Bergische Universität Wuppertal

Fakultät für Mathematik und Naturwissenschaften

Institute of Mathematical Modelling, Analysis and Computational Mathematics (IMACM)

Preprint BUW-IMACM 26/02

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March 8, 2026

http://www.imacm.uni-wuppertal.de

Nonstandard Finite Difference Schemes for Delayed Credit Risk Contagion Models

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Abstract

We investigate structure-preserving, nonstandard finite-difference (NSFD) schemes for nonlinear, compartmental, credit risk contagion models of the SEIR-type. These models include a delayed formulation and a generalized model with nonlinear incidence and state-dependent transition rates. For continuous systems, we derive default-free and endemic equilibria, compute the basic reproduction number, and establish local and global stability results.

We construct NSFD discretizations that preserve positivity, boundedness, invariant regions, equilibria, and the exact threshold parameter independently of the time step. The schemes reproduce the same default-free and endemic equilibria as the continuous models and inherit their stability properties near equilibrium. Numerical experiments confirm that the proposed schemes remain dynamically consistent for large time steps, unlike standard explicit methods.

These results demonstrate that NSFD methods provide reliable, structure-preserving discretizations for nonlinear delay differential equations that arise in financial contagion modeling.

Keywords: Credit risk contagion, Nonstandard finite difference scheme, Time delay, Positivity-preserving discretization, Mimetic numerical methods.
2020 Mathematics Subject Classification: 92D30, 91G40, 65M06, 37N30.

1. Introduction

Our main contribution to the literature about credit risk contagion is given by the application of the SIR model [1–3, 5–7]. The model under consideration, introduced by Fanelli and Maddalena [7], provides a deterministic framework for describing contagion phenomena in *credit risk transfer* (CRT) markets. Inspired by epidemiological compartmental models, the model groups financial institutions into three classes: susceptible (defaultable), infected (defaulted), and recovered (those that have undergone debt restructuring and are temporarily protected from default).

A key feature of the model is the presence of a time delay representing the immunity period following recovery. During this interval, agents are not assumed to immediately re-enter the defaultable class. Additionally, the transmission mechanism is described by a nonlinear saturated incidence rate reflecting the fact that the intensity of financial interactions is limited by market frictions, regulatory constraints, or behavioral effects and cannot grow indefinitely.

This formulation captures several realistic aspects of systemic risk propagation. In particular, it allows one to analyze how temporary protection, recovery intensity, and the inflow of

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new participants influence the persistence or extinction of default events. The analytical study in Fanelli and Maddalena [7] establishes the positivity and boundedness of solutions, identifies threshold conditions for the stability of the *default-free equilibrium* (DFE), and demonstrates how the delay can significantly impact the long-term market configuration.

The continuous-time model has important qualitative properties, such as positive solutions, boundedness, and a conservation law for the total population. However, standard numerical discretizations do not automatically preserve these features. For instance, [15] demonstrated that a standard Runge-Kutta scheme yielded negative values for typical parameter configurations in an epidemiological model. Thus, there is a need for structure-preserving numerical schemes that can faithfully reproduce the qualitative dynamics of the continuous model at the discrete level. The presence of nonlinear incidence terms and time delays, in particular, poses nontrivial challenges for constructing positivity-preserving and dynamically consistent discretizations. We refer interested readers to [11] for a recent review on discretization techniques for epidemiological models.

Exact finite difference methods (FDMs) have been defined for different particular differential problems without delay. The use of *nonstandard finite difference* (NSFD) numerical schemes has gained increasing interest in recent years [17, 18]. The NSFD schemes are designed to provide dynamically consistent solutions. In other words, these discrete solutions inherit the structural properties of the underlying continuous ordinary differential equations (ODEs).

NSFD methods have also been extended to classes of *delay differential equations* (DDEs), particularly in the context of epidemic and population dynamics. For linear DDEs with a constant delay, NSFD schemes were proposed in [4, 8, 9]. First NSFD schemes for nonlinear DDEs were proposed and analyzed by Su, Li & Ding [20], Suryanto [21], Wang [22] and Xu, Geng & Hou [23, 24]. These works typically focus on model-specific constructions that preserve qualitative properties, such as positivity, boundedness, and stability of equilibria. Examples include NSFD schemes for delayed SIS and SIR-type epidemic models, delayed viral infection models, and coupled delay systems with nonlinear incidence rates [4, 16, 21–24]. The authors usually discretize the delay terms by combining backward or mixed time-level evaluations with appropriately chosen denominator functions. This approach allows the discrete model to reproduce key dynamical features of the underlying continuous system.

Although NSFD methods have been successfully applied to epidemic and population models, systematic treatment of nonlinear delay systems remains limited. This work addresses this issue for a class of financial contagion models by developing Mickens-type NSFD schemes that independently preserve positivity, boundedness, equilibria, threshold dynamics, and elementary stability, regardless of the time step.

The remainder of the paper is organized as follows: Section 2 briefly reviews the credit risk contagion model and its main qualitative properties in the continuous-time setting. Section 3 provides some analysis of the considered models. Section 4 introduces the proposed nonstandard finite difference schemes for the non-delayed and delayed cases, analyzing their structural properties. Section 5 provides the analysis of the proposed NSFD scheme. Section 6 presents numerical experiments to illustrate the performance and long-time behavior of the proposed schemes. Finally, Section 7 concludes the paper with remarks on future work.

2. The Model Problem

We consider the (reduced) credit risk contagion model introduced by Fanelli and Maddalena [7], describing the dynamics of defaultable and defaulted financial agents.

To model this contagion process, we use a time-delay SIR model that categorizes agents into three states over time: healthy (S), infected (I), and recovered (R). $S(t)$ represents healthy agents currently susceptible to default, $I(t)$ represents "infected" agents that have defaulted and can spread risk, and $R(t)$ represents recovered agents undergoing or completing restructuring. By incorporating temporary immunity and a nonlinear time-delay incidence rate, this model provides a more nuanced perspective on the evolution of financial distress, recognizing that the impact of a default often lingers and propagates with a lag rather than affecting the entire system instantaneously.

2.1. The Credit Risk Contagion Model

Here, we consider the following ODE system with a constant time delay τ:

$$\begin{cases} \dot{S}(t) &= B - \gamma S(t) - acS(t)\frac{I(t-\tau)}{1+\alpha I(t-\tau)} + \delta I(t-\tau)\, e^{-\mu\tau}, \\ \dot{I}(t) &= acS(t)\frac{I(t-\tau)}{1+\alpha I(t-\tau)} - \delta I(t) - \gamma I(t), \\ \dot{R}(t) &= \delta I(t) - \delta I(t-\tau)\, e^{-\mu\tau} - \gamma R(t). \end{cases} \tag{1}$$

All parameters in (1) are assumed to be nonnegative. The constant B denotes the rate at which new financial agents enter the market and are assigned to the susceptible (defaultable) class. The parameter γ represents the natural exit rate from the system and affects all compartments uniformly. The coefficient c denotes the contagion (contact) rate among market participants, whereas a is the probability of transmission of distress upon contact; their product $\beta = a\,c$ therefore determines the overall intensity of the infection mechanism. The coefficient α appears in the saturated incidence function and reflects limitations in the effective transmission of risk when the number of distressed agents is large. This can be due to market frictions or regulatory constraints, for instance. Defaulted agents leave the infected class at a rate of δ, which can be interpreted as the intensity of restructuring or recovery. After recovery, agents experience a temporary immunity period of average length τ, during which they cannot become susceptible again. The parameter μ accounts for the possibility that agents exit the recovered class during this immunity interval.

The nonlinear incidence term

$$ac\, S(t)\, \frac{I(t-\tau)}{1+\alpha I(t-\tau)}$$

therefore captures the exposure of defaultable institutions to previously defaulted ones, while also preventing unrealistically high transmission rates at high infection levels. We show the positivity and boundedness of solutions to (1) in Theorem 3 in Appendix A.

The *total CRT market population* is $N(t) := S(t)+I(t)+R(t)$ and summing up the equations in (1) yields

$$\dot{N}(t) = B - \gamma N(t), \tag{2}$$

with the solution

$$N(t) = \frac{B}{\gamma} + \left(N(0) - \frac{B}{\gamma}\right) e^{-\gamma t}, \tag{3}$$

that tends to B/γ for $t \to \infty$.

2.2. A Generalization with Endogenous Delay (Pre-default Fragility)

In equation (1), the immunity delay parameter τ models the average time during which recovered agents remain temporarily protected before re-entering the defaultable class S. However, in credit risk transfer (CRT) markets, an additional, economically relevant latency arises before a default is realized: institutions typically pass through a phase of financial fragility (e.g., liquidity stress, rating deterioration, covenant breaches, margining pressure, and renegotiation) prior to becoming effectively defaulted. This motivates the introduction of an endogenous delay mechanism [14, 19] via an intermediate compartment $E(t)$ of exposed/fragile agents so that time-to-default is generated by the internal transition $E \to I$ rather than by a fixed, exogenous delay.

Specifically, we consider the following SEIR-extension of model (1):

$$\begin{cases} \dot{S}(t) = B - \gamma S(t) - \beta\, S(t)\dfrac{I(t)}{1+\alpha I(t)} + \rho\, R(t), \\ \dot{E}(t) = \beta\, S(t)\dfrac{I(t)}{1+\alpha I(t)} - \big(\kappa(I(t)) + \gamma\big)E(t), \\ \dot{I}(t) = \kappa\big(I(t)\big)\, E(t) - (\delta+\gamma)I(t), \\ \dot{R}(t) = \delta I(t) - (\rho+\gamma)R(t), \end{cases} \tag{4}$$

where $\beta := ac$ is the effective contagion intensity and $E(t)$ represents agents that are already exposed to distress but not yet in default. The function $\kappa(\cdot)$ is the state-dependent activation rate from fragility to default; it captures the empirical fact, under stressed conditions, the progression to default accelerates due to tighter funding constraints, higher haircuts/margin calls, and reduced rollover. A simple choice is

$$\kappa(I) = \kappa_0\,(1 + kI), \qquad \kappa_0 > 0,\ k \geq 0, \tag{5}$$

for which the mean residence time in the fragile class is approximately $1/\kappa(I)$ and thus decreases endogenously as systemic distress I increases. The parameter $\rho \geq 0$ models the return of recovered agents to the defaultable class, recovering an SIRS-type feedback without explicitly prescribing a fixed immunity delay.

