SeMA Journal
https://doi.org/10.1007/s40324-025-00404-9

Differential equation models for infectious diseases: Mathematical modeling, qualitative analysis, numerical methods and applications

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Received: 28 April 2025 / Accepted: 22 July 2025

Abstract
Mathematical epidemiology has a long history of origin and development. In particular, mathematical modeling and analysis of infectious diseases has become a fundamental and indispensable approach to discovering the characteristics and mechanisms of the transmission dynamics of epidemics, thereby effectively predicting possible scenarios in reality, as well as controlling and preventing diseases. In recent decades, differential equations have been widely used to model many important infectious diseases. The study of these differential equation models is very useful in both theory and practice, especially in proposing appropriate strategies for disease control and prevention. This is of great benefit to public health and health care. In this survey article, we review many recent developments and real-world applications of deterministic ordinary and partial differential equations (ODEs and PDEs) in modeling major infectious diseases, particularly focusing on the following aspects: mathematical modeling, qualitative analysis, numerical methods, and real-world applications. We also present and discuss some open problems and future directions that research in differential equation models for infectious diseases can take. This article provides a comprehensive introduction to epidemic modeling and insights into nonstandard finite difference methods.

Keywords Mathematical modeling · Epidemiology · Epidemics · Infectious diseases · Numerical methods

Mathematics Subject Classification Primary 34A34 · 35Q92 · 37N25 · 37N30 · 92B05 · 65L05 · 65L12 · 92Dxx

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Published online: 05 August 2025

1 Introduction

Infectious diseases have always been a major and constant threat to public health. Mankind has always had to face and fight many infectious diseases with varying degrees of danger, such as influenza, hepatitis, Zika, malaria, measles, tuberculosis, hepatitis, vector-borne diseases, Ebola, and most recently the COVID-19 pandemic.

The well-known SIR model, proposed by Kermack and McKendrick [252], can be considered one of the first epidemic models and is usually used to introduce epidemic modeling. The study of mathematical models of infectious diseases is very useful in both theory and practice, especially in proposing appropriate strategies for disease control and prevention. This is of great benefit to public health and health care.

It is well known that differential equations, including ordinary differential equations (ODEs) and partial differential equations (PDEs), have several useful applications in real life. They are widely used to describe many important phenomena and processes in science and engineering (see e.g. [30, 55, 78–83, 258, 323, 360, 454]). One of its prominent applications is the mathematical modeling and analysis of infectious diseases. Over the past few decades, a large number of differential equation models have been extensively developed to explore the transmission dynamics of major infectious diseases. These models have confirmed the important role of differential equations in epidemic modeling.

Nowadays, epidemic models based on differential equations have always been an important and indispensable approach in modeling infectious diseases, especially in the context that epidemics are constantly changing and posing new challenges. For differential equation models of infectious diseases, the following aspects are mainly focused:

- *Mathematical modeling:* The use of differential equations and the foundations of mathematical epidemiology to propose mathematical models that describe the transmission of infectious diseases.
- *Qualitative study:* Investigate mathematical properties of the proposed differential equation models, including existence and uniqueness of solutions, positivity and boundedness of solutions, asymptotic stability properties, conservation laws, physical properties, and basic reproduction number.
- *Numerical methods:* Construction of efficient numerical methods, especially numerical methods that preserve important mathematical features of the proposed differential equation models.
- *Practical applications:* Applying the theoretical results to provide scenarios of disease spread, to suggest anti-epidemic measures and strategies, to evaluate the effectiveness of vaccines and existing anti-epidemic measures, to study the spread of computer viruses, rumors and malware on the Internet, and to model animal diseases. and animal disease modeling with applications in agriculture.

The aim of this review article is to review many recent developments and real-life applications of deterministic differential equation models in modeling major infectious diseases, focusing mainly on the following aspects: mathematical modeling, qualitative analysis, numerical methods, and real-life applications. We also present and discuss some open problems and future directions that research in differential equation models for infectious diseases can take.

The manuscript is expected to cover not only the latest developments in deterministic ODE and PDE models for infectious diseases, but also future research and open problems in this area. Unlike some previous review articles (see, for example, [82, 84, 87, 115, 208, 315, 363, 375, 392, 514]) that focus only on the mathematical modeling of specific diseases, this

review provides a comprehensive analysis of all four aspects, where the main differences are outlined as follows:

- In the mathematical modeling aspect: The selected references are systematically reviewed based on common and dangerous diseases. Many common and dangerous diseases (e.g., basic models of virus dynamics, influenza, severe acute respiratory syndrome (SARS), Ebola, hepatitis B and C, tuberculosis, vector-borne diseases, malaria, measles, Zika virus, dengue fever, COVID-19 pandemic, HIV/AIDS, …) have been mentioned, providing readers with a comprehensive and in-depth insight into infectious disease modeling with applications.
- In the qualitative study aspect: We list in detail the essential qualitative properties for the proposed models and, in particular, the tools and methods used in qualitative research are rigorously analyzed.
- In the numerical methods aspect: We provide a detailed overview of numerical methods, including standard and nonstandard methods, for solving differential equations, with an emphasis on those used to solve disease transmission models. This section also provides an introduction to NSFD methods for mathematical models arising in real-world situations and recent advances in this area.
- In the practical application aspect: We focus on important applications of differential equations for infectious diseases: modeling animal diseases with applications in agriculture, chemostat models to represent microbial growth and competition, modeling the spread of computer viruses and rumors on the Internet, modeling addictions (e.g., alcohol, tobacco, heroin, opioids, cocaine, drug use, etc.), understanding disease dynamics and potential scenarios, informing data-driven public health initiatives.

In general, this survey provides a systematic overview of infectious disease modeling for mathematicians, epidemiologists, and all researchers of all experience levels, whether they are experienced or new to the field, that can help them understand:

- Recent advances in modeling of major diseases.
- Methods, methodologies, approaches, and tools for modeling infectious diseases.
- Techniques for extracting insights and shaping public health strategies.
- Exciting future directions in infectious disease research.

