

University of Naples Federico II

Ph.D. Programme in Mathematics and Applications

XXXVIII Cycle

Ph.D. Thesis

**Numerical Analysis of Integral Epidemic Models
Incorporating Human Behaviour**

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A.A. 2024–2025

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Preface

This thesis is the result of the research I have carried out during my Ph.D. studies in Mathematics and Applications at University of Naples Federico II, between 2022 and 2025. Over the past three years, my work has focused on numerical solution of integral models with applications in epidemiology. Specifically, the aim of my research has been twofold: on the one hand, the development of epidemic models that incorporate behavioural feedback mechanisms, reflecting changes in individual behaviour in response to an ongoing epidemic, together with their formulation and mathematical analysis; and on the other hand, the design of numerical methods intended to accurately preserve the dynamical properties of the corresponding continuous systems.

The thesis is based on a selection of the works I developed during this period. In particular, it includes three published research papers and one manuscript currently in preparation.

The choice of including these works was motivated by their coherence with the overarching theme of the thesis and their contribution to addressing the research questions defined at the beginning of my doctoral journey. Some additional results obtained during this period, although not presented here in detail, have also contributed to shaping the overall perspective of my research.

I hope that the collection of studies presented in this thesis not only provides a consistent overview of my scientific contributions during these years but also offers a starting point for further investigations in this field.

Finally, I would like to acknowledge all the people and institutions that have supported my research during these three years, which made this work possible.

Claudia Panico
Naples, October 2025

Chapter 1

Introduction

1.1 Motivation

When solving a continuous mathematical model numerically, it is nowadays widely believed that convergence is no longer the only objective to be considered. Indeed, a robust numerical method should preserve the properties of the model, such as the positivity of the solutions and/or the conservativity of certain quantities, under weak or no conditions on the step-size [18, 30, 65]. In this way, the classical concept of numerical stability is generalised to include more general aspects such as the preservation of equilibria and their asymptotic stability. A matter of interest is that the numerical method could introduce spurious outcomes like the creation of ghost equilibrium points and change in the stability nature of equilibria, and would therefore fail to correctly represent the dynamics of the model at hand [66, 67]. The theory on *conservative* numerical methods is mostly applied to study the dynamics of differential equations [4, 35, 36, 37] and it is less developed in the case of evolution problems with memory [29, 45, 49]. Integral equations with memory are particularly well suited to modelling the temporal evolution of epidemics of infectious diseases, as they naturally capture cumulative effects over time.

The use of models in *Mathematical Epidemiology* (ME) dates back to the celebrated paper of Kermack and McKendrick, published in 1927, who introduced an integral renewal equation describing the epidemic spread in terms of individual infectiousness [38, 39]. Since this pioneering contribution, models of ME have become essential tools for understanding epidemic dynamics and for identifying optimal intervention and mitigation strategies. Many researchers have made substantial contributions to the development of integral epidemic models in the field of epidemiology. Applications of these models to actual diseases, such as influenza and Severe Acute Respiratory Syndrome (SARS), were provided by Roberts [59]; Roberts et al.

[60]. O. Diekmann and coworkers proposed mathematical formulations for a wide range of epidemic models that incorporate demography and non-permanent immunity as a scalar renewal equation for the *Force of Infection* (FoI), i.e. the rate at which a susceptible becomes infected [11]. In such a paper, the concept of *Infectivity Function* (IF) is introduced, which represents the expected contribution to the FoI by an individual infected since τ units of time. Comprehensive treatments of integral epidemic models can be found in [9, 21]. Kermack and McKendrick’s integral renewal equation reduces to compartmental models when the infectivity function takes specific forms [20, 23]. Compartmental models, such as the classical SIR (Susceptible–Infectious–Removed) and SEIR (Susceptible–Exposed–Infectious–Removed), are formulated as system of *Ordinary Differential Equations* (ODEs) and divide the individuals of a given population into mutually exclusive groups (the *compartments*) according to their disease status. Many noteworthy cases can be cited where compartmental models of ME have been applied to recent diseases and parametrised with official data. Clear examples are the recent COVID-19 pandemic [19, 31, 33, 57], the transmission of Middle East Respiratory Syndrome (MERS) in South Korea and Saudi Arabia [61], and the spread of the West Nile virus in Italy [32].

Both integral and compartmental models of ME do exhibit a notable limitation, however, that in the context of contemporary societies cannot be disregarded: they do not take into account the feedback effects produced by the behavioural change of individuals during an epidemic, such as increased social distancing, voluntary isolation, or the adoption of protective measures when perceived risk rises. Classical compartmental models have been revisited in recent years within the framework of a new field called *Behavioural Epidemiology of Infectious Disease* (BEID) [46, 70]. Among the possible approaches within the BEID, the one based on the so-called *information index* (i.e. an additional variable which describes the opinion-driven changes of behaviour of individuals) is particularly promising and increasingly used [3, 40, 43]. The first studies on compartmental epidemic models embedding the information index date back to 2007 with A. d’Onofrio and coworkers [27]. Generally speaking, researchers working on models of BEID focus on potential complex dynamics caused by the presence of behavioural effects. For example, it has been proven that the opinion-driven change of behaviour, modelled through the information index, can induce oscillatory dynamics that go uncaptured by analogous non-behavioural models [24, 27, 46, 70]. A key factor in the occurrence of oscillations is the type of *memory kernel* adopted (i.e. the function used to model how a population keeps memory of information and rumours regarding a disease [24, 26, 27]). It is worth mentioning that in several papers an *awareness variable*

is employed to represent behavioural responses due to information. Such a variable can be considered as a special case of the information index [41, 62, 73].

Motivated by the recent promising advances of BEID, we propose new formulations of integral *behavioural* epidemic models where the FoI incorporates the information index [17]. In this context, *Volterra Integral Equations* (VIEs), and the associated mathematical theory and numerical techniques, provide a powerful framework for both the formulation and the analysis of memory-dependent epidemic models [12, 13, 42, 56]. Their structure is especially appropriate for capturing transmission processes that rely on past states of the system, including individual infectiousness profiles and behavioural responses. Despite their theoretical appeal, however, numerical methods for Volterra-type epidemic models remain relatively underdeveloped. Standard schemes often fail to preserve properties such as positivity and boundedness of the solution, or asymptotic dynamics of the continuous model, thus limiting their practical applicability. This highlights the need for tailored numerical approaches specifically designed to preserve key structural and qualitative properties of the model.

According to this considerations, we build a numerical method based on non-standard discretizations of the nonlinear terms in the equations. This idea was originally introduced by Mickens in [54] for systems of ordinary differential equations. Only recently, the Mickens' approach has been extended to integral problems. Examples include the integral SIS model studied in [45] and an epidemic model for a closed population discussed in [51, 53]. Compared to standard numerical techniques such as collocation, direct quadrature, and Runge-Kutta methods [13, 42], *Non-Standard Finite Difference* (NSFD) methods offer simplicity of implementation and facilitate a priori analysis of quantitative and asymptotic properties of the numerical solution as a function of the discretization step h . NSFD methods are semi-implicit schemes characterized by a nonlocal discretization of nonlinear terms, tailored specifically to the model to preserve the positivity of the continuous solution. Moreover, the discrete system associated to the method can be viewed, for any fixed value of the step-size $h > 0$, as a discrete time model. It is possible to study the numerical equilibria and their stability by leveraging results on the linearization of discrete VIEs. The primary objective of this analysis is to establish the unconditional preservation of stability properties with respect to the step-size $h > 0$, as well as the recovery of the full dynamic behaviour of the continuous model as $h \rightarrow 0$.

1.2 Thesis organisation

The scientific work presented in this thesis is devoted to the study of integral models with applications in epidemiology. The focus lies on the development of epidemic models incorporating human behaviour and its numerical solution [15, 16, 17]. This study includes the formulation of such models, their mathematical analysis, and the development of numerical methods aimed at investigate the system's dynamics, providing accurate and efficient approximations of the solutions. The thesis is structured into four main chapters, followed by a concluding chapter.

Chapter 2. Numerical Methods for Volterra Integral Equations.

Volterra integral equations arise naturally in the mathematical modelling of processes involving memory, and are widely encountered in applications across physics, biology, epidemiology, engineering, and other scientific disciplines.

This chapter focuses on Volterra integral equations of the second kind, presenting their basic structure and properties. We begin by introducing the general formulation of the problem, followed by an overview of classical and modern approaches to their numerical resolution. Particular attention is given to equations of convolution type, which frequently appear in applications and admit simplified treatment through tailored numerical schemes.

The development of numerical methods is accompanied by an analysis of their accuracy. We discuss the notions of consistency, and convergence, and derive error bounds that allow for a rigorous assessment of the reliability of the approximations. These results are essential for understanding how the discrete solutions behave in relation to the exact continuous ones.

To handle nonlinear problems, we also present the linearization theory for continuous and discrete Volterra equations. This theoretical framework provides insight into the asymptotic behaviour of the numerical solutions and establishes connections between the stability properties of linear and nonlinear models.

Chapter 3. An Integral Behavioural Epidemic Model: Formulation and Mathematical Analysis.

In this chapter we formulate a new integral epidemic model that incorporates human behaviour [17]. It is formulated using Volterra-type

integral equations, allowing us to account for memory effects that influence disease transmission in a behaviourally driven context.

After introducing and describing the model, we examine some fundamental properties of its solutions, such as positivity, boundedness, and the existence of equilibria. Through the application of linearization theory for VIEs, we obtain the characteristic equation, which constitutes the foundation for the subsequent stability analysis. Building on this result, we further examine the local stability of the equilibria of the system.

Chapter 4. A Numerical Method for the Integral Behavioural Epidemic Model.

In this chapter, we describe in details the numerical method that we specially designed to deal with the integral epidemic model presented in Chapter 3. The method has been built with the aim to preserve, in the numerical solution, the relevant properties of the model. It is based on non-standard discretizations of the nonlinear terms introduced in Section 2.3. Moreover, for any fixed value of the step-size $h > 0$, the discrete system associated to the method can be viewed as a discrete time model [16].

We will approach the analysis of the discrete problem from two related perspectives. The first concerns the numerical method, focusing on the convergence of the discrete solution to the solution of the continuous model as $h \rightarrow 0$. The second perspective treats the discrete-time model itself, investigating positivity properties, the existence of equilibrium solutions, and the analysis of their asymptotic behaviour for any fixed $h > 0$. In particular, we perform a qualitative analysis based on linearization theory for discrete Volterra equations described in Chapter 2. When the discrete equilibria and their properties are related to those of the continuous model, this second aspect can also be regarded as a study of numerical stability. Another point of interest is the examination of the existence and behaviour of equilibrium solutions of the discrete model in the limit as the step-size $h \rightarrow 0$. This analysis, which represents an investigation into the approximation properties of numerical equilibria, allows us to deepen and extend the concept of convergence.

At the end, we present some numerical examples based on the non-

standard discretization of the model, with the aim of illustrating the theoretical results.

Chapter 5. Connections Between Compartmental and Integral Epidemic Models.

In Chapters 3 and 4, we examine an integral model that incorporates behavioural aspects of human populations, thereby enriching the classical epidemiological modelling framework. This integral formulation, however, should not be regarded as an entirely separate approach. On the contrary, it constitutes a natural generalisation of classical compartmental models, as it allows for the inclusion of general infectivity functions rather than being restricted to the exponential forms typically assumed in traditional models.

The goal of this chapter is twofold [15]. First, we aim to prove that the integral models introduced in the previous chapters are not conceived in isolation from established approaches. Instead, they are rooted in the well-known compartmental tradition and extend it by embedding more general assumptions on infectivity profiles. In particular, we show how suitable choices of the infectivity function and memory kernel allow for the recovery of classical compartmental structures, thereby illustrating the coherence and unifying nature of the integral approach.

Second, we introduce a novel model directly formulated in integral form, to further highlight the connection between integral and compartmental models. This new model incorporates incidence-dependent contact patterns and extends the ODE-based model available in literature. We analyze the asymptotic behaviour of the solutions and derive sufficient conditions for the stability of steady states, expressed in terms of the infectivity function and memory kernel. Numerical simulations are then presented to illustrate the impact of different memory kernels on the qualitative behaviour of the epidemic, under specific functional forms for infectivity.

Chapter 6. Conclusions.

A summary of the main results is provided together with possible directions for future research.

Chapter 2

Numerical Methods for Volterra Integral Equations

This chapter provides an introduction to Volterra Integral Equations (VIEs) of the second kind, presenting their basic structure, properties and their numerical resolution [12, 13, 42, 56]. The results presented here provide the foundation for the subsequent developments. The focus is placed on convolution-type equations, which play a central role in the epidemiological applications discussed later in the thesis. Particular attention is devoted to reviewing both standard and non-standard approaches to their numerical treatment.

2.1 Volterra integral equations of the second kind

An integral equation is a functional equation in which the unknown function occurs under one or several integral signs. Integral equations are typically classified based on the position of the unknown, the limits of integration, and the linearity of the kernel. A particularly important class of such equations is given by the Volterra integral equations, where the integration domain is variable, with the upper limit depending on the independent variable.

VIEs are generally classified into two principal categories, based on the position of the unknown function within the equation. The first category includes Volterra equations of the first kind, which take the form

$$\int_a^t K(t, s, y(s)) ds = f(t), \quad t \geq a, \quad (2.1)$$

where the unknown function $y(s)$ appears only under the integral sign. In this formulation, the goal is to determine $y(s)$ such that the integral equation is satisfied

for each t in the domain. Here, $K(t, s, u)$ is a known function referred to as the *kernel*, and $f(t)$ is a given function. To simplify notation, we can, without loss of generality, assume the lower limit of integration to be zero and focus on the equation

$$\int_0^t K(t, s, y(s)) ds = f(t), \quad t \geq 0, \quad (2.2)$$

an assumption that will be maintained throughout the remainder of the text.

The second category consists of Volterra equations of the second kind, typically written as

$$y(t) = f(t) + \int_0^t K(t, s, y(s)) ds, \quad t \geq 0, \quad (2.3)$$

in which the unknown function $y(t)$ appears both outside and inside the integral sign.

This classification naturally extends to systems of equations of dimension d , by treating y , K , and f as vector-valued functions. In this setting, equation (2.3) takes the form

$$\mathbf{y}(t) = \mathbf{f}(t) + \int_0^t \mathbf{K}(t, s, \mathbf{y}(s)) ds, \quad t \geq 0, \quad (2.4)$$

where

$$\mathbf{y}(t) = \begin{bmatrix} y_1(t) \\ y_2(t) \\ \vdots \\ y_d(t) \end{bmatrix} \quad \mathbf{f}(t) = \begin{bmatrix} f_1(t) \\ f_2(t) \\ \vdots \\ f_d(t) \end{bmatrix} \quad (2.5)$$

$$\mathbf{K}(t, s, \mathbf{y}(s)) = \begin{bmatrix} k_1(t, s, y_1(s), y_2(s), \dots, y_d(s)) \\ k_2(t, s, y_1(s), y_2(s), \dots, y_d(s)) \\ \vdots \\ k_d(t, s, y_1(s), y_2(s), \dots, y_d(s)) \end{bmatrix} \quad (2.6)$$

The focus of the present thesis will be on nonlinear Volterra integral equations of the second kind, as these will constitute the mathematical framework for the application-oriented developments that follow.

Let us consider equation (2.3), the following result prove the existence and uniqueness of the solution under the assumption that $K(t, s, y)$ satisfies a simple Lipschitz condition with respect to the third argument [42].

Theorem 2.1.1. *Assume that in equation (2.3) the functions $g(t)$ and $K(t, s, y)$ are continuous in $0 \leq s \leq t \leq T$ and $-\infty < y < \infty$ and exists a constant $L > 0$ such*

that

$$|K(t, s, u) - K(t, s, v)| \leq L|u - v|. \quad (2.7)$$

Then (2.3) has a unique continuous solution for all finite T .