Summing the equations in (4) yields the same total-population law as in (2),

$$N(t) := S(t) + E(t) + I(t) + R(t), \qquad \dot{N}(t) = B - \gamma N(t), \tag{6}$$

so that the boundedness mechanism and the asymptotic level $\lim_{t\to\infty} N(t) = B/\gamma$ remain unchanged. This generalized formulation therefore provides a natural economic interpretation of endogenous credit risk contagion delays while maintaining the essential structural property that underpins the structure-preserving NSFD discretization presented in the subsequent sections.

To incorporate also the immunity-delay mechanism of (1) within the SEIR framework, we retain the same delayed return flux from the defaulted class to the defaultable one as in (1). Moreover, in line with the original delayed formulation, we allow the contagion incidence to depend on the past default level $I(t-\tau)$. In particular, we consider the delayed extension

$$\begin{cases} \dot{S}(t) = B - \gamma S(t) - \beta\, S(t)\dfrac{I(t-\tau)}{1+\alpha I(t-\tau)} + \delta\, I(t-\tau)\, e^{-\mu\tau}, \\ \dot{E}(t) = \beta\, S(t)\dfrac{I(t-\tau)}{1+\alpha I(t-\tau)} - \big(\kappa(I(t)) + \gamma\big)E(t), \\ \dot{I}(t) = \kappa(I(t))\, E(t) - (\delta+\gamma)I(t), \\ \dot{R}(t) = \delta I(t) - \delta\, I(t-\tau)\, e^{-\mu\tau} - \gamma R(t), \end{cases} \tag{7}$$

where $\tau > 0$ is the (post-recovery) immunity delay and the survival factor $e^{-\mu\tau}$ is retained as in (1). In (7), the additional compartment $E(t)$ generates an endogenous time-to-default through the internal transition $E \to I$, while the delayed incidence and return terms model, respectively, the lagged impact of distress and the fraction of agents that survive the immunity interval and re-enter the defaultable class. Obviously, we have again the same evolution law of total population (6) for the generalized delayed formulation (7).

3. Analysis of the Models

Regarding the fundamental theory of functional differential equations in [10], for any initial condition, the systems (1) and (7) have a unique solution.

3.1. Analysis for the non-delayed problem

We first consider the case without delay, i.e. $\tau = 0$. At a default-free steady state there are no defaulted agents, i.e. $I^* = 0$. Hence, the recovered class disappears at equilibrium $R^* = 0$ and from the first equation in (1), we obtain $S^* = B/\gamma$. The *default-free equilibrium* (DFE) of the Fanelli-Maddalena system (1) is

$$E_{\text{DFE}} = \Big(\frac{B}{\gamma}, 0, 0\Big). \tag{8}$$

For the nontrivial steady state, the so-called *endemic equilibrium* (EE), of the Fanelli-Maddalena system we search for a solution with $I^* > 0$. From the second equation in (1) we get

$$acS^* \frac{I^*}{1+\alpha I^*} = (\gamma+\delta)I^*,$$

and dividing by $I^* > 0$ yields

$$S^* = \frac{(\gamma+\delta)(1+\alpha I^*)}{ac}. \tag{9}$$

From the third equation we have $R^* = 0$, so in the endemic equilibrium E_{EE} the recovered class vanishes. Now, inserting (9) into the first equation of (1) yields $S^* + I^* = B/\gamma$, and we substitute (9) in this equation to determine I^*:

$$I^* = \frac{acB - \gamma(\gamma+\delta)}{\gamma\big(ac + \alpha(\gamma+\delta)\big)}. \tag{10}$$

Thus we obtain

$$S^* = \frac{B}{\gamma} - I^* = \frac{(\gamma+\alpha B)(\gamma+\delta)}{\gamma\big(ac+\alpha(\gamma+\delta)\big)}, \tag{11}$$

and we need for the existence of the non-trivial equilibrium $I^* > 0$, i.e. from (10) follows

$$\Theta = \frac{acB}{\gamma(\gamma+\delta)} > 1. \tag{12}$$

3.2. Analysis for the problem with delay

The default-free equilibrium (DFE) of the Fanelli-Maddalena system (1) does not depend on the delay τ nor on the immunity decay μ, since for $I^* = 0$, all infection-related fluxes vanish and we obtain as before

$$E^{\tau}_{\text{DFE}} = \Big(\frac{B}{\gamma}, 0, 0\Big). \tag{13}$$

For the second non-trivial equilibrium we consider the system (1) in the form

$$\begin{aligned} acS^*\frac{I^*}{1+\alpha I^*} &= B - \gamma S^* + \delta I^* \, e^{-\mu\tau}, \\ acS^*\frac{I^*}{1+\alpha I^*} &= (\gamma+\delta)I^*, \\ \gamma R^* &= \delta I^* \,(1-e^{-\mu\tau}). \end{aligned} \tag{14}$$

From the first two equations we obtain

$$S^* + I^* = \frac{B - \delta I^* \left(1 - e^{-\mu\tau}\right)}{\gamma}, \tag{15}$$

and inserting (9) (from the infected equation) yields

$$I^* = \frac{acB - \gamma(\gamma+\delta)}{\gamma\big(ac + \alpha(\gamma+\delta)\big) + ac\delta\left(1 - e^{-\mu\tau}\right)}. \tag{16}$$

Thus, the non-trivial ('endemic') steady state $E^\tau_{\text{EE}} = (S^*, I^*, R^*)$ reads

$$E^\tau_{\text{EE}} = \Big(\frac{\gamma+\delta}{ac}\left(1+\alpha I^*\right), \frac{acB - \gamma(\gamma+\delta)}{\gamma\big(ac + \alpha(\gamma+\delta)\big) + ac\delta\left(1 - e^{-\mu\tau}\right)}, \frac{\delta}{\gamma} I^* \left(1 - e^{-\mu\tau}\right)\Big), \tag{17}$$

which exists (i.e. is financially meaningful) for $\Theta > 1$.

Next, we will compute the Jacobians evaluated at an equilibrium (S^*, I^*, R^*), i.e. the linearization of the system (1). Because of the delay, this linearization involves two matrices: J includes the derivatives w.r.t. the present state $x(t) = (S(t), I(t), R(t))$ and J^τ the derivatives w.r.t. the delayed state $x(t-\tau) = (S(t-\tau), I(t-\tau), R(t-\tau))$. We obtain

$$J = \begin{pmatrix} -\gamma & 0 & 0 \\ 0 & -(\gamma+\delta) & 0 \\ 0 & \delta & -\gamma \end{pmatrix}, \qquad J^\tau = J^\tau(S^*, I^*) = \begin{pmatrix} 0 & -\frac{acS^*}{(1+\alpha I^*)^2} + \delta e^{-\mu\tau} & 0 \\ 0 & \frac{acS^*}{(1+\alpha I^*)^2} & 0 \\ 0 & -\delta e^{-\mu\tau} & 0 \end{pmatrix}, \tag{18}$$

and finally the linearized delay differential equation

$$\dot{x} = Jx(t) + J^\tau x(t-\tau). \tag{19}$$

If $\tau = 0$, then effectively J^τ merges into J, and we recover the classical ODE Jacobian

$$J = J(S^*, I^*) = \begin{pmatrix} -\gamma & -\frac{acS^*}{(1+\alpha I^*)^2} + \delta & 0 \\ 0 & \frac{acS^*}{(1+\alpha I^*)^2} - (\gamma+\delta) & 0 \\ 0 & 0 & -\gamma \end{pmatrix}. \tag{20}$$

First, recall that the DFE (13) is independent of the delay τ and the immunity decay μ, and thus we obtain at the DFE

$$J = \begin{pmatrix} -\gamma & 0 & 0 \\ 0 & -(\gamma+\delta) & 0 \\ 0 & \delta & -\gamma \end{pmatrix}, \qquad J^\tau = J^\tau_{\text{DFE}} = \begin{pmatrix} 0 & -\frac{acB}{\gamma} + \delta e^{-\mu\tau} & 0 \\ 0 & \frac{acB}{\gamma} & 0 \\ 0 & -\delta e^{-\mu\tau} & 0 \end{pmatrix}. \tag{21}$$

To analyze local stability, one looks for exponential solutions $u(t) = e^{\lambda t} v$. In this case the delayed term becomes $x(t-\tau) = e^{\lambda(t-\tau)} v = e^{-\lambda\tau} e^{\lambda t} v$, so that substitution into the linearized equation (19) yields $(\lambda I - J - J^\tau e^{-\lambda\tau})v = 0$. Hence nontrivial solutions exist if and only if

$$\det(\lambda I - J - J^\tau e^{-\lambda\tau}) = 0. \tag{22}$$

This construction of the characteristic matrix for delay differential equations is standard; see, for instance, [10, Section 1.4]. Here, the characteristic matrix is given by

$$\lambda I - J - J^\tau_{\text{DFE}} e^{-\lambda\tau} = \begin{pmatrix} \lambda+\gamma & (\frac{acB}{\gamma} - \delta e^{-\mu\tau}) e^{-\lambda\tau} & 0 \\ 0 & \lambda + (\gamma+\delta) - \frac{acB}{\gamma} e^{-\lambda\tau} & 0 \\ 0 & -\delta + \delta e^{-\mu\tau} e^{-\lambda\tau} & \lambda+\gamma \end{pmatrix}. \tag{23}$$

The first column has only one nonzero element, we have a factorization

$$\det(\lambda I - J - J^\tau_{\mathrm{DFE}} e^{-\lambda\tau}) = (\lambda+\gamma)\cdot\det\begin{pmatrix} \lambda + (\gamma+\delta) - \frac{acB}{\gamma}e^{-\lambda\tau} & 0 \\ -\delta + \delta e^{-\mu\tau}e^{-\lambda\tau} & \lambda+\gamma \end{pmatrix}$$
$$= (\lambda+\gamma)^2\big[\lambda + (\gamma+\delta) - \frac{acB}{\gamma}e^{-\lambda\tau}\big].$$

Hence, we immediately obtain a double eigenvalue $\lambda_{1,2} = -\gamma < 0$, and the third eigenvalue is given by the solution of the scalar transcendental equation

$$0 = \lambda + (\gamma+\delta) - \frac{acB}{\gamma}e^{-\lambda\tau}. \tag{24}$$

An equilibrium is *locally asymptotically stable* (LAS), if all eigenvalues have negative real part. For $\tau = 0$ we obtain the stability condition $\Theta < 1$, with Θ given in (12). In the case with delay, stability changes can occur only through this last exponential factor in (24).

