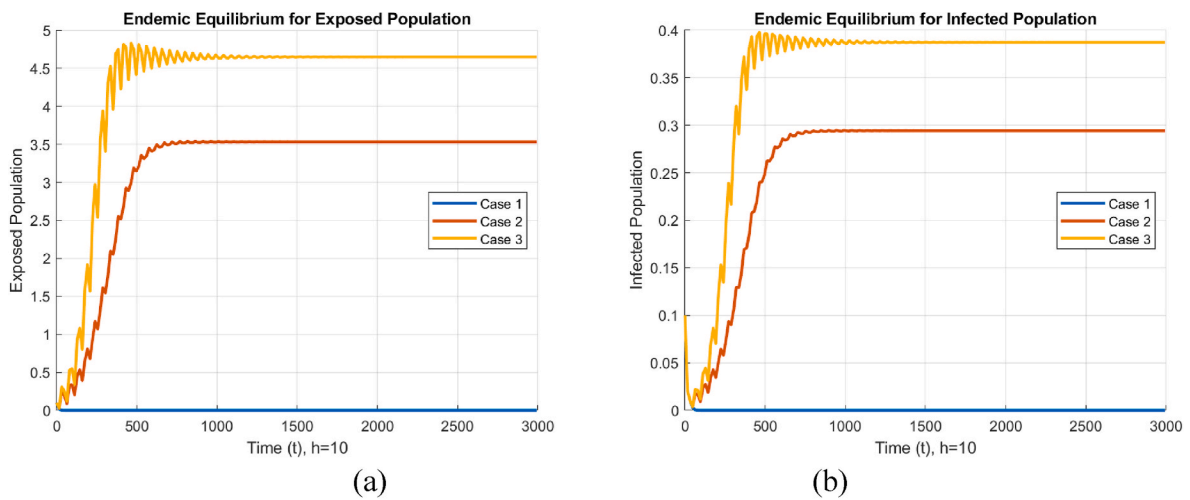


Fig. 11. Exposed and infected population in each case using the NSFD approach.

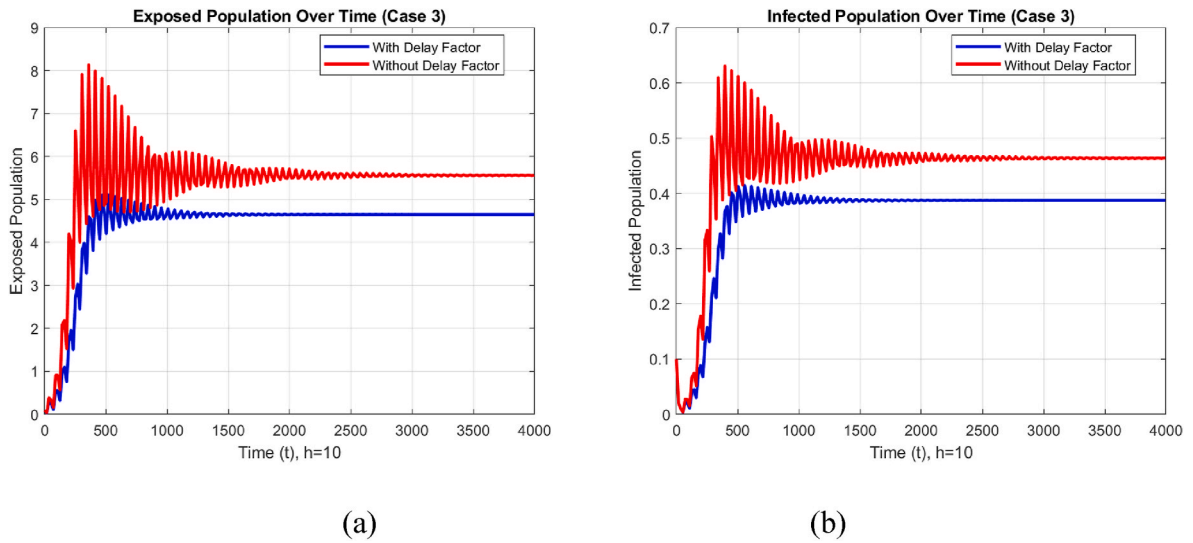


Fig. 12. Influence of time delay on infection dynamics using the NSFD approach.

