Journal of Nonlinear Modeling and Analysis
Volume 7, Number 2, April 2025, 453–462
https://journal.global-sci.org/jnma
DOI:10.12150/jnma.2025.453

Global Stability of a Parabolic System Involving a Nonlinear Diffusion Operator with an Example in Epidemiology

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Abstract The purpose of this work is to study the global stability of specific nonlinear parabolic equations incorporating the p-Laplacian operator, with a primary emphasis on their application in biology, particularly in the context of epidemiology. This investigation entails the construction of Lyapunov functions derived from the associated ODEs. To elucidate our approach, we provide an illustrative example from the field of epidemiology.

Keywords Epidemiological model, Lyapunov function, parabolic systems, p-Laplacian operator

MSC(2010) 92D30, 93D05, 35K40, 35K92.

1. Introduction

Mathematical models have long been indispensable tools in understanding and predicting the dynamics of infectious diseases. By quantifying the complex interactions between pathogens, hosts, and populations, these models enable researchers and policymakers to gain insights into the spread of diseases and evaluate potential control strategies. One of the pioneering works in epidemic modeling can be attributed to Kermack and McKendrick [2], who introduced the influential SIR model in 1927. Over time, epidemic mathematical models have evolved and expanded to incorporate additional complexities. Researchers recognized the importance of accounting for factors such as age structure, spatial heterogeneity, and varying transmission rates. This led to the development of more sophisticated compartmental models, such as the SEIR model that introduced an exposed compartment [9]. Moreover, spatial epidemic models and network-based models emerged to capture the influence of geographical locations and social connections on disease spread [1]. In recent years, advanced mathematical techniques have further enhanced the capabilities of epidemic models.

In recent studies, a multitude of researchers in the fields of epidemiology and virology have employed spatiotemporal equations. The research [3], for instance, analyzed an epidemic model described as a parabolic system of PDEs, incorporating the p-Laplacian operator. Their primary objective was to devise a well-thought-out optimal control approach within a specified spatiotemporal context. This approach

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was meticulously crafted to mitigate both the propagation of infections and the associated vaccination expenses. Through a Lyapunov function, [4] demonstrated the global asymptotic stability of the endemic equilibrium in a graph Laplacian reaction-diffusion SIR system when the basic reproduction number $\mathcal{R}_0$ is greater than 1. Conversely, they also established the global asymptotic stability of the disease-free equilibrium when the basic reproduction number is less than 1.

An independent investigation was carried out by [7], where an Ebola epidemic model was developed, taking into account constraints posed by limited medical resources, immunity loss, and the implementation of measures such as tracking and quarantining susceptible individuals. Meanwhile, the stability analysis of the disease-free equilibrium is presented, and the existence of multiple endemic equilibria as well as the occurrence of bifurcation are deduced. Utilizing the next-generation matrix method, the study computed the basic reproduction number $\mathcal{R}_0$ and subsequently analyzed the model's stability. A separate study by [10] explored an SIRS epidemic model incorporating logistic growth and information intervention. The study introduced the basic reproduction number $\mathcal{R}_0$ and presented key findings about local stability. Furthermore, the research derived sufficient conditions for the global stability of the endemic equilibrium. In the study by [6], an intracellular time delay was incorporated into an HBV model originally proposed in [5]. This time delay accounts for the lag between cell infection and the generation of new virus particles. The investigation was conducted within a one-dimensional interval with Neumann boundary conditions, with the simplifying assumption of homogeneous space to facilitate the establishment of global stability for equilibrium solutions. Furthermore, [8] extended the model introduced by [6] by introducing a saturation response mechanism and derived the necessary conditions for ensuring the global stability of the infected steady state.

The motivation for this paper lies in the profound significance of understanding and establishing the global stability of reaction-diffusion systems featuring the p-Laplacian operator, with a particular focus on systems incorporating Neumann boundary conditions and the potential inclusion of delay. These systems serve as powerful mathematical models that find wide-ranging applications across numerous scientific and practical domains. By investigating their global stability, our study addresses several critical aspects. First, the dynamics of such systems are intrinsic to a multitude of real-world phenomena, including the spread of infectious diseases, the diffusion of chemicals in biological tissues, and heat conduction in materials. The inclusion of the p-Laplacian operator, known for capturing nonlinear effects, allows for a more faithful representation of the complex interactions and diffusion processes inherent in these phenomena.