In addition, this manuscript provides an overview of nonstandard finite difference (NSFD) methods and their applications in disease modeling.

It is important to note that there are many other types of epidemiological models, such as integro-differential models, delayed differential equation models, fractional-order and stochastic differential models (see, for example, [16, 46, 47, 59, 90, 97, 159, 184, 352, 388, 413, 426, 460, 497, 516]). However, the manuscript focuses only on ODEs and PDEs because the approaches, methodologies, and methods for constructing ODE and PDE models of infectious diseases are very similar. In fact, they share many common features that should be included in a single systematic review. The other types of epidemiological models will be considered in future studies.

The outline of this article is as follows: In Sect. 2, we provide an overview of epidemic models based on differential equations, considering basic models and their variants and extensions. In Sect. 3 we focus on the qualitative analysis aspect and its practical applications. Numerical methods are presented in Sect. 4. Future research and open problems are discussed in Sect. 5. The last section contains concluding remarks and discussions.

2 Mathematical modeling

In this section, we review results on mathematical modeling based on deterministic ODEs and PDEs for infectious diseases.

2.1 ODE models: basic epidemic models

We start with one of the first and basic epidemic models introduced by Kermack and McKendrick in 1927 [252]. For this purpose, let us consider general autonomous dynamical systems described by ODEs of the form

$$\dot{y}(t) = f\big(y(t)\big), \quad t > 0, \qquad y(0) = y_0 \in \mathbb{R}^n, \tag{2.1}$$

where $y = [y_1, y_2, \ldots, y_n]^\top : [0, \infty) \to \mathbb{R}^n$, $f = [f_1, f_2, \ldots, f_n]^\top : \mathbb{R}^n \to \mathbb{R}^n$ and $\dot{y}$ stands for the time derivative of y. Here it is assumed that the right-hand-side function f satisfies all necessary smoothness assumptions so that solutions of (2.1) exist and are unique (see e.g. [55, 258, 454]).

Many mathematical models based on (2.1) have been proposed to study epidemic models. In these models, diseases caused by viruses or bacteria are not modelled directly in the population model, but only indirectly through the number of infected individuals. For example, the classical SI, SIS and SIR epidemic models classify individuals in the population according to their status with respect to the disease: healthy, infected and immune. More clearly, the disease states S, I and R are defined as follows [30, 323]:

- *susceptible* S: Individuals who are not infected but are susceptible to acquiring the disease and becoming contagious.
- *infected* I: Individuals who have been infected, are currently contagious, and have the potential to spread the disease to others.
- *removed* R: Individuals who have experienced the disease, recovered, and achieved permanent immunity, or are isolated until both recovery and permanent immunity are achieved.

Models with these states are called *SIR models*, adapted to the characteristics of the infectious disease, for example:

- SI implies the absence of any possible recovery: $S \to I$;
- SIS indicates the possibility of recovery, but does not guarantee immunity: $S \to I \to S$;
- SIR represents a temporary state of immunity: $S \to I \to R \to S$.

One of the simplest models involves the dynamics of $S-$, $I-$, $R-$ individuals, first introduced by Kermack and McKendrick in 1927 [252] (see also [323]):

$$\begin{aligned} \dot{S}(t) &= -\beta I(t) S(t), \\ \dot{I}(t) &= \beta I(t) S(t) - \alpha I, \\ \dot{R}(t) &= \alpha I(t), \quad t > 0, \end{aligned} \tag{2.2}$$

where

- β is the proportionality constant ('transmission rate');
- α is the recovery rate;
- $\beta I(t)$ is called the force of infection.
- βSI represents the number of new infections per unit of time (incidence).

Although the SIR model (2.2) looks analytically simple, finding its exact analytical solution is an interesting problem. Some analytical techniques used to find the solution of (2.2) can be found in [101, 207, 261].

It is not difficult to analyze basic mathematical properties of the Kermack-McKendrick SIR model [252, 323]. More clearly, it can be shown that

$$\lim_{t\to\infty} S(t) = S_\infty > 0, \quad \lim_{t\to\infty} R(t) = R_\infty > 0, \quad \lim_{t\to\infty} I(t) = I_\infty = 0.$$

The quantity S_∞ is called the *final size of the epidemic*. In particular, the function $I(t)$ of infected individuals can monotonically decrease to zero, or first monotonically increase to some maximum value $I_{\max}$ and then decrease to zero. Here, a necessary and sufficient condition for the initial increase of $I(t)$ is easily determined and is given by

$$S(0) > \frac{\alpha}{\beta}.$$

On the other hand, $I_{\max}$ can be computed as

$$I_{\max} = -\frac{\alpha}{\beta} + \frac{\alpha}{\beta}\ln\frac{\alpha}{\beta} + S_0 + I_0 - \frac{\alpha}{\beta}\ln S_0.$$

The quantity $I_{\max}$ is very useful in estimating the progression of epidemics since it indicates when the number of infections will begin to decline.

Note that the Kermack-McKendrick SIR epidemic model, for example, uses some hypotheses:

- Infected individuals are also infectious;
- the total population remains constant;
- the population experiences no births or deaths;
- the population is closed, that is, no outside individuals enter or leave the population;
- all recovered individuals have complete immunity and are impervious to reinfection.

The above assumptions may seem rather restrictive, but they can be satisfied within certain limits. For example, several childhood diseases such as chickenpox, smallpox, rubella, mumps, scarlet fever, hand-foot-and-mouth disease lead to permanent immunity, or many vaccines can create long-lasting or even lifelong immunity [323].

Although the Kermack-McKendrick SIR epidemic model is simple and under some strict assumptions, it is still appropriate and effective for modeling many infectious diseases. In fact, once we have given disease-specific time series data, the parameter estimation problem for the SIR model can be solved by comparing its solution to the given data. Examples of parameter estimation from data can be found in [78, 80, 323]. Recently, the Kermack-McKendrick SIR epidemic model was used to study and predict the transmission dynamics of the COVID-19 pandemic [250, 268, 278, 322, 359, 442, 483].