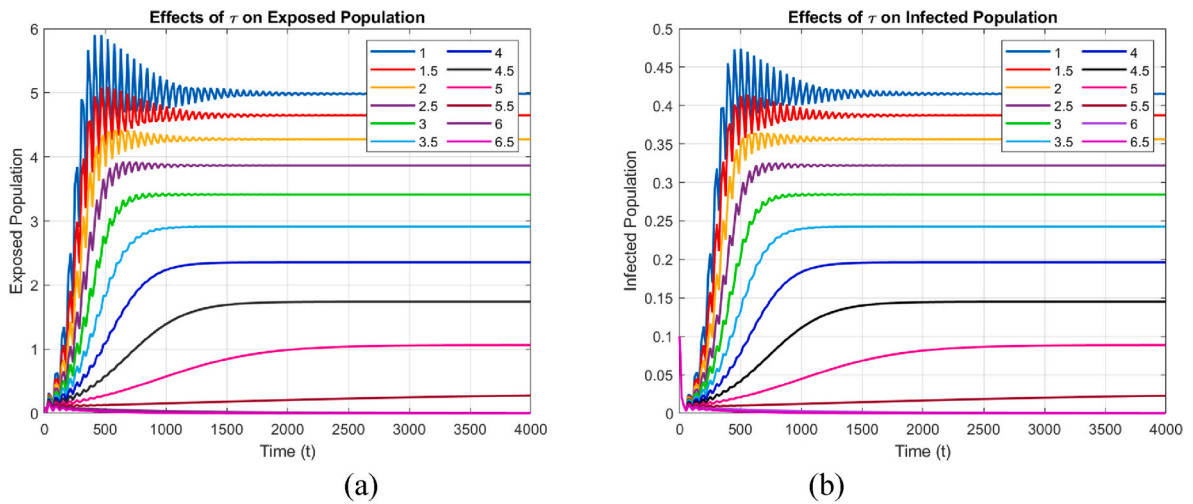


Fig. 13. Influence of time delay τ on the dynamics of exposed and infected populations.

$$\left(I^n + h \frac{dI}{dt} + \frac{h^2}{2!} \frac{d^2 I}{dt^2} + \frac{h^3}{3!} \frac{d^3 I}{dt^3} + \dots \right) (1 + h k_3) = I^n + h \lambda V^n.$$

Apply $h \rightarrow 0$, we get

$$I^n k_3 + \frac{dI}{dt} = \lambda V^n,$$

$$\frac{dI}{dt} = \lambda V^n - I^n k_3,$$

$$\Rightarrow \frac{dI}{dt} = \lambda V - I k_3,$$

Utilizing the fifth expression in the NSFD scheme, it follows that:

$$R^{n+1} (1 + h k_4) = R^n + h \theta I^n. \quad (34)$$

Taylor series expansion to the R^{n+1} is as follows

$$R^{n+1} = \left(R^n + h \frac{dR}{dt} + \frac{h^2}{2!} \frac{d^2 R}{dt^2} + \frac{h^3}{3!} \frac{d^3 R}{dt^3} + \dots \right), \quad (35)$$

From equation (34), we obtain

$$\left(R^n + h \frac{dR}{dt} + \frac{h^2}{2!} \frac{d^2 R}{dt^2} + \frac{h^3}{3!} \frac{d^3 R}{dt^3} + \dots \right) (1 + h k_4) = R^n + h \theta I^n.$$

Apply $h \rightarrow 0$, we get

$$k_4 R^n + \frac{dR}{dt} = \theta I^n,$$

$$\frac{dR}{dt} = \theta I^n - k_4 R^n,$$

$$\Rightarrow \frac{dR}{dt} = \theta I - k_4 R.$$

From the sixth equation of the NSFD scheme, we have

$$T^{n+1} (1 + h \mu) = T^n + h k_5 I^n, \quad (36)$$

Taylor's series expansion of the T^{n+1} is as follows

$$T^{n+1} = \left(T^n + h \frac{dT}{dt} + \frac{h^2}{2!} \frac{d^2 T}{dt^2} + \frac{h^3}{3!} \frac{d^3 T}{dt^3} + \dots \right). \quad (37)$$

