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Nonlinear Mathematical Modeling of Infectious Disease Progression in Biological Systems

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Abstract

The dynamics of infectious disease development within a population of interacting organisms are investigated using a mathematical modeling framework. The epidemic process is described by a system of nonlinear ordinary differential equations that account for susceptible, asymptomatically infected, symptomatic, and unreported infected individuals. The model incorporates time-dependent parameters and delay effects to capture realistic features of disease transmission and progression. An analytical approach based on the method of averaging of functional corrections is proposed and applied to obtain approximate solutions of the governing system. First- and second-order approximations are derived, enabling a detailed analysis of the temporal evolution of the epidemic process. The accuracy of the analytical results is confirmed through comparison with numerical simulations, showing strong agreement between the two approaches. The proposed model allows for the investigation of how variations in epidemiological parameters influence the speed and pattern of disease spread. In particular, transmission, recovery, and progression rates significantly affect the amplitude and duration of the epidemic outbreak. The results also highlight the important role of asymptomatic and unreported infections in sustaining disease transmission within the population.

Keywords: Infectious dynamics, Epidemiological model, Nonlinear ODEs, Analytical approximation, Averaging method, Epidemic modeling, Delay systems.

1 | Introduction

The infectious process represents a complex, multicomponent phenomenon arising from dynamic interactions between pathogenic agents and the host organism. These interactions involve a wide spectrum of biological mechanisms, including cellular and molecular responses, systemic physiological changes, and regulatory feedback processes. The infectious process is characterized not only by typical pathological manifestations but also by systemic functional alterations, hormonal imbalances, activation of specific immune responses, and engagement of nonspecific resistance mechanisms [1–5]. Such complexity reflects the

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inherently dynamic and adaptive nature of host–pathogen relationships, which evolve over time and under varying environmental conditions. A comprehensive understanding of these interactions is fundamental, as the infectious process constitutes the basis for the development and progression of infectious diseases. Despite significant advances in modern medicine, including vaccination, antimicrobial therapies, and improved sanitation, infectious diseases continue to pose substantial challenges to global health systems. Their persistence is attributed to multiple factors, including pathogen evolution, environmental changes, globalization, and increasing population density. Consequently, the study of infectious disease dynamics remains a critical area of interdisciplinary research, integrating biology, medicine, mathematics, and public health [6], [7]. From an epidemiological standpoint, infectious diseases remain among the leading causes of morbidity and mortality worldwide. Historically, they have ranked alongside cardiovascular and oncological diseases as major contributors to global disease burden. The successful eradication or control of several infectious diseases, such as smallpox and diphtheria, highlights the effectiveness of coordinated public health interventions and advances in biomedical science [8–10]. However, these achievements are counterbalanced by the continuous emergence of new pathogens, the re-emergence of previously controlled diseases, and the rapid spread of antimicrobial resistance [11]. These trends underscore the limitations of traditional approaches and emphasize the necessity for innovative tools capable of predicting and managing infectious disease outbreaks. In recent decades, mathematical and analytical modeling has emerged as a powerful approach for studying the dynamics of infectious diseases [12–15]. These models provide a systematic framework for representing complex biological processes in a quantitative and interpretable manner. By incorporating key variables such as transmission rates, recovery rates, population structure, and environmental influences, mathematical models enable researchers to investigate how infectious agents propagate within and between populations. Moreover, they facilitate the identification of critical thresholds, such as the basic reproduction number, that determine whether an infection will spread or decline. Analytical models, in particular, play a crucial role in enhancing our understanding of infection dynamics. Unlike purely numerical simulations, analytical approaches allow for the derivation of explicit relationships between model parameters and system behavior. This provides deeper insights into the mechanisms governing disease progression and enables the formulation of general principles applicable across different infectious systems. Furthermore, analytical models are especially valuable in situations where empirical data are limited or uncertain, as they can offer qualitative predictions and guide experimental and observational studies. One of the key challenges in modeling infectious diseases lies in capturing the nonlinear and often stochastic nature of host–pathogen interactions. The progression of an infection within an organism or population is influenced by a multitude of factors, including immune system variability, pathogen virulence, environmental conditions, and social behavior. These factors interact in complex ways, leading to diverse outcomes ranging from rapid disease clearance to chronic infection or widespread epidemics. Therefore, the development of robust models capable of accounting for such complexity is essential for advancing both theoretical and applied aspects of infectious disease research. In this study, we propose an analytical model designed to describe and predict the development of infectious diseases within a population of organisms. The proposed framework is grounded in dynamical systems theory, which provides a natural and rigorous foundation for analyzing time-dependent processes governed by interacting variables. By formulating the infectious process as a system of coupled equations, we aim to capture the essential features of disease progression while maintaining analytical tractability. A central objective of this work is to identify the conditions under which the progression of an infectious disease may accelerate or decelerate. These conditions are determined by the interplay of biological, environmental, and epidemiological factors, which are explicitly incorporated into the model. By analyzing the stability and behavior of the system under different parameter regimes, we seek to determine the key drivers of disease dynamics and to establish criteria for effective intervention. In addition to its theoretical contributions, the proposed modeling approach has important practical implications. Predictive models of infectious diseases are invaluable tools for public health planning and decision-making. They can be used to estimate the potential impact of intervention strategies, such as vaccination, quarantine, and treatment programs, and to optimize resource allocation in healthcare systems. Furthermore, in the early stages of an outbreak, when empirical data are scarce, analytical models can provide rapid assessments of transmission