In [253], the limitation of the SIR model (2.2) was improved by considering the effect of the continuous introduction of new susceptible individuals into the population. However, the results presented in [253] had two important limitations. One was that the disease of interest was the only cause of death, and the second was that the age of the individuals did not affect their infectivity, susceptibility, or reproductive capacity. In [254], the first of the above limitations was overcome by the introduction of constant non-specific mortality rates, which, for the sake of generality, are assumed to be different. are assumed to be different for virgins (individuals who have never been infected), sick, and recovered.

In general, the classical SIR model should be adapted to the characteristics of each epidemic.

2.2 Variants and extensions of the basic models

The classical epidemic models have played an important role in epidemic modeling. Inspired by basic epidemic models and principles of mathematical epidemiology, many mathematical models models have been proposed and developed to study infectious diseases.

There are several types of incidence, depending on the assumption made about the force of infection. One of the simplest forms is the mass action incidence or bilinear incidence function, which is $f(S, I) = \beta SI$. In the model (2.2), the *interaction term* βIS is a linearly increasing function of the number of infected individuals. As analyzed in [99], while this interaction term may be true for small I, it seems rather unrealistic that it can still hold for large I. For this reason, Capasso and Serio modified (2.2) by replacing the linear interaction term βIS by a non-linear function $g(I)S$, where $g(I)$ satisfies

1. $\forall x \in \mathbb{R}_+ : g(x) \geq 0$;
2. $g(0) = 0$;
3. $\exists c \in \mathbb{R}_+ \backslash \{0\}$ s.t. $\forall x \in \mathbb{R}_+ : g(x) \leq c$;
4. $g'(x) : \mathbb{R}_+ \to \mathbb{R}$, the derivative of g, exists and is bounded on any compact interval of $\mathbb{R}_+$, with $g'(0) > 0$;
5. $\forall x \in \mathbb{R}_+ : g(x) \leq xg'(0)$, where $\mathbb{R}_+ := [0, \infty)$.

The function $g(I)$ takes into account the "saturation" phenomenon or the other "psychological" effects. Two famous nonlinear incidence functions are the saturated incidence rate $f(S, I) = \beta SI/(1 + \gamma I)$ and the standard incidence function $f(S, I) = \beta SI/(S + I)$. Epidemic models using generalized nonlinear incidence rate can be found in [165, 166, 179, 211, 231, 285, 294, 296, 329, 415, 440, 468].

In the SIR model, it was assumed (2.2) that the rate of contacts per infective is proportional to the total population size N, which was widely used in all early epidemic models. As mentioned in [78, 82], this assumption is quite unrealistic except in the early stages of an epidemic occurring within a moderately sized population. It is more realistic to consider a contact rate that is a non-increasing function of total population size. The SIR model can then be generalized by assuming that an average member of the population makes $C(N)$ contacts per unit time, with $C'(N) \geq 0$, and defining

$$\beta(N) = \frac{C(N)}{N},$$

where $\beta'(N)$ is assumed to be negative to express the idea of saturation in the number of contacts. The following are some special cases of $C(N)$ that have been widely used in epidemic modeling with *general contact rates*.

- Standard incidence: $C(N) = \lambda$;
- Mass action incidence: $C(N) = \beta N$;
- Interaction of Michaelis-Menten type:

$$C(N) = \frac{aN}{1 + bN},$$

  which was used in [147].
- Saturating contact rate based on a mechanistic derivation for pair formation [209]

$$C(N) = \frac{aN}{1 + bN + \sqrt{1 + 2bN}}.$$

- $C(N) = \lambda N^{\alpha}$ with $\alpha = 0.05$ was used in [332]. It has been shown that this function works quite well for data on contact-borne diseases in medium-sized cities.

In recent decades, the basic classical epidemic models and their variants have been extensively developed to describe the transmission dynamics of many major infectious diseases:

- Basic virus dynamics models and outbreak spread models in epidemiology [36, 75, 163, 215, 370, 392, 471, 514];
- Influenza [8, 10, 102, 198, 434];
- Severe acute respiratory syndrome (SARS) [77, 121, 195, 229, 366, 518];
- Ebola [1, 65, 142, 288, 317, 364];
- Hepatitis B and C [168, 216, 221, 319, 324, 351, 369, 393, 452, 458, 491, 507, 515];
- Tuberculosis [24, 72, 197, 208, 299, 361, 408, 446, 466, 469];
- Vector-borne diseases [70, 85, 129, 236, 287, 418, 496];
- Malaria [3, 16, 18, 175, 235, 264, 315, 368, 424, 464, 472];
- Measles [19]
- Zika virus [11, 177, 206, 240, 263, 310, 365, 425, 432, 493, 519];
- Dengue fever [14, 107, 155, 239, 365, 395, 401];
- COVID-19 pandemic [12, 17, 33, 57, 66, 119, 123, 130, 154, 222, 248, 250, 262, 265, 268, 278, 283, 303, 314, 322, 355, 358, 359, 367, 375, 377, 387, 403, 406, 411, 442, 456, 464, 483];
- HIV/AIDS [169, 194, 242, 297, 305, 371, 416, 479].

Besides, epidemic models are widely used in

- Diabetes Mellitus [105, 361];
- cancer: malignant invasion of tumor cells [321];
- cervical cancer: human papillomavirus model [89];
- animal disease modeling with applications in agriculture [2, 9, 50, 471];
- chemostat models to represent microbial growth and competition [20, 21, 23, 444];
- modeling the spreading of computer viruses and rumors on the Internet [179, 230, 238, 292, 389, 390, 402, 404, 430, 510–512, 523, 524];
- modeling addictions, e.g. alcohol drinking [133, 233, 256, 257, 386, 419, 420, 436, 495, 503, 504], tobacco [181, 284, 298, 433, 478, 485], heroin [125, 298, 356, 451, 498], opioids [63, 88, 95, 125, 521], cocaine [421, 423], drug consumption [141, 188, 486], obesity [51, 93, 164, 244, 422], etc.