Second, global stability analysis plays a crucial role in epidemiological applications, particularly in the control and predictability of diseases. It ensures that equilibrium states within the systems are robust and that the dynamics tend to stabilize under various conditions. This insight has profound implications for optimizing processes, managing epidemics, controlling chemical reactions, and enhancing the reliability of materials and structural components, which is pertinent in different fields like engineering and others.

Furthermore, our approach to constructing Lyapunov functionals for PDEs based on those developed for ODEs represents a unifying and systematic method for analyzing and predicting the behavior of diverse dynamical systems. This methodological contribution not only advances the theoretical foundations of nonlinear systems

but also enhances the toolbox available to researchers and practitioners across various disciplines.

In essence, the present study focuses on demonstrating the global stability of reaction-diffusion systems with the p-Laplacian operator, Neumann boundary conditions, and the potential inclusion of delay, addressing fundamental questions with far-reaching implications, spanning mathematical theory and practical applications. It promises to provide valuable insights, tools, and solutions for a broad and interdisciplinary audience, underscoring its significance and relevance.

The organization of this research is outlined as follows. In Section 2, we conduct a qualitative analysis of the nonlinear parabolic model, with a primary objective of establishing global stability. Moreover, we apply our method to study the global stability of a reaction-diffusion biological model in Section 3. To encapsulate our findings and contributions, Section 4 offers a conclusion of our study.

2. Qualitative analysis of the proposed model

Consider the positive solution $\vartheta = (\vartheta_1, \vartheta_2, \cdots, \vartheta_n)$ of the following ODE

$$\begin{cases} \partial_t \vartheta(t) = \Phi(\vartheta(t)), \\ \vartheta(0) = \vartheta^0, \end{cases} \qquad t \in \mathcal{I}_{\mathcal{T}} := [0, \mathcal{T}], \tag{2.1}$$

where $\mathcal{T} \in \mathbb{R}_+^*$ and Φ is a $\mathcal{C}^1$ function defined on $\mathbb{R}^n$ into $\mathbb{R}^n$.

Assuming that ϑ^* represents a positive equilibrium of (2.1), it also serves as a spatially homogeneous solution to the following spatiotemporal system

$$\begin{cases} \partial_t \vartheta(t,x) - \kappa \Delta_p \vartheta(t,x) = \Phi(\vartheta(t,x)), & \text{in } \mathcal{I}_{\mathcal{T}} \times \mathcal{U}, \\ \nabla \vartheta_i \cdot \vec{\nu} = 0, \ 1 \le i \le n, & \text{on } \mathcal{I}_{\mathcal{T}} \times \partial\mathcal{U}, \\ \vartheta(0,x) = \vartheta^0(x), & \text{in } \mathcal{U}, \end{cases} \tag{2.2}$$

with $p \ge 2, \mathcal{U} \subset \mathbb{R}^m$ is a bounded domain with smooth boundary $\partial\mathcal{U}$, the diffusion coefficient is denoted as $\kappa = (\kappa_1, \cdots, \kappa_n)$, Δ_p be the p-Laplacian operator defined by

$$\Delta_p \vartheta = \nabla \cdot \left(|\nabla \vartheta|^{p-2} \nabla \vartheta \right) = div \left(|\nabla \vartheta|^{p-2} \nabla \vartheta \right),$$

and the normal vector to $\partial\mathcal{U}$ is represented by $\vec{\nu}$.

Let $\mathcal{Y}$ represents a Lyapunov function associated with (2.1), defined over a domain within $\mathbb{R}_+^n$. When ϑ serves as a solution to (2.1), it becomes imperative to calculate the time-derivative of $\mathcal{Y}(\vartheta(t))$. Then

$$d_t \mathcal{Y}(\vartheta) = \nabla \mathcal{Y}(\vartheta) \cdot \Phi(\vartheta). \tag{2.3}$$