potential and outbreak severity, thereby supporting timely and informed responses. Another important aspect of infectious disease modeling is the incorporation of population heterogeneity. Real-world populations are not homogeneous; they differ in terms of age, immunity, behavior, and susceptibility to infection. Environmental factors, such as climate and geographical conditions, also play a significant role in shaping disease dynamics. By integrating these sources of variability into analytical frameworks, models can achieve greater realism and predictive accuracy. This, in turn, enhances their utility in designing targeted and context-specific intervention strategies. Moreover, analytical modeling serves as a bridge between theoretical biology and applied epidemiology. It allows researchers to test hypotheses, explore hypothetical scenarios, and evaluate the potential consequences of different policy decisions without the ethical and logistical constraints associated with real-world experimentation. In this way, models contribute not only to scientific understanding but also to the development of evidence-based strategies for disease prevention and control.

The purpose of this paper is to develop a comprehensive analytical framework capable of describing the fundamental patterns of infectious disease development and providing actionable insights into their control. Through systematic analysis, we investigate the influence of key parameters on the rate of disease progression and identify both accelerating and decelerating factors. Particular attention is given to the conditions that lead to qualitative changes in system behavior, such as the transition from endemic stability to epidemic outbreaks.

The findings presented in this study contribute to a deeper understanding of infectious disease dynamics and highlight the importance of quantitative approaches in modern epidemiology. By combining rigorous mathematical analysis with biological interpretation, the proposed model offers a valuable tool for both researchers and public health practitioners. It provides a basis for further theoretical developments and can be adapted to a wide range of infectious diseases and epidemiological contexts. In conclusion, infectious diseases continue to exert a profound impact on human health despite decades of scientific and medical progress. The complexity of the infectious process, coupled with the evolving nature of pathogens and host responses, necessitates the use of advanced analytical tools for effective study and management. Mathematical modeling, and analytical approaches in particular, play a central role in addressing these challenges by enabling the prediction of disease dynamics, the evaluation of intervention strategies, and the improvement of outbreak preparedness. By elucidating the intricate interplay between pathogens and hosts, and by identifying the factors that govern disease spread, such models contribute significantly to the prevention and mitigation of infectious disease threats worldwide.