In the case of an endemic equilibrium (EE) we have $I^* > 0$ and the Jacobians evaluated at the endemic equilibrium (17) are

$$J = \begin{pmatrix} -\gamma & 0 & 0 \\ 0 & -(\gamma+\delta) & 0 \\ 0 & \delta & -\gamma \end{pmatrix}, \qquad J^\tau = J^\tau_{\mathrm{EE}}(I^*) = \begin{pmatrix} 0 & -\frac{\gamma+\delta}{1+\alpha I^*} + \delta e^{-\mu\tau} & 0 \\ 0 & \frac{\gamma+\delta}{1+\alpha I^*} & 0 \\ 0 & -\delta e^{-\mu\tau} & 0 \end{pmatrix}, \tag{25}$$

and the characteristic matrix reads

$$\lambda I - J - J^\tau_{\mathrm{EE}}(I^*)e^{-\lambda\tau} = \begin{pmatrix} \lambda+\gamma & (\frac{\gamma+\delta}{1+\alpha I^*} - \delta e^{-\mu\tau})e^{-\lambda\tau} & 0 \\ 0 & \lambda + (\gamma+\delta) - \frac{\gamma+\delta}{1+\alpha I^*}e^{-\lambda\tau} & 0 \\ 0 & -\delta + \delta e^{-\mu\tau}e^{-\lambda\tau} & \lambda+\gamma \end{pmatrix}. \tag{26}$$

Again, a factorization leads to

$$\det(\lambda I - J - J^\tau_{\mathrm{EE}}(I^*)e^{-\lambda\tau}) = (\lambda+\gamma)\cdot\det\begin{pmatrix} \lambda + (\gamma+\delta) - \frac{\gamma+\delta}{1+\alpha I^*}e^{-\lambda\tau} & 0 \\ -\delta + \delta e^{-\mu\tau}e^{-\lambda\tau} & \lambda+\gamma \end{pmatrix}$$
$$= (\lambda+\gamma)^2\big[\lambda + (\gamma+\delta) - \frac{\gamma+\delta}{1+\alpha I^*}e^{-\lambda\tau}\big],$$

with a double eigenvalue $\lambda_{1,2} = -\gamma < 0$, and the third eigenvalue as solution of

$$0 = \lambda + (\gamma+\delta) - \frac{\gamma+\delta}{1+\alpha I^*}e^{-\lambda\tau},$$

and inserting I^* from (16) gives

$$0 = \lambda + (\gamma+\delta) - (\gamma+\delta)\frac{\gamma\big(1+\alpha\frac{\gamma+\delta}{ac}\big) + \delta\left(1 - e^{-\mu\tau}\right)}{\alpha B + \gamma + \delta\left(1-e^{-\mu\tau}\right)}e^{-\lambda\tau}. \tag{27}$$

For $\tau = 0$ we obtain the stability condition

$$\frac{ac\alpha B + ac\gamma}{\gamma[ac + \alpha(\gamma+\delta)]} > 1. \tag{28}$$

In general, the characteristic equation (27) is transcendental due to the exponential term $e^{-\lambda\tau}$ and therefore cannot be solved explicitly for λ. Nevertheless, qualitative information on stability can be obtained by standard techniques, such as investigating the sign of the real root at $\lambda = 0$ or searching for purely imaginary solutions $\lambda = \mathrm{i}\omega$.

3.3. Analysis of the Generalized Model

We consider the system (4) with incidence parameter $\beta = ac$ and $\kappa(I)$ chosen as (5). Obviously, for the default-free equilibrium (DFE) we have $E^* = I^* = R^* = 0$ and $S^* = B/\gamma$. For the endemic equilibrium (EE), let $I^* > 0$, and from the last equation of (4) we obtain

$$R^* = \frac{\delta}{\rho+\gamma} I^*.$$

From the infected equation in (4) we get immediately $E^* = (\delta+\gamma)I^*/\kappa(I^*)$, and inserting E^* in the exposed equation yields

$$S^* = S^*(I^*) = \frac{(1+\alpha I^*)(\kappa(I^*)+\gamma)(\delta+\gamma)}{\beta\,\kappa(I^*)}.$$

The value I^* is then determined by substituting the relation above into the susceptible balance

$$0 = B - \gamma S^* - \beta S^* \frac{I^*}{1+\alpha I^*} + \frac{\rho\delta}{\rho+\gamma} I^*,$$

which provides a scalar nonlinear equation for I^*

$$0 = B - \gamma \frac{(1+\alpha I^*)(\kappa(I^*)+\gamma)(\delta+\gamma)}{\beta\,\kappa(I^*)} - \frac{(\kappa(I^*)+\gamma)(\delta+\gamma)I^*}{\kappa(I^*)} + \frac{\rho\delta}{\rho+\gamma} I^*. \tag{29}$$

Whenever this equation (29) admits a positive solution, an EE exists.

Next, the Jacobian of the right-hand side $F(S,E,I,R)$ of the generalized model (4) is

$$J = J(S,E,I) = \begin{pmatrix} -\gamma - \dfrac{\beta I}{1+\alpha I} & 0 & -\dfrac{\beta S}{(1+\alpha I)^2} & \rho \\ \dfrac{\beta I}{1+\alpha I} & -(\kappa(I)+\gamma) & \dfrac{\beta S}{(1+\alpha I)^2} - E\kappa'(I) & 0 \\ 0 & \kappa(I) & E\kappa'(I) - (\delta+\gamma) & 0 \\ 0 & 0 & \delta & -(\rho+\gamma) \end{pmatrix},$$

where $\kappa'(I) = \kappa_0 k$.

At the DFE we have $\kappa(0) = \kappa_0$ and $\kappa'(0) = \kappa_0 k$. For $I^* = 0$ the Jacobian becomes block triangular and one eigenvalue is $\lambda_1 = -\gamma < 0$ and another one is $\lambda_2 = -(\rho+\gamma) < 0$. The remaining eigenvalues are those of the (E,I) block

$$M = \begin{pmatrix} -(\kappa_0+\gamma) & \dfrac{\beta B}{\gamma} \\ \kappa_0 & -(\delta+\gamma) \end{pmatrix}.$$

The eigenvalues of M satisfy $\lambda^2 + a_1\lambda + a_0 = 0$, with

$$a_1 = -\operatorname{Tr} M = (\kappa_0+\gamma) + (\delta+\gamma), \qquad a_0 = \det M = (\kappa_0+\gamma)(\delta+\gamma) - \kappa_0 \frac{\beta B}{\gamma}.$$

Since $a_1 > 0$, both roots have negative real part iff $a_0 > 0$, which is equivalent to

$$\frac{\beta B}{\gamma} < \frac{(\kappa_0+\gamma)(\delta+\gamma)}{\kappa_0},$$

Hence, the DFE is locally asymptotically stable if for the basic reproduction number Θ holds

$$\Theta = \frac{\beta B}{\gamma(\delta+\gamma)} \frac{\kappa_0}{\kappa_0+\gamma} < 1 \tag{30}$$

and unstable if $\Theta > 1$.

Now assume $\Theta > 1$ so that a (positive) endemic equilibrium exists. At the EE one eigenvalue remains $\lambda_1 = -(\rho+\gamma) < 0$. The remaining eigenvalues are determined by the 3×3 block associated with (S, E, I). Let the characteristic polynomial be $\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0$. Using the equilibrium identities

$$\kappa(I^*)E^* = (\delta+\gamma)I^*, \qquad \beta S^* \frac{I^*}{1+\alpha I^*} = (\kappa(I^*)+\gamma)E^*,$$

the coefficients simplify and are positive under the admissible parameter range. By the *Routh–Hurwitz criterion*, all roots have negative real part provided

$$b_1 > 0, \qquad b_3 > 0, \qquad b_1 b_2 > b_3.$$

These inequalities are satisfied for standard monotone incidence functions and positive parameters. Therefore the endemic equilibrium is locally asymptotically stable whenever it exists.

3.4. Equilibria and Stability of the Delayed Generalized System

We consider the generalized system with delay (7) and proceed analogously to Section 3.2. The default-free equilibrium (DFE) is again independent of the delay τ and the immunity decay μ, and thus we have

$$E^{\tau}_{\mathrm{DFE}} = \left(\frac{B}{\gamma}, 0, 0, 0\right). \tag{31}$$

Next, the computation of the two Jacobians at the equilibria yields ($\kappa'(I) = \kappa_0 k$)

$$J = J(E^*, I^*) = \begin{pmatrix} -\gamma & 0 & 0 & 0 \\ 0 & -(\kappa(I^*)+\gamma) & -\kappa'(I^*)E^* & 0 \\ 0 & \kappa(I^*) & \kappa'(I^*)E^* - (\delta+\gamma) & 0 \\ 0 & 0 & \delta & -\gamma \end{pmatrix},$$

$$J^{\tau} = J^{\tau}(S^*, I^*) = \begin{pmatrix} -\dfrac{\beta I^*}{1+\alpha I^*} & 0 & -\dfrac{\beta S^*}{(1+\alpha I^*)^2} + \delta e^{-\mu\tau} & 0 \\ \dfrac{\beta I^*}{1+\alpha I^*} & 0 & \dfrac{\beta S^*}{(1+\alpha I^*)^2} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -\delta e^{-\mu\tau} & 0 \end{pmatrix}.$$

Linearizing the infected subsystem (E, I) yields the same next-generation matrix as in the non-delayed ODE case. Hence, we have the same condition (30) for stability of the DFE. Linearization yields a system of the form (19) with delay only appearing in infection terms. The characteristic equation at the DFE reads

$$\det(\lambda I - J_{\mathrm{DFE}} - J^{\tau}_{\mathrm{DFE}} e^{-\lambda\tau}) = 0,$$

with

$$J_{\mathrm{DFE}} = J(0,0) = \begin{pmatrix} -\gamma & 0 & 0 & 0 \\ 0 & -(\kappa_0+\gamma) & 0 & 0 \\ 0 & \kappa_0 & -(\delta+\gamma) & 0 \\ 0 & 0 & \delta & -\gamma \end{pmatrix},$$

$$J^{\tau}_{\text{DFE}} = J^{\tau}\Big(\frac{B}{\gamma}, 0\Big) = \begin{pmatrix} 0 & 0 & -\frac{\beta B}{\gamma} + \delta e^{-\mu\tau} & 0 \\ 0 & 0 & \frac{\beta B}{\gamma} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -\delta e^{-\mu\tau} & 0 \end{pmatrix}.$$

One eigenvalue is immediately $\lambda_1 = -\gamma < 0$. The remaining spectrum is determined by

$$(\lambda+\gamma)\Big[\lambda^2 + a_1\lambda + a_0 - \kappa_0\frac{\beta B}{\gamma}e^{-(\lambda+\mu)\tau}\Big] = 0,$$

where

$$a_1 = (\kappa_0+\gamma) + (\delta+\gamma), \qquad a_0 = (\kappa_0+\gamma)(\delta+\gamma).$$

The DFE is locally asymptotically stable for all $\tau \geq 0$ if $\Theta < 1$. If $\Theta > 1$, it is unstable.