In inequality (2.7), the symbol $|\cdot|$ stands for the absolute value. If we introduce the vector norm

$$\|\mathbf{y}(t)\| = \max_{1 \leq i \leq d} |y_i(t)|, \quad (2.8)$$

and the induced matrix norm

$$\|\mathbf{K}(t, s)\| = \max_{1 \leq i \leq d} \sum_{j=1}^d |K_{ij}(t, s)|,$$

then Theorem 2.1.1 can be easily extended to the system of equations (2.4), replacing absolute values with norms.

A wide class of integral equations encountered in applications involves kernels that depend solely on the difference of the variables, that is,

$$K(t, s) = K(t - s). \quad (2.9)$$

Integral equations of the form (2.3) with a kernel satisfying (2.9) are commonly referred to as *convolution equations*.

In the context of the applications addressed in this thesis, particular attention is devoted to systems of d nonlinear Volterra equations of convolution type, given by

$$\mathbf{y}(t) = \mathbf{f}(t) + \int_0^t \mathbf{K}(t - s) \mathbf{g}(\mathbf{y}(s)) ds, \quad t \geq 0, \quad (2.10)$$

where the kernel $\mathbf{K}$ is a $d \times d$ matrix and $\mathbf{g}$ is a nonlinear function of the unknown $\mathbf{y}$, which is assumed to satisfy the condition $\mathbf{g}(\mathbf{0}) = \mathbf{0}$. In many practical contexts, the analysis focuses on stability properties of the solution of system (2.10). A standard approach is to linearize the system around a given solution and then investigate the stability of the resulting linear problem. In [55], Miller developed a general linearization theory for equations of the form (2.10). His approach allows to replace the original local, nonlinear problem with a corresponding linear equation and a linear equation for its resolvent, both of which can then be analyzed using classical tools such as integral transforms. Since this constitutes a starting point for the study we will carry out in the following chapters, we present below the main results.

Let us consider the system of equations (2.10) and the linear system

$$\mathbf{x}(t) = \mathbf{f}(t) + \int_0^t \mathbf{K}(t - s) \mathbf{J} \mathbf{x}(s) ds, \quad t \geq 0, \quad (2.11)$$

where $\mathbf{x}$ is a d -vector, $\mathbf{J}$ is the Jacobian matrix $\mathbf{g}'(\mathbf{0}) = [\partial g_i(0)/\partial x_j]_{ij}$, $i, j = 1, \dots, d$, and the following assumptions are satisfied for $i, j = 1, \dots, d$:

$$[\mathbf{K}]_{ij} \in L^1(0, +\infty), \quad \mathbf{f}_i \in C[0, +\infty), \quad \mathbf{g} \in C^1(\mathbb{R}^n), \quad \mathbf{g}(\mathbf{0}) = \mathbf{0}, \quad \det \mathbf{J} \neq 0. \quad (2.12)$$

Since $\mathbf{J}$ is nonsingular, it is no loss of generality to assume $\mathbf{J}$ is the $d \times d$ identity matrix $\mathbb{I}$ and the linear system (2.11) can be rewritten as

$$\mathbf{x}(t) = \mathbf{f}(t) + \int_0^t \mathbf{K}(t-s) \mathbf{x}(s) ds, \quad t \geq 0. \quad (2.13)$$

System (2.13) admits the unique solution of the form

$$\mathbf{x}(t) = \mathbf{f}(t) - \int_0^t \mathbf{R}(t-s) \mathbf{f}(s) ds, \quad t \geq 0, \quad (2.14)$$

where the $d \times d$ matrix $\mathbf{R}$ is the resolvent kernel determined by the matrix equation

$$\mathbf{R}(t) = -\mathbf{K}(t) + \int_0^t \mathbf{R}(t-s) \mathbf{K}(s) ds, \quad t \geq 0. \quad (2.15)$$

The following result characterizes the integrability of the solution to equation (2.15).

Theorem 2.1.2. *Let $\mathbf{K} \in L^1(0, \infty)$. Then the solution $\mathbf{R}$ of equation (2.15) is $L^1(0, \infty)$ if and only if the determinant*

$$\det \left(\mathbb{I} - \int_0^\infty \mathbf{K}(t) e^{-wt} dt \right) \neq 0, \quad (2.16)$$

in the right half plane $\operatorname{Re}(w) \geq 0$.

Theorem 2.1.2 was proposed by Miller in [55] as a straightforward modification of the original theorem by Paley and Wiener [58], in which they analyze the asymptotic behaviour of solutions of (2.13) in case $\mathbf{f}(t) \rightarrow 0$ as $t \rightarrow \infty$.

In the next, we present some results that illustrate how the behaviour of the solution of the nonlinear system (2.10) relates to that of its linear counterpart (2.13). The details of proofs can be found in [55]. We denote by $\|\cdot\|_0$ the norm on the space of bounded continuous functions $BC[0, \infty)$, defined as

$$\|\mathbf{h}\|_0 = \sup_{0 \leq t < \infty} \|\mathbf{h}(t)\|, \quad (2.17)$$

where $\|\cdot\|$ denotes the norm in $\mathbb{R}^d$ introduced in (2.8).

Theorem 2.1.3. *If assumptions (2.12) are satisfied then there exists $\epsilon_0 > 0$ and $\epsilon_1 > 0$ such that when the solution $\mathbf{x}(t)$ of (2.13) satisfies $\|\mathbf{x}\|_0 \leq \epsilon_0$, the solution $\mathbf{y}(t)$ of (2.10) exists for all $t \geq 0$ and $\|\mathbf{y}\|_0 \leq \epsilon_1$.*

Corollary 2.1.1. *If (2.12) are satisfied, then there exist $\epsilon_1 > 0$ and $\epsilon_2 > 0$ such that when $\|\mathbf{f}\|_0 \leq \epsilon_2$, the solution $\mathbf{y}(t)$ of (2.10) exists for all $t \geq 0$ and satisfies $\|\mathbf{y}\|_0 \leq \epsilon_1$.*

Theorem 2.1.4. *Let (2.12) hold and let ϵ_0 and ϵ_1 be given by Theorem 2.1.3 above. If $\|\mathbf{x}\|_0 \leq \epsilon_0$ and $\mathbf{x}(t) \rightarrow 0$ as $t \rightarrow \infty$, then $\mathbf{y}(t) \rightarrow 0$ as $t \rightarrow \infty$.*

Corollary 2.1.2. *Let (2.12) hold and let ϵ_1 and ϵ_2 be given by Corollary 2.1.1 above. If $\|\mathbf{f}\|_0 \leq \epsilon_2$ and $\mathbf{x}(t) \rightarrow 0$ as $t \rightarrow \infty$, then $\mathbf{y}(t) \rightarrow 0$ as $t \rightarrow \infty$.*

Theorem 2.1.5. *Suppose (2.12) hold, and ϵ_2 is given by Corollary 2.1.1 above. If $\|\mathbf{f}\|_0 \leq \epsilon_2$ and $\mathbf{f}(t) \rightarrow 0$ as $t \rightarrow \infty$, then $\mathbf{y}(t) \rightarrow 0$ as $t \rightarrow \infty$.*

An extension of the Theorem 2.1.2 to the case where the matrix kernel does not belong to $L^1(0, \infty)$ is provided by Lubich [44]. Here, we report this result for the sake of completeness.

Theorem 2.1.6. *Consider the system of Volterra integral equations (2.13), where the matrix $\mathbf{K}(\cdot)$ and the function $\mathbf{f}(t)$ satisfy*

$$\mathbf{K}(\cdot) = \mathbf{K}_1(\cdot) + \mathbf{K}_\infty, \quad \mathbf{K}_\infty = \underbrace{\begin{bmatrix} \mathbf{K}_\infty^1 \\ 0 \end{bmatrix}}_d \begin{matrix} \} l_1 \\ \} l_2 \end{matrix}, \quad \mathbf{f}(t) = \begin{bmatrix} \mathbf{f}_0(t) \\ \mathbf{f}_1(t) \end{bmatrix} \begin{matrix} \} l_1 \\ \} l_2 \end{matrix}. \quad (2.18)$$

In (2.18), $\mathbf{K}_1(\cdot) \in L^1(0, \infty)$ and $\mathbf{K}_\infty$ is an arbitrary $d \times d$ constant matrix constituted of two block, a null block of dimension $l_2 = d - l_1$ and a constant block, denoted as $\mathbf{K}_\infty^1$. Then $\mathbf{x}(t) \rightarrow 0$, whenever $\mathbf{f}'_0(t), \mathbf{f}_1(t) \rightarrow 0$ for $t \rightarrow \infty$, if and only if the equation

$$w^{l_1} \det \left(\mathbb{I} - \int_0^\infty \mathbf{K}(t) e^{-wt} dt \right) = 0, \quad (2.19)$$

has no solution for $\operatorname{Re}(w) \geq 0$.

Remark 2.1.1. *For $\operatorname{Re}(w) = 0$, equation (2.19) must be interpreted as limit for $\tilde{w} \rightarrow w$, $\operatorname{Re}(\tilde{w}) > 0$.*

It follows from Theorem 2.1.6 that the resolvent kernel $\mathbf{R}$ defined in (2.15) belongs to $L^1(0, \infty)$ even when the kernel $\mathbf{K}$ does not. Consequently, all previous results remain valid for kernels $\mathbf{K}$ satisfying condition (2.18).

2.2 Newton-Cotes and Gregory type integration rules

In this section, we present numerical techniques for solving Volterra integral equations of the second kind [13, 42]. To this end, we focus on the following scalar equation

$$y(t) = f(t) + \int_0^t K(t, s, y(s)) ds, \quad 0 \leq t \leq T, \quad (2.20)$$

under the assumptions of Theorem 2.1.1. This setting will serve as the basis for all subsequent developments and analyzes in the chapter.

In order to compute an approximate solution to (2.20), we consider an uniform mesh $t_n = nh$, where $n = 0, 1, \dots, N$, $T = Nh$, and $h > 0$ denotes the step-size. Assume that the solution is known at the points $t_i = ih$, for $i = 0, 1, \dots, n-1$. An approximation of $y(t_n)$ can then be obtained by replacing the integral on the right-hand-side (r.h.s.) of (2.20) with a numerical quadrature rule, using the values of the integrand at t_i , $i = 0, 1, \dots, n$, and solving the resulting equation for $y(t_n)$. It follows that the specific form of the algorithm depends primarily on the choice of the integration rule. Let us assume that we have an integration rule of the form

$$\int_0^{nh} \varphi(t) dt \cong h \sum_{i=0}^n w_{ni} \varphi(t_i), \quad (2.21)$$

where $\varphi(t)$ denotes any continuous integrand, and the coefficients w_{ni} are referred to as the *integration weights*. By substituting the integral term in equation (2.20) with the quadrature rule provided in (2.21), the following numerical scheme is obtained, where $y_n \approx y(t_n)$:

$$y_n = f(t_n) + h \sum_{i=0}^n w_{ni} K(t_n, t_i, y_i), \quad r \leq n \leq N. \quad (2.22)$$

It follows that equation (2.22) is valid only for $n \geq r$, where r is a fixed integer. This condition reflects the requirement that higher-order integration formulas depend on a minimum number of previous points. As a result, the values $y_1, y_2, \dots, y_{r-1}$ cannot be computed directly using (2.22) and must instead be obtained through alternative means [42].

In the analysis that follows, we assume a constant step-size $h > 0$ and we consider two classes of integration rules: the Newton–Cotes and Gregory-type methods. Moreover, we demonstrate how these quadrature techniques lead to different numerical schemes for approximating the solution of equation (2.20), illustrating the

approach with selected examples.

We begin our analysis by considering the first numerical method arising from the application of the composite trapezoidal rule to the integral term. This leads to the following discretization scheme:

$$y_n = f(t_n) + h \left\{ \frac{1}{2} K(t_n, t_0, y_0) + \sum_{i=1}^{n-1} w_{ni} K(t_n, t_i, y_i) + \frac{1}{2} K(t_n, t_n, y_n) \right\}, \quad (2.23)$$

with $n = 1, 2, \dots, N$, and the initial condition is $y_0 = f(0)$. In equation (2.23), the coefficients w_{ni} represent the integration weights associated with the composite trapezoidal quadrature formula. Specifically, for all $n \geq 1$, they are given by $w_{n0} = w_{nn} = \frac{1}{2}$, and $w_{ni} = 1$ for $i = 1, \dots, n-1$. Equation (2.23) defines the unknown y_n implicitly. However, for sufficiently small values of h , the equation admits a unique solution. In the case where the kernel K is linear in y , this solution can be obtained explicitly. Conversely, when K is nonlinear, iterative schemes are typically employed to compute y_n to the desired accuracy.

Another important example within the class of Newton–Cotes integration rules is composite Simpson’s rule, which corresponds to a composite quadrature formula based on quadratic polynomial interpolation over equally spaced nodes. We now apply the composite Simpson’s rule to the integral in equation (2.20), which leads to a more accurate discretization, under the assumption that $[0, T]$ is split up into an even number of subintervals. The resulting numerical method takes the form:

$$y_n = f(t_n) + \frac{h}{3} \left\{ K(t_n, t_0, y_0) + 4 \sum_{\substack{i=1 \\ i \text{ odd}}}^{n-1} K(t_n, t_i, y_i) + 2 \sum_{\substack{i=2 \\ i \text{ even}}}^{n-2} K(t_n, t_i, y_i) + K(t_n, t_n, y_n) \right\}, \quad (2.24)$$

valid for $0 < n \leq N$. The integration weights w_{ni} associated with this scheme follow the classical Simpson pattern: $w_{n0} = w_{nn} = \frac{1}{3}$, $w_{ni} = \frac{4}{3}$ for odd indices $i = 1, 3, \dots, n-1$, and $w_{ni} = \frac{2}{3}$ for even indices $i = 2, 4, \dots, n-2$. These weights are derived from the interpolation of the integrand by a second-degree polynomial on each subinterval. As in the trapezoidal case, the presence of y_n on the r.h.s. implies an implicit formulation, which may require iteration if the kernel K is nonlinear in its third argument.

In the case of odd number of subintervals, one can combine different quadrature

rules by applying the *three-eighths rule* to the initial four nodes and employing Simpson's rule over the remaining portion of the interval. Specifically, when the three-eighths rule is applied to the points t_0, t_1, t_2, t_3 , the resulting weights for $n \geq 2$ are given by:

$$\begin{aligned} w_{n0} &= \frac{3}{8}, & w_{n1} &= w_{n2} = \frac{9}{8}, & w_{n3} &= \frac{17}{24} - \frac{1}{3}\delta_{n3}, \\ w_{n,2i} &= \frac{4}{3}, & & \text{for } i = 2, 3, \dots, (n-1)/2, \\ w_{n,2i+1} &= \frac{2}{3}, & & \text{for } i = 2, 3, \dots, (n-3)/2, \\ w_{nn} &= \frac{1}{3}, & & \text{for } n \geq 5, \end{aligned} \tag{2.25}$$

where δ_{ij} denotes the Kronecker delta, defined as $\delta_{ij} = 1$ if $i = j$ and $\delta_{ij} = 0$ otherwise.

Among the integration rules of the form (2.21), the Gregory formulas constitute an important class that leads to numerical methods for solving equation (2.20). Gregory's quadrature method provides a systematic way to improve the accuracy of numerical integration by introducing a sequence of end corrections. As an example, the Gregory formula of order four is characterized by the following set of weights, valid for $n \geq 5$:

$$\begin{aligned} w_{n0} = w_{nn} &= \frac{3}{8}, & w_{n1} = w_{n,n-1} &= \frac{7}{6}, & w_{n2} = w_{n,n-2} &= \frac{23}{24}, \\ w_{ni} &= 1, & & \text{for } i = 3, 4, \dots, n-3. \end{aligned} \tag{2.26}$$

When the numerical scheme in (2.22) is implemented using the weights in (2.26) with $r = 5$, the resulting method is referred to as the fourth-order Gregory method for the Volterra integral equation (2.20). The requirement $r = 5$ reflects the fact that at least six grid points are necessary for the weight structure in (2.26) to be applicable. To initiate the method, the values y_1, y_2, y_3, y_4 must be provided, while y_0 is conventionally set as $y_0 = f(0)$.