From equation (36), we obtain

$$\left(T^n + h \frac{dT}{dt} + \frac{h^2}{2!} \frac{d^2T}{dt^2} + \frac{h^3}{3!} \frac{d^3T}{dt^3} + \dots\right)(1 + h\mu) = T^n + hk_5I^n.$$

Apply $h \rightarrow 0$, we get

$$\mu T^n + \frac{dD}{dt} = k_5 I^n,$$

$$\frac{dT}{dt} = k_5 I^n - \mu T^n,$$

$$\Rightarrow \frac{dT}{dt} = k_5 I - \mu T.$$

4. Mathematical simulations

The forward Euler method illustrates the population exposed at several step sizes in Fig. 4. The graph shows the disease-free and endemic stable states and non-physical oscillations in node values, with larger step sizes resulting in some negative values unsuitable for population dynamics. This suggests that increased step sizes commonly lead to incorrect and negative results. This is shown in Fig. 5, highlighting the infected population utilizing a variety of step sizes while also applying the Forward Euler method. Like the population under exposure, oscillations and adverse results are noted, leading to the same findings regarding step sizes and model reliability. In Fig. 6, the SEVIRT model for conjunctivitis illustrates the functions of all compartments, with a focus on compartmental dynamics. The representation in Fig. 7, achieved using the NSFD scheme, displays the exposed population at different step sizes, offering enhanced results by removing negative values. As illustrated in Fig. 8, the NSFD method successfully keeps populations approximately positive for all step sizes, particularly concerning the infected population. Fig. 9 illustrates a comparison of the SEVIRT model compartments using the NSFD method, and Fig. 10 shows a contrast between the results of the SEVIRT model achieved by both the Euler and NSFD methods. Fig. 11 evaluates the population infected in every situation, pointing out changes in the rate of infection increase or decrease. The disease prevalence level is about zero in case 1, but it stays stable at an endemic rate in cases 2 and 3, where case 3 records more infections than case 2. Fig. 12 represents the number of infected persons in circumstances with and without incorporating a delay factor into the fuzzy delayed NSFD model. The graph proves that adding the delay factor greatly facilitates the control of the disease, lowering the incidence of conjunctivitis.

Finally, Fig. 13 examines how the delay parameter (τ) impacts exposed and infected populations within the proposed NSFD framework. In terms of performance, the NSFD approach outperforms the Forward Euler method. It delivers realistic and accurate results while avoiding the non-physical outcomes typical of the Euler method, making it especially effective for modeling population dynamics related to conjunctivitis.

The comparative findings indicate that the NSFD method offers a distinct advantage over the forward Euler method by preserving critical properties such as positivity, boundedness, and convergence more effectively. Simple by design, the Forward Euler method generates unstable numerical results when used with longer time steps or under uncertain modeling conditions. The NSFD approach exhibits excellent compatibility with the continuous system and maintains high levels of accuracy and stability, regardless of fuzzy parameters and time delays. Experimental findings strengthen the NSFD scheme, demonstrating that it is a superior numerical tool for complex infectious disease transmission.

5. Conclusion

This research underlines the necessity of using fuzzy mathematical modeling to accurately depict CVD, also known as pink eye transmission

dynamics. Employing fuzzy theory in the model allows researchers to address real-world disease transmission with uncertainty, circumventing limitations imposed by classical deterministic systems. It should be made clear that including a vaccination compartment in the model does not imply that an actual vaccine for CVD currently exists. Therefore, the compartment is defined as a general representation for various safety measures to prevent infections. These may include regular hand and face washing, cleaning surfaces with disinfectants, wearing safety glasses, taking medicines, and mass media campaigns promoting behavior change. Since the model interprets infection more broadly, it remains useful for numerous interventions, regardless of whether a vaccine is developed. This abstraction enables researchers and public health experts to simultaneously test multiple strategies within the model, aiding in the formulation of policy and emergency preparedness plans. Asymptomatic carriers, along with environmental variables, are regarded as critical infection rate factors that the model addresses with special focus, while integrating the incubation period and other temporal elements through delay features. The fuzzy modeling framework encompasses equilibrium, sensitivity, and stability analytic tools that address both the basic reproduction number and disease transmission parameter sensitivity.