Next, we assume $\Theta > 1$. A positive equilibrium ($I^* > 0$) of (7) satisfies

$$R^* = R^*(I^*) = \frac{\delta}{\gamma}\big(1 - e^{-\mu\tau}\big)I^*, \qquad E^* = E^*(I^*) = \frac{(\delta+\gamma)}{\kappa(I^*)}\,I^*,$$

and from the first two equations of (7) we obtain

$$S^* = S^*(I^*) = \frac{B}{\gamma} + \frac{\delta}{\gamma}\,I^* e^{-\mu\tau} - \frac{(\delta+\gamma)(\kappa(I^*)+\gamma)}{\gamma\kappa(I^*)}\,I^*.$$

Substitution into the susceptible balance yields a scalar nonlinear equation

$$\Phi(I^*) = \frac{(\delta+\gamma)(\kappa(I^*)+\gamma)}{\kappa(I^*)}\,I^* - \beta\, S^*(I^*)\frac{I^*}{1+\alpha I^*} = 0. \tag{32}$$

Now, we will show the existence of a positive solution of (32). To do so, we define $\Phi\colon (0,\infty) \to \mathbb{R}$ by the right-hand side of (32). First, Φ is continuous for $I > 0$ since $\kappa(I)$, the saturated incidence function, and $S^*(I)$ are smooth.

First, we have $\Phi(0) = 0$ and differentiating Φ and evaluating at $I = 0$ shows

$$\Phi'(0) = (\delta+\gamma) - \frac{\beta B}{\gamma}\frac{\kappa_0}{\kappa_0+\gamma} < 0 \quad \text{for } \Theta > 1.$$

Moreover, using $\kappa(I) = \kappa_0(1+kI)$, we see the asymptotic behaviour $\Phi(I) \sim CI$, as $I \to \infty$, with $C > 0$. Thus, $\lim_{I\to\infty}\Phi(I) = +\infty$. Therefore, if $\Theta > 1$, at least one positive root exists.

Differentiating $\Phi = \Phi(I)$ shows that all nonlinear terms are strictly increasing functions of I since both the saturated incidence $I/(1+\alpha I)$ and $\kappa(I) = \kappa_0(1+kI)$ are increasing. Consequently, $\Phi'(I) > 0$ for all sufficiently large I, and the function can cross zero at most once. Hence the EE is unique whenever $\Theta > 1$.

We have seen that the basic reproduction number Θ is unaffected by the delay. Hence the onset of contagion depends solely on structural parameters of transmission and progression. However, the delay may destabilize the endemic state. Economically, this corresponds to cyclical waves of financial distress driven by delayed recovery and immunity decay.

After analyzing the qualitative dynamics of the continuous model, we will construct in the next section numerical schemes that preserve these structural properties.

4. The Nonstandard Finite Difference Scheme

4.1. The NSFD scheme for the non-delayed problem

First, we propose the following NSFD discretization for solving the ODE system (1) with $\tau = 0$

$$\begin{aligned}\frac{S^{n+1}-S^n}{\phi(h)} &= B - \gamma S^{n+1} - acS^{n+1}\frac{I^n}{1+\alpha I^n} + \delta I^n,\\ \frac{I^{n+1}-I^n}{\phi(h)} &= acS^{n+1}\frac{I^n}{1+\alpha I^n} - (\delta+\gamma)I^{n+1},\\ \frac{R^{n+1}-R^n}{\phi(h)} &= \delta I^{n+1} - \delta I^n - \gamma R^{n+1}.\end{aligned} \tag{33}$$

Here, in (33), the *denominator function* $\phi(h) > 0$ is given by

$$\phi(h) = \frac{e^{\gamma h}-1}{\gamma}. \tag{34}$$

In the sequel, let us briefly motivate the choice (34). We can rewrite (3) in the form

$$N(t) = N(0) + \left(N(0) - \frac{B}{\gamma}\right)(e^{-\gamma t} - 1), \tag{35}$$

From (35) we immediately deduce that we have in the long term $\lim_{t\to\infty} N(t) = B/\gamma$.

Next, adding the equations in the discrete NSFD model (33) yields

$$\frac{N^{n+1}-N^n}{\phi(h)} = B - \gamma\, N^{n+1}, \tag{36}$$

i.e.

$$\begin{aligned}N^{n+1} &= \frac{N^n + \phi(h)B}{1+\phi(h)\,\gamma} = N^n - \left(N^n - \frac{B}{\gamma}\right)\frac{\phi(h)\,\gamma}{1+\phi(h)\,\gamma}\\ &= N^n + \left(N^n - \frac{B}{\gamma}\right)\left(\frac{1}{1+\phi(h)\,\gamma} - 1\right).\end{aligned} \tag{37}$$

The denominator function $\phi(h)$ can be derived by comparing Equation (37) with the discrete version of Equation (35), that is

$$N^{n+1} = N^n + \left(N^n - \frac{B}{\gamma}\right)(e^{-\gamma h} - 1), \quad h = \Delta t, \tag{38}$$

such that the (positive) denominator function is defined by

$$\frac{1}{1+\phi(h)\,\gamma} = e^{-\gamma h}, \tag{39}$$

i.e.

$$\phi(h) = \frac{e^{\gamma h}-1}{\gamma} = \frac{1+\gamma h + \frac{1}{2}\gamma^2 h^2 + \cdots - 1}{\gamma} = h + \frac{\gamma h^2}{2} + \cdots = h + \mathcal{O}(h^2), \quad h \to 0. \tag{40}$$

Remark 1 (Structural Conflict)**.** *The denominator function (34) (in the sense of Mickens) is derived from the decay rate γ of the total population N, mimicking the exact preservation of total population law. However, the infected compartment has a higher decay rate $\gamma + \delta$, due to the recovery, which motivates an individual denominator function*

$$\phi_I(h) = \frac{e^{(\gamma+\delta)h} - 1}{\gamma + \delta} \tag{41}$$

only for the I compartment. So, there is a fundamental incompatibility: compartment-wise exact decay vs. the exact preservation of total population law. For epidemiological and credit contagion models preserving the invariant region and asymptotic total population is usually more important than reproducing the exact decay rate of a single compartment. So, we will choose only one denominator function (34). A similar conflict will appear for the E and I compartment in the generalized problem (46).

We will briefly comment on the discretization of the nonlinear terms. In the first line of equation (33), for example, we have replaced the nonlinear contact term $acS(t)\frac{I(t)}{1+\alpha I(t)}$ in equation (1) with $acS^{n+1}\frac{I^n}{1+\alpha I^n}$ rather than with $acS^n\frac{I^n}{1+\alpha I^n}$ or $acS^{n+1}\frac{I^{n+1}}{1+\alpha I^{n+1}}$. The rule is that exactly one factor of the variable appearing in the time derivative (S) must be evaluated at the new time level, $n+1$ and due to the minus sign we have to choose S here. This selection is necessary to obtain a positivity-preserving scheme (42). In order to maintain explicit sequential evaluation, all other variables are taken from the previous time level unless they are already known from a previous step, such as I^{n+1} in the third line in (33). If possible, discrete conservation properties must also be taken into account (here, the total population N^{n+1}).

Observe that although the initial scheme (33) can be considered implicit, the variables at the $(n+1)$-th discrete-time level can be explicitly calculated in terms of the previously known variable values as given in the sequence of the equations above, i.e. we can rewrite (33) as an explicit scheme:

$$\begin{aligned} S^{n+1} &= \frac{S^n + \phi(h)\left(B + \delta I^n\right)}{1 + \phi(h)\left(\gamma + ac\frac{I^n}{1+\alpha I^n}\right)}, \\ I^{n+1} &= \frac{I^n + \phi(h)acS^{n+1}\frac{I^n}{1+\alpha I^n}}{1 + \phi(h)(\delta + \gamma)}, \\ R^{n+1} &= \frac{R^n + \phi(h)\delta(I^{n+1} - I^n)}{1 + \phi(h)\gamma}, \end{aligned} \tag{42}$$

and equation (36) is rewritten as

$$N^{n+1} = \frac{N^n + \phi(h)B}{1 + \phi(h)\gamma}. \tag{43}$$

The calculations in (42) must be done in exactly this order. By convention, all parameters appearing in these type of epidemic models are always non-negative. From the explicit representation (42) it is easy to deduce that this scheme preserves the positivity for S^n and I^n; for the recovered class R^n we show this property in Corollary 1 in Appendix A.