By adopting this strategy, one can derive various schemes for the numerical approximation of equation (2.20). Establishing their validity and evaluating their comparative efficiency necessitates a rigorous investigation of their error behaviour. This analysis will be the focus of the next section, where a more detailed error analysis will be presented.

2.3 Non-standard finite difference methods

Non-Standard Finite Difference (NSFD) methods, originally introduced and developed by Mickens in [54], aim to construct discrete schemes that better preserve the qualitative features of the differential equations. Unlike standard schemes, which may fail to maintain properties such as positivity, boundedness, or conservation laws, NSFD methods are designed to overcome these limitations. In the following, the formulation of non-standard finite difference methods is first examined in the context of ordinary differential equations, in order to clarify their underlying structure and guiding principles, and then extended to integral problems.

2.3.1 NSFD methods for ODEs: an introduction

Consider a generic first-order ODE of the form

$$\frac{dy}{dt} = f(y, t), \quad y(0) = y_0. \quad (2.27)$$

A standard explicit Euler discretization takes the form

$$y_{n+1} = y_n + hf(y_n, t_n), \quad n = 0, 1, \dots, N-1, \quad (2.28)$$

where $h > 0$ is the step-size. While this method is simple and widely used, it may not preserve critical properties of the continuous solution, particularly for nonlinear or stiff equations. Mickens proposed a modified approach, introducing a non-trivial discretization of the derivative and nonlinear terms:

$$\frac{y_{n+1} - y_n}{\phi(h)} = \tilde{f}(y_n, y_{n+1}, t_n, t_{n+1}), \quad n = 0, 1, \dots, N-1, \quad (2.29)$$

where the denominator function $\phi(h)$ is a non-standard function of the step-size h , satisfying the property

$$\phi(h) = h + \mathcal{O}(h^2), \quad (2.30)$$

where the term $\mathcal{O}(h^2)$ indicates that there exists a constant $C > 0$, independent of h , such that

$$|\phi(h) - h| \leq C h^2,$$

for sufficiently small h . The big- $\mathcal{O}$ notation is commonly used to describe the asymptotic behaviour of functions.

The left-hand-side (l.h.s.) of (2.29) represents the discrete derivative and it is obtained from a generalization of the traditional definition of derivative:

$$\frac{dy}{dt} = \lim_{h \rightarrow 0} \frac{y(t + \psi_1(h)) - y(t)}{\psi_2(h)},$$

where $\psi_i(h) = h + \mathcal{O}(h^2)$ for $i = 1, 2$. A classical example of function $\phi(h)$ that satisfy this condition is

$$\phi(h) = \frac{1 - e^{-ch}}{c}, \quad c \neq 0, \quad (2.31)$$

which guarantees positivity preservation for specific models (e.g., population dynamics).

In the r.h.s. of (2.29), $\tilde{f}$ is a nonlocal approximation of f that may depend on multiple time levels or nonlinear terms treated implicitly. Moreover, Mickens established that nonlinear terms involving products or rational expressions should not be discretized using standard approximations, i.e. $xy \mapsto x_n y_n$, but rather by carefully constructed nonlocal forms, often involving mixed time levels, e.g. $xy \mapsto x_n y_{n+1}$.

Remark 2.3.1. *NSFD methods are designed to satisfy several important qualitative properties of the original continuous system. First, they ensure positivity, meaning that if the continuous system guarantees $y(t) > 0$ for all $t \geq 0$, then the discrete solution should also satisfy $y_n > 0$ for all $n \geq 0$. Second, they preserve boundedness, reproducing the upper and lower bounds of the continuous solution. Third, they maintain dynamic consistency, so that the equilibrium points and their stability properties in the discrete system match those of the continuous one. Finally, they are designed to capture the long-time behaviour of the system, such as the correct asymptotic limits and steady states.*

2.3.2 NSFD method for Volterra integral equations

The methodology of NSFD schemes can also be extended to Volterra integral equations. In particular, consider a Volterra integral equation of the second kind, as given in (2.3), and its discretization (2.22), which was discussed in detail in the previous section. For convenience, we recall it here:

$$y_n = f(t_n) + h \sum_{i=0}^n w_{ni} K(t_n, t_i, y_i), \quad r \leq n \leq N. \quad (2.32)$$

To construct an NSFD scheme, the step-size h in (2.32) is substituted with a non-standard mesh function $\phi(h)$, specifically designed to reflect the features of the problem under consideration. In addition, the kernel approximation is modified so as to incorporate nonlocal effects:

$$y_n = f(t_n) + \phi(h) \sum_{i=0}^n w_{ni} \tilde{K}(t_n, t_i, y_i, y_{i-1}), \quad r \leq n \leq N, \quad (2.33)$$

where $\tilde{K}$ includes implicit or averaged approximations of $y(s)$ to preserve positivity or other features. Such an approach ensures that the numerical approximation to the integral equation reflects the structure and constraints of the original problem.

Mickens' NSFD methodology has found broad applications in epidemiological models, population dynamics, chemical kinetics, and reaction-diffusion systems, and its versatility suggests potential relevance in many other areas of mathematical modelling.

2.4 Convergence of the approximate solution

The numerical methods introduced in the previous sections yield approximate solutions exclusively at the points t_i . Consequently, any error analysis is inherently limited to these points. To formalize this, we define the sequence

$$\varepsilon_n = y_n - y(t_n), \quad n = 0, 1, 2, \dots, N, \quad (2.34)$$

which we refer to as the *discretization error*. Our primary interest lies in examining how this error behaves as a function of the step-size h .

For a fixed step-size h , the interval $[0, T]$ is partitioned into N subintervals such that $Nh = T$. Consequently, as $h \rightarrow 0$, the number of nodes N tends to infinity in a manner that preserves the identity $Nh = T$. This implies that h must belong to a specific set of admissible values, those satisfying $h \leq T$ and for which $T/h \in \mathbb{N}$. Since this limiting process will recur frequently in the upcoming analysis, we adopt the simplified notation $\lim_h$ to denote limits taken under the constraint $Nh = T$ as $h \rightarrow 0$.

Definition 2.4.1. *A method of the form (2.22) is said to be a convergent approximation method for equation (2.20) if*

$$\lim_h \left(\max_{0 \leq n < N} |\varepsilon_n| \right) = 0. \quad (2.35)$$

Definition 2.4.2. *If, for all admissible h , there exists a number $M < \infty$, independent of h , such that*

$$\max_{0 \leq n < N} |\varepsilon_n| \leq Mh^p, \quad (2.36)$$

and if p is the largest number for which such an inequality holds, then p is called the

order of convergence of the method.

Definition 2.4.3. Let y be the solution of (2.20). Then the function

$$\delta(h, t_n) = \int_0^{t_n} K(t_n, s, y(s)) ds - h \sum_{i=0}^n w_{ni} K(t_n, t_i, y(t_i)) \quad (2.37)$$

is the local consistency error for (2.20).

Specifically, local consistency error reflects the discrepancy introduced when the continuous integral operator is replaced by its discrete counterpart, independently of any accumulation of errors over successive steps. In the context of Volterra integral equations, the local consistency error serves as a fundamental indicator of the intrinsic accuracy of the quadrature rule employed. A precise characterization of this error provides insight into the overall convergence properties of the method. Therefore, before analysing the global error behaviour, it is crucial to establish bounds on the local consistency error and understand how it scales with h .

Definition 2.4.4. Let $\mathcal{C}$ be a class of equations of the form (2.20). If for every equation in $\mathcal{C}$

$$\lim_h \max_{0 \leq n \leq N} |\delta(h, t_n)| = 0, \quad (2.38)$$

then the approximation method (2.22) is said to be consistent with (2.20) for the class of equations $\mathcal{C}$. If for every equation in $\mathcal{C}$, there exists a constant c , independent of h , such that

$$\max_{0 \leq n \leq N} |\delta(h, t_n)| \leq ch^p, \quad (2.39)$$

then the method is said to be consistent of order p in $\mathcal{C}$.

The next result provides a crucial tool for the subsequent convergence theorem.

Proposition 2.4.1. Let the sequence $\xi_0, \xi_1, \dots$ satisfy

$$|\xi_n| \leq A \sum_{i=0}^{n-1} |\xi_i| + B_n, \quad n = r, r+1, \dots, \quad (2.40)$$

where

$$A > 0, \quad |B_n| \leq B, \quad \sum_{i=0}^{r-1} |\xi_i| \leq \eta. \quad (2.41)$$

Then

$$|\xi_n| \leq (1 + A)^{n-r} (B + A\eta), \quad \eta = r, r+1, \dots \quad (2.42)$$

Proof. We prove the result by induction on $n \geq r$. Let us define

$$\zeta_n := \max_{r \leq k \leq n} |\xi_k| \quad \text{for } n \geq r.$$

We start by considering the base case: for $n = r$, we have

$$|\xi_r| \leq A \sum_{i=0}^{r-1} |\xi_i| + B_r \leq A\eta + B.$$

This confirms that the result holds for $n = r$, completing the base case of the induction. Suppose now that for some $n \geq r$, the inequality

$$|\xi_k| \leq (1 + A)^{k-r}(B + A\eta), \quad \text{for all } r \leq k \leq n,$$

holds. We will show that it also holds for $k = n + 1$. From (2.40), we have:

$$|\xi_{n+1}| \leq A \sum_{i=0}^n |\xi_i| + B_{n+1}.$$

Split the sum:

$$\sum_{i=0}^n |\xi_i| = \sum_{i=0}^{r-1} |\xi_i| + \sum_{i=r}^n |\xi_i| \leq \eta + \sum_{i=r}^n |\xi_i|.$$

Applying the induction hypothesis for each $i \in [r, n]$, it follows that

$$|\xi_i| \leq (1 + A)^{i-r}(B + A\eta),$$

which allows us to estimate the sum as:

$$\sum_{i=r}^n |\xi_i| \leq (B + A\eta) \sum_{i=0}^{n-r} (1 + A)^i = (B + A\eta) \frac{(1 + A)^{n-r+1} - 1}{A}.$$

Thus,

$$\begin{aligned} |\xi_{n+1}| &\leq A \left(\eta + \frac{(B + A\eta)((1 + A)^{n-r+1} - 1)}{A} \right) + B \\ &= A\eta + (B + A\eta)((1 + A)^{n-r+1} - 1) + B \\ &= (B + A\eta)(1 + A)^{n-r+1}. \end{aligned}$$

This completes the induction, and the bound (2.42) follows. $\square$

Theorem 2.4.2. *Consider the approximate solution of (2.20) by (2.22), and assume that the following conditions hold:*

1. *The solution $y(t)$ of (2.20) and the kernel $K(s, t, y)$ are such that the approximation method is consistent of order p with (2.20).*

2. The weights satisfy

$$\sup_{n,i} |w_{ni}| \leq W < \infty.$$

3. The starting errors $y_i - y(t_i)$, for $i = 0, 1, \dots, r-1$, vanish as $h \rightarrow 0$. Since r is fixed, this implies that

$$\lim_{h \rightarrow 0} \sum_{i=0}^{r-1} |y_i - y(t_i)| = 0.$$

Then the method is a convergent approximation method. Moreover, in the absence of starting errors, the order of convergence is at least p .

Proof. Evaluating equation (2.20) at $t = t_n$ and subtracting it from the numerical method (2.22), we obtain, for $n = r, r+1, \dots$,

$$\varepsilon_n = h \sum_{i=0}^n w_{ni} \{K(t_n, t_i, y_i) - K(t_n, t_i, y(t_i))\} - \delta(h, t_n),$$

where ε_n denotes the discretization error and $\delta(h, t_n)$ is the local consistency error.

Invoking the Lipschitz condition (2.7) on the kernel k in its third argument, and using assumption 2, we get:

$$\begin{aligned} |\varepsilon_n| &\leq hL \sum_{i=0}^n |w_{ni}| |y_i - y(t_i)| + |\delta(h, t_n)| \\ &\leq hLW \sum_{i=0}^n |\varepsilon_i| + |\delta(h, t_n)|. \end{aligned} \tag{2.43}$$

To express this in a recursive form, we isolate the term $|\varepsilon_n|$ by removing it from the summation on the r.h.s., yielding:

$$|\varepsilon_n| \leq hLW \sum_{i=0}^{n-1} |\varepsilon_i| + hLW |\varepsilon_n| + |\delta(h, t_n)|.$$

Rearranging terms, and assuming $h < \frac{1}{LW}$ so that $1 - hLW > 0$, we derive:

$$|\varepsilon_n| \leq \frac{hLW}{1 - hLW} \sum_{i=0}^{n-1} |\varepsilon_i| + \frac{|\delta(h, t_n)|}{1 - hLW}.$$

Now, applying Proposition 2.4.1 to this recursive inequality yields the following estimate:

$$|\varepsilon_n| \leq \frac{1}{1 - hLW} \left[\max_{r \leq i \leq n} |\delta(h, t_i)| + hLW \sum_{i=0}^{r-1} |y_i - y(t_i)| \right] e^{\frac{WLt_n}{1 - hLW}}, \tag{2.44}$$

where the factor $(1 + A)^{n-r}$ in inequality (2.42) is estimated via the inequality $(1 + \alpha)^m \leq e^{\alpha m}$, yielding:

$$\left(1 + \frac{hLW}{1 - hLW}\right)^{n-r} \leq \exp\left(\frac{LW t_n}{1 - hLW}\right).$$

Due to the assumptions of the theorem, both the starting errors $|y_i - y(t_i)|$ and the consistency errors $\delta(h, t_i)$ in equation (2.44) tend to zero as $h \rightarrow 0$. Consequently, we obtain

$$\lim_h |\varepsilon_n| = 0,$$

which establishes convergence of the method. In the special case where no starting errors are present, inequality (2.44) simplifies to:

$$|\varepsilon_n| = \mathcal{O}\left(\max_{0 \leq i \leq n} |\delta(h, t_i)|\right),$$

which shows that the global error behaves as the maximum local consistency error, up to a constant factor. This concludes the proof. $\square$

An additional implication of (2.44) is that the influence of the initial errors is mitigated by a factor of h , indicating that these methods exhibit relative insensitivity to errors at the starting points. Specifically, it suffices to assume that

$$|y_i - y(t_i)| = \mathcal{O}(h^{p-1}), \quad i = 0, 1, \dots, r-1,$$

in order to guarantee that a method with consistency order p achieves a corresponding convergence order p .

2.5 Discrete stability analysis

In this section, we present the linearization theory for discrete Volterra equations, following the approach introduced by Song and Baker in [64]. In this paper, the authors employ fixed point theory to analyze nonlinear discrete Volterra equations, treating them as perturbations of corresponding linear systems.

This discrete setting reflects, in an analogous approach, the core ideas introduced in Section 2.1 for the continuous case. Since the continuous analysis was carried out in a vector-valued framework, we adopt the same perspective in the discrete case, providing a foundation for studying the asymptotic behaviour of solutions to the following nonlinear system in $\mathbb{R}^d$

$$\mathbf{y}_n = \mathbf{f}_n + \sum_{j=0}^n \mathbf{K}_{n-j}[\mathbf{y}_j + \mathbf{G}_j(\mathbf{y}_j)], \quad n = 0, 1, \dots, \quad (2.45)$$

where the sequence of matrices $\mathbf{K}_n \in \mathbb{R}^{d \times d}$, $n \geq 0$, incorporates the step-size and integration weights of the discretization scheme, and each function $\mathbf{G}_j(\cdot)$ is Lipschitz continuous, capturing the nonlinear contributions of the original equation (2.22). Additionally, it is assumed that $\mathbf{G}_j(\mathbf{0}) = \mathbf{0}$ for all $j \geq 0$.