It should be noted that the ODE models of the form (2.1) are also used in the context of:

- Delayed systems [7, 104, 127, 163, 173, 174, 214, 381, 407, 445, 474, 506];
- Time fractional-order systems [7, 38, 115, 313, 455, 471];
- Stochastic Systems [29, 32, 37, 84, 92, 114, 189, 429]

for infectious disease modeling. These extended models provide an additional powerful approach to disease analysis.

2.3 PDE models

In addition to ODE models of the form (2.1), PDE models, which extend ODE models, have also been extensively studied for the analysis of infectious diseases [30, 79, 83, 323, 360, 427].

More specifically, compartmental models in epidemiology can be extended by using spatial reaction-diffusion systems, where each compartment, representing a different species, is allowed to invade a spatial domain $\Omega \subset \mathbb{R}^m$ (or a metric graph network) with a space-dependent density. The densities interact with each other according to the same mathematical laws as for the space-independent case, but are individually subject to a spatial diffusion mechanism, usually associated with the Laplace operator [48]. Then a system of n interacting species, each with a spatial density

$$\big\{u_i(x,t)\colon x \in \Omega, \quad t \geq 0\big\}, \quad i = 1, 2, \ldots, n$$

can be described by a system of semilinear parabolic PDEs of the form

$$\frac{\partial u}{\partial t}(x,t) = D\Delta u(x,t) + f\big(u(x,t)\big) \tag{2.3}$$

supplied with suitable boundary conditions, where $D = \operatorname{diag}(d_1, d_2, \ldots, d_n)$, $f\colon \mathbb{R}^n \to \mathbb{R}$ is the interaction law among the species via their densities, and

$$\Delta u(x,t) = \frac{\partial^2 u}{\partial x_1^2}(x,t) + \ldots + \frac{\partial^2 u}{\partial x_n^2}(x,t).$$

Spatial models of the form (2.3) have been used to study the transmission of infection, depending on how a particular disease is transmitted between different populations or subpopulations.

Allen et al. [31] proposed an SIS reaction-diffusion model in a heterogeneous environment to understand the impact of spatial heterogeneity of the environment and movement of individuals on the persistence and extinction of a disease. This model is given in the form:

$$\begin{aligned}
\frac{\partial}{\partial t}S(t,x) &= d_S\Delta S(t,x) - \frac{\beta(x)S(t,x)I(t,x)}{S(t,x)+I(t,x)} + \gamma(x)I(t,x), \quad t > 0,\ x \in \Omega,\\
\frac{\partial}{\partial t}I(t,x) &= d_I\Delta I(t,x) + \frac{\beta(x)S(t,x)I(t,x)}{S(t,x)-I(t,x)} - \gamma(x)I(t,x), \quad t > 0,\ x \in \Omega,
\end{aligned} \tag{2.4}$$

with the coupling condition

$$\frac{\partial}{\partial \mathbf{n}}S(t,x) = \frac{\partial}{\partial \mathbf{n}}I(t,x) = 0, \tag{2.5}$$

where

- $S(t,x)$ and $I(t,x)$ denote the density of susceptible and infectious individuals at location x and time t in a given spatial region Ω, which is assumed to be a bounded domain in $\mathbb{R}^n (n \geq 1)$ with a smooth boundary $\partial\Omega$;
- Ω is isolated from the outside for the host, implying the homogeneous Neumann boundary condition; $\mathbf{n}$ is the outward unit normal vector on $\partial\Omega$, and $\partial/\partial\mathbf{n}$ denotes the normal derivative along $\mathbf{n}$ on $\partial\Omega$.
- d_S and d_I are the dispersion for susceptible and infectious individuals, respectively;
- the positive functions $\beta(x)$ and $\gamma(x)$ are the spatially dependent transmission and recovery rates at position $x \in \Omega$, respectively.

The existence, uniqueness and asymptotic profile of the equilibria are then analyzed. First, a basic reproduction number is defined for this PDE-SIS model (2.4), which is based on the next generation approach for heterogeneous populations [145, 146]. It is then shown that if the basic reproduction number is less than 1, a unique disease-free equilibrium is globally asymptotically stable and there is no endemic equilibrium, while if the basic reproduction

number is greater than 1, the disease-free equilibrium is unstable and there is a unique endemic equilibrium. It is also pointed out that the disease-free equilibrium is always unstable for high-risk domains, and for low-risk domains, the disease-free equilibrium is stable if and only if infected individuals have mobility above a threshold. These results have several useful implications for real-world situations.

In [383], Peng provided further understanding of how large and small diffusion rates of the susceptible and infected populations affect disease persistence and extinction. In another paper [384], Peng and Yi considered a more complicated heterogeneous environment in which the moderate risk area occurs, and dealt with two cases: (i) only the moderate and high risk areas exist; (ii) the low, moderate, and high risk areas coexist. In both works, the asymptotic profile of the positive steady state was rigorously investigated, and optimal strategies for eradicating the epidemic disease were proposed.

In [232], Huang et al. proposed and studied two modified SIS diffusion models of the form (2.4) but they are associated with the Dirichlet boundary condition $S(t,x) = I(t,x) = 0$ for $x \in \partial\Omega$ and $t > 0$, reflecting a hostile environment in the boundary. The analysis of the basic reproduction number and a partial result on the global stability of the endemic equilibrium are also performed.