4.2. The NSFD scheme for the problem with delay

We design a NSFD discretization for solving the ODE system (1) with delay $\tau > 0$. Let the time delay be given by $\tau = mh > 0$ and $I^{n-m} \approx I(t-\tau)$:

$$\begin{aligned}
\frac{S^{n+1}-S^n}{\phi(h)} &= B - \gamma S^{n+1} - acS^{n+1}\frac{I^{n-m}}{1+\alpha I^{n-m}} + \delta I^{n-m}\, e^{-\mu m h}, \\
\frac{I^{n+1}-I^n}{\phi(h)} &= acS^{n+1}\frac{I^{n-m}}{1+\alpha I^{n-m}} - (\delta+\gamma)I^{n+1}, \\
\frac{R^{n+1}-R^n}{\phi(h)} &= \delta I^{n+1} - \delta I^{n-m}\, e^{-\mu m h} - \gamma R^{n+1}.
\end{aligned} \tag{44}$$

Again, this formally implicit scheme can be rewritten in an explicit form:

$$\begin{aligned}
S^{n+1} &= \frac{S^n + \phi(h)\,(B + \delta I^{n-m}\, e^{-\mu m h})}{1+\phi(h)\big(\gamma + ac\frac{I^{n-m}}{1+\alpha I^{n-m}}\big)}, \\
I^{n+1} &= \frac{I^n + \phi(h)\, acS^{n+1}\frac{I^{n-m}}{1+\alpha I^{n-m}}}{1+\phi(h)(\delta+\gamma)}, \\
R^{n+1} &= \frac{R^n + \phi(h)\,\delta(I^{n+1} - I^{n-m}\, e^{-\mu m h})}{1+\phi(h)\gamma},
\end{aligned} \tag{45}$$

and for the total population N^{n+1} the same equation (37). The arguments for the positivity of the solution follows the ones in Corollary 1.

4.3. The NSFD scheme for the generalized problem

Finally, we propose a NSFD discretization for the generalized ODE system (7) with $\beta = ac$. Again, the time delay is $\tau = mh > 0$ and we have $I^{n-m} \approx I(t-\tau)$:

$$\begin{aligned}
\frac{S^{n+1}-S^n}{\phi(h)} &= B - \gamma S^{n+1} - \beta S^{n+1}\frac{I^{n-m}}{1+\alpha I^{n-m}} + \delta I^{n-m}\, e^{-\mu m h}, \\
\frac{E^{n+1}-E^n}{\phi(h)} &= \beta S^{n+1}\frac{I^{n-m}}{1+\alpha I^{n-m}} - (\kappa(I^n)+\gamma)E^{n+1}, \\
\frac{I^{n+1}-I^n}{\phi(h)} &= \kappa(I^n)E^{n+1} - (\delta+\gamma)I^{n+1}, \\
\frac{R^{n+1}-R^n}{\phi(h)} &= \delta I^{n+1} - \delta I^{n-m}\, e^{-\mu m h} - \gamma R^{n+1}.
\end{aligned} \tag{46}$$

As before, we rewrite this scheme (46) in a sequential explicit version:

$$\begin{aligned}
S^{n+1} &= \frac{S^n + \phi(h)\,(B + \delta I^{n-m}\, e^{-\mu m h})}{1+\phi(h)\big(\gamma + \beta\frac{I^{n-m}}{1+\alpha I^{n-m}}\big)}, \\
E^{n+1} &= \frac{E^n + \phi(h)\beta S^{n+1}\frac{I^{n-m}}{1+\alpha I^{n-m}}}{1+\phi(h)(\kappa(I^n)+\gamma)}, \\
I^{n+1} &= \frac{I^n + \phi(h)\kappa(I^n)E^{n+1}}{1+\phi(h)(\delta+\gamma)}, \\
R^{n+1} &= \frac{R^n + \phi(h)\delta(I^{n+1} - I^{n-m}\, e^{-\mu m h})}{1+\phi(h)\gamma},
\end{aligned} \tag{47}$$

and for the total population $N^{n+1} = S^{n+1} + E^{n+1} + I^{n+1} + R^{n+1}$ the same discrete decay equation (36) holds.

5. Analysis of the NSFD schemes

The proposed NSFD discretization reproduces the main structural features of the continuous credit risk contagion model at the discrete level. In particular, for arbitrary step sizes and nonnegative initial data, the scheme guarantees the following: (i) well-posedness of the updates, (ii) unconditional positivity of the susceptible and defaulted populations, (iii) nonnegativity of the recovered class via the discrete conservation relation, (iv) boundedness of all compartments derived from the exact evolution law of the total population, (v) first-order consistency with the differential system, (vi) exact correspondence between the continuous and discrete equilibria, and (vii) preservation of local stability properties in the sense of elementary stability. These results confirm that the numerical method is dynamically compatible with the qualitative behavior of the underlying model.

5.1. Analysis for the model without delay

We will show that any equilibrium of the continuous system is a fixed point of the NSFD scheme, independently of the step size $h = \Delta t$. A discrete equilibrium (S_d^*, I_d^*, R_d^*) of the NSFD scheme satisfies

$$S^{n+1} = S^n =: S_d^*, \quad I^{n+1} = I^n =: I_d^*, \quad R^{n+1} = R^n =: R_d^*.$$

Plugging these equilibrium values into the scheme (33) yields algebraic identities that reduce precisely to the equilibrium equations of the continuous system. We have $R_d^* = 0$ and

$$\begin{aligned} acS_d^* \frac{I_d^*}{1+\alpha I_d^*} &= B - \gamma S_d^* + \delta I_d^*, \\ acS_d^* \frac{I_d^*}{1+\alpha I_d^*} &= (\delta+\gamma) I_d^*, \end{aligned} \tag{48}$$

i.e. for $I_d^* > 0$ the same values as (10), (11). Since the denominator function $\phi(h)$ cancels out, the fixed point is independent of the time step. This holds obviously also for the default-free equilibrium E_{DFE}.

5.2. Analysis for the model with delay

We consider the NSFD scheme (45) to solve (1) with a positive denominator function satisfying $\phi(h) = h + O(h^2)$ to preserve the first order consistency. Throughout, we assume nonnegative initial data. First, we quickly realize that the steady-state equations of the scheme (45) coincide exactly with the equilibrium equations of the continuous model. In other words, the scheme preserves the equilibria exactly. Hence, the default-free equilibrium E_{DFE}^τ is given by (13) and linearizing (45) around E_{DFE}^τ yields

$$I^{n+1} = \frac{I^n + \phi(h)\, ac\frac{B}{\gamma} I^{n-m}}{1+\phi(h)(\delta+\gamma)}.$$

Let

$$\beta_d = \frac{\phi(h)acB/\gamma}{1+\phi(h)(\delta+\gamma)} = \frac{\phi(h)(\delta+\gamma)}{1+\phi(h)(\delta+\gamma)}\Theta.$$

The characteristic equation becomes

$$\lambda^{m+1} - \frac{1}{1+\phi(h)(\delta+\gamma)}\lambda^m - \beta_d = 0,$$

and setting $\lambda = 1$ in the characteristic equation gives

$$1 - \frac{1}{1+\phi(h)(\delta+\gamma)} - \frac{\phi(h)(\delta+\gamma)}{1+\phi(h)(\delta+\gamma)}\Theta = 0,$$

i.e. the discrete basic reproduction number (threshold parameter) coincides exactly with that of the continuous system (12).

Theorem 1. *The default-free equilibrium (DFE) is locally asymptotically stable if $\Theta < 1$ and unstable if $\Theta > 1$, independently of $h > 0$.*

Proof. If $\Theta < 1$, the characteristic roots satisfy $|\lambda| < 1$. Because the linear part is discretized using $\phi(h)$, the mapping $\lambda = e^{\sigma h}$ transforms the discrete characteristic equation into the continuous one. Therefore stability properties coincide. □

Remark 2. *The basic reproduction number Θ is independent of the delay τ. The delay only affects the stability of the endemic equilibrium.*

5.3. Analysis for the generalized four-compartment NSFD scheme

We consider the NSFD scheme for the generalized problem (47), where $\phi(h) > 0$ satisfies $\phi(h) = h + O(h^2)$ and $\kappa(I) \geq 0$ is a continuous incidence progression rate. Assuming non-negative initial data for $n = -m, \ldots, 0$, one verifies from the scheme (47) that it is positivity-preserving, i.e. the solution satisfies $S^n, E^n, I^n, R^n \geq 0$, for all $n \geq 0$.

Define the total population $N^n = S^n + E^n + I^n + R^n$. Summing the four equations yields

$$N^{n+1} = \frac{N^n + \phi(h)B}{1+\phi(h)\gamma}.$$

If $N^n \leq B/\gamma$, then

$$N^{n+1} \leq \frac{B/\gamma + \phi(h)B}{1+\phi(h)\gamma} = \frac{B}{\gamma},$$

i.e. the set $\Omega = \{(S,E,I,R) \geq 0 : \ N \leq B/\gamma\}$ is a *positively invariant region.*

Once more, the steady states of the discrete scheme (47) are exactly the same as those of the corresponding continuous delayed SEIR system, regardless of h. Thus, the default-free equilibrium E^{τ}_{DFE} is (31), and linearizing (47) around E^{τ}_{DFE} gives

$$E^{n+1} = \frac{E^n + \phi(h)\beta\frac{B}{\gamma}I^{n-m}}{1+\phi(h)(\kappa(0)+\gamma)}, \quad I^{n+1} = \frac{I^n + \phi(h)\kappa(0)E^{n+1}}{1+\phi(h)(\delta+\gamma)}.$$

Combining gives the linear delayed system of the form

$$\begin{pmatrix} E^{n+1} \\ I^{n+1} \end{pmatrix} = J \begin{pmatrix} E^{n} \\ I^{n} \end{pmatrix} + J^{\tau} \begin{pmatrix} E^{n-m} \\ I^{n-m} \end{pmatrix}.$$

After elimination, the threshold parameter becomes

$$\Theta = \frac{\beta B \kappa(0)}{\gamma(\kappa(0)+\gamma)(\delta+\gamma)},$$

i.e. the discrete basic reproduction number coincides exactly with the continous one (30).