Let ϑ represent a positive solution to (2.2). We define

$$\mathcal{X} = \int_{\mathcal{U}} \mathcal{Y}(\vartheta)\, dx. \tag{2.4}$$

Lemma 2.1. *The time-derivative of $\mathcal{X}$ is given by*

$$d_t \mathcal{X} = \int_{\mathcal{U}} \mathcal{Y}(\vartheta)\, dx - \sum_{i=1}^{n} \kappa_i \int_{\mathcal{U}} |\nabla \vartheta_i|^{p-2} \nabla \vartheta_i \cdot \nabla \left(\frac{\partial \mathcal{Y}}{\partial \vartheta_i} \right) dx. \tag{2.5}$$

Proof. Given that ϑ is a non-negative solution to (2.2), we can express the time-derivative of $\mathcal{X}$ as follows

$$\begin{aligned} d_t\mathcal{X} &= \int_{\mathcal{U}} \nabla\mathcal{Y}(\vartheta)\cdot(\kappa\Delta_p\vartheta+\Phi(\vartheta))\,dx \\ &= \int_{\mathcal{U}} \nabla\mathcal{Y}(\vartheta)\cdot\Phi(\vartheta)\,dx + \int_{\mathcal{U}} \nabla\mathcal{Y}(\vartheta)\cdot\kappa\Delta_p\vartheta dx \\ &= \int_{\mathcal{U}} \nabla\mathcal{Y}(\vartheta)\cdot\Phi(\vartheta)\,dx + \sum_{i=1}^{n}\kappa_i\int_{\mathcal{U}}\nabla\cdot\left(|\nabla\vartheta_i|^{p-2}\nabla\vartheta_i\right)\frac{\partial\mathcal{Y}(\vartheta)}{\partial\vartheta_i}. \end{aligned}$$

According to the homogeneous boundary conditions of Neumann, we get

$$d_t\mathcal{X} = \int_{\mathcal{U}} \nabla\mathcal{Y}(\vartheta)\cdot\Phi(\vartheta)\,dx - \sum_{i=1}^{n}\kappa_i\int_{\mathcal{U}}|\nabla\vartheta_i|^{p-2}\nabla\vartheta_i\cdot\nabla\left(\frac{\partial\mathcal{Y}(\vartheta)}{\partial\vartheta_i}\right)dx.$$

□

Based on this significant finding, we can establish the following theorem

Theorem 2.1. *If the condition*

$$\forall i\in\{1,2,\cdots,n\},\ \int_{\mathcal{U}}|\nabla\vartheta_i|^{p-2}\nabla\vartheta_i\cdot\nabla\left(\frac{\partial\mathcal{Y}(\vartheta)}{\partial\vartheta_i}\right)dx\geq 0, \tag{2.6}$$

holds, then the function $\mathcal{X}$, defined as in (2.4)*, serves as a Lyapunov function associated with the proposed spatiotemporal problem* (2.2)*.*

In the field of mathematical epidemiology, numerous studies have devised explicit Lyapunov functions with the following structure

$$\mathcal{Y}(\vartheta)=\sum_{j=1}^{n}\alpha_j\left(\vartheta_j-\vartheta_j^*\ln(\vartheta_j)+\delta_j\right), \tag{2.7}$$

with α_j and δ_j are constants. A straightforward computation yields

$$\int_{\mathcal{U}}|\nabla\vartheta_j|^{p-2}\nabla\vartheta_j\cdot\nabla\left(\frac{\partial\mathcal{Y}(\vartheta)}{\partial\vartheta_j}\right)dx=\alpha_j\vartheta_j^*\int_{\mathcal{U}}\frac{|\nabla\vartheta_j|^p}{\vartheta_j^2}dx\geq 0. \tag{2.8}$$

We can encapsulate this outcome in the following corollary.

Corollary 2.1. *If $\mathcal{Y}$ is established as a Lyapunov function of* (2.1)*, defined as shown in* (2.7)*, then it follows that $\mathcal{X}$ serves as a Lyapunov function of the reaction-diffusion system described in* (2.2)*.*

3. Application

In this section, we utilize our approach to analyze the global stability of equilibrium of a spatiotemporal SIR mathematical model within the field of biology. The entire population N in this model is categorized into three compartments corresponding to pathological conditions. These compartments are denoted as S (susceptible), I