In [279], a spatially diffusive SIR epidemic model with the mass action infection mechanism and homogeneous Neumann boundary condition was considered in the form

$$\begin{aligned}
\frac{\partial}{\partial t}S(t,x) &= k_S\Delta S(t,x) + b(x) - \beta(x)S(t,x)I(t,x) - \mu(x)S(t,x), \quad t > 0,\ x \in \Omega,\\
\frac{\partial}{\partial t}I(t,x) &= k_I\Delta I(t,x) + \beta(x)S(t,x)I(t,x) + \big(\mu(x)+\gamma(x)\big)I(t,x), \quad t > 0,\ x \in \Omega,\\
\frac{\partial}{\partial t}R(t,x) &= k_R\Delta R(t,x) + \gamma(x)I(t,x) - \mu(x)R(t,x), \quad t > 0, \quad x \in \Omega,
\end{aligned} \tag{2.6}$$

with initial data

$$S(0,x) = S_0(x), \quad I(0,x) = I_0(x), \quad R(0,x) = R_0(x), \quad x \in \Omega, \tag{2.7}$$

and boundary conditions

$$\frac{\partial}{\partial \mathbf{n}}S(t,x) = \frac{\partial}{\partial \mathbf{n}}I(t,x) = \frac{\partial}{\partial \mathbf{n}}R(t,x)0, \tag{2.8}$$

where

- $S(t,x)$, $I(t,x)$ and $R(t,x)$ denote the populations of susceptible, infective and recovered individuals at position x and time t, respectively;
- k_S, k_I and k_R denote the dissemination rates for susceptible, infectious and recovered individuals, respectively;
- $b(x)$, $\beta(x)$, $\mu(x)$ and $\gamma(x)$ denote the birth rate, the transmission rate, the mortality rate and the recovery rate at position x, respectively.

By discretizing the PDE model (2.6) with respect to the space variable and constructing Lyapunov functions for the corresponding ODE models, the global asymptotic stability of (2.6) has been established [279].

In [280], the model (2.6) is extended by a new more realistic model with nonlocal diffusion.

In a recent paper, some extensions of the classical SIR model with non-symmetric spatial dependence are introduced to study the spread of some diseases [461]. The proposed model yields a system of partial integro-differential equations. Also, two methods that handle the integrals of the equations have been provided.

In addition to the above PDE models, a large number of spatial reaction-diffusion models of major infectious diseases such as HBV, malaria, influenza, West Nile virus transmission, Zika, etc. can be found in [56, 100, 118, 134, 255, 266, 267, 290, 300, 301, 354, 405, 437, 447, 457, 482, 488–490, 502, 505, 507, 522], in which the models proposed in [118, 255, 266, 300, 437, 482, 502, 522] can be directly used to study the COVID-19 epidemic.

3 Qualitative analysis and applications

Qualitative analysis of differential equations modeling infectious diseases is very important since it can have many useful applications in reality, such as suggesting appropriate strategies for disease control and prevention; evaluating the effects of vaccines; waning immunity; parameter estimation problems; parameter sensitivity analysis and optimal control strategies (usually w.r.t. vaccination strategies, stakeholder decisions (wearing masks, physical isolation, curfews, etc.).

In this section, we emphasize qualitative analytical aspects of differential equation models and their applications, where methods, approaches, and tools used in qualitative analysis are discussed in detail.

3.1 Analysis of ODE models

The first property of interest for ODE models of infectious diseases is well-posedness, including existence, uniqueness of solutions, and continuous dependence on initial data. Well-posedness is easy to establish and is often automatically satisfied due to the smoothness of the right-hand-side functions [30, 55, 258, 454]. In general, in addition to well-posedness, qualitative analysis aspects of ODE models of infectious diseases focus mainly on the following issues.

3.1.1 The positivity and boundedness of the solutions

Obviously, positivity should be an obvious property of the solutions of ODE models for infectious diseases, i.e. $y(t) \in \mathbb{R}^n_+ = \{(y_1, y_2, \ldots, y_n) \in \mathbb{R}^n | y_1, y_2, \ldots, y_n \geq 0\}$ for $t > 0$ whenever $y(0) \in \mathbb{R}^n_+$. In this case, the set $\mathbb{R}^n_+$ is called a *positively invariant set*. This property can be easily verified using well-known theorems on the positivity of ODEs [228, Lemma 1], [444]. Meanwhile, boundedness can be established on the basis of comparison theorems for differential equations [330]. Note that positively invariant sets and feasible sets of ODE models follow from their positivity and boundedness.

3.1.2 Conservation laws

Many ODE models in population dynamics and also in epidemiology can satisfy some *conservation laws*, such as direct, generalized and subconservation laws [344, 347]. Conservation laws for ODE models of infectious diseases can be established based on the theory of ODEs [20, 30, 258, 444, 454] or comparison theorems for differential equations [330].

3.1.3 Equilibrium points

Equilibrium points of ODE models of the form (2.1) are solutions of the equation $f(y) = 0$. An equilibrium point is also called a *fixed point, constant solution, steady state, critical point* or a *steady-state solution* [30, 258, 454]. In general, it is not difficult to determine the set of equilibrium points, except when the ODE model under consideration has high dimensions and contains many parameters. Two common types of equilibria are *disease-free equilibrium* (DFE) and *endemic equilibrium* (EE) points, which correspond to the possibility of the epidemic being suppressed or remaining in the community.

3.1.4 Local asymptotic stability (LAS)

An equilibrium y^* is said to be *locally stable* if for every $\epsilon > 0$ there exists a $\delta > 0$ with the property that every solution $y(t)$ starting from the initial condition $y(0) = y_0$ with $\|y_0 - y^*\| < \delta$ satisfies $\|y(t) - y_0\| < \epsilon$ for all $t \geq 0$. It is said to be *locally asymptotically stable* if it is sable and there exists $\gamma > 0$ such that $\|y_0 - y^*\| < \gamma$ implies $\lim_{t\to\infty} y(t) = y^*$ (see, e.g., [30, 258, 454]). The local dynamics of dynamical systems has several important implications in the real world. The LAS of equilibrium points can be studied by the *Lyapunov indirect method* using the *Routh-Hurwitz criteria* [30, 258, 454]. This approach analyzes the LAS of an equilibrium point by considering the position of the eigenvalues of the Jacobian matrix evaluated at the equilibrium point with respect to the left-half plane. More specifically, an equilibrium point y^* is locally asymptotically stable if all eigenvalues λ of the Jacobian $J(y^*) = (\partial f/\partial y)(y^*)$ satisfy $\mathrm{Re}(\lambda) < 0$, and it is unstable if $\mathrm{Re}(\lambda) > 0$ for one or more of the eigenvalues of J. Note that the direct Lyapunov method is only applicable to *hyperbolic equilibrium* points. Here, an equilibrium point y^* is said to be *hyperbolic* if none of the eigenvalues of the matrix J lie on the imaginary axis, and *non-hyperbolic* otherwise, cf. [454].