To investigate the asymptotic behaviour of the solutions to (2.45), we consider the corresponding linear system:

$$\mathbf{x}_n = \mathbf{f}_n + \sum_{j=0}^n \mathbf{K}_{n-j} \mathbf{x}_j, \quad n = 0, 1, \dots \quad (2.46)$$

Assume that $\det(\mathbb{I} - \mathbf{K}_n) \neq 0$, $\forall n \geq 0$, then the unique solution of the system (2.46) is given by

$$\mathbf{x}_n = \mathbf{f}_n - \sum_{j=0}^n \mathbf{R}_{n-j} \mathbf{f}_j, \quad n \geq 0, \quad (2.47)$$

where $\{\mathbf{R}_n\}_n$ are the resolvent matrices of the kernel $\{\mathbf{K}_n\}_n$, such that

$$\mathbf{R}_{n-m} = \sum_{j=m}^n \mathbf{R}_{n-j} \mathbf{K}_{j-m} - \mathbf{K}_{n-m}, \quad 0 \leq m \leq n. \quad (2.48)$$

The following results represent the basis for studying the stability properties of the solution to the system (2.45). More details about the proof can be found in [64].

Theorem 2.5.1. *Let $\{\mathbf{y}_n\}_n$ be the solution of (2.45) and $\{\mathbf{x}_n\}_n$ the solution of (2.46) given by (2.47). Then*

$$\mathbf{y}_n = \mathbf{x}_n - \sum_{j=0}^n \mathbf{R}_{n-j} \mathbf{G}_j(\mathbf{x}_j), \quad \forall n \geq 0.$$

Theorem 2.5.2. *Assume that*

$$\begin{aligned} \sup_{n \geq 0} \|\mathbf{x}_n\| &< \infty, \quad \lim_{n \rightarrow \infty} \mathbf{x}_n = 0, \\ \sum_{n=0}^{\infty} \|\mathbf{R}_n\| &< \infty, \quad \lim_{n \rightarrow \infty} \|\mathbf{R}_n\| = 0, \end{aligned}$$

where $\{\mathbf{x}_n\}_n$ is the solution to (2.46) and $\{\mathbf{R}_n\}_n$ is defined in (2.48). Then the solution $\{\mathbf{y}_n\}_n$ of (2.45) also satisfies $\lim_{n \rightarrow \infty} \mathbf{y}_n = 0$.

The next result, originally proposed by Lubich in [44], serves as the discrete

analogue of Theorem 2.1.6, presented in Section 2.1.

Theorem 2.5.3. *Consider the discrete system of Volterra equations*

$$\mathbf{x}_n = \mathbf{f}_n + \sum_{j=0}^n \mathbf{K}_{n-j} \mathbf{x}_j, \quad (2.49)$$

for $n = 0, 1, \dots$, where $\sum_{n=0}^{\infty} \|\mathbf{K}_n - \bar{\mathbf{K}}_{\infty}\| < \infty$ for some $\bar{\mathbf{K}}_{\infty}$. $\bar{\mathbf{K}}_{\infty}$ and $\mathbf{f}_n$ can be written as

$$\bar{\mathbf{K}}_{\infty} = \underbrace{\begin{bmatrix} \bar{\mathbf{K}}_{\infty}^1 \\ \mathbf{0} \end{bmatrix}}_d \begin{matrix} \} l_1 \\ \} l_2 \end{matrix}, \quad \mathbf{f}_n = \begin{bmatrix} \mathbf{f}_n^0 \\ \mathbf{f}_n^1 \end{bmatrix} \begin{matrix} \} l_1 \\ \} l_2 \end{matrix}, \quad (2.50)$$

where $\bar{\mathbf{K}}_{\infty}$ is an arbitrary constant matrix constituted of two block, a null block and a constant block, denoted as $\bar{\mathbf{K}}_{\infty}^1$.

Then

$$\mathbf{x}_n \longrightarrow 0, \quad \text{whenever} \quad \mathbf{f}_n^1, \Delta \mathbf{f}_n^0 \longrightarrow 0 \quad (n \rightarrow \infty),$$

if and only if

$$(1 - z)^{l_1} \det \left(\mathbb{I} - \sum_{n=0}^{\infty} \mathbf{K}_n z^n \right) = 0 \quad (2.51)$$

has no solution for $|z| \leq 1$.

For $|z| = 1$, (2.51) has been interpreted as limit for $\tilde{z} \rightarrow z$, $|\tilde{z}| < 1$.

Chapter 3

An Integral Behavioural Epidemic Model: Formulation and Mathematical Analysis

This chapter is devoted to the introduction of an integral epidemic model that incorporates human behavioural responses described in [17]. We present the model in detail, describe the functions involved, and highlight the novelties of this formulation arising from the inclusion of the information index. Furthermore, we analyze the properties of the solutions, establish the existence of equilibria, and investigate their local asymptotic stability.

3.1 The model

In the formulation of the Kermack and McKendrick's integral renewal equation [11], the FoI at time t , denoted by $\hat{F}(t)$, is introduced through the following constitutive equation:

$$\hat{F}(t) = \int_0^\infty S(t - \tau) \hat{F}(t - \tau) A_\mu(\tau) d\tau. \quad (3.1)$$

Here, S denotes the size of the susceptible compartment; $A_\mu(\tau) = e^{-\mu\tau} A(\tau)$, where $A(\tau)$ is the Infectivity Function, that is the expected contribution to the Force of Infection (FoI) by an individual that was itself infected τ units of time ago; the positive parameter μ is the per capita natural death rate, and $e^{-\mu\tau}$ represents the probability that an individual stays alive for at least τ units of time [7]. We call $A_\mu(\tau)$ the *Infectivity Function with Demography* (IFD). The IF is assumed to be an L^1 function from $[0, \infty)$ to $[0, \infty)$.

The balance equation describing the changes of the variable S , when demographic turnover is taking into account, is given by:

$$\dot{S}(t) = \Lambda - \mu S(t) - \hat{F}(t)S(t), \quad (3.2)$$

where the upper dot denotes the time derivative, Λ represents the inflow of susceptibles, μ is the natural death rate and the quantity $\hat{F}(t)S$ is the *incidence*, that is the number of new cases per unit time and area [11]. Therefore, the constitutive equation (3.1) describes how the current FoI depends on past incidence.

It is well known that compartmental models can be derived from equation (3.1) under specific hypothesis on the IF (see [20, 23]). For example, if $A(\tau)$ is an exponential function or a linear combination of exponential functions, from (3.1) one can derive the classical SIR and SEIR compartmental models ruled by three and four ODEs, respectively. The connection between compartmental formulations and the integral approach will be addressed in more detail later in Chapter 5.

In the behavioural formulation of epidemic models based on the information index, it is explicitly taken into account that the transmission of information and rumours about the disease are often preceded by articulated routine procedures so that public awareness regarding the disease takes time to build [46, 70]. Moreover, people keep memory of the past values of the infection. Therefore, delay and memory are two major aspects of the opinion-driven change of behaviour. The information index, $M(t)$, summarizes these aspects and is defined as

$$M(t) = \int_0^\infty H(t - \tau) g(x_1(t - \tau), x_2(t - \tau), \dots, x_d(t - \tau)) K(\tau) d\tau, \quad (3.3)$$

where $H(\cdot)$ is the Heaviside step function and

- (i) the quantities $x_1, x_2, \dots, x_d$ are a subset of the state variables of the epidemic model under consideration;
- (ii) the function g (*message function*) describes the individuals' perceived risks associated to the disease. In principle, it may depend on all the state variables, but, in practice, it is assumed to depend only on some specific quantities as, for example, the incidence and the prevalence of the disease;
- (iii) $K(\cdot)$ is the memory kernel, such that $K(t) > 0$ and $\int_0^\infty K(t)dt = 1$. For example, the memory kernel can be chosen to be a Dirac delta $K(t) = \delta(t)$, which represents the instantaneous propagation of the information, or an Erlang distribution with rate parameter a and shape parameter n ,

$$K(t) = \text{Erl}_{n,a}(t) = \frac{a^n}{(n-1)!} t^{n-1} e^{-nt}, \quad a \in \mathbb{R}^+, \quad n \in \mathbb{N}^+, \quad (3.4)$$

which includes both the cases of unimodal kernels (when $n > 1$) and of exponentially fading memory (when $n = 1$). In this last case the memory of the mean individual decays with characteristic length $T = 1/a$, see [46, 70]. An Erlangian kernel offers the advantage of simplifying the corresponding integro-differential system into ordinary differential equations via the so-called *linear chain trick* [63]. Since

$$\text{Erl}'_{1,a} = -a \text{Erl}_{1,a}, \quad \text{Erl}'_{n,a} = a(\text{Erl}_{n-1,a} - \text{Erl}_{n,a}), \quad n > 1,$$

it follows that, by defining the auxiliary variables

$$M_j(t) = \int_0^\infty g(x_1(t-\tau), x_2(t-\tau), \dots, x_n(t-\tau)) \text{Erl}_{j,a}(\tau) d\tau,$$

the following additional equations must be included into the model at hand:

$$\begin{aligned} \dot{M}_1 &= a(g(x_1, x_2, \dots, x_n) - M_1), \\ &\vdots \\ \dot{M}_n &= a(M_{n-1} - M_n). \end{aligned} \quad (3.5)$$

Now, going back to the balance equation (3.2), we assume that the FoI is made up of two terms:

$$\hat{F}(t) = \beta(M(t)) F(t).$$

The first term, $\beta(M(t))$, is a function that describes the inhibitory contribution to the FoI due to the information and rumours regarding the disease status. The function β is assumed to verify the following conditions: $\beta(M) > 0$, $\beta(0) = 1$ and $\beta'(M) < 0$. The second term, $F(t)$, is the *driving factor of the FoI*, which we introduce through the following constitutive law:

$$F(t) = \int_0^\infty \beta(M(t-\tau)) S(t-\tau) F(t-\tau) A_\mu(\tau) d\tau. \quad (3.6)$$

The balance equation for the susceptibles is still equation (3.2), which can now be written:

$$\dot{S}(t) = \Lambda - \mu S(t) - \beta(M(t)) F(t) S(t). \quad (3.7)$$

Note that the formulation (3.6) is a “behavioural” variant of (3.1) and tells us how the driving factor of the FoI depends on the past incidence. We underline again that

Symbol	Meaning
Λ	Net inflow of susceptibles
μ	Natural death rate
β_0	Constant transmission rate
ν	Average duration of the infectious period
R_0	Basic Reproduction Number
N	Total population size ($N = \Lambda/\mu$)
S_0	Initial susceptible population

Table 3.1: Description of the parameters and quantities related to model (3.9).

F is not a FoI. However, it becomes a FoI in the “non-behavioural” case $M = 0$.

It remains now to discuss the message function g in (3.3). We propose that such function depends on the quantity F . In other words, we assume that the perception of individuals regarding the disease is based on the awareness regarding the driving factor of the FoI. This assumption rests on the idea that the quantity F generalizes, in a certain sense, the prevalence of the disease (i.e. the size of the infectious population, say I). Therefore, the assumption that g depends on F generalizes the idea of prevalence-dependent behavioural epidemic models studied through ODEs in previous papers by d’Onofrio and Manfredi [24, 25].

The discussion above leads to the following form of the information index:

$$M(t) = \int_0^\infty H(t - \tau) g(F(t - \tau)) K(\tau) d\tau, \quad (3.8)$$

where $g(0) = 0$, $g(F) \geq 0$ and $g'(F) > 0$. Collecting together (3.6), (3.7) and (3.8), the complete model reads

$$\begin{aligned} \dot{S}(t) &= \Lambda - \mu S(t) - \beta(M(t)) S(t) F(t), \\ F(t) &= \int_0^\infty \beta(M(t - \tau)) S(t - \tau) F(t - \tau) A_\mu(\tau) d\tau, \\ M(t) &= \int_0^\infty H(t - \tau) g(F(t - \tau)) K(\tau) d\tau. \end{aligned} \quad (3.9)$$

For the sake of clarity, the parameters and the functions used in model (3.9), with their respective meaning and related assumptions, are listed in Tables 3.1 and 3.2.

Remark 3.1.1. *In the early epidemic model introduced by Kermack and McKendrick [38, 39], it is assumed that factors such as infectivity, recovery, and mortality depend on the age-of-infection, i.e. the time elapsed by the infection. It is worth to mention that the infectious individuals may be further distinguished (say, “struc-*

Function	Meaning	Assumptions
A	Infectivity function	$A(t) \geq 0$
A_μ	Infectivity function with demography	$\hat{A}_\mu(0) := \int_0^\infty A_\mu(t)dt < +\infty$
β	Inhibition function	$\beta(0) = 1, \beta(M) > 0, \beta'(M) < 0$
g	Message function	$g(0) = 0, g(\cdot) \geq 0, g'(\cdot) > 0$
K	Memory kernel	$K(\tau) > 0, \hat{K}(0) := \int_0^\infty K(\tau)d\tau = 1$

Table 3.2: Functions involved in model (3.9) and the related assumptions.

tured”) according to their age-of-infection. This assumption lead to consider the age-of-infection as an independent variable and the corresponding models are integro-differential models involving partial differential equations. Time-since-infection (and time-since-recovery) structured epidemic models have been studied by several authors (see e.g. [28, 47]). An intriguing time-since-infection behavioural model where the individuals’ decisions regarding vaccination is driven by imitation dynamics can be found in [71].

3.2 Basic properties and existence of equilibria

In order to study the solution $\mathbf{u}(t) = (S(t), F(t), M(t))$ of model (3.9), we assume that, over the interval $(-\infty, 0]$, the state variable S is such that $S(t) \in (0, \Lambda/\mu]$ and $F(t), M(t)$ are non-negative bounded functions. We show below that these properties hold true for all $t \in \mathbb{R}$, provided that $A(\tau)$ satisfies reasonable biological conditions.