(infected), and R, (recovered). Consider the reaction-diffusion SIR model characterized by

$$\begin{cases} \partial_t S - \kappa_1 \Delta_p S = \mu N - \beta SI - \eta S, & \\ \partial_t I - \kappa_2 \Delta_p I = \beta SI - (\eta + \sigma) I, & \text{in } \mathcal{I}_{\mathcal{T}} \times \mathcal{U}, \\ \partial_t R - \kappa_3 \Delta_p R = \sigma I - \eta R, & \\ \nabla S \cdot \vec{\nu} = \nabla I \cdot \vec{\nu} = \nabla R \cdot \vec{\nu} = 0, & \text{on } \mathcal{I}_{\mathcal{T}} \times \partial\mathcal{U}, \\ (S(0,\cdot), I(0,\cdot), R(0,\cdot)) = \left(S^0(\cdot), I^0(\cdot), R^0(\cdot)\right), & \text{in } \mathcal{U}, \end{cases} \tag{3.1}$$

where β denotes the effective contact rate, σ signifies the removal rate, μ characterizes the birth rate, and η represents the natural mortality rate.

The following Figure shows how the epidemic spreads from one compartment to another in system (3.1)

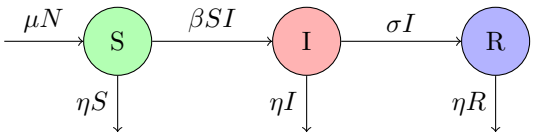

Figure 1. Transfer diagram for the proposed SIR problem.

The problem (3.1), can be rewriten in the form (2.2), with $\mathcal{U} \subset \mathbb{R}^2$, $\vartheta = (S, I, R)$, $\vartheta^0(\cdot) = (S^0(\cdot), I^0(\cdot), R^0(\cdot))$, and

$$\Phi(\vartheta) = \begin{pmatrix} \mu(\vartheta_1 + \vartheta_2 + \vartheta_3) - \beta\vartheta_1\vartheta_2 - \eta\vartheta_1 \\ \beta\vartheta_1\vartheta_2 - (\eta + \sigma)\vartheta_2 \\ \sigma\vartheta_2 - \eta\vartheta_3 \end{pmatrix}.$$

Applying the methodology outlined in page 4 from [3], it can be demonstrated that (3.1) possesses a unique non-negative weak solution in

$$\left(C(0,T;L^2(\Omega)) \cap L^p(0,T;W_0^{1,p}(\Omega))\right)^3.$$

The basic reproduction number of (3.1) in the absence of spatial effects, is expressed as

$$\mathcal{R}_0 = \frac{\mu\beta N}{\eta(\eta + \sigma)}, \tag{3.2}$$

To identify equilibria, we equate the right-hand side of (3.1) to zero. This results in the determination of two equilibria in the coordinate space (S, I, R). Specifically, the disease-free equilibrium $E^f(\frac{\mu N}{\eta}, 0, 0)$, and the endemic equilibrium $E^*(S^*, I^*, R^*)$, with

$$S^* = \frac{\mu N}{\eta \mathcal{R}_0}, \quad I^* = \frac{\eta}{\beta}(\mathcal{R}_0 - 1) \quad \text{and} \quad R^* = \frac{\sigma}{\beta}(\mathcal{R}_0 - 1). \tag{3.3}$$

It's important to note that the variable R is not present in the first two equations of (3.1). This observation allows us to focus our analysis on the associated system to (3.1) without R.

$$\begin{cases} \partial_t S - \kappa_1 \Delta_p S = \mu N - \beta SI - \eta S, & \text{in } \mathcal{I}_{\mathcal{T}} \times \mathcal{U}, \\ \partial_t I - \kappa_2 \Delta_p I = \beta SI - (\eta + \sigma) I, & \\ \nabla S \cdot \vec{\nu} = \nabla I \cdot \vec{\nu} = 0, & \text{on } \mathcal{I}_{\mathcal{T}} \times \partial\mathcal{U}, \\ (S(0,\cdot), I(0,\cdot)) = \left(S^0(\cdot), I^0(\cdot)\right), & \text{in } \mathcal{U}, \end{cases} \tag{3.4}$$

3.1. Global stability of E^*

We introduce a Goh-Volterra-type nonlinear Lyapunov function as follows

$$\tilde{\mathcal{Y}}(\vartheta) = S^* \left(\frac{S}{S^*} - \ln \frac{S}{S^*} - 1 \right) + I^* \left(\frac{I}{I^*} - \ln \frac{I}{I^*} - 1 \right). \tag{3.5}$$