Theorem 2. *The DFE is locally asymptotically stable if $\Theta < 1$ and unstable if $\Theta > 1$, independently of the step size h.*

Proof. The characteristic equation of the linearized system satisfies $\lambda = e^{\sigma h}$, where σ solves the continuous characteristic equation. Hence stability properties coincide. □

Next, we will study the stability of the endemic equilibrium. Assume $\Theta > 1$ so that a unique endemic equilibrium $E^{\tau}_{\text{EE}} = (S^*, E^*, I^*, R^*)$ exists with $I^* > 0$. Linearizing system (7) at E^{τ}_{EE} yields a linear delay differential equation of the form

$$\dot{x}(t) = Jx(t) + J^{\tau}x(t-\tau), \tag{49}$$

where

$$J = \frac{\partial F}{\partial x}\Big|_{E^{\tau}_{\text{EE}}}, \qquad J^{\tau} = \frac{\partial F}{\partial x_{\tau}}\Big|_{E^{\tau}_{\text{EE}}},$$

and F denotes the right hand side of the DDE system. Since only the infected component $I(t-\tau)$ appears with a delay, the matrix B has nonzero entries only in the first and fourth equations. A direct computation gives

$$J = \begin{pmatrix} -\gamma - \Phi_1 & 0 & -\Phi_2 & 0 \\ \Phi_1 & -(\kappa(I^*) + \gamma) & \Phi_3 & 0 \\ 0 & \kappa(I^*) & -(\delta+\gamma) & 0 \\ 0 & 0 & \delta & -\gamma \end{pmatrix}, \quad J^{\tau} = \begin{pmatrix} -\frac{\beta S^*}{(1+\alpha I^*)^2} & 0 & 0 & 0 \\ \frac{\beta S^*}{(1+\alpha I^*)^2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -\delta e^{-\mu\tau} & 0 & 0 & 0 \end{pmatrix},$$

where

$$\Phi_1 = \beta\frac{I^*}{1+\alpha I^*}, \quad \Phi_2 = \beta S^*\frac{1}{(1+\alpha I^*)^2}, \quad \Phi_3 = \beta S^*\frac{1}{(1+\alpha I^*)^2} + E^*\kappa'(I^*).$$

Next, we seek exponential solutions $x(t) = e^{\lambda t}v$, $v \neq 0$, in (49). This yields

$$\lambda v = Jv + J^{\tau}e^{-\lambda\tau}v.$$

Hence, $(\lambda I - J - J^{\tau}e^{-\lambda\tau})v = 0$. Nontrivial solutions exist if and only if

$$\det(\lambda I - J - J^{\tau}e^{-\lambda\tau}) = 0. \tag{50}$$

This is the characteristic equation of the linearized delay system, cf. [10, Section 1.4]. Because of the block triangular structure, one eigenvalue is immediately $\lambda = -\gamma$. The remaining eigenvalues satisfy a three-dimensional transcendental equation.

The following simulations in Section 6 illustrate the theoretical results and demonstrate the robustness of the proposed NSFD schemes.

6. Numerical Results

In this section, we demonstrate the performance and qualitative behavior of the proposed NSFD schemes for the non-delayed and delayed versions of the credit risk contagion model introduced in Fanelli and Maddalena [7]. These numerical experiments highlight the structure-preserving properties of the schemes, such as positivity, boundedness, and correct long-time dynamics of the susceptible, infected, and recovered populations.

Following the modeling framework of [7], the parameter B represents the constant inflow rate of new susceptible (defaultable) agents into the market ('bank growth rate'), and parameter γ denotes the natural exit rate common to all compartments ('medium rate for default'). The contagion mechanism is determined by the contact rate c and the credit risk transmission probability a, whose product ac governs the effective interaction between susceptible and defaulted agents. The parameter α moderates the nonlinear incidence term, limiting the effective rate of infection for large defaulted populations. Defaulted agents recover at a rate of δ, and in the delayed formulation, they are temporarily immune to reinfection for a period of τ. The parameter μ quantifies this immunity decay and determines the fraction of recovered agents that return to susceptible dynamics after the delay.

Unless otherwise stated, the baseline parameter values and initial conditions match those in [7] to facilitate direct comparison with the continuous-time model behavior. Specifically, we select values for B, γ, a, c, α, δ, τ, and μ that yield both subcritical and supercritical regimes in the continuous model, examining how NSFD schemes (45), (47) reproduce these regimes.

6.1. The base model with delay

We selected a time interval of $[0, 30]$ and a time step of $h = 0.1$. In Figure 1 we consider in the Case 1 and the model (1) with delays of $\tau = 0.5$, and $\tau = 2$, respectively. We fix a constant solution history $S(t) = I(t) = R(t) = 0.5$ for the time interval $t \in [-\tau, 0]$.

In Table 1 we summarize the baseline parameter values used in the Case 1. In this case we obtain for the factor $\Theta = 8/11 < 1$. In our numerical simulations of the model (1), we found that the default state $I(t)$ dies out of the banks and the solution approaches the globally stable, default-free equilibrium E_{DFE}.

Parameter	Meaning	Baseline Value
B	Bank growth rate	0.8
γ	Medium rate for default	0.5
a	Transmission probability	0.5
c	Contact rate	1
α	Saturation coefficient	1
δ	Recovery rate	0.6
τ	Immunity delay	0.5, 2
μ	Immunity decay	0.5

Table 1: Case 1: Baseline parameter values used in numerical simulations, cf. [7].

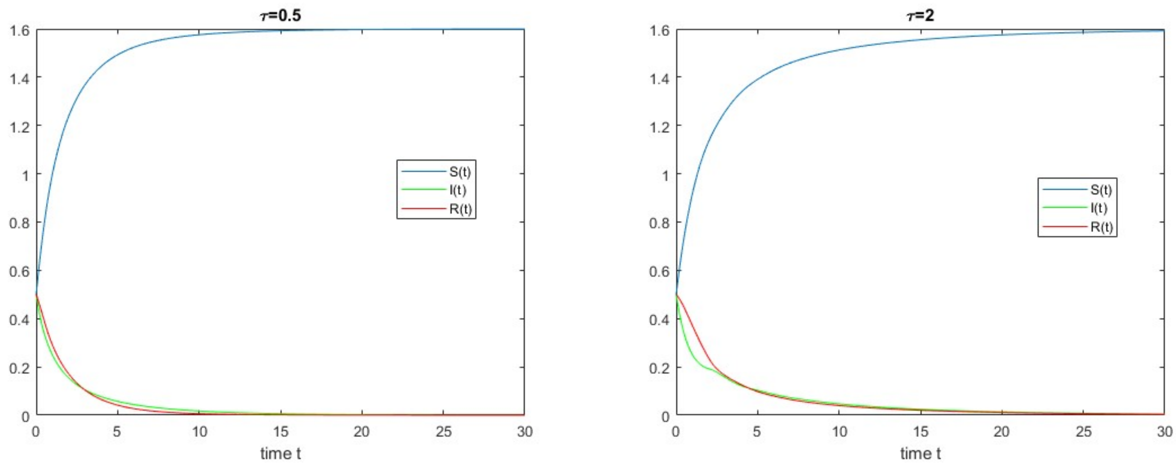

Figure 1: Case 1: NSFD solution of model (1) for $h = 0.1$, $\Theta < 1$ and $\tau = 0.5$, $\tau = 2$.

On the other hand, in Case 2, we consider a smaller default probability ($\gamma = 0.2$) and a smaller recovery rate ($\delta = 0.3$), while keeping the other parameter values unchanged, see

Parameter	Meaning	Baseline Value
B	Bank growth rate	0.8
γ	Medium rate for default	0.2
a	Transmission probability	0.5
c	Contact rate	1
α	Saturation coefficient	1
δ	Recovery rate	0.3
τ	Immunity delay	0.5, 1, 2
μ	Immunity decay	0.5

Table 2: Case 2: Baseline parameter values used in numerical simulations, cf. [7].

Table 2. In this Case 2, we have a different regime, since the rate $\Theta = 4 > 1$. Figure 2 shows the solutions to the model (1) for different values of τ: 0.5, 1, and 2. As the delay τ increases, we observe that the equilibrium values S^* and I^* decrease and the number of recovered agents after default $R(t)$ increases, cf. [7, Table 1].

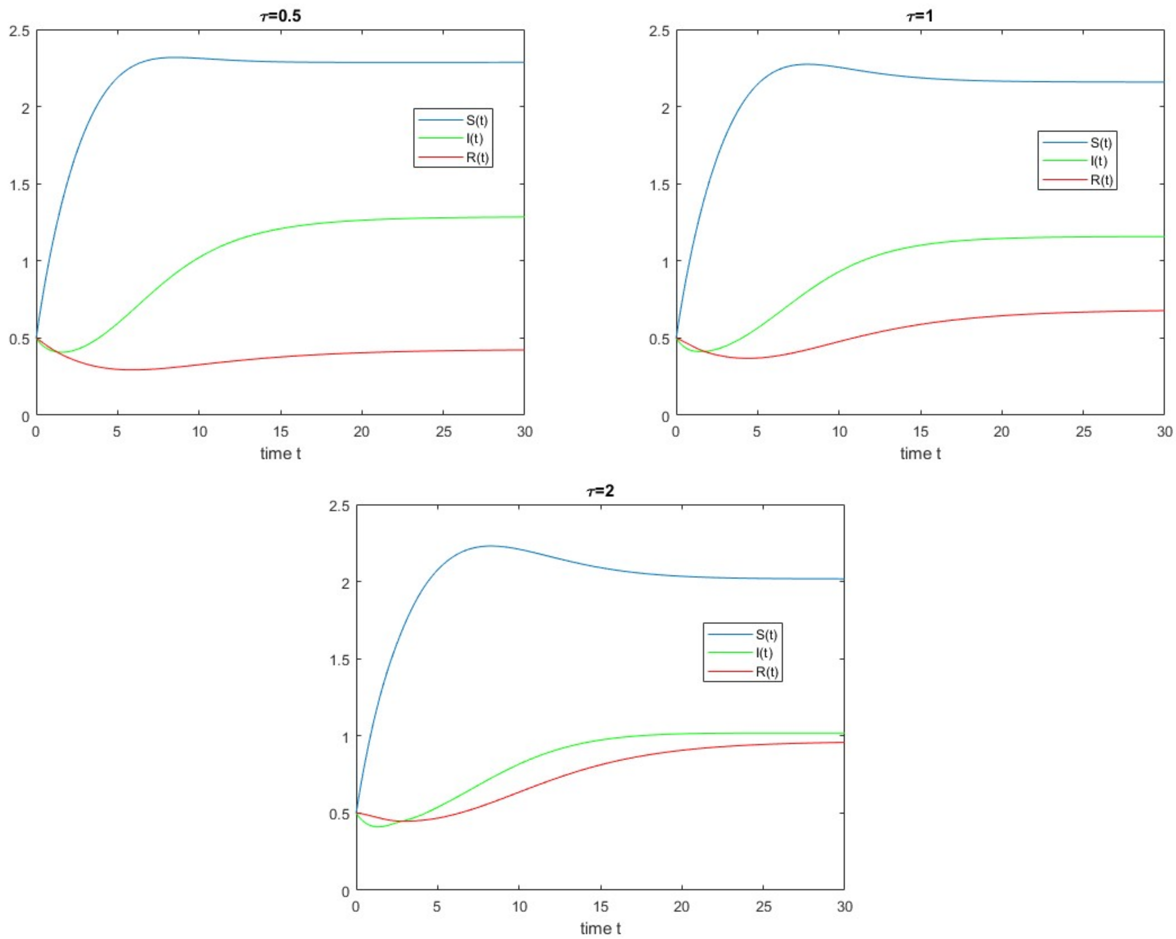

Figure 2: Case 2: NSFD solution of model (1) for $h = 0.1$, $\Theta > 1$ and $\tau = 0.5$, $\tau = 1$, $\tau = 2$.