Theorem 3.2.1. *The inequalities $S(t) > 0, F(t) \geq 0, M(t) \geq 0$ hold true for all $t > 0$.*

Proof. From the first equation of (3.9) we deduce that

$$S(t) = e^{-\int_0^t [\mu + \beta(M(\tau))F(\tau)]d\tau} \left(S_0 + \int_0^t \Lambda e^{\int_0^s [\mu + \beta(M(\tau))F(\tau)]d\tau} ds \right).$$

Being $S(0) = S_0 > 0$, it easily follows that $S(t) > 0, \forall t \geq 0$. The non-negativity of $F(t)$ and $M(t)$ follows from the non-negativity of the functions A, β, g and K (see the assumptions in Table 3.2). $\square$

Theorem 3.2.2. *Assume that $A'(\tau) \in L^1([0, \infty))$ and $\lim_{\tau \rightarrow \infty} A(\tau) = 0$, then $S(t) \leq S_{\max}, F(t) \leq F_{\max}, M(t) \leq M_{\max}$, for all $t > 0$, where*

$$S_{\max} = \frac{\Lambda}{\mu}, \quad F_{\max} = \Lambda \hat{A}_\mu(0) + \frac{\Lambda}{\mu} \int_0^\infty |A'_\mu(\tau)| d\tau, \quad M_{\max} = g(F_{\max}). \quad (3.10)$$

Proof. The non-negativity of $S(t)$, $F(t)$ and $\beta(M(t))$ implies that $\dot{S}(t) \leq \Lambda - \mu S(t)$. Thus, using the comparison theorem, we obtain

$$S(t) \leq e^{-\mu t} \left(S_0 + \Lambda \int_0^t e^{\mu s} ds \right) = \frac{\Lambda}{\mu} + \left(S_0 - \frac{\Lambda}{\mu} \right) e^{-\mu t}.$$

Since $S_0 \leq \Lambda/\mu$, we obtain the bound for $S(t)$. From the first and the second equation in (3.9), we have

$$F(t) = \Lambda \int_0^\infty A_\mu(\tau) d\tau - \mu \int_0^\infty S(t-\tau) A_\mu(\tau) d\tau - \int_0^\infty \dot{S}(t-\tau) A_\mu(\tau) d\tau.$$

Thus, since $\int_0^\infty S(t-\tau) A_\mu(\tau) d\tau \geq 0$, we deduce that

$$F(t) \leq \Lambda \int_0^\infty A_\mu(\tau) d\tau - \int_0^\infty \dot{S}(t-\tau) A_\mu(\tau) d\tau. \quad (3.11)$$

Integrating by parts the second term in the r.h.s of (3.11), taking into account the non-negativity of the functions involved and that $\lim_{\tau \rightarrow \infty} A(\tau) = 0$, we obtain the following bound for $F(t)$:

$$F(t) \leq \Lambda \hat{A}_\mu(0) + \frac{\Lambda}{\mu} \int_0^\infty |A'_\mu(\tau)| d\tau,$$

where $\hat{A}_\mu(0) := \int_0^\infty A_\mu(\tau) d\tau$. Finally, from the third equation in (3.9), since g is increasing and $\int_0^\infty K(\tau) d\tau = 1$, we have

$$M(t) = \int_0^\infty H(t-\tau) g(F(t-\tau)) K(\tau) d\tau \leq g(F_{\max}) \int_0^\infty K(\tau) d\tau = g(F_{\max}).$$

□

Now, looking for equilibria, we begin by observing that model (3.9) admits a unique *Disease-Free Equilibrium* (DFE) given by

$$\text{DFE} = (\bar{S}_0, 0, 0), \quad (3.12)$$

where $\bar{S}_0 = \Lambda/\mu$. Denote by

$$\text{EE} = (S_e, F_e, M_e), \quad (3.13)$$

the generic equilibrium with $F_e > 0$, i.e. the *Endemic Equilibrium* (EE). Endemic equilibria are obtained from conditions

$$\Lambda - \mu S - \beta(M)SF = 0, \quad \beta(M)S\hat{A}_\mu(0) = 1, \quad M = g(F). \quad (3.14)$$

Introduce

$$R_0 = \frac{\Lambda}{\mu} \hat{A}_\mu(0). \quad (3.15)$$

Theorem 3.2.3. *If the assumptions of Theorem 3.2.2 hold, then: (i) if $R_0 < 1$, then model (3.9) has no endemic equilibria; (ii) if $R_0 > 1$, then model (3.9) has a unique endemic equilibrium.*

Proof. The EE is defined by the following conditions

$$M_e = g(F_e), \quad S_e = \frac{\Lambda}{\mu R_0 \beta(g(F_e))}, \quad (3.16)$$

and $F_e > 0$ is the zero of the nonlinear function

$$G(x) = x - \mu R_0 + \frac{\mu}{\beta(g(x))}, \quad (3.17)$$

in the interval $[0, F_{\max}]$, where $F_{\max}$ is introduced in (3.10). We observe that

$$\begin{aligned} G(0) &= -\mu(R_0 - 1), \\ G(F_{\max}) &= \Lambda \int_0^\infty |A'_\mu(\tau)| d\tau + \frac{\mu}{\beta(g(F_{\max}))} > 0, \\ G'(x) &= 1 - \frac{\mu \beta'(g(x)) g'(x)}{[\beta(g(x))]^2} > 0, \end{aligned}$$

where the last inequality holds due to the assumptions $\beta'(\cdot) < 0$ and $g'(\cdot) > 0$. Thus, when $R_0 > 1$, it is $G(0) < 0$ and $G(x)$ in (3.17) has a unique zero in $[0, F_{\max}]$. Furthermore, if $R_0 < 1$, then $G(x)$ has no zeros in $[0, F_{\max}]$.

□

Remark 3.2.1. In (3.15), $\hat{A}_\mu(0)$ represents the ability to generate secondary cases of infection from an individual within a given population (in fact, if $A_\mu(\tau) = \beta_0 e^{-(\mu+\nu)\tau}$, then the quantity $\hat{A}_\mu(0)$ reduces to the transmission rate β_0 multiplied by the average infectious period $1/(\mu + \nu)$). When $\hat{A}_\mu(0)$ is multiplied by the total population size $N = \Lambda/\mu$, one recognizes the biological meaning of Basic Reproduction Number, that is the average number of secondary cases produced by one primary infection over the course of the infectious period in a fully susceptible population.

3.3 Preliminaries for stability

In this section we derive the characteristic equation as preliminary step of the local stability analysis of the equilibria. To this end, we employ the linearization theory for Volterra integral equations, as presented in Section 2.1. We begin by observing that model (3.9) can be written in integral form as

$$\begin{aligned} S(t) &= S_0 + \int_0^t (\Lambda - \mu S(t-\tau) - \beta(M(t-\tau))S(t-\tau)F(t-\tau))d\tau, \\ F(t) &= F_0(t) + \int_0^t \beta(M(t-\tau))S(t-\tau)F(t-\tau)A_\mu(\tau)d\tau, \\ M(t) &= M_0(t) + \int_0^t g(F(t-\tau))K(\tau)d\tau, \end{aligned} \quad (3.18)$$

where the functions $F_0(t)$ and $M_0(t)$ are defined by

$$\begin{aligned} F_0(t) &= \int_t^\infty \beta(M(t-\tau))S(t-\tau)F(t-\tau)A_\mu(\tau)d\tau, \\ M_0(t) &= \int_t^\infty H(t-\tau)g(F(t-\tau))K(\tau)d\tau = g(F(0))K(t). \end{aligned} \quad (3.19)$$

Consider the perturbation (W, V, Z) to the generic equilibrium $\tilde{E} = (\tilde{S}, \tilde{F}, \tilde{M})$,

$$S = \tilde{S} + W, \quad F = \tilde{F} + V, \quad M = \tilde{M} + Z.$$

Linearization of (3.18) about the equilibrium leads to the following system:

$$\mathbf{y}(t) = \mathbf{f}(t) + \int_0^t \mathbf{Q}(t-\tau)\mathbf{y}(\tau)d\tau, \quad (3.20)$$

where

$$\mathbf{y}(t) = \begin{bmatrix} W(t) \\ V(t) \\ Z(t) \end{bmatrix}, \quad \mathbf{f}(t) = \begin{bmatrix} W(0) \\ V_0(t) \\ Z_0(t) \end{bmatrix},$$

and

$$\mathbf{Q}(t) = \begin{bmatrix} -\mu - \beta(\tilde{M})\tilde{F} & -\beta(\tilde{M})\tilde{S} & -\beta'(\tilde{M})\tilde{F}\tilde{S} \\ \beta(\tilde{M})\tilde{F}A_\mu(t) & \beta(\tilde{M})\tilde{S}A_\mu(t) & \beta'(\tilde{M})\tilde{S}\tilde{F}A_\mu(t) \\ 0 & K(t)g'(\tilde{F}) & 0 \end{bmatrix}.$$

Furthermore, the forcing terms are

$$V_0(t) = \int_t^\infty \left[\beta(M(t-\tau))S(t-\tau)F(t-\tau) - \beta(\tilde{M})\tilde{S}\tilde{F} \right] A_\mu(\tau) d\tau,$$

$$Z_0(t) = [g(F(0)) - g(\tilde{F})]K(t).$$

In order to study the stability of $\tilde{E}$, we make use of the extended version of the classical Paley–Wiener result presented in Theorem 2.1.6, to which we refer for the notation and meaning of the symbols involved. System (3.20) fits into the form (2.13)-(2.18) with $l_1 = 1$, $l_2 = 2$, and

$$\begin{aligned} \mathbf{f}_0(t) &= W(0), \\ \mathbf{f}_1(t) &= \begin{bmatrix} \int_t^\infty [\beta(M(t-\tau))S(t-\tau)F(t-\tau) - \beta(\tilde{M})\tilde{S}\tilde{F}] A_\mu(\tau) d\tau \\ [g(F(0)) - g(\tilde{F})]K(t) \end{bmatrix}, \\ \mathbf{Q}_\infty^1 &= [-\mu - \beta(\tilde{M})\tilde{F}, \quad -\beta(\tilde{M})\tilde{S}, \quad -\beta'(\tilde{M})\tilde{F}\tilde{S}]. \end{aligned}$$

Let $\hat{A}_\mu$ and $\hat{K}$ denote the Laplace transforms

$$\hat{A}_\mu(w) = \int_0^\infty e^{-wt} A_\mu(t) dt, \quad \hat{K}(w) = \int_0^\infty e^{-wt} K(t) dt, \quad (3.21)$$

with $w \in \mathbb{C}$. If $\operatorname{Re}(w) > 0$, the characteristic equation of system (3.20) takes the form

$$\det \begin{bmatrix} w + \mu + \beta(\tilde{M})\tilde{F} & \beta(\tilde{M})\tilde{S} & \beta'(\tilde{M})\tilde{F}\tilde{S} \\ -\beta(\tilde{M})\tilde{F}\hat{A}_\mu(w) & 1 - \beta(\tilde{M})\tilde{S}\hat{A}_\mu(w) & -\beta'(\tilde{M})\tilde{S}\tilde{F}\hat{A}_\mu(w) \\ 0 & -g'(\tilde{F})\hat{K}(w) & 1 \end{bmatrix} = 0. \quad (3.22)$$

Under the assumptions of Theorem 3.2.2, we have that the functions $\mathbf{f}_0$ and $\mathbf{f}_1$ satisfy the hypothesis of Theorem 2.5.3. The following theorem gives a sufficient condition for the local asymptotic stability of the equilibria.

Theorem 3.3.1. *Any equilibrium $\tilde{E} = (\tilde{S}, \tilde{F}, \tilde{M})$ of model (3.9) is locally asymp-*

totically stable if the characteristic equation

$$\beta(\tilde{M})\tilde{S}\hat{A}_\mu(w)\left(1 + \frac{g'(\tilde{F})\beta'(\tilde{M})\tilde{F}\hat{K}(w)}{\beta(\tilde{M})}\right) = 1 + \frac{\beta(\tilde{M})\tilde{F}}{w + \mu}, \quad (3.23)$$

has no solution for $Re(w) \geq 0$.

Proof. (Sketch) The proof is a straightforward application of Theorem 2.5.3, after observing that the transcendental equation (3.23) is equivalent to (3.22). $\square$

We conclude the section by noting that the information dependent parameters affect the location of the equilibrium EE only through the message function (see the term $g(F_e)$ in (3.16), (3.17)), while its stability properties depend also on the memory kernel K .

3.4 Local stability analysis of equilibria

In this section we analyze the stability properties of the equilibria DFE and EE, by applying Theorem 3.3.1 to some special cases of interest. In our analysis, we assume that $Re(w) > 0$. All the results can be easily extended to $Re(w) \geq 0$, by passing to the limit for $\tilde{w} \rightarrow w$ with $Re(\tilde{w}) > 0$.

At the DFE (3.12) the characteristic equation (3.23) simply reduces to $\bar{S}_0\hat{A}_\mu(w) = 1$, and $Re(w) > 0$ implies

$$\left| \frac{\Lambda}{\mu} \hat{A}_\mu(w) \right| \leq \frac{\Lambda}{\mu} \hat{A}_\mu(0).$$

Therefore, from Theorem 3.3.1 and (3.15) the DFE is locally asymptotically stable provided that $R_0 < 1$.

Regarding the equilibrium EE in (3.13), from the characteristic equation (3.23) we obtain sufficient conditions for the stability in some specific cases. Generally speaking, the study of the roots of the characteristic equation is based on the following considerations: when $Re(w) > 0$, it is

$$|\beta(M_e)S_e\hat{A}_\mu(w)| \leq 1, \quad Re\left(1 + \frac{\beta(M_e)F_e}{w + \mu}\right) > 1, \quad (3.24)$$

where the first inequality comes from condition (3.14)₂ and the second one is a direct consequence of $Re(w) > 0$. Given (3.24), a sufficient condition guaranteeing that the characteristic equation (3.23) does not have roots w such that $Re(w) \geq 0$ (as required by Theorem 3.3.1) is given by

$$\left| 1 + \frac{g'(\tilde{F})\beta'(\tilde{M})\tilde{F}\hat{K}(w)}{\beta(\tilde{M})} \right| \leq 1. \quad (3.25)$$

In the following, we discuss three noteworthy cases according to the form of the memory kernel K : no-delay memory; exponentially fading memory; unimodal memory.

Case (i): no-delay memory.

Here we assume that the information propagates instantaneously, with the memory kernel represented by a Dirac delta:

$$K(\tau) = \delta(\tau). \quad (3.26)$$

From (3.23) we get

$$g'(F_e)\beta'(M_e)F_eS_e\hat{A}_\mu(w) + \beta(M_e)S_e\hat{A}_\mu(w) = 1 + \frac{\beta(M_e)F_e}{w + \mu}. \quad (3.27)$$

Theorem 3.4.1. *If $K(\tau) = \delta(\tau)$ and $A_\mu(\tau)$ is a positive definite function¹, then EE is LAS for any choice of the functions β and g .*

Proof. Let be $Re(w) > 0$. Due to the Bochner characterization of positive definite functions (see Section 8.2 in [44]), it is $Re(\hat{A}_\mu(w)) \geq 0$.

Thus, in view of (3.24), since $g'(\cdot) > 0$ and $\beta'(\cdot) < 0$, the real part of the l.h.s. of (3.27) is less than 1, while the real part of the r.h.s. is strictly greater than 1. Therefore equation (3.27) can never be satisfied. $\square$

Remark 3.4.1. *An example of positive definite infectivity function is $A_\mu(\tau) = e^{-(\mu+\nu)\tau}$. In this case we obtain that the endemic equilibrium is always locally asymptotically stable, in agreement with d'Onofrio and Manfredi [24]. Note also that Theorem 3.4.1 applies with any non-negative, non-increasing infectivity function, convex on $[0, \infty)$, since such functions are positive definite [44].*

Theorem 3.4.2. *If $K(\tau) = \delta(\tau)$, $A'(\tau) < 0$, and the functions β and g are such that*

$$g'(F_e)\beta'(M_e)F_e \leq -\beta(M_e),$$

then EE is LAS.

Proof. Let be $\omega = x + iy$, with $x > 0$. We discuss the three cases: $y = 0$, $y > 0$, and $y < 0$.

¹A continuous function $a : \mathbb{R} \rightarrow \mathbb{C}$ is called a positive definite function if $\sum_{i,j} a(x_i - x_j)z_i \bar{z}_j \geq 0$ for any choice of finite sequences $\{x_n\}_n$, $\{z_n\}_n$, with $x_n \in \mathbb{R}$ and $z_n \in \mathbb{C}$ (see [44]).

- If $y = 0$, we consider equation (3.27) for which the inequalities (3.24) hold. Since in this case $g'(F_e)\beta'(M_e)F_eS_e\hat{A}_\mu(w) < 0$, for the assumptions on β and g in Table 3.2, the l.h.s. is less than one and cannot be equal to the r.h.s., which is in turn greater than one. So, equation (3.27) has no roots with positive real part.
- If $y > 0$, we write the imaginary part of equation (3.27) as

$$\left(1 + \frac{g'(F_e)\beta'(M_e)F_e}{\beta(M_e)}\right)y \int_0^\infty e^{-xt} \sin(yt) A_\mu(t) dt = \frac{\beta(M_e)F_e y^2}{(x + \mu)^2 + y^2}. \quad (3.28)$$

Inspired by the proof of Lemma 1 in [68], we observe that since, for the theorem assumption, $e^{-xt} A_\mu(t)$ is a strictly decreasing function, it is

$$\begin{aligned} & \int_0^\infty e^{-xt} \sin(yt) A_\mu(t) dt \\ &= \sum_{j=0}^{+\infty} \int_{\frac{2\pi j}{y}}^{\frac{2\pi(j+1/2)}{y}} (e^{-xt} A_\mu(t) - e^{-x(t+\pi/y)} A_\mu(t + \pi/y)) \sin(yt) dt > 0. \end{aligned}$$

It follows that

$$y \int_0^\infty e^{-xt} \sin(yt) A_\mu(t) dt > 0.$$

The positivity of the r.h.s. of (3.28) implies that the characteristic equation (3.27) can never be satisfied if

$$1 + \frac{g'(F_e)\beta'(M_e)F_e}{\beta(M_e)} \leq 0.$$

- If $y = -z < 0$, then $\sin(yt) = -\sin(zt)$. This case can be discussed as the previous one since

$$\int_0^\infty e^{-xt} \sin(yt) A_\mu(t) dt = - \int_0^\infty e^{-xt} \sin(zt) A_\mu(t) dt < 0.$$

□

Case (ii): exponentially fading memory.