2 | Method of Solution

In this section, we consider the following model based on the solution of the following system of ordinary differential equations.

$$\begin{cases} \dfrac{dS(t)}{dt} = \gamma(t) - \beta(t)S(t)\left[I(t) - U(t)\right] - \mu(t)S(t), \\ \dfrac{dI(t)}{dt} = \beta(t)S(t-\tau)\left[I(t-\tau) - U(t-\tau)\right] - \left[\nu_1(t) + \nu_2(t) + \mu(t)\right]I(t), \\ \dfrac{dR(t)}{dt} = \nu_1(t)I(t) - \left[\eta(t) + \mu(t)\right]R(t), \\ \dfrac{dU(t)}{dt} = \nu_2(t)I(t) - \left[\eta(t) + \mu(t)\right]U(t), \end{cases} \tag{1}$$

where $S(t)$ is the number of organisms susceptible to infection; $I(t)$ is the number of asymptomatically infected organisms; $R(t)$ is the number of infected organisms with associated symptoms; $U(t)$ is the number of infected organisms with relevant symptoms but not reported; t is the current time; τ is the delay; $\gamma(t)$, $\beta(t)$, $\eta(t)$, $\mu(t)$, $\nu_i(t)$ are the parameters of the system. Initial conditions for the functions $S(t)$, $I(t)$, $R(t)$ and $U(t)$ could be written as *Eq. (2)*:

$$S(0) = S_0,\ I(0) = I_0,\ R(0) = R_0,\ U(0) = U_0. \quad (2)$$

We solved the system of *Eqs. (1)* by the method of averaging of functional corrections. In the framework of the considered method, we replace the required functions with their not yet known average values α_1 in the right sides of the *Eqs. (1)*. Next integration of the obtained relations on time leads to the following relations to determine the first-order approximations of the required functions in the following form:

$$\begin{aligned}
S_1(t) &= \int_0^t \gamma(\theta)d\theta - \alpha_{1S}(\alpha_{1I} - \alpha_{1U})\int_0^t \beta(\theta)d\theta - \alpha_{1S}\int_0^t \mu(\theta)d\theta + S_0.\\
I_1(t) &= \alpha_{1S}(\alpha_{1I} - \alpha_{1U})\int_0^t \beta(\theta)d\theta - \alpha_{1I}\int_0^t \left[\nu_1(\theta) + \nu_2(\theta) + \mu(\theta)\right]d\theta + I_0.\\
R_1(t) &= \alpha_{1I}\int_0^t \nu_1(\theta)d\theta - \alpha_{1R}\int_0^t \left[\eta(\theta) + \mu(\theta)\right]d\theta + R_0.\\
U_1(t) &= \alpha_{1I}\int_0^t \nu_2(\theta)d\theta - \alpha_{1U}\int_0^t \left[\eta(\theta) + \mu(\theta)\right]d\theta + U_0.
\end{aligned} \quad (3)$$

Not yet known average values were determined by the following standard equation.

$$\alpha_{1\rho} = \frac{1}{\Theta}\int_0^\Theta \rho_1(t)\,dt. \quad (4)$$

Substitution of *Eq. (3)* into *Eq. (4)* and solution of the obtained equations gives a possibility to obtain relations for the required average values.

$$\begin{aligned}
\alpha_{1S} &= \frac{\frac{1}{\Theta}\int_0^\Theta (\Theta - t)\gamma(t)dt + S_0}{\frac{1}{\Theta}\int_0^\Theta (\Theta - t)\beta(t)dt + \frac{1}{\Theta}\int_0^\Theta (\Theta - t)\mu(t)dt}.\\
\alpha_{1I} &= \frac{I_0}{\frac{1}{\Theta}\int_0^\Theta (\Theta - t)\beta(t)dt + \frac{1}{\Theta}\int_0^\Theta (\Theta - t)\left[\nu_1(t) + \nu_2(t) + \mu(t)\right]dt}.\\
\alpha_{1R} &= \frac{R_0 + \frac{1}{\Theta}\int_0^\Theta (\Theta - t)\nu_1(t)dt}{\frac{1}{\Theta}\int_0^\Theta (\Theta - t)\left[\eta(t) + \mu(t)\right]dt}.\\
\alpha_{1U} &= \frac{U_0 + \frac{1}{\Theta}\int_0^\Theta (\Theta - t)\nu_2(t)dt}{\frac{1}{\Theta}\int_0^\Theta (\Theta - t)\left[\eta(t) + \mu(t)\right]dt}.
\end{aligned} \quad (5)$$

The second-order approximations were determined by replacement by required functions on the following sum $\rho(t) \to \alpha_2 + \rho_1(t)$ in the right sides of *Eqs. (1)*. The replacement and integration of the obtained relations

on time gives a possibility to obtain the second-order approximations of the required functions in the following form.