By utilizing the ODE associated with (3.1), the time-derivative of (3.5) is expressed as follows

$$\begin{aligned} d_t \tilde{\mathcal{Y}} = & \left((\mu N - \beta SI - \eta S) - \frac{S^*(\mu N - \beta SI - \eta S)}{S} \right) \\ & + \left((\beta SI - (\eta + \sigma) I) - \frac{I^*(\beta SI - (\eta + \sigma) I)}{I} \right). \end{aligned} \tag{3.6}$$

At a steady state, as determined by the ODE associated with (3.1), the following relationships are satisfied

$$\mu N = \beta S^* I^* + \eta S^* \quad \text{and} \quad \eta + \sigma = \beta S^*. \tag{3.7}$$

Substituting these expressions from (3.7) into (3.6), further simplification yields

$$d_t \tilde{\mathcal{Y}} = -\frac{\eta + \beta I^*}{S} \left(S - S^* \right)^2. \tag{3.8}$$

Put

$$\tilde{\mathcal{X}} = \int_\Omega \tilde{\mathcal{Y}}(\vartheta(t,x)) dx. \tag{3.9}$$

Upon evaluating the time-derivative of $\tilde{\mathcal{X}}$, we deduce the ensuing inequality

$$d_t \tilde{\mathcal{X}} = -(\eta + \beta I^*) \int_\Omega \frac{\left(S - S^* \right)^2}{S} dx - \kappa_1 S^* \int_\Omega \frac{|\nabla S|^p}{S^2} dx - \kappa_2 I^* \int_\Omega \frac{|\nabla I|^p}{I^2} dx. \tag{3.10}$$

This establishes that $\tilde{\mathcal{X}}$ serves as a Lyapunov functional for (3.1) at the endemic equilibrium E^*.

3.2. Global stability of E^f

To investigate the global stability of the disease-free equilibrium E^f, we utilize the following Lyapunov function

$$\hat{\mathcal{Y}}(\vartheta) = \left(S - S^f \ln \frac{S}{S^f} \right) + I. \tag{3.11}$$

By examining the associated ODE of (3.1), we derive

$$d_t \tilde{\mathcal{Y}} = \left(\beta S^f - (\eta + \sigma) \right) I + \frac{1}{S} \left(\mu N S - \eta S^2 - \mu N S^f + \eta S S^f \right). \tag{3.12}$$

Given that

$$\mu N = \beta S^f, \tag{3.13}$$

then, we can simplify (3.12) as follows

$$d_t\tilde{\mathcal{Y}} = -\frac{\eta}{S}\left(S - S^f\right)^2 + (\eta + \sigma)\left(\mathcal{R}_0 - 1\right) I. \tag{3.14}$$

Introduce

$$\hat{\mathcal{X}} = \int_\Omega \hat{\mathcal{Y}}(\vartheta(t,x))dx. \tag{3.15}$$

From (3.14) and (3.15), further simplification gives

$$d_t\hat{\mathcal{X}} = \int_\Omega \left(\frac{-\eta\left(S - S^f\right)^2}{S} + (\eta + \sigma)\left(\mathcal{R}_0 - 1\right) I \right) dx - \kappa_1 S^f \int_\Omega \frac{|\nabla S|^p}{S^2} dx. \tag{3.16}$$

It is evident that $\mathcal{R}_0 \leq 1$ ensures $d_t\hat{\mathcal{X}} \leq 0$. Therefore, $\hat{\mathcal{X}}$ serves as a Lyapunov functional for (3.1) at equilibrium E^f.

3.3. Numerical results

The given mathematical model (3.1) has been computationally solved by using an FDM implemented in MatLab. For our simulations, we adopted specific parameter values $\kappa = 0.1$, $\mu = 0.09/\mu = 0.05$, $\beta = 0.01/\beta = 0.009$, $\sigma = 0.1$, $\eta = 0.09$. The initial conditions were specified as $\left(S^0, I^0, R^0\right) = (30, 5, 0)$.

Figures 2 and 3 depicts time series of the model solution when $\mathcal{R}_0 \leq 1$, signifying the global asymptotic stability of the disease-free equilibrium E^f.