In this case, the memory of the mean individual decays with characteristic length $T = 1/a$, and the memory kernel is assumed to follow a weak Erlang distribution:

$$K(\tau) = ae^{-a\tau}. \quad (3.29)$$

The Laplace transform in (3.21) reads $\hat{K}(w) = a/(a + w)$. Therefore, the sufficient condition (3.25) can be written as

$$\left| 1 + \frac{ag'(F_e)\beta'(M_e)F_e}{\beta(M_e)(a + w)} \right| \leq 1. \quad (3.30)$$

Theorem 3.4.3. *If $K(\tau) = ae^{-a\tau}$ and the functions β and g satisfy the inequality*

$$g'(F_e)\beta'(M_e)F_e \geq -2\beta(M_e), \quad (3.31)$$

then EE is LAS for any choice of $A_\mu(\tau)$.

Proof. Let us set $w = x + iy$, with $x > 0$, and

$$c = \frac{g'(F_e)\beta'(M_e)F_e}{\beta(M_e)}. \quad (3.32)$$

Note that $c < 0$ and the inequality (3.31) becomes $c \geq -2$. It is

$$\left| 1 + \frac{ca}{(a + w)} \right|^2 = 1 + \frac{ca}{(a + x)^2 + y^2} \left(ca + 2(a + x) \right).$$

Therefore, the sufficient condition (3.30) is satisfied if and only if

$$-1 \leq \frac{ca}{(a + x)^2 + y^2} \left(ca + 2(a + x) \right) \leq 0.$$

The left inequality is straightforward. To verify the right inequality for all $x \geq 0$ and $y \in \mathbb{R}$, we observe that $\frac{ca}{(a+x)^2+y^2} < 0$. Since $(ca + 2(a + x)) \geq 0$, for $c \geq -2$, the product of the two factors is less than or equal to zero.

□

Remark 3.4.2. *The result given in Theorem 3.4.3 remains valid in the no-delay case $K(\tau) = \delta(\tau)$. As matter of fact, the sufficient condition (3.25) turns out to be*

$$|1 + c| \leq 1,$$

which is equivalent to (3.31).

Case (iii): unimodal memory.

In this case we assume that the memory kernel is given by a unimodal Erlang distribution:

$$K(\tau) = a^2 \tau e^{-a\tau}. \quad (3.33)$$

This formulation means that current information is not available to individuals and that the maximum weight is assigned to the information that arrives to them after a characteristic time $T = 2/a$. The Laplace transform in (3.21) reads $\hat{K}(w) = \frac{a^2}{(a+w)^2}$. Therefore, the sufficient condition (3.25) is

$$\left| 1 + \frac{ca^2}{(a+w)^2} \right| \leq 1. \quad (3.34)$$

Reasoning as in the proof of Theorem 3.4.3, it is

$$\left| 1 + \frac{ca^2}{(a+w)^2} \right|^2 = 1 + 2ca^2 \frac{((a+x)^2 - y^2)}{((a+x)^2 + y^2)^2} + \frac{c^2 a^4}{((x+a)^2 + y^2)^2},$$

where c is given in (3.32). Since $\beta'(\cdot) < 0$, it follows that (3.34) is satisfied if and only if

$$-1 \leq 2ca^2 \frac{((a+x)^2 - y^2)}{((a+x)^2 + y^2)^2} + \frac{c^2 a^4}{((x+a)^2 + y^2)^2} \leq 0.$$

We observe that, while the left inequality is obviously accomplished, the right one reads

$$\frac{2a^2((a+x)^2 - y^2)}{((a+x)^2 + y^2)^2} > \frac{-ca^2}{((a+x)^2 + y^2)^2},$$

which is satisfied if

$$2(a+x)^2 \geq -ca^2 + 2y^2.$$

The last inequality cannot be true for any $y \in \mathbb{R}$, then Theorem 3.3.1 does not apply. Therefore, instability of the equilibrium EE may take place, in principle.

In the next chapter we introduce a numerical method specially designed to deal with the behavioural integral epidemic model (3.9). Through numerical simulations, we demonstrate that self-sustained oscillations can emerge when the memory kernel is unimodal.

Chapter 4

A Numerical Method for the Integral Behavioural Epidemic Model

In this chapter, we present the numerical method specially designed to deal with the integral epidemic model presented in Chapter 3. We analyze its consistency and convergence, proving that it preserves fundamental properties such as positivity and boundedness for any step-size. Furthermore, the discrete system associated with the method is shown to admit two equilibria under specific assumptions, whose local stability is also investigated. We further examine how the properties of the discrete model relate to those of the continuous system, including a study of its approximation capabilities. Finally, numerical experiments illustrate the performance of the method for selected cases of infectivity functions and memory kernels [16, 17].

4.1 The discretization scheme

In order to numerically solve the system (3.9), we first write $(3.9)_2$ and $(3.9)_3$ in the forms $(3.18)_2$ and $(3.18)_3$, respectively, where the forcing terms $F_0(t)$ and $M_0(t)$ are known non-negative functions given by (3.19).

The numerical method we propose is the result of combining several distinct approaches. First, in equation $(3.9)_1$, the derivative is approximated using forward differences. Moreover, the nonlinear terms $\beta(M)SF$ are discretized through a non-local technique which is explicit in M and F and implicit in S .

Second, the integral equations $(3.9)_2$ and $(3.9)_3$ are solved using a direct quadrature method [13] based on non-local quadrature formulas for the integrals involved (see Section 2.3). Specifically, left approximations are used for K , F , and M , while

right approximations are employed for A_μ and S .

Finally, the discretisation process incorporates *weighted step-sizes* of the form $h\gamma_i(h)$ for $i = 1, 2, 3$. The method formulation is the following:

$$\begin{aligned} S_{n+1} &= S_n + h\gamma_1(h)\left(\Lambda - \mu S_{n+1} - \beta(M_n)S_{n+1}F_n\right), \\ F_{n+1} &= F_0(t_{n+1}) + h\gamma_2(h)\sum_{j=0}^n A_\mu(t_{n+1-j})\beta(M_j)S_{j+1}F_j, \\ M_{n+1} &= M_0(t_{n+1}) + h\gamma_3(h)\sum_{j=0}^n K(t_{n-j})g(F_j), \end{aligned} \quad (4.1)$$

with $n = 0, 1, 2, \dots$. The vector $\mathbf{u}_n = (S_n, F_n, M_n)$ denotes the numerical approximation of the continuous solution $\mathbf{u}(t) = (S(t), F(t), M(t))$ to system (3.9). The approximation is evaluated at the discrete time points $t_n = nh$, where $h > 0$ is the discretization step-size. The weight factors are positive functions of h defined by $\gamma_i(h) = 1 + \varphi_i(h)$, where $\varphi_i(h) = O(h)$ as $h \rightarrow 0$, for all $i = 1, 2, 3$. Here, $S_0 = S(0)$, $F_0 = F(0) = F_0(0)$ and $M_0 = M(0) = M_0(0)$.

The reasoning that led to the formulation (4.1) will be made clear in the following sections, here we point out that, in analogy to what was said in the introduction, we will approach the analysis of the discrete problem (4.1) from two different, though related, points of view:

- numerical method: convergence of the numerical solution $\mathbf{u}_n$ to the solution $\mathbf{u}(t)$ of (3.9), as $h \rightarrow 0$;
- discrete-time model: positivity properties, existence of equilibrium solutions and analysis of their asymptotic properties, for any fixed $h > 0$.

When the discrete equilibria and their properties are related to those of the continuous model, the last item can be regarded as a numerical stability study. Another point of interest is the study of existence and behaviour of equilibrium solutions of (4.1), in the limit as the step-size $h \rightarrow 0$. This study, that represents an investigation into the approximation properties of numerical equilibria, allows to deepen and enlarge the concept of convergence.

4.2 Consistency and convergence

Since a central objective in the analysis of numerical schemes is to establish their convergence, we introduce two key error measures. The global discretization error at time t_n , defined in (2.34), is given by the vector $\mathbf{e}(t_n, h) = \mathbf{u}(t_n) - \mathbf{u}_n$, where $\mathbf{u}(t_n)$ is

the exact solution and $\mathbf{u}_n$ its numerical approximation. The local truncation error, denoted by $\boldsymbol{\delta}(t_n, h)$ and defined in (2.37), corresponds to the residual obtained by substituting the exact solution $\mathbf{u}(t)$ into the discrete system (4.1).

In what follows we use the integral formulation of equation (3.9)₁

$$S(t) = S_0 + \int_0^t \left(\Lambda - \mu S(t - \tau) - \beta(M(t - \tau))S(t - \tau)F(t - \tau) \right) d\tau, \quad (4.2)$$

and its discrete counterpart in (4.1)₁

$$S_{n+1} = S_0 + h \sum_{j=0}^n \left(\Lambda - \mu S_{j+1} - \beta(M_j)S_{j+1}F_j \right), \quad n = 0, 1, 2, \dots \quad (4.3)$$

We begin by establishing a consistency result, recalling the general definition of consistency introduced in Section 2.4 (see Definition 2.4.4). This result provides the first step toward establishing convergence for the numerical scheme. In particular, Lemma 4.2.1 verifies that the scheme satisfies a consistency condition tailored to our model, which will be used in the subsequent convergence analysis.

Lemma 4.2.1. *Let $\bar{n}$ be a fixed positive integer. Assume that the given functions $A_\mu, K \in C^1([0, T])$, with $T = \bar{n}h$, then*

$$\max_{0 \leq n \leq \bar{n}} \|\boldsymbol{\delta}(t_n, h)\| \leq \bar{C}h, \quad \text{with } \bar{C} > 0. \quad (4.4)$$

That is, the method (4.1) is consistent with model (3.9) of order 1.

Proof. The assumptions on $A_\mu(t)$ and $K(t)$ imply that also $S(t)$, $F(t)$ and $M(t)$ are continuously differentiable on $[0, T]$. Let us define the local truncation error $\boldsymbol{\delta}(t_n, h)$ as:

$$\begin{aligned} \boldsymbol{\delta}(t_n, h) = \sum_{j=0}^{n-1} \left(\begin{pmatrix} \int_{t_j}^{t_{j+1}} (\Lambda - \mu S(\tau) - \beta(M(\tau))S(\tau)F(\tau)) d\tau \\ \int_{t_j}^{t_{j+1}} A_\mu(t_n - \tau) \beta(M(\tau))S(\tau)F(\tau) d\tau \\ \int_{t_j}^{t_{j+1}} K(t_n - \tau) g(F(\tau)) d\tau \end{pmatrix} \right. \\ \left. - h \begin{pmatrix} \gamma_1(h)(\Lambda - \mu S(t_{j+1}) - \beta(M(t_j))S(t_{j+1})F(t_j)) \\ \gamma_2(h)A_\mu(t_{n-j})\beta(M(t_j))S(t_{j+1})F(t_j) \\ \gamma_3(h)K(t_{n-j-1})g(F(t_j)) \end{pmatrix} \right). \end{aligned} \quad (4.5)$$

Given the form of $\gamma_i(h) = 1 + \varphi_i(h)$, for $i = 1, 2, 3$, we have that

$$\boldsymbol{\delta}(t_n, h) = \tilde{\boldsymbol{\delta}}(t_n, h) + h\varphi(h) \sum_{j=0}^{n-1} \begin{pmatrix} (\Lambda - \mu S(t_{j+1}) - \beta(M(t_j))S(t_{j+1})F(t_j)) \\ A_\mu(t_{n-j})\beta(M(t_j))S(t_{j+1})F(t_j) \\ K(t_{n-j-1})g(F(t_j)) \end{pmatrix}, \quad (4.6)$$

for $n = 0, \dots, \bar{n}$, where $\varphi(h) = \text{diag}(\varphi_1(h), \varphi_2(h), \varphi_3(h))$ and $\tilde{\boldsymbol{\delta}}(t_n, h)$ is the local truncation error of the *unweighted* numerical method. A slight generalization of the proof of Lemma 3.1 in [51], leads to the following bound for $\tilde{\boldsymbol{\delta}}(t_n, h)$,

$$\max_{0 \leq n \leq \bar{n}} \|\tilde{\boldsymbol{\delta}}(t_n, h)\| \leq \tilde{C}h, \quad \tilde{C} > 0.$$

It then follows, from (4.6), that

$$\|\boldsymbol{\delta}(t_n, h)\| \leq \tilde{C}h + h \|\varphi(h)\| \sum_{j=0}^{n-1} \left\| \begin{pmatrix} (\Lambda - \mu S(t_{j+1}) - \beta(M(t_j))S(t_{j+1})F(t_j)) \\ A_\mu(t_{n-j})\beta(M(t_j))S(t_{j+1})F(t_j) \\ K(t_{n-j-1})g(F(t_j)) \end{pmatrix} \right\|,$$

for $n = 0, \dots, \bar{n}$. As a consequence, due to the regularity of the given functions in $[0, T]$ and also to the fact that $\varphi_i(h) = O(h)$, for $i = 1, 2, 3$, the bound (4.4) holds, where $\bar{C}$ is a positive constant depending on the parameters of the problem but not on h . $\square$

We now prove the convergence result.

Theorem 4.2.2. *Let $\bar{n}$ be a fixed positive integer. Assume $A_\mu, K \in C^1([0, T])$, with $T = \bar{n}h$. Then,*

$$\lim_{h \rightarrow 0} \max_{0 \leq n \leq \bar{n}} \|\mathbf{e}(t_n, h)\| = 0.$$

Furthermore, $\|\mathbf{e}(t_n, h)\| = O(h)$ as $h \rightarrow 0$.

Proof. Subtracting (4.1) from (3.9), with (4.1)₁ and (3.9)₁ formulated as (4.3) and (4.2), gives the following error recursion

$$\begin{aligned}
\mathbf{e}(t_n, h) \leq \boldsymbol{\delta}(t_n, h) + h \sum_{j=0}^{n-1} & \left(\begin{pmatrix} \gamma_1(h)(\Lambda - \mu S(t_{j+1}) - \beta(M(t_j))S(t_{j+1})F(t_j)) \\ \gamma_2(h)A_\mu(t_{n-j})\beta(M(t_j))S(t_{j+1})F(t_j) \\ \gamma_3(h)K(t_{n-j-1})g(F(t_j)) \end{pmatrix} \right) \\
& - \begin{pmatrix} \gamma_1(h)(\Lambda - \mu S_{j+1} - \beta(M_j)S_{j+1}F_j) \\ \gamma_2(h)A_\mu(t_{n-j})\beta(M_j)S_{j+1}F_j \\ \gamma_3(h)K(t_{n-j-1})g(F_j) \end{pmatrix} \right), \tag{4.7}
\end{aligned}$$

for $n = 0, \dots, \bar{n}$. Here $\boldsymbol{\delta}(t_n, h)$ is the local truncation error defined in (4.5). Using the bound (4.4) for $\boldsymbol{\delta}(t_n, h)$ and standard techniques for the second term in the r.h.s. of (4.7), we have

$$\|\mathbf{e}(h; t_n)\| \leq \bar{C}h + hL(1 + \|\varphi(h)\|) \sum_{j=0}^{n-1} \|\mathbf{e}(h; t_j)\|, \quad n = 0, \dots, \bar{n},$$

where $\varphi(h) = \text{diag}(\varphi_1(h), \varphi_2(h), \varphi_3(h))$, and $\bar{C}$, L are positive constants depending on the parameters of the problem but not on h . Thanks to the Gronwall discrete inequality (see Proposition 2.4.1), we finally have

$$\|\mathbf{e}(h; t_n)\| \leq Ch e^{TL(1+\varphi(h))},$$

for $C > 0$ and $n = 0, \dots, \bar{n}$, which yields the result. $\square$

4.3 Basic properties and existence of discrete equilibria

In order to investigate the qualitative and asymptotic behaviour of the solution to (4.1) for any fixed discretization step-size $h > 0$, and to assess its coherence with the properties of the continuous model (3.9), we henceforth assume that all conditions on the parameters and functions, as outlined in Section 3.1, are satisfied. From this point onward, we also assume that

$$A'_\mu(\tau) \in L^1[0, \infty), \quad K'(\tau) \in L^1[0, \infty). \tag{4.8}$$

Two quantities that will play an important role in our analysis are:

$$\bar{A}_\mu(1, h) = h \gamma_2(h) \sum_{n=1}^{\infty} A_\mu(t_n), \tag{4.9}$$

$$\bar{K}(1, h) = h \gamma_3(h) \sum_{n=0}^{\infty} K(t_n), \quad (4.10)$$

for which we prove the following result.