$$\begin{cases} S_2(t) = \int\limits_0^t \gamma(\theta)d\theta - \int\limits_0^t \beta(\theta)\left[\alpha_{2S} + S_1(\theta)\right]\left[\alpha_{2I} + I_1(\theta) - \alpha_{2U} - U_1(\theta)\right]d\theta - \\ \qquad \int\limits_0^t \mu(\theta)\left[\alpha_{2S} + S_1(\theta)\right]d\theta + S_0, \\ I_2(t) = \int\limits_0^t \beta(\theta)\left[\alpha_{2S} + S_1(\theta-\tau)\right]\left[\alpha_{2I} + I_1(\theta-\tau) - \alpha_{2U} - U_1(\theta-\tau)\right]d\theta - \\ \qquad \int\limits_0^t \left[\nu_1(\theta) + \nu_2(\theta) + \mu(\theta)\right]\left[\alpha_{2I} + I_1(\theta)\right]d\theta + I_0, \\ R_2(t) = \int\limits_0^t \nu_1(\theta)\left[\alpha_{2I} + I_1(\theta)\right]d\theta - \int\limits_0^t \left[\eta(\theta) + \mu(\theta)\right]\left[\alpha_{2R} + R_1(\theta)\right]d\theta + R_0, \\ U_2(t) = \int\limits_0^t \nu_2(\theta)\left[\alpha_{2I} + I_1(\theta)\right]d\theta - \int\limits_0^t \left[\eta(\theta) + \mu(\theta)\right]\left[\alpha_{2U} + U_1(\theta)\right]d\theta + U_0. \end{cases} \tag{6}$$

Average values of the second-order approximations of the required functions were determined by the following standard relations.

$$\alpha_{2\rho} = \frac{1}{\Theta}\int\limits_0^\Theta \left[\rho_2(t) - \rho_1(t)\right]dt. \tag{7}$$

Substitution of *Eq. (6)* into *Eq. (7)* and solution of the obtained equations gives a possibility to obtain relations for the required average values.

$$\alpha_{2S} = \left[\int\limits_0^\Theta (\Theta - t)\beta(t)S_1(t)\left[I_1(t) - U_1(t)\right]dt + \int\limits_0^\Theta (\Theta - t)\mu(t)S_1(t)dt - \alpha_{1S}(\alpha_{1I} - \alpha_{1U}) \times \right.$$

$$\left. \frac{1}{\Theta}\int\limits_0^\Theta (\Theta - t)\beta(t)\,dt - \alpha_{1S}\int\limits_0^\Theta (\Theta - t)\mu(t)dt\right]\left[\int\limits_0^\Theta (\Theta - t)\mu(t)dt\right]^{-1}.$$

$$\alpha_{2I} = \left\{\int\limits_0^\Theta (\Theta - t)\beta(t)S_1(t-\tau)\left[I_1(t-\tau) - U_1(t-\tau)\right]dt + \int\limits_0^\Theta (\Theta - t)\left[\nu_1(t) + \nu_2(t) + \mu(t)\right] \times \right.$$

$$\left. I_1(t)\,dt - \int\limits_0^\Theta (\Theta - t)\beta(t)S_1(t)\left[I_1(t) - U_1(t)\right]dt\right\}\left\{\int\limits_0^\Theta (\Theta - t)\beta(t)\left[I_1(t) - U_1(t)\right]dt + \right. \tag{8}$$

$$\left. \int\limits_0^\Theta (\Theta - t)\beta(t)S_1(t-\tau)\left[\alpha_{2I} + I_1(t-\tau) - \alpha_{2U} - U_1(t-\tau)\right]dt\right\}^{-1}.$$

$$\alpha_{R2} = \left\{\int\limits_0^\Theta (\Theta - t)\nu_1(t)I_1(t)dt - \int\limits_0^\Theta (\Theta - t)\left[\eta(t) + \mu(t)\right]R_1(t)dt\right\}\left\{\int\limits_0^\Theta (\Theta - t)\nu_1(t)dt - \right.$$