Conversely, Figures 4 and 5 illustrate time series of the solution when $\mathcal{R}_0 \geq 1$, indicating the global asymptotic stability of the endemic equilibrium E^*.

Furthermore, it is noteworthy that higher values of the parameter p contribute to the accelerated propagation of the disease and hasten the convergence of the involved compartments towards their respective equilibria.

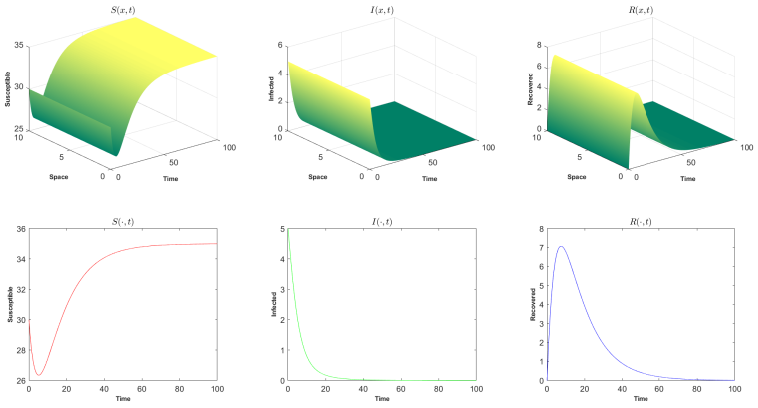

Figure 2. The global behavior of the solution of (3.1), with $\mu = 0.05$ and $\beta = 0.009$ ($\mathcal{R}_0 \leq 1$) for $p = 5$.

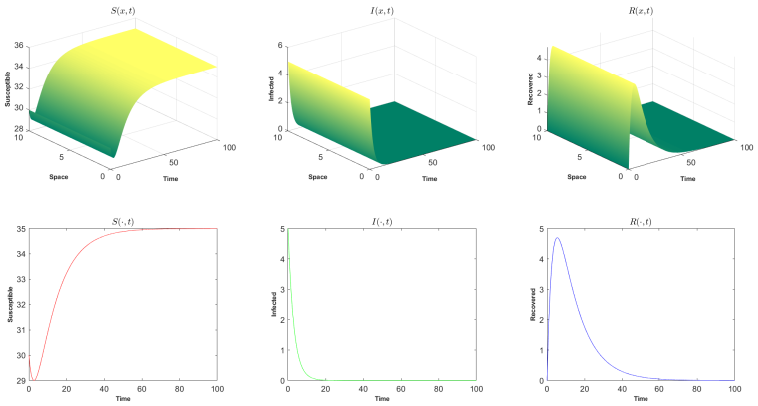

Figure 3. The global behavior of the solution of (3.1), with $\mu = 0.05$ and $\beta = 0.009$ ($\mathcal{R}_0 \leq 1$) for $p = 10$.

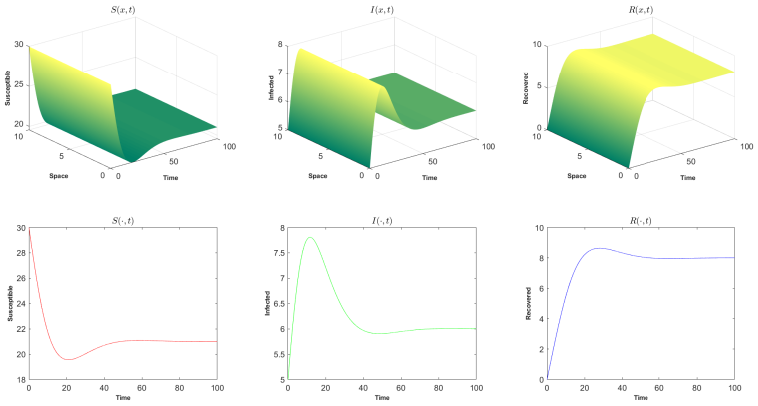

Figure 4. The global behavior of the solution of (3.1), with $\mu = 0.09$ and $\beta = 0.01$ ($\mathcal{R}_0 \geq 1$) for $p = 5$.