Lemma 4.3.1. *For any $h > 0$, $\bar{A}_\mu(1, h) < +\infty$ and $\bar{K}(1, h) < +\infty$. Furthermore, we have*

$$\lim_{h \rightarrow 0} \bar{A}_\mu(1, h) = \hat{A}_\mu(0), \quad \lim_{h \rightarrow 0} \bar{K}(1, h) = \hat{K}(0) = 1, \quad (4.11)$$

where $\hat{A}_\mu(0)$ and $\hat{K}(0)$ are defined in Table 3.2.

Proof. We prove the result for K . The proof similarly applies to A_μ . Consider

$$\int_{t_j}^{t_{j+1}} K(\tau) d\tau = hK(t_j) + \int_{t_j}^{t_{j+1}} K(\tau) d\tau - \int_{t_j}^{t_{j+1}} K(t_j) d\tau.$$

Thus,

$$h \sum_{j=0}^{\infty} K(t_j) \leq \int_0^{\infty} K(\tau) d\tau + h \int_0^{\infty} |K'(\tau)| d\tau < +\infty, \quad (4.12)$$

thanks to the assumption (4.8) and the fact that $\int_0^{\infty} K(\tau) d\tau = 1$. Therefore, also $\bar{K}(1, h) < +\infty$. Moreover, from (4.12), we deduce that

$$\left| \int_0^{\infty} K(\tau) d\tau - h \gamma_3(h) \sum_{j=0}^{\infty} K(t_j) \right| \leq h \int_0^{\infty} |K'(\tau)| d\tau + |\varphi_3(h)| h \sum_{j=0}^{\infty} K(t_j),$$

and, for $h \rightarrow 0$, since $h \sum_{j=0}^{\infty} K(t_j)$ is bounded, the second of (4.11) is satisfied. $\square$

Let $h > 0$ and

$$D(h) = \{(x, y, z) : 0 < x \leq S_{max}(h), 0 \leq y \leq F_{max}(h), 0 \leq z \leq M_{max}(h)\}, \quad (4.13)$$

where

$$\begin{aligned} S_{max}(h) &= S_{max} = \frac{\Lambda}{\mu}, \\ F_{max}(h) &= \Lambda \left(\hat{A}_\mu(0) + \bar{A}_\mu(1, h) \right) + \frac{\Lambda}{\mu} \int_0^{\infty} |A'_\mu(\tau)| d\tau, \\ M_{max}(h) &= g(F_{max}) + g(F_{max}(h)) \bar{K}(1, h). \end{aligned} \quad (4.14)$$

The following theorem ensures that the solutions of model (4.1) remain in $D(h)$ of any $n \geq 0$.

Theorem 4.3.2. Assume that $\lim_{\tau \rightarrow \infty} A(\tau) = 0$ and that the weight functions $\varphi_1(h)$ and $\varphi_2(h)$ satisfy

HP1. $\varphi_2(h) = c\varphi_1(h)$ with $|c| \leq 1$.

Then the region $D(h)$ defined in (4.13) is positively invariant for the discrete model (4.1).

Proof. We proceed by induction on n with base case $n = 0$, which is trivially true. Let us consider (4.1)₁ written in the following form

$$S_{n+1} = \frac{S_n + h\gamma_1(h)\Lambda}{1 + h\gamma_1(h)(\mu + \beta(M_n)F_n)}. \quad (4.15)$$

If we assume that $S_j > 0$ and $F_j, M_j \geq 0$ for $j = 1, \dots, n$, the expression (4.15) implies that $S_{n+1} > 0$. From (4.1)₁ and (4.1)₃ it easily follows that $F_{n+1}, M_{n+1} \geq 0$.

Let us now prove the boundedness and assume that $S_j \leq \Lambda/\mu$, for $j = 0, \dots, n$. Taking into account that $h\gamma_1(h)\beta(M_n)F_n \geq 0$, from equation (4.15) one has

$$S_{n+1} \leq \frac{\frac{\Lambda}{\mu} + h\gamma_1(h)\Lambda}{1 + h\gamma_1(h)\mu} = \frac{\Lambda}{\mu}.$$

By using (4.1)₁ in (4.1)₂, since S_n is positive, it is easily seen that

$$F_{n+1} \leq F_0(t_{n+1}) + \frac{\gamma_2(h)}{\gamma_1(h)} \sum_{j=0}^n A_\mu(t_{n+1-j})(S_j - S_{j+1}) + \Lambda h \gamma_2(h) \sum_{j=1}^{n+1} A_\mu(t_j). \quad (4.16)$$

Summation by parts in the second term of the r.h.s. of (4.16) leads to

$$F_{n+1} \leq F_0(t_{n+1}) + \frac{\gamma_2(h)}{\gamma_1(h)} \left(A_\mu(t_{n+1})S_0 + \frac{\Lambda}{\mu} \int_0^{t_n} |A'_\mu(\tau)| d\tau \right) + \Lambda h \gamma_2(h) \sum_{j=1}^{n+1} A_\mu(t_{n+1-j}).$$

For the forcing term $F_0(t)$ defined in (3.19)₁, one has

$$F_0(t_{n+1}) \leq \Lambda \int_{t_{n+1}}^{\infty} A_\mu(\tau) d\tau - A_\mu(t_{n+1})S_0 + \frac{\Lambda}{\mu} \int_{t_{n+1}}^{\infty} |A'_\mu(\tau)| d\tau.$$

Using the last two inequalities, we have that

$$\begin{aligned}
F_{n+1} \leq & \Lambda \left(\int_{t_{n+1}}^{\infty} A_{\mu}(\tau) d\tau + h\gamma_2(h) \sum_{j=1}^{n+1} A_{\mu}(t_j) \right) + \left(\frac{\gamma_2(h)}{\gamma_1(h)} - 1 \right) A_{\mu}(t_{n+1}) S_0 \\
& + \frac{\Lambda}{\mu} \left(\int_{t_{n+1}}^{\infty} |A'_{\mu}(\tau)| d\tau + \frac{\gamma_2(h)}{\gamma_1(h)} \int_0^{t_n} |A'_{\mu}(\tau)| d\tau \right).
\end{aligned} \tag{4.17}$$

Since HP1 holds, then the r.h.s. in (4.17) is, for any $h > 0$, less than or equal to $F_{max}(h)$, defined in (4.14)₂, and the desired conclusion follows. Finally, by considering (4.1)₃ and the fact that, from (3.19)₂, it is $M_0(t_{n+1}) \leq g(F_{max}) \int_{t_{n+1}}^{+\infty} K(\tau) d\tau$, the bound for M_n is a consequence of the following inequality:

$$M_{n+1} \leq g(F_{max}) \int_{t_{n+1}}^{+\infty} K(\tau) d\tau + g(F_{max}(h)) h\gamma_3(h) \sum_{j=1}^{n+1} K(t_j).$$

□

We now define

$$R_0(h) = \frac{\Lambda}{\mu} \bar{A}_{\mu}(1, h),$$

as the discrete basic reproduction number. Since $\bar{A}_{\mu}(1, h)$, given in (4.9), represents a weighted quadrature formula for $\hat{A}_{\mu}(0)$, $R_0(h)$ is the numerical approximation to the basic reproduction number R_0 introduced for the model problem (3.9) in Section 3.2. From system (4.1) we derive the following equations for equilibria

$$\begin{aligned}
\Lambda - \mu S - \beta(M)SF &= 0, \\
F &= \bar{A}_{\mu}(1, h)\beta(M)SF, \\
M &= \bar{K}(1, h)g(F).
\end{aligned}$$

If $F = 0$, for any $h > 0$ there is exactly one equilibrium, the disease-free equilibrium

$$\text{DFE}(h) = \left(\frac{\Lambda}{\mu}, 0, 0 \right). \tag{4.18}$$

If $F > 0$, an endemic equilibrium

$$\text{EE}(h) = (S_e(h), F_e(h), M_e(h)), \tag{4.19}$$

exists, for any $h > 0$, if and only if there exists a zero $F_e(h)$ of the nonlinear function

$$\Phi(h, x) = x - \mu R_0(h) + \frac{\mu}{\beta(\bar{K}(1, h)g(x))}, \tag{4.20}$$

in $[0, F_{max}(h)]$, and

$$S_e(h) = \frac{\Lambda}{\mu R_0(h) \beta(M_e(h))}, \quad M_e(h) = \bar{K}(1, h) g(F_e(h)). \quad (4.21)$$

Theorem 4.3.3. *Let $h > 0$. If $R_0(h) < 1$, then model (4.1) has no endemic equilibria; if $R_0(h) > 1$, then model (4.1) has a unique endemic equilibrium.*

Proof. Consider the function $\Phi(h, x)$ defined in (4.20) and let $x \in [0, F_{max}(h)]$. We observe that

$$\begin{aligned} \Phi(h, 0) &= -\mu(R_0(h) - 1), \\ \Phi(h, F_{max}(h)) &= \Lambda \hat{A}_\mu(0) + \frac{\Lambda}{\mu} \int_0^\infty |A'_\mu(\tau)| d\tau + \frac{\mu}{\beta(\bar{K}(1, h) g(F_{max}(h)))} > 0, \\ \Phi'(h, x) &= 1 - \frac{\mu \beta'(\bar{K}(1, h) g(x)) \bar{K}(1, h) g'(x)}{[\beta(\bar{K}(1, h) g(x))]^2} > 0, \end{aligned}$$

where the last inequality holds since $\beta'(\cdot) < 0$. Thus, when $R_0(h) > 1$, it is $\Phi(h, 0) < 0$ and, being $\Phi(h, x)$ in (4.20) an increasing function, it has a unique zero in $[0, F_{max}(h)]$. For the same reason it is clear that if $R_0(h) < 1$, then $\Phi(h, x)$ has no zeros in $[0, F_{max}(h)]$. $\square$

All the results reported in this and in the previous section apply whatever the choice of the weight factors γ_1 , γ_2 , and γ_3 . However, in order to match the numerical endemic equilibrium $EE(h)$ with the equilibrium EE of system (3.9), we choose

$$\text{HP2. } \gamma_3(h) = \frac{1}{h \sum_{n=0}^\infty K(t_n)},$$

that acts as a normalization factor, yielding $\bar{K}(1, h) = \hat{K}(0) = 1$. From the proof of Lemma 4.3.1 we conclude that also $\lim_{h \rightarrow 0} h \sum_{n=0}^\infty K(t_n) = 1$, therefore the weight defined by HP2 satisfies the property $\gamma_3(h) = 1 + O(h)$, as required in the definition of the weight factors. Since the form of γ_1 and γ_2 does not play a significant role, we set

$$\text{HP3. } \gamma_1(h) = \gamma_2(h) = 1.$$

4.4 Asymptotic properties of discrete equilibria

The aim of this section is to derive the characteristic equation in order to study the local asymptotic stability of the equilibria of the discrete model (4.1), for any $h > 0$.

In this regard, we assume that HP3 holds for the weight factors $\gamma_1(h)$ and $\gamma_2(h)$. Thus, we refer to the following formulation of the numerical method (4.1)

$$\begin{aligned} S_{n+1} &= S_0 + h \sum_{j=0}^n \left(\Lambda - \mu S_{j+1} - \beta(M_j) S_{j+1} F_j \right), \\ F_{n+1} &= F_0(t_{n+1}) + h \sum_{j=0}^n A_\mu(t_{n+1-j}) \beta(M_j) S_{j+1} F_j, \\ M_{n+1} &= M_0(t_{n+1}) + h \gamma_3(h) \sum_{j=0}^n K(t_{n-j}) g(F_j), \end{aligned} \quad (4.22)$$

for $n = 0, 1, \dots$, where the functions $F_0(t)$ and $M_0(t)$ are defined in (3.19). Here and in the following we assume that the weight factor $\gamma_3(h)$ is the one given in HP2. Consider the perturbations (W_n, V_n, Z_n) to a generic equilibrium $\tilde{E}(h) = (\tilde{S}(h), \tilde{F}(h), \tilde{M}(h))$. Since,

$$S_n = \tilde{S}(h) + W_n, \quad F_n = \tilde{F}(h) + V_n, \quad M_n = \tilde{M}(h) + Z_n,$$

we have that, for $n = 0, 1, \dots$, (W_n, V_n, Z_n) satisfy:

$$\begin{bmatrix} W_{n+1} \\ V_n \\ Z_n \end{bmatrix} = \begin{bmatrix} W(0) \\ V_0(t_n) \\ Z_0(t_n) \end{bmatrix} + h \sum_{j=0}^n \mathbf{Q}_{n-j} \left(\begin{bmatrix} W_{j+1} \\ V_j \\ Z_j \end{bmatrix} + \mathbf{G}_j \left(\begin{bmatrix} W_{j+1} \\ V_j \\ Z_j \end{bmatrix} \right) \right), \quad (4.23)$$

where $\mathbf{G}_j(\cdot)$, $j \geq 0$, are given mappings that include all the nonlinear terms of (4.22). In (4.23) the 3×3 matrices $\mathbf{Q}_n$ are defined by

$$\mathbf{Q}_n = \begin{cases} \begin{bmatrix} -\mu - \beta(\tilde{M}(h))\tilde{F}(h) & -\beta(\tilde{M}(h))\tilde{S}(h) & -\beta'(\tilde{M}(h))\tilde{S}(h)\tilde{F}(h) \\ \beta(\tilde{M}(h))\tilde{F}(h)A_\mu(t_n) & \beta(\tilde{M}(h))\tilde{S}(h)A_\mu(t_n) & \beta'(\tilde{M}(h))\tilde{S}(h)\tilde{F}(h)A_\mu(t_n) \\ 0 & K(t_n)g'(\tilde{F}(h)) & 0 \end{bmatrix}, & n > 0, \\ \begin{bmatrix} -\mu - \beta(\tilde{M}(h))\tilde{F}(h) & -\beta(\tilde{M}(h))\tilde{S}(h) & -\beta'(\tilde{M}(h))\tilde{S}(h)\tilde{F}(h) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, & n = 0, \end{cases} \quad (4.24)$$

and the forcing terms are

$$V_0(t_{n+1}) = F_0(t_{n+1}) - \beta(\tilde{M}(h))\tilde{S}(h)\tilde{F}(h)h \sum_{j=n+1}^{\infty} A_\mu(t_j),$$

and

$$Z_0(t_{n+1}) = \left(g(F_0) - hg(\tilde{F}(h)) \right) K(t_{n+1}).$$

The analysis of the asymptotic behaviour of the solutions to the nonlinear perturbed system (4.23) goes through the linearization theory developed in [64] and described in Section 2.5. Let $\mathbf{y}_n = [W_{n+1}, V_n, Z_n]^T$ be the solution of system (4.23) and $\mathbf{x}_n$ be the solution of the linear system

$$\mathbf{x}_n = \mathbf{f}_n + h \sum_{j=0}^n \mathbf{Q}_{n-j} \mathbf{x}_j, \quad n = 0, 1, \dots, \quad (4.25)$$

related to (4.23), with $\mathbf{Q}_n$ defined in (4.24) and $\mathbf{f}_n = \left(W(0), V_0(t_n), Z_0(t_n) \right)^T$. We observe that the condition $\det(\mathbb{I} - h\mathbf{Q}_n) \neq 0$, is always satisfied provided h sufficiently small. Therefore, by Theorem 2.5.1, the perturbation $\mathbf{y}_n$ can be expressed in terms of $\mathbf{x}_n$ as

$$\mathbf{y}_n = \mathbf{x}_n - \sum_{j=0}^n \mathbf{R}_{n-j} \mathbf{G}_j(\mathbf{y}_j), \quad n = 0, 1, \dots,$$

where $\mathbf{R}_n$ are the resolvent matrices of $h\mathbf{Q}_n$. Since Theorem 2.5.2 states that $\mathbf{y}_n$ asymptotically vanishes whenever $\lim_{n \rightarrow \infty} \mathbf{x}_n = 0$, we study the asymptotic behaviour of the solutions to the linear system (4.25) and thus we refer to Theorem 2.5.3 for the stability analysis. System (4.25) fits into the form (2.49), (2.50) with $l_1 = 1$, $l_2 = 2$ and

$$\begin{aligned} \mathbf{f}_n^0 &= W(0), \\ \mathbf{f}_n^1 &= \begin{bmatrix} V_0(t_n) \\ Z_0(t_n) \end{bmatrix}, \\ \mathbf{B}_n &= h\mathbf{Q}_n, \\ \mathbf{B}_\infty^1 &= h[-\mu - \beta(\tilde{M}(h))\tilde{F}(h), \quad -\beta(\tilde{M}(h))\tilde{S}(h), \quad -\beta'(\tilde{M}(h))\tilde{S}(h)\tilde{F}(h)]. \end{aligned}$$