$$\int_0^{\Theta}(\Theta-t)\left[\eta(t)+\mu(t)\right]dt\Bigg\}.$$

$$\alpha_{U2}=\left\{\int_0^{\Theta}(\Theta-t)\nu_2(t)I_1(t)dt-\int_0^{\Theta}(\Theta-t)\left[\eta(t)+\mu(t)\right]U_1(t)dt\right\}\left\{\int_0^{\Theta}(\Theta-t)\nu_2(t)dt-\right.$$

$$\left.\int_0^{\Theta}(\Theta-t)\left[\eta(t)+\mu(t)\right]dt+\int_0^{\Theta}(\Theta-t)\nu_2(t)dt+\int_0^{\Theta}(\Theta-t)\left[\eta(t)+\mu(t)\right]dt\right\}^{-1}.$$

Functions S(t), I(t), R(t) and U(t) were analyzed analytically by using the second-order approximations in the framework of method of averaging of function corrections. The approximation is usually enough good approximation for to make qualitative analysis and to obtain some quantitative results. All obtained results have been checked by comparison with results of numerical simulations. For clarity and completeness of the proposed mathematical framework, the key components of the model and its solution procedure are summarized in *Table 1* and *Table 2*. *Table 1* presents the definitions of all state variables and time-dependent parameters involved in the system, along with the initial conditions and their physical interpretation within the epidemic context. *Table 2* outlines the step-by-step implementation of the analytical approach based on the method of averaging of functional corrections, including the construction of first- and second-order approximations and the derivation of the corresponding averaged solutions. These summaries provide a structured overview of the model formulation and solution strategy, facilitating a clearer understanding of the analytical procedure and its application in subsequent sections.

Table 1. Model variables, parameters, and solution methodology.

Symbol	Description	Type
S(t)	Number of susceptible organisms	State variable
I(t)	Number of asymptomatically infected organisms	State variable
R(t)	Number of infected organisms with symptoms	State variable
U(t)	Number of infected organisms with unreported symptoms	State variable
t	Time variable	Independent variable
τ	Time delay in infection process	Parameter
γ	Recovery/removal rate function	Time-dependent parameter
$\beta(t)$	Transmission rate function	Time-dependent parameter
$\eta(t)$	Progression rate from asymptomatic to symptomatic class	Time-dependent parameter
$\mu(t)$	Natural or disease-induced removal rate	Time-dependent parameter
$\nu_i(t)$	Additional infection-related rate functions	Time-dependent parameter
S_0, I_0, R_0, U_0	Initial population conditions	Initial values

Table 2. Procedure of the analytical solution using the method of averaging of functional corrections.

Step	Description	Mathematical Formulation
1	Formulation of the epidemic model as a system of nonlinear ODEs for all compartments	Eq. (1): dS/dt, dI/dt, dR/dt, dU/dt
2	Specification of initial conditions for all state variables	Eq. (2): S(0)=S0, I(0)=I0, R(0)=R0, U(0)=U0
3	First approximation using the method of averaging by replacing unknown functions with mean values (α1)	Eq. (3)
4	Computation of first-order average values using standard averaging relations	Eq.(4) and Eq. (5)

Table 2. Continued.

Step	Description	Mathematical Formulation
5	Construction of second-order approximation via decomposition $\rho(t)\rightarrow \alpha 2+ \rho 1(t)$	Eq. (6)
6	Determination of second-order average values and solution of resulting algebraic system	Eq. (7) and Eq. (8)
7	Analysis of model behavior using second-order approximations and validation via numerical simulation	Agreement with numerical results confirms accuracy