6.2. The Generalized model

Finally, we simulate the generalized model with delay (7) using the NSFD method (47). For the state-dependent activation rate $\kappa(I)$ we use the choice (5), with $\kappa_0 = 0.8$ and $k = 1$. We revisit the previous two cases and label them Cases 3 and 4.

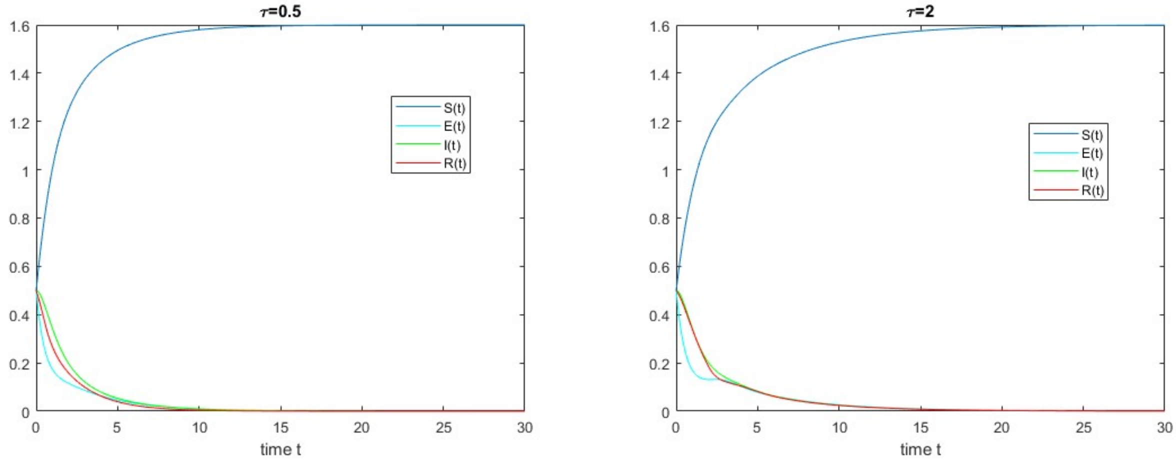

Figure 3: Case 3: NSFD solution of model (7) for $h = 0.1$, $\Theta < 1$ and $\tau = 0.5$, $\tau = 2$.

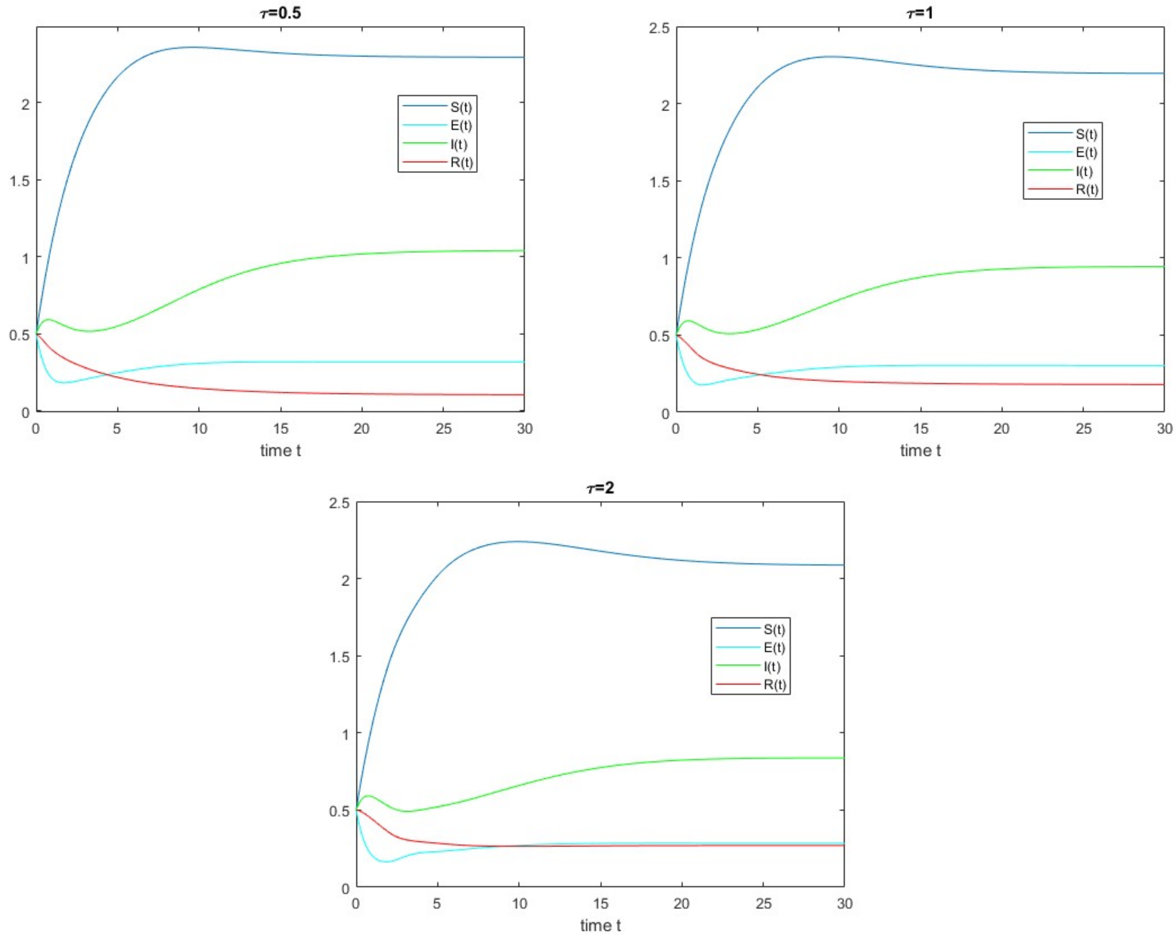

Figure 4: Case 4: NSFD solution of model (7) for $h = 0.1$, $\Theta > 1$ and $\tau = 0.5$, $\tau = 1$, $\tau = 2$.

The qualitative behavior changes precisely at $\Theta = 1$ in all simulations, confirming the analytical threshold condition. The qualitative behavior remains consistent across all tested step sizes, demonstrating the fundamental stability of the NSFD scheme approach. From a financial perspective, the delay parameter, tau, represents the time required for contagion effects to materialize. Large delays may induce oscillatory credit cycles.

Finally we want to illustrate what might happen, when using a standard FDM approach. Here selected the simple explicit Euler scheme with a coarser step size $h = 0.9$ and simulated

Cases 3 and 4, i.e. the generalized model (7). One observes in Figure 5 that the solution in both cases is oscillatory and becomes negative at some time instance.

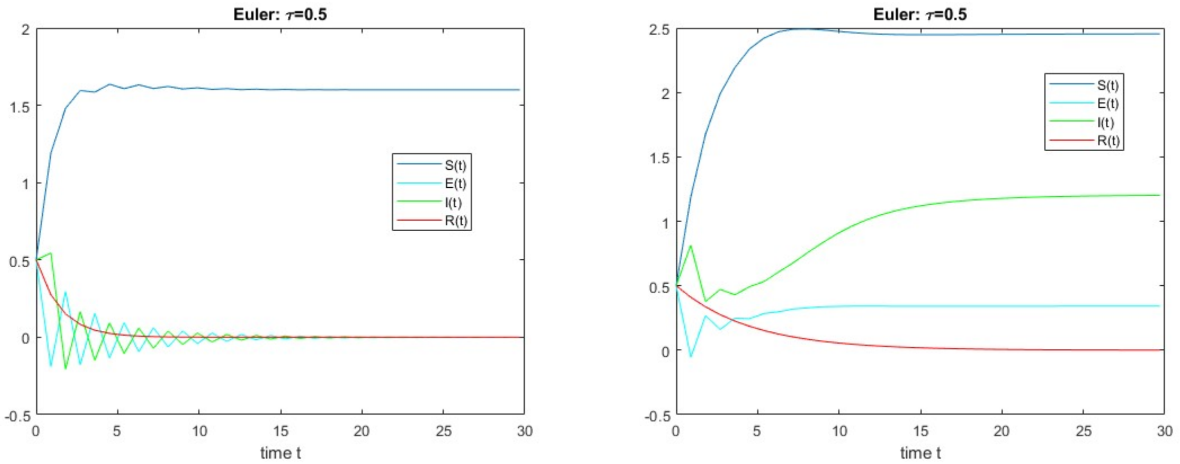

Figure 5: Cases 3 (left) / 4 (right): Euler scheme solution of model (7) for $h = 0.9$, $\Theta < 1$ and $\tau = 0.5$.

7. Conclusion

In this work, we developed structure-preserving, nonstandard, finite-difference (NSFD) schemes for the credit-risk contagion model introduced by Fanelli and Maddalena [7] and a generalized four-compartment model. These schemes address both the non-delayed and delayed formulations. Our approach was guided by the requirement that the discrete dynamics reproduce the essential qualitative properties of the continuous system, such as positivity of solutions, boundedness, and the evolution law for the total population.

Our approach ensures the positivity of the susceptible and defaulted compartments. The recovered population is obtained from the discrete conservation principle, which prevents the occurrence of negative values that may arise in standard discretizations. A central outcome of our study is that unconditional positivity of the recovered compartment is not automatic in the discrete setting but follows from an exact survival matching principle. By choosing the denominator function $\phi(h) = (e^{\gamma h} - 1)/\gamma$, the positivity independently of the step size is guaranteed. The result highlights that structure-preserving discretization of delayed compartment models requires matching the intrinsic exponential survival mechanisms of the continuous system.