3.1.5 Global asymptotic stability (GAS)

An equilibrium point y^* is said to be *globally asymptotically stable* if it is stable and *globally attractive*, i.e. $\lim_{t\to\infty} y(t, y_0) = y^*$ for all initial conditions y_0 (see e.g. [30, 258, 454]). The GAS analysis of equilibrium points is a very important problem because it can reveal the future evolution of epidemics. In particular, the GAS of free-disease equilibrium points indicates that epidemics will be extinguished, while the GAS of endemic-equilibrium points indicates that epidemics will exist stably in the population. In general, the GAS problem is not an easy one. One of the most successful approaches to this problem is the Lyapunov stability theory [286, 309]. This approach requires suitable candidate Lyapunov functions that must satisfy some specific conditions. In general, it is not easy to determine a Lyapunov function for a given dynamical system. However, several classes of Lyapunov functions have been proposed to analyze the GAS of ODE models in epidemiology [98, 272–274, 374, 406, 438, 480, 508], where common classes of Lyapunov functions are linear, quadratic and Volterra-type Lyapunov functions or combinations of them. In particular, Cangiotti [98] provided an overview of Lyapunov functions for epidemic compartmental models.

On the other hand, the geometric method is a remarkable approach to the GAS analysis of ODEs [293–295]. Also, the Poincaré-Bendixson Theorem in combination with the Bendixson-Dulac Criterion is very useful in studying the GAS of two-dimensional dynamical systems governed by ODEs [30, 323].

In [103], Castillo-Chavez et al. discussed some conditions that clarify the connections between the basic reproduction number and its relation to the GAS of disease-free equilibrium points of epidemiological models. Then, global stability conditions for disease-free equilibrium points were given, which are easy to verify.

3.1.6 Basic reproduction number

One of the most important concerns about any infectious disease is its reproductive number $\mathcal{R}_0$, which is useful in guiding control strategies [145, 146, 475–477]. The basic reproduction number can be defined as the expected number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a fully susceptible population [145]. It can also be considered as a threshold parameter for the local asymptotic stability of the disease-free equilibrium [475]. The basic reproduction number of epidemic models is very useful in guiding control strategies with the help of sensitivity analysis.

3.1.7 Optimal control problems

Epidemic models based on differential equations are often combined with optimal control strategies to find effective disease control measures [58, 60, 68, 86, 113, 260, 307, 432, 435, 463]. The proposed optimal control problems can be solved using Pontryagin's maximum principle [391].

3.1.8 Epidemic models with effect of vaccines

It is well known that vaccines are effective tools to combat infectious diseases and to protect people against disease. For this reason, epidemic models with the effect of vaccines are often considered [8, 25, 156, 157, 163, 167, 180, 193, 196, 234, 277, 414, 439]. The study of vaccination models [34, 192, 363, 473] can evaluate the efficacy of certain vaccines and suggest effective vaccination strategies.

3.1.9 Parameter estimation problem

ODE models for infectious diseases can be combined with real data of diseases to predict possible scenarios in reality. Therefore, the parameter estimation problem is very important to find best-fit parameters [78, 80, 323]. Following this approach, the parameter estimation problem has been extensively studied for several epidemic models [122, 311, 357, 397, 417], especially for the COVID-19 pandemic [250, 268, 307, 322, 359].

3.1.10 Bifurcation analysis and chaos

It is well-known that bifurcation theory studies qualitative changes in the state of a system as a parameter is varied [106, 282]. In general, applications of bifurcation analysis in epidemiology are very diverse, especially in studying the evolution and determining factors that may be associated with the suppression or outbreak of disease. For example, the forward bifurcation phenomenon, first noted by Kermack and McKendrick in [252], can be observed in several disease transmission models [160]. For epidemic models that exhibit *forward bifurcation*, the condition $\mathcal{R}_0 < 1$ is a necessary and sufficient condition for disease elimination [160, 199].

For many years, bifurcation analysis for epidemic models has been studied extensively with many useful applications, including forward bifurcation, *backward bifurcation, Hopf bifurcation, Bogdanov-Takens bifurcation, saddle-node bifurcation, flip bifurcation* are mainly focused [26, 28, 54, 76, 120, 185, 202, 243, 281, 304, 327, 412, 431].

Chaos theory has many useful applications in many fields such as physics, biology, ecology and epidemiology, economics, etc. [73, 217, 328, 428]. In recent decades, chaos theory has been developed and studied with the aim of discovering chaotic phenomena/dynamics, complicated or even unpredictable dynamical behavior in epidemic models [67, 74, 87, 161, 185, 190, 246, 316–318, 372, 373].

3.2 Analysis of PDE models

In general, the qualitative analysis aspects for PDE models of infectious diseases are very similar to those for ODE models.

In particular, the qualitative analysis of PDE models also focuses on well-posedness of mathematical models, positivity and boundedness of the solution, conservation laws, equilibria and their asymptotic stability, basic reproduction numbers and their implications, optimal control problems, parameter estimation, vaccination models, bifurcations and chaos [31, 56, 61, 62, 91, 108, 109, 128, 129, 134, 158, 178, 187, 232, 241, 255, 267, 279, 280, 290, 291, 301, 306, 312, 353, 354, 385, 405, 437, 447, 448, 450, 457, 461, 482, 487–490, 494, 502, 505, 507, 509, 517, 520, 522, 525].

Several methods and tools used in the qualitative analysis of ODE models, such as basic reproduction number, Lyapunov stability theory, optimal control, bifurcation and chaos analysis, can be developed and extended for PDE models. However, the qualitative study for PDE models is more challenging due to the complexity of their structures.

4 Numerical methods

4.1 Standard and nonstandard numerical methods

It is well known that both ODEs and PDEs can be solved exactly only in a small number of cases, and that in most real-world situations it is almost inevitable to find approximate solutions. For this reason, numerical methods for differential equations have become one of the most fundamental and practically important research tasks [55, 203, 204, 289, 443, 453, 454, 467].