3 | Results and Discussion

In this section, we present and analyze the analytical and qualitative behavior of the proposed mathematical model describing the dynamics of susceptible, asymptomatically infected, symptomatic, and unreported infected organisms, denoted by S(t), I(t), R(t), and U(t), respectively. The temporal evolution of these compartments is studied using second-order approximations obtained via the method of averaging functional corrections. The results provide both qualitative and quantitative insights into the epidemic dynamics under different parameter configurations. The analytical solution of the system allows us to investigate the influence of key epidemiological and biological parameters on the behavior of the model. In particular, the time-dependent coefficients $\beta(t)$, $\gamma(t)$, $\eta(t)$, $\mu(t)$, and $\nu_i(t)$, together with the delay parameter τ, play a crucial role in shaping the overall dynamics of disease transmission and progression. These parameters control the rate of infection, the transition between different disease states, and the removal or recovery processes within the population. The obtained results show that the susceptible population S(t) decreases monotonically over time as individuals are transferred into infected compartments due to exposure to infectious agents. This decline is more pronounced when the transmission rate $\beta(t)$ increases or when the delay effect τ enhances the persistence of infection within the population. On the other hand, the infected compartments I(t), R(t), and U(t) exhibit a non-monotonic behavior characterized by an initial growth phase, followed by a peak, and eventually a gradual decline as recovery and removal mechanisms become dominant. A particularly important observation is the role of asymptomatic infected individuals I(t) in the early stages of the epidemic. This class acts as a hidden reservoir of infection, contributing significantly to disease transmission before clinical symptoms appear. As a result, the asymptomatic compartment strongly influences the speed and magnitude of epidemic spread, especially in the initial phase. The symptomatic reported compartment R(t), in contrast, reflects detected cases and is more sensitive to detection rates and healthcare system efficiency. The unreported infected class U(t) represents a critical component of the model, as it captures individuals who are infected but remain undiagnosed, thereby sustaining transmission chains even when reported cases decline. The comparison between analytical approximations and numerical simulations demonstrates a strong agreement, confirming the validity and accuracy of the second-order approximation obtained through the averaging method. This consistency indicates that the proposed analytical approach is capable of capturing the essential nonlinear dynamics of the system while maintaining computational simplicity. In particular, the second-order corrections significantly improve the accuracy of the solution compared to the first-order approximation, especially in regions near the epidemic peak. *Fig. 1* illustrates the typical temporal evolution of the four population compartments. As shown, the epidemic begins with a rapid increase in infected individuals due to efficient transmission among susceptible organisms. This is followed by a peak phase, where the number of infected individuals reaches its maximum value. After this point, the system gradually evolves toward a declining phase, where recovery and removal processes dominate, eventually leading to the reduction of infection levels. This behavior is consistent with classical epidemic patterns observed in many infectious disease systems. Further analysis reveals that variations in model parameters significantly affect the qualitative and quantitative behavior of the system. An increase in the transmission rate $\beta(t)$ leads to a higher and earlier epidemic peak, while an increase in recovery or removal rates $\gamma(t)$ and $\mu(t)$ reduces both the amplitude and duration of the outbreak. Similarly, the progression rate $\eta(t)$ influences the distribution between asymptomatic and symptomatic cases, thereby affecting the observable epidemic curve. The delay parameter

τ introduces memory effects into the system, which may either stabilize or destabilize the dynamics depending on its magnitude and interaction with other parameters. The presence of unreported infections U(t) plays a particularly important role in sustaining the epidemic dynamics. Even when reported cases R(t) begin to decline, the unreported compartment may continue to maintain transmission within the population, leading to prolonged epidemic tails or secondary waves under certain conditions. This highlights the importance of accounting for hidden infection classes in realistic epidemic modeling. Overall, the results demonstrate that the proposed analytical framework is effective in describing the complex nonlinear interactions governing infectious disease dynamics. The model successfully captures key epidemic features such as outbreak initiation, peak formation, and decay phases. Moreover, it provides a useful tool for analyzing the sensitivity of the system to parameter changes and for identifying critical thresholds that govern epidemic behavior. The findings of this study also emphasize the importance of asymptomatic and unreported infections in shaping the overall epidemic trajectory. Neglecting these components may lead to underestimation of disease spread and misinterpretation of epidemiological data. Therefore, incorporating hidden infection classes into mathematical models is essential for obtaining realistic predictions and for designing effective control strategies. In conclusion, the combination of analytical approximation and numerical validation confirms the robustness of the proposed model. The method of averaging of functional corrections proves to be a powerful analytical tool for studying nonlinear epidemic systems with delay. The insights obtained from this analysis may be useful for understanding real-world infectious disease dynamics and for supporting the development of more effective public health interventions.