These explicit NSFD schemes are easy to implement and remain reliable for large time steps, making them ideal for long-time simulations and sensitivity analyses in systemic risk applications. From a methodological perspective, our results underscore the inherent challenge of preserving both transfer mechanisms and positivity in nonlinear models with delay. The NSFD methodology provides a flexible framework to overcome these obstacles by embedding qualitative information of the continuous dynamics directly into the discrete structure.

Future research may include extending the present techniques to more detailed financial networks, heterogeneous agent populations, or stochastic contagion mechanisms. Another area of investigation may be higher-order, structure-preserving discretizations, cf. [12].

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Appendix A. Positivity of the Systems

Theorem 3 (Invariant region, positivity and boundedness)**.** *Consider the delayed system* (1) *with positive parameters,* $\tau \geq 0$*. Let the initial history satisfy* $S(t), I(t), R(t) \geq 0$*,* $t \in [-\tau, 0]$*.*
Then:

(i) The solution exists globally and remains nonnegative: $S(t), I(t), R(t) \geq 0$*, for all,* $t \geq 0$*.*

(ii) The region

$$\Omega := \left\{(S, I, R) \in \mathbb{R}^3_+ : S + I + R \leq \frac{B}{\gamma}\right\}$$

is positively invariant.

(iii) All solutions satisfy

$$0 \leq S(t), I(t), R(t) \leq \max\left\{S(0) + I(0) + R(0), \frac{B}{\gamma}\right\}.$$

Proof. The right-hand side of (1) is locally Lipschitz in $(S(t), I(t), R(t), I(t-\tau))$, hence local existence follows from standard DDE theory (see Hale and Lunel [10, Sect. 2, Thm. 2.3]).

In order to show the positivity of the solutions, we show that the vector field is quasi-positive: whenever one component vanishes and the others are nonnegative, its derivative is nonnegative. Hence the nonnegative cone is positively invariant Thus solutions remain nonnegative.

We want to thoroughly investigate this and step through the three compartments. We will prove the invariance of the nonnegative cone by contradiction.

1. **Positivity of $I(t)$.**
   Suppose $I(t)$ attains the value zero at a first time $t_0 > 0$. Then $I(t_0) = 0$ and $I(t) \geq 0$ for $t < t_0$. Evaluating the equation at t_0 gives

   $$\dot{I}(t_0) = acS(t_0)\,\frac{I(t_0 - \tau)}{1 + \alpha I(t_0 - \tau)} \geq 0,$$

   since $S(t_0) \geq 0$ and $I(t_0 - \tau) \geq 0$. Hence $I(t)$ cannot cross into negative values. Therefore $I(t) \geq 0$ for all $t \geq 0$.
2. **Positivity of $S(t)$.**
   Assume $S(t)$ reaches zero for the first time at $t_0 > 0$. Then $S(t_0) = 0$ and

   $$\dot{S}(t_0) = B + \delta I(t_0 - \tau)e^{-\mu\tau} > 0,$$

   since $B > 0$ and $I(t_0 - \tau) \geq 0$. Hence $S(t)$ cannot become negative, and thus $S(t) \geq 0$ for all $t \geq 0$.
3. **Step 3: Positivity of $R(t)$.**
   The R-equation is linear in R:

   $$\dot{R}(t) + \gamma R(t) = \delta I(t) - \delta I(t - \tau)\,e^{-\mu\tau}.$$

   Multiplying by the integrating factor $e^{\gamma t}$ yields

   $$\frac{d}{dt}\big(e^{\gamma t}R(t)\big) = e^{\gamma t}\big(\delta I(t) - \delta I(t - \tau)\,e^{-\mu\tau}\big).$$

   Integrating from 0 to t gives

   $$R(t) = e^{-\gamma t}R(0) + \delta \int_0^t e^{-\gamma(t-s)}\big(I(s) - I(s - \tau)\,e^{-\mu\tau}\big)\,ds.$$

   Since $I(s) \geq 0$ and $I(s - \tau) \geq 0$, the solution remains well defined for all t. Moreover, the equation is linear in R with nonnegative initial data; hence $R(t)$ cannot cross into negative values. Therefore $R(t) \geq 0$ for all $t \geq 0$.

For the recovered compartment, we can also give a *cohort interpretation*: at a first hitting time of zero we have $R(t_0) = 0$ and $R(t) > 0$ for $t < t_0$. At that instant, the model structure ensures the delayed term corresponds to individuals who entered R at time $t_0 - \tau$. Those individuals cannot exceed the mass that is currently present in R.

Combining the three steps shows that $(S(t), I(t), R(t))$ remains in $\mathbb{R}^3_+$ for all $t \geq 0$.

Next, we investigate the boundedness of solutions. Consider the total population $N(t) = S(t) + I(t) + R(t)$. Summing the equations yields (2), since the infection and delayed transition terms cancel exactly. The explicit solution is, cf. (3),

$$N(t) = N(0)\,e^{-\gamma t} + \frac{B}{\gamma}(1 - e^{-\gamma t}).$$

Hence,

$$0 \leq N(t) \leq \max\Big\{N(0), \frac{B}{\gamma}\Big\}.$$

Therefore Ω is positively invariant. □

Corollary 1 (Positivity and invariant region for the NSFD scheme)**.** *Consider the NSFD discretization (44) constructed with a positive denominator function $\phi(h)$ given by (34) and nonlocal approximations preserving the exact cancellation structure in the total population equation. Assume $S^n, I^n, R^n \geq 0$ for $n = 0, \ldots, m$, where m corresponds to the delay index, i.e. $mh = \tau$.*

Then for all $n \geq 0$:

(i) Positivity: $S^n, I^n, R^n \geq 0$.

(ii) Discrete total population equation:

$$\frac{N^{n+1} - N^n}{\phi(h)} = B - \gamma N^{n+1}, \quad N^n = S^n + I^n + R^n.$$

(iii) Invariant region: $0 \leq N^n \leq \max\{N^0, \frac{B}{\gamma}\}$, $n \geq 0$.

(iv) The discrete analogue of Ω, namely $\Omega_h := \{(S, I, R) \in \mathbb{R}^3_+ : S + I + R \leq \frac{B}{\gamma}\}$, is positively invariant for every step size $h > 0$.

Proof. By construction of the NSFD scheme (45):

1. Each equation is written in a nonstandard form where negative contributions are evaluated at level $n + 1$, yielding

$$S^{n+1} = \frac{S^n + \phi(h) \cdot (\text{nonnegative terms})}{1 + \phi(h)\gamma + \cdots},$$

and similarly for I^{n+1}. Hence positivity holds for any $h > 0$. Only for R^{n+1} the positivity in (44) is less obvious since the update formula contains a delayed subtraction term. It will hold if one assumes $I^{n-m} e^{-\mu m h} \leq I^{n+1}$, $m \geq 0$, which is plausible as a *discrete cohort interpretation*: at time t_{n+1-m}, a cohort enters the compartment R via δI^{n+1-m} and after m steps, a fraction $e^{\mu\tau}$ leaves. That cohort must still be part of R^n, so the delayed subtraction cannot exceed the stored mass.

Nevertheless, we will prove the positivity of R^n in the discrete setting by mimicking the continuous boundary (barrier) argument of Theorem 3. This requires proving that R^n is a discrete weighted sum of past infected states, rather than merely manipulating the update formula (45). The key question is whether we can derive a representation of R^n as a weighted sum of past infected states. If so, positivity would follow immediately. The R-equation is a linear, nonhomogeneous recurrence with delay forcing that can be solved by iteration.

Consider the discrete recovered equation from the NSFD scheme (45), with $m = \tau/h$:

$$\big(1 + \phi(h)\gamma\big) R^{n+1} = R^n + \phi(h)\delta I^{n+1} - \phi(h)\delta I^{n+1-m} e^{-\mu\tau}, \tag{A.1}$$

Writing $\eta = \frac{1}{1+\phi(h)\gamma}$, the linear recurrence with delayed forcing (A.1) becomes

$$R^{n+1} = \eta R^n + \phi(h)\delta\eta\big(I^{n+1} - I^{n+1-m} e^{-\mu\tau}\big).$$

Iterating yields the explicit representation

$$R^n = \eta^n R^0 + \phi(h)\delta \sum_{j=0}^{n-1} \eta^{n-1-j} \big(I^{j+1} - I^{j+1-m} e^{-\mu\tau}\big). \tag{A.2}$$

The key structural observation is that positivity is guaranteed if the discrete decay factor over one delay interval matches the continuous survival factor, i.e.,

$$\eta^m = e^{-\gamma\tau}.$$

This condition holds if and only if the denominator function is chosen as

$$\phi(h) = \frac{e^{\gamma h} - 1}{\gamma}. \tag{A.3}$$

which is exactly our choice of a Mickens-type denominator function (34). Thus the discrete decay of each cohort entering R reproduces exactly the continuous exponential survival. The delayed subtraction term in (A.1) then removes precisely those individuals that have survived m discrete steps, and no more.

2. Summing the three discrete equations of (44) preserves the exact cancellation of infection and delayed transfer terms, yielding

$$\frac{N^{n+1} - N^n}{\phi(h)} = B - \gamma N^{n+1}.$$

3. Solving this linear recursion above gives

$$N^{n+1} = \frac{N^n + \phi(h)B}{1 + \phi(h)\gamma},$$

which implies

$$0 \le N^n \le \max\Big\{N^0, \frac{B}{\gamma}\Big\}.$$

Thus Ω_h is invariant independently of h. □

We obtained the structural condition "exact survival matching implies unconditional positivity". The positivity of the R-compartment in the NSFD scheme (44) is guaranteed provided the denominator function reproduces the exact exponential survival factor, i.e. $\phi(h) = (e^{\gamma h} - 1)/\gamma$. This Corollary 1 shows that unconditional positivity in the delayed case is not automatic but follows from a structurally consistent choice of the denominator function.

The analogous theorem for the extended system (4) and the corresponding corollary for the NSFD scheme (46) follow exactly the same arguments.