The stability order within fuzzy systems serves as a key component that provides robustness and biological plausibility to the predictive model. The stability analysis ensures prediction reliability when different parameter values are applied, thus enabling applications in real-world scenarios. The fuzzy system demonstrates flexibility through its capacity to process biological data with uncertainties and imprecision, making it effective for modeling epidemic complexity. The stability order helps preserve the model's biological validity through behaviors that align with observed epidemic patterns in real-life situations. Numerical results from Forward Euler and NSFD calculations show stable outcomes with wide step sizes. The NSFD method stands out because it retains essential continuous model characteristics such as positivity, boundedness, and convergence properties. The NSFD method functions effectively as a modeling tool for realistic epidemiological systems that must handle fuzzy conditions and delays, as it maintains numerical stability while capturing dynamic behavior.

The application parameters from the NSFD scheme demonstrate their ability to accurately predict real-world system behavior while determining their extensive usefulness in disease epidemiology modeling. These methods enhance prediction accuracy by addressing uncertainties and environmental influences while developing effective public health plans. It should be made clear that having a vaccination compartment in the model does not imply that an actual vaccine for CVD currently exists. For this reason, the compartment is defined as a general model for different safety actions to prevent infections. These may consist of regular hand and face washing, cleaning surfaces with disinfectants, using safety glasses, taking medicines, and engaging in mass media campaigns promoting behavior change. Since the model interprets infection more broadly, it is still useful for many interventions, regardless of whether a vaccine is developed. With this abstraction, researchers and public health experts can test multiple strategies within the model simultaneously, helping to shape policy and emergency preparation plans.

To validate the presented results, the model can be extended for future work by incorporating real data from reported conjunctivitis outbreaks. This will strengthen the model's applicability and value in informing public health policies. Further refinements could involve stratifying populations (e.g., by age or geography), introducing stochastic elements, or modeling behavioral responses to outbreaks.

6. Limitations

Although the approach presented in this study is as systematic as possible, much of the subject merits further investigation. The analysis provided is based on generalized parameters to demonstrate that the model performs well across various situations. Future research should

incorporate real-world epidemiological data to enhance model accuracy and usability in practical contexts. Additionally, this model assumes a closed and well-mixed population, which simplifies the analysis while preserving the core transmission dynamics. Consequently, simpler population models can be developed to support more detailed future studies. Furthermore, the model integrates various public health interventions to emphasize the broader impact of protective measures on the population. By using this approach as a foundation, various sub-models can be created and evaluated to assess the effectiveness of different interventions.

CRedit authorship contribution statement

Muhammad Tashfeen: Conceptualization, Methodology, Software, Writing – original draft. **Hothefa Shaker Jassim:** Funding acquisition, Data curation, Writing – review & editing. **Fazal Dayan:** Validation, Resources, Project administration. **Muhammad Azizur Rehman:** Visualization, Investigation, Writing – review & editing. **Alwahab Dhulfiqar Zoltán:** Validation, Funding acquisition, Writing – review & editing. **Husam A. Neamah:** Formal analysis, Writing – review & editing, Methodology, Software.

Consent to participate

Informed consent was obtained from all individual participants included in the study.

Ethics approval

No ethical approval was required for this observational study.

Consent for publication

All authors consent to the publication of this manuscript.

Ethical statement - studies in humans and animals

This research did not involve any studies in humans or animals. Therefore, no ethical approval or consent was required or obtained for this manuscript.

Declaration of competing interest

The authors declare that they have no conflicts of interest or competing interests.

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Data availability

No data was used for the research described in the article.

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