Numerical solutions for ODE models can be easily obtained using standard numerical methods such as the Runge–Kutta and Taylor (one-step) methods and multistep methods, while finite difference methods are appropriate and efficient for the numerical solution of PDE models [55, 203, 204, 289, 445, 453, 454, 467]. However, mathematical models arising in real-world applications in general, and in infectious disease modeling in particular, often possess several essential qualitative features, such as positivity, boundedness, asymptotic stability properties, conservation laws, periodicity and physical properties, etc., which must be respected by corresponding numerical schemes. Therefore, an important requirement for numerical methods is that they correctly preserve the essential properties of the corresponding differential equations. However, it has been shown by Mickens in [335, 338, 342, 343, 346, 348] that standard numerical methods cannot preserve the mathematical properties of ODEs for all values of the temporal step size.

In the 1980s, Mickens proposed the concept of *nonstandard finite difference (NSFD) methods* to compensate for drawbacks and shortcomings of standard numerical methods [335, 338, 342, 343, 346, 348]. One of the main and outstanding advantages of NSFD methods is that they can preserve essential mathematical properties of differential equations independently of the values of the step size. Such NSFD methods are said to be *dynamically consistent*. Thus, dynamically consistent NSFD methods are efficient and suitable for simulating the behavior of dynamic differential equation models over long periods of time.

In addition to NSFD methods for ODEs, geometric numerical integration [96, 191, 205] (or both [93]) and positivity-preserving Runge–Kutta methods [69, 183, 228, 462] and modified Patankar-Runge–Kutta schemes [270, 271] have also been developed to construct reliable numerical methods that preserve the positivity as well as other dynamical properties of ODE models.

In the next subsection, we provide an overview of NSFD methods for mathematical models of infectious diseases and their applications.

4.2 Nonstandard finite difference methods for epidemiological models of infectious diseases

In numerical analysis, *numerical instabilities* are solutions of finite difference models that do not correspond to any solution of the counterpart differential equation [346]. Mickens, the creator of the concept of NSFD methods, wrote: "Numerical instabilities are an indication that the discrete models are unable to model the correct mathematical properties of the solutions to the differential equations of interest" [335, 338, 342, 343, 346, 348]. The concept of NSFD schemes was first introduced by Mickens in the 1980s to overcome the usual numerical instabilities associated with standard finite-difference schemes [335, 338, 342, 343, 346, 348]. A finite difference scheme is said to be *nonstandard* if it is constructed based on a set of basic rules proposed by Mickens [335, 338, 342, 343, 346, 348]. In particular, NSFD schemes for the ODE models of the form (2.1) can be defined as follows.

Consider a general finite difference scheme for (2.1) of the form

$$D_{\Delta t}(y_k) = F_{\Delta t}(f; y_k), \tag{4.1}$$

where $D_{\Delta t}(y_k) \approx dy/dt$, $F_{\Delta t}(f; y_k) \approx f(y)$ and $t_k = k\Delta t$, Δt is the step size.

Definition 4.1 [39, 44, 151] The finite difference scheme (4.1) is called an NSFD scheme if at least one of the following conditions is satisfied:

- $D_{\Delta t}(y_k) = \dfrac{y_{k+1} - y_k}{\phi(\Delta t)}$, where $\phi(\Delta t) = \Delta t + \mathcal{O}(\Delta t^2)$ is a non-negative function and is called a nonstandard denominator function;
- $F_{\Delta t}(f; y_k) = g(y_k, y_{k+1}, \Delta t)$, where $g(y_k, y_{k+1}, \Delta t)$ is a non-local approximation of the right-hand side of the system (2.1).

NSFD schemes for (parabolic) PDEs [35, 111, 117, 144, 212, 249, 276, 331, 340, 362, 379, 394], fractional-order differential equations [90], delay differential equations are similarly defined based on the Mickens' methodology.

The main advantage of NSFD schemes over standard schemes is expressed in the following definitions.

Definition 4.2 [39, 44] Assume that the solutions of the equation (2.1) satisfy some property $\mathcal{P}$. The numerical scheme (4.1) is said to be (qualitatively) stable with respect to the property

$\mathcal{P}$ (or $\mathcal{P}$-stable), if for every value of $\Delta t > 0$ the set of solutions of (4.1) satisfies the property $\mathcal{P}$.

Definition 4.3 [41, 302, 342] Consider the differential equation $dy/dt = f(y)$. Let a finite difference scheme for the equation be $y_{k+1} = F(y_k; \Delta t)$. Let the differential equation and/or its solutions have the property $\mathcal{P}$. The discrete model equation is dynamically consistent with the differential equation if it and/or its solutions also have the property $\mathcal{P}$.

Nowadays, NSFD methods based on the Mickens' methodology have become an efficient approach for numerically solving ODE models arising in real-world problems [5, 39, 40, 44, 131, 132, 135–140, 148–153, 172, 200, 225, 226, 334, 335, 338, 339, 342–344, 346–348, 350, 380, 382, 409, 410, 449, 499–501]. In particular, NSFD schemes have been extensively studied for epidemic models, such as

- General epidemiological models [44, 52, 53, 110, 201, 325, 326]
- Influenza disease [176, 245, 259];
- Ebola [17, 45, 65, 237, 459];
- Hepatitis B [220, 221];
- Visceral Leishmaniasis [4, 441];
- Malaria [43, 170];
- Measles [13, 171];
- Zika [310, 465];
- COVID-19 [66, 130, 210, 213, 222, 314, 396, 403, 470];
- Cancer: malignant invasion of tumor cells [49];
- Computer virus propagation models [139, 218, 402].