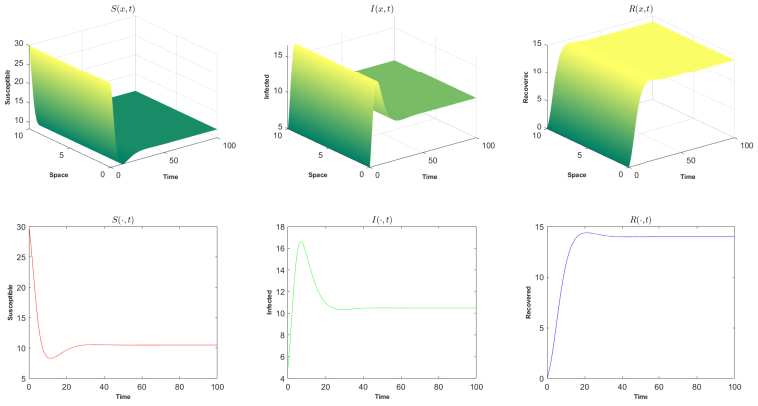

Figure 5. The global behavior of the solution of (3.1), with $\mu = 0.09$ and $\beta = 0.01$ ($\mathcal{R}_0 \geq 1$) for $p = 10$.

4. Conclusion

In this study, we have undertaken a thorough exploration of the global stability of specific nonlinear parabolic equations featuring the p-Laplacian operator, with Neumann boundary conditions. Our approach involves the construction of Lyapunov functions derived from the associated ODEs. We dedicated to a qualitative analysis of the nonlinear parabolic model, with the overarching goal of establishing global stability. Furthermore, we showcased the application of our method in studying the global stability of a reaction-diffusion SIR biological model, providing valuable insights into its implications for real-world scenarios.

Declarations

Acknowledgments

The authors express their appreciation to the reviewers for their valuable, constructive comments, and suggestions. This work is carried out under the supervision of CNRST as part of the PASS program.

CRediT author statement

A. Zinihi: Conceptualization, Methodology, Software, Formal analysis, Investigation, Writing – Original Draft, Writing – Review & Editing, Visualization.
M. R. Sidi Ammi: Conceptualization, Methodology, Validation, Formal analysis, Investigation, Writing – Original Draft, Writing – Review & Editing, Supervision.

Funding

The authors claim that the preparation of this manuscript execution received no financing.

Data availability

All information analyzed or generated, which would support the results of this work are available in this article. No data was used for the research described in the article.

Conflict of interest

The authors declare that there are no problems or conflicts of interest between them that may affect the study in this paper.

References

[1] M. J. Keeling and P. Rohani, *Modeling infectious diseases in humans and animals*, Princeton University Press, 2007.

[2] W. O. Kermack and A. G. McKendrick, *A contribution to the mathematical theory of epidemics*, Proceedings of the Royal Society A, 1927, 115(772), 700–721.

[3] M. R. Sidi Ammi, A. Zinihi, A. A. Raezah and Y. Sabbar, *Optimal control of a spatiotemporal SIR model with reaction-diffusion involving p-laplacian operator*, Results in Physics, 2023, 52, 106895.

[4] C. Tian, Q. Zhang and L. Zhang, *Global stability in a networked SIR epidemic model*, Applied Mathematics Letters, 2020, 107, 106444.

[5] K. Wang and W. Wang, *Propagation of HBV with spatial dependence*, Mathematical Biosciences, 2007, 210(1), 78–95.

[6] K. Wang, W. Wang and S. Song, *Dynamics of an HBV model with diffusion and delay*, Journal of Theoretical Biology, 2008, 253(1), 36–44.

[7] X. Wang, J. Li, S. Guo and M. Liu, *Dynamic analysis of an Ebola epidemic model incorporating limited medical resources and immunity loss*, Journal of Applied Mathematics and Computing, 2023, 69(6), 4229–4242.

[8] R. Xu and Z. Ma, *An HBV model with diffusion and time delay*, Journal of Theoretical Biology, 2009, 257(3), 499–509.

[9] A. Zinihi, M. R. Sidi Ammi and M. Ehrhardt, *Optimal control of a diffusive epidemiological model involving the Caputo-Fabrizio fractional time-derivative*, 2024. ArXiv:2403.00364.

[10] I. Çay, *On the local and global stability of an SIRS epidemic model with logistic growth and information intervention*, Turkish Journal of Mathematics, 2021, 45, 1668–1677.