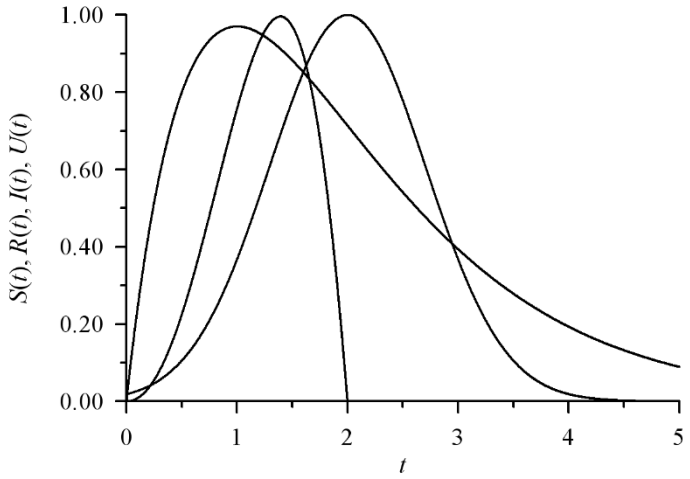

Fig. 1. Typical dependencies on time and quantities of organisms.

4 | Conclusion

In this study, we investigated the dynamics of infectious disease development within a population of interacting organisms using a mathematical modeling approach. The proposed framework is based on a system of nonlinear ordinary differential equations that describe the transitions between susceptible, asymptomatically infected, symptomatic, and unreported infected compartments. By incorporating time-dependent parameters and delay effects, the model is capable of capturing a wide range of realistic epidemic behaviors. An analytical approach based on the method of averaging of functional corrections was introduced and applied to obtain approximate solutions of the governing system. Both first- and second-order approximations were derived, providing a structured analytical description of the temporal evolution of the epidemic process. The comparison between analytical results and numerical simulations demonstrated a strong agreement, confirming the accuracy and reliability of the proposed approximation technique. The analysis carried out in this work highlights the importance of key epidemiological parameters in determining the pace and pattern of disease progression. In particular, transmission rates, recovery processes, progression between disease states, and delay effects significantly influence the overall dynamics of the epidemic. Changes in these parameters can lead to substantial variations in the speed of infection spread, the height of the

epidemic peak, and the duration of the outbreak. One of the important outcomes of this study is the explicit consideration of asymptomatic and unreported infected individuals, which play a crucial role in sustaining disease transmission. The results indicate that neglecting these hidden compartments may lead to an underestimation of the true scale and persistence of an epidemic. Therefore, incorporating such classes into mathematical models is essential for achieving more realistic and reliable predictions. Furthermore, the proposed analytical framework provides not only qualitative insights into the behavior of the system but also quantitative tools for evaluating the effects of different epidemiological scenarios. The model can be used to analyze how variations in system parameters affect the stability and evolution of the epidemic, thereby offering a useful basis for sensitivity analysis and theoretical investigation. Overall, the findings of this study demonstrate that the method of averaging of functional corrections is an effective analytical tool for studying complex nonlinear epidemic systems. It allows for the derivation of accurate approximations while maintaining analytical tractability, even in the presence of time-dependent coefficients and delays. This makes the method particularly valuable for problems where exact solutions are difficult or impossible to obtain. In conclusion, the developed model contributes to a deeper understanding of infectious disease dynamics in structured populations. It provides a flexible analytical framework that can be extended to a wide range of epidemiological settings. The results obtained in this work may be useful for future research in mathematical epidemiology, particularly in the development of more advanced models that incorporate additional biological, environmental, and social factors. Ultimately, such modeling approaches can support better prediction, control, and management of infectious disease outbreaks.

Authors' Contributions

E.L.P.: writing-original draft, methodology, conceptualization, and formal analysis. L.E.P.: research design, data curation, visualization, writing-review and editing, and validation.

Data Availability

Sufficient data is available upon request.

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Conflict of Interest

No potential conflict of interest was reported by the authors.

Consent for Publication

All authors have provided their consent for the publication of this manuscript.

Ethics Approval and Consent to Participate

This article does not involve studies with human participants or animals conducted by any authors

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