Compared to numerical methods for ODE models, numerical methods for PDE models are more challenging. Finite difference methods are one of the most common and efficient approaches for numerical simulation of PDEs [55, 289, 453, 467]. It is important to note that positivity should be an obvious property of the solutions of both ODE and PDE models for infectious diseases. Therefore, positivity preserving numerical methods are essential. To the best of our knowledge, numerical methods that preserve positivity and other dynamical properties for the PDE models are few. However, NSFD methods based on Mickens' methodology have been shown to be suitable and effective for constructing such numerical methods [39, 41, 42, 112, 126, 162, 335–338, 341–343, 345, 346, 348, 349, 492]. In particular, dynamically consistent NSFD schemes have been applied to solve some PDE models of infectious diseases [182, 319, 320, 378, 393, 457, 458, 513].

Even though NSFD methods have several advantages, most of the existing dynamically consistent NSFD methods are only first-order convergent [116, 124, 131, 218, 220, 221], which can be considered as an inherent drawback of NSFD methods. For this reason, the problem of improving the accuracy of NSFD methods has attracted the attention of many researchers [22, 116, 138, 186, 219, 223, 224, 226, 227, 269, 325, 326]. However, it is very challenging to construct dynamically consistent NSFD methods, especially high-order methods, for differential equations.

In recent years, there has been an increased interest in solving PDEs using *Deep Learning* (see e.g. [64, 71, 210, 398]). More recently, in [275], a deep learning approach has been proposed to improve numerical methods for PDEs. This approach is based on an approximation of the local truncation error of the numerical method used to approximate the spatial derivatives of a given PDE.

In general, the construction of numerical methods, especially those that preserve important properties of differential models, is an important problem but not easy to solve. In addition,

high-order numerical methods are still an important problem that has not been fully solved, and the reduced spatial accuracy of NSFD methods for PDEs is still an open problem.

NSFD methods for PDEs have lacked guaranteed first-order temporal accuracy and consistency for key models such as diffusion and reaction-diffusion systems. In a recent paper Pasha, Nawaz and Arif [378] proposed a novel NSFD scheme that overcomes this limitation and guarantees first-order temporal accuracy and second-order spatial accuracy while preserving positivity. The question remains as to how one can develop compact higher-order schemes with the NSFD concept.

5 Future research and open problems

Although research on differential equation models for infectious diseases has been extensively developed over the past decades and has achieved many important successes, these models still need to be studied and expanded for the following reasons.

First, mankind is always facing and fighting many infectious diseases, which are not only constantly changing but also difficult to predict, and thus always pose a great and constant threat to public health. In this context, the development of mathematical models of infectious diseases remains a fundamental and effective approach to discover the characteristics and mechanisms of transmission of epidemics, and thus effectively predict possible scenarios in reality. On the other hand, as the existing differential equation models are built based on observations, experience, and understanding of the diseases, they often become outdated and therefore need to be updated and modified to keep up with the constant changes in epidemics. Therefore, in addition to building new models, improving existing models is also very important.

Second, once mathematical models have been formulated, aspects of qualitative study and approximate solutions are raised. Addressing these issues is useful for finding appropriate strategies for disease prevention and control, as well as for predicting disease spread scenarios. In addition, infectious diseases often need to be monitored over very long periods of time. This leads to the rapid solution of differential equation models over long time periods. Therefore, efficient numerical methods are urgently needed. However, the construction of efficient high-order numerical methods in general, and numerical methods that preserve essential qualitative properties of differential equation models in particular, is still an important problem that has not been fully solved.

Lastly, the practical application of mathematical models of infectious diseases is essential, but has not been widely used. In particular, theoretical studies should be combined with observed real-world epidemic data to calibrate the mathematical models and find optimal parameters, thereby building scenarios that better reflect reality and proposing appropriate anti-epidemic strategies.

For the above reasons, differential equation models for infectious diseases need to be studied and developed. To achieve this, it is also necessary to develop and extend research methods to keep pace with the complexity of the proposed models.

Another future direction is to use one NSFD scheme not exclusively, but as one element in a *hybrid scheme approach*, e.g. using operator splitting [13, 17], Chebyshev collocation [6], Hermite Polynomials [400] wavelets [399, 481] or a predictor corrector NSFD approach [171].

A special challenge are mimetic / fitted operator schemes for singular perturbed problems, due to the necessary resolution of boundary layers having different scales, e.g. convection-diffusion equations [46, 47, 251], Burgers-Huxley equation [143], differential difference equations [381] or boundary value ODE problems [308, 376].

Finally, most recent research directions for NSFD schemes are integro-differential equations [333], the GPU acceleration of the (serial) NSFD code [247] and geometric numerical integration, symmetrization of NSFD schemes [93].

6 Concluding remarks and discussions

In this work, we have reviewed many but not all recent developments and real-life applications of deterministic ODEs and PDEs of major infectious diseases, mainly focusing on mathematical modeling, qualitative analysis, numerical methods and real-life applications. We have also presented and discussed some open problems and future directions that research in differential equation models for infectious diseases can take. In the presentation, we focus only on deterministic differential equation models associated with the integer-order derivatives. Delayed models [352], stochastic models [16, 59, 388, 516], and fractional-order models, especially for PDEs [46, 47, 90, 460], will be considered in future work.

All the results presented demonstrate the important role of differential equation models in disease modeling. Moreover, they remain an effective and indispensable approach to study the characteristics of infectious diseases and thereby suggest effective measures for disease prevention and public health protection.

Acknowledgements The first author, Manh Tuan Hoang, wishes to thank Vietnam Institute for Advanced Study in Mathematics (VIASM) for the financial support and the excellent working condition. This work was completed while he was working at the VIASM.

Author Contributions Manh Tuan Hoang (M.T.H.) initiated the concept of the review, defined the scope, conceived the study, designed the methodology, performed critical analysis and comparison of mathematical models and methods. Matthias Ehrhardt (M.E.) supervised the project and the overall structure and content development, drafted significant portions of the manuscript, focusing on theoretical frameworks and methodological discussions. All authors contributed to manuscript editing, to final revisions, conducted an extensive literature search, and approved the final manuscript.

Funding Open Access funding enabled and organized by Projekt DEAL. Manh Tuan Hoang was supported by the Vietnam Institute for Advanced Study in Mathematics (VIASM).

Data Availability Does not apply.

Declarations

Conflict of interest The authors declare that they have no Conflict of interest.

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