For $|z| \leq 1$, the characteristic equation takes the form

$$(1 - z) \cdot \det \begin{bmatrix} 1 + (\mu + \beta(\tilde{M}(h))\tilde{F}(h))\frac{h}{1-z} & \beta(\tilde{M}(h))\tilde{S}(h)\frac{h}{1-z} & \beta'(\tilde{M}(h))\tilde{S}(h)\tilde{F}(h)\frac{h}{1-z} \\ -\beta(\tilde{M}(h))\tilde{F}(h)\bar{A}_\mu(z, h) & 1 - \beta(\tilde{M}(h))\tilde{S}(h)\bar{A}_\mu(z, h) & -\beta'(\tilde{M}(h))\tilde{S}(h)\tilde{F}(h)\bar{A}_\mu(z, h) \\ 0 & -g'(\tilde{F}(h))\bar{K}(z, h) & 0 \end{bmatrix} = 0, \quad (4.26)$$

where

$$\bar{A}_\mu(z, h) = h \sum_{n=1}^{\infty} A_\mu(t_n) z^n, \quad \bar{K}(z, h) = h \gamma_3(h) \sum_{n=0}^{\infty} K(t_n) z^n,$$

with $\gamma_3(h)$ given by HP2, are the discrete and the *weighted* discrete Laplace transforms of A_μ and K , respectively. The following theorem gives a sufficient condition for the local asymptotic stability of the equilibria.

Theorem 4.4.1. *Any equilibrium $\tilde{E}(h) = (\tilde{S}(h), \tilde{F}(h), \tilde{M}(h))$ of model (4.22) is locally asymptotically stable, for $h > 0$, if the characteristic equation*

$$\beta(\tilde{M}(h)) \tilde{S}(h) \bar{A}_\mu(z, h) \left(1 + \frac{g'(\tilde{F}(h)) \beta'(\tilde{M}(h)) \tilde{F}(h) \bar{K}(z, h)}{\beta(\tilde{M}(h))} \right) = 1 + h \frac{\beta(\tilde{M}(h)) \tilde{F}(h)}{1 - z + \mu h}, \quad (4.27)$$

has no solution for $|z| \leq 1$.

Proof. The proof is a straightforward application of Theorem 2.5.2, after observing that equation (4.27) is equivalent to (4.26) and that $\mathbf{f}_n^0$ and $\mathbf{f}_n^1$ satisfy the assumptions of Theorem 2.5.2. $\square$

At the DFE (4.18), the characteristic equation (4.27) becomes

$$\frac{\Lambda}{\mu} \bar{A}_\mu(z, h) = 1.$$

Note that $\frac{\Lambda}{\mu} \bar{A}_\mu(1, h) = R_0(h)$. Suppose that $R_0(h) < 1$ and there is a root z with $|z| < 1$ then

$$\left| \frac{\Lambda}{\mu} \bar{A}_\mu(z, h) \right| \leq R_0(h) < 1,$$

that is a contradiction. Thus, stability follows.

In the next we present and analyze characteristic equations for the endemic equilibria $EE(h)$ (4.19) of some submodels obtained by specific choices for the IFD A_μ and the memory kernel K , which have been introduced in Section 3.1 as the more common in the literature and significative in the applications. Here the step-size $h > 0$ is fixed. In our analysis we assume that $|z| < 1$, all the results can be easily extended to $|z| \leq 1$, by passing to the limit for $\tilde{z} \rightarrow z$, with $|\tilde{z}| < 1$.

As main consideration, we observe that the assumption $|z| < 1$ implies that

$$|\beta(M_e(h)) S_e(h) \bar{A}_\mu(z, h)| \leq 1, \quad \operatorname{Re} \left(1 + h \frac{\beta(M_e(h)) F_e(h)}{1 - z + \mu h} \right) > 1, \quad (4.28)$$

where the first bound is a consequence of the equilibrium condition (4.21)₁.

Theorem 4.4.2. *If $A_\mu(\tau)$ is positive definite and $K(\tau) = \delta(\tau)$, then $EE(h)$ is LAS for any choice of the functions β and g .*

Proof. When $K(\tau) = \delta(\tau)$ the characteristic equation (4.27) reads

$$\beta(M_e(h))S_e(h)\bar{A}_\mu(z, h)\left(1 + \frac{g'(F_e(h))\beta'(M_e(h))F_e(h)}{\beta(M_e(h))}\right) = 1 + h\frac{\beta(M_e(h))F_e(h)}{1 - z + \mu h}. \quad (4.29)$$

If $A_\mu(\tau)$ is a positive definite function then $\{A_\mu(t_n)\}_n$ is a positive definite sequence and the discrete analogue of the Bochner characterization holds (see Lemma 8.2 in [44]). Then

$$\operatorname{Re}(\bar{A}_\mu(z, h)) \geq 0, \quad \text{in } |z| < 1.$$

Thus, being $g' < 0$ and $\beta' > 0$, considering (3.24), the real part of the left side of (4.29) is less than or equal to 1, while the real part of the right side is strictly greater than 1. It is not possible for (4.29) to have root inside the unit circle and then the local stability is guaranteed by Theorem 4.4.1. $\square$

Examples of infectivity functions that satisfy Theorem 3.4.1 are non-negative, non-increasing, convex functions on $[0, \infty)$ [44]. Among them, a notable case is $A_\mu(\tau) = e^{-(\mu+\nu)\tau}$, which leads to the SIR-M compartment model introduced in [24].

In a more general context for A_μ we derive that, given (4.28), a sufficient condition for equation (4.27) not to have roots with $|z| < 1$ is

$$\left|1 + \frac{g'(F_e(h))\beta'(M_e(h))F_e(h)\bar{K}(z, h)}{\beta(M_e(h))}\right| \leq 1. \quad (4.30)$$

In the following we analyze how condition (4.30) impacts on the model parameters when the memory kernel K is a Dirac delta or a weak Erlang distribution.

Theorem 4.4.3. *Assume that $K(\tau) = \delta(\tau)$ or $K(\tau) = ae^{-a\tau}$, with $a \in \mathbb{R}^+$, and that the functions β and g satisfy the inequality*

$$g'(F_e(h))\beta'(M_e(h))F_e(h) \geq -2\beta(M_e(h)), \quad (4.31)$$

then $EE(h)$ is LAS for any choice of $A_\mu(\tau)$.

Proof. When $K(\tau) = \delta(\tau)$ the sufficient condition (4.30) reads

$$\left|1 + \frac{g'(F_e(h))\beta'(M_e(h))F_e(h)}{\beta(M_e(h))}\right| \leq 1,$$

which is equivalent to (4.31) since $\beta' < 0$.

If $K(\tau) = ae^{-a\tau}$ then

$$\bar{K}(z, h) = \frac{e^{ah} - 1}{e^{ah} - z}.$$

Set $c = \frac{g'(F_e(h))\beta'(M_e(h))F_e(h)}{\beta(M_e(h))}$, with $z = x + iy$ consider

$$\left| 1 + c \frac{e^{ah} - 1}{e^{ah} - z} \right|^2 = 1 + \frac{c(e^{ah} - 1)}{(e^{ah} - x)^2 + y^2} \left(c(e^{ah} - 1) + 2(e^{ah} - x) \right).$$

Condition (4.30) is satisfied if

$$\left| 1 + c \frac{e^{ah} - 1}{e^{ah} - z} \right|^2 \leq 1,$$

that is

$$\frac{c(e^{ah} - 1)}{(e^{ah} - x)^2 + y^2} \left(c(e^{ah} - 1) + 2(e^{ah} - x) \right) \leq 0. \quad (4.32)$$

The negativity of β' implies that $c < 0$ and then (4.32) is satisfied by $c \geq -2$. $\square$

When the memory kernel has the unimodal form $K(\tau) = a^2\tau e^{-a\tau}$, condition (4.30) reduces to

$$\left| 1 + \frac{z(e^{ah} - 1)^2 g'(\tilde{F}(h))\beta'(\tilde{M}(h))\tilde{F}(h)}{(e^{ah} - z)^2 \beta(\tilde{M}(h))} \right| \leq 1. \quad (4.33)$$

Let $z = x + iy$ and $c = \frac{g'(\tilde{F}(h))\beta'(\tilde{M}(h))\tilde{F}(h)}{\beta(\tilde{M}(h))}$, then the condition (4.33) becomes

$$|1 + c(e^{ah} - 1)^2(X + iY)| \leq 1, \quad (4.34)$$

where

$$X = \frac{x[(e^{ah} - x)^2 - y^2] - 2y^2(e^{ah} - x)}{[(e^{ah} - x)^2 + y^2]^2 + 4y^2(e^{ah} - x)^2},$$

$$Y = \frac{y[(e^{ah} - x)^2 - y^2] - 2xy(e^{ah} - x)}{[(e^{ah} - x)^2 + y^2]^2 + 4y^2(e^{ah} - x)^2}.$$

From (4.34) we derive

$$-1 \leq 2c(e^{ah} - 1)^2 X + c^2(e^{ah} - 1)^4(X^2 + Y^2) \leq 0, \quad (4.35)$$

which is not true for any x and y in the interval $(-1, 1)$.

Remark 4.4.1. *The fact that the sufficient condition (4.30) is not satisfied suggests that the choice of unimodal memory kernels may lead to instability of the equilibrium $EE(h)$, in accordance to what observed for the continuous model in Section 3.4.*

4.5 Qualitative relation and approximation properties

In the previous sections we have addressed the problem to construct a numerical method that, for any step-size $h > 0$, replicates all the properties of the continuous model described in Chapter 3.

Here, we consider the effect of the approximation (4.1) on equilibria under the assumptions (4.8) on A_μ and K . First of all we observe that, because of Lemma 4.3.1, it is $R_0(h) \rightarrow R_0$ as $h \rightarrow 0$. For the bounds (3.10) and (4.14) on the continuous and numerical solution respectively, the following result holds.

Theorem 4.5.1. *For any $h > 0$, $S_{max} = S_{max}(h)$, $F_{max} < F_{max}(h) < \bar{F}$, and $M_{max} < M_{max}(h) < \bar{M}$, with $\bar{F}$ and $\bar{M}$ positive constants. Furthermore,*

$$\begin{aligned}\lim_{h \rightarrow 0} F_{max}(h) &= F_{max} + \Lambda \hat{A}_\mu(0), \\ \lim_{h \rightarrow 0} M_{max}(h) &= M_{max} + g(F_{max} + \Lambda \hat{A}_\mu(0)).\end{aligned}$$

Proof. The proof is straightforward by considering, in (4.14), that $\bar{A}_\mu(1, h), \bar{K}(1, h) < +\infty$ and that $\bar{A}_\mu(1, h) \rightarrow \hat{A}_\mu(0)$ and $\bar{K}(1, h) \rightarrow 1$, as $h \rightarrow 0$ (see Lemma 4.3.1) $\square$

Theorem 4.5.2. *Consider the endemic equilibria $EE = (S_e, F_e, M_e)$ and $EE(h) = (S_e(h), F_e(h), M_e(h))$ of the continuous and numerical models, respectively. Then*

$$\lim_{h \rightarrow 0} EE(h) = EE.$$

Proof. The function $\Phi(h, x)$ defined in (4.20), is a continuous function in $[0, +\infty) \times [0, \bar{F}]$ and, from Theorem 4.3.3, it has a unique zero $F_e(h)$. Furthermore, since $R_0(h) \rightarrow R_0$ and $\bar{K}(1, h) \rightarrow \hat{K}(0) = 1$, as $h \rightarrow 0$, it is $\Phi(0, x) = \Phi(x)$, where $\Phi(x)$ is the nonlinear function defined in (3.17), for which F_e is the unique zero (see Section 3.2). Thus, $\Phi(h, x)$ satisfies the assumptions of Theorem 3.5 in [52], which states the continuous dependence of solutions to nonlinear equations with respect to parameters. Therefore, we have that $F_e(h) \rightarrow F_e$ and, from (4.21), also $M_e(h) \rightarrow M_e$ and $S_e(h) \rightarrow S_e$, as $h \rightarrow 0$. $\square$

Thanks to Theorems 4.5.1 and 4.5.2, we can assert that the discrete characteristic equation (4.27) is consistent as $h \rightarrow 0$ with the characteristic equation (3.23) of the model (3.9). This is clear upon the change of variable $z = e^{-wh}$, with $\operatorname{Re}(w) > 0$, in (4.27) and passing to the limit as $h \rightarrow 0$.

Concerning the local stability analysis, it is clear that the sufficient conditions established in Theorems 4.4.2 and 4.4.3 for the numerical equilibria are consistent, as $h \rightarrow 0$, with the continuous analogous described by Theorems 3.4.1-3.4.3 and Remark 3.4.2.

All the considerations and results reported in this section prove that not only the numerical model behaves in agreement with the continuous model in terms of preservation of the main properties such as positivity invariance, existence of equilibrium solutions and their stability, independently of the step-size, but also all the numerical parameters that describe the numerical dynamics converge, as $h \rightarrow 0$, to their continuous counterparts.

4.6 Numerical experiments

4.6.1 Preliminaries

Here we present some numerical examples, based on the non-standard discretization (4.1) of model (3.9), with the aim to show experimentally the theoretical results of the previous sections. More specifically, we aim both to illustrate important properties of the numerical method, like its convergence order and its ability to preserve positivity, and to offer explanations of the behaviour of the solution in situations where the theoretical analysis alone is not sufficient.

The discrete model described by equations (4.1) needs the knowledge of the initial condition $S_0 = S(0)$ and the forcing functions $F_0(t)$, $M_0(t)$ defined in (3.19), which depend on the entire past history of S , F and M over $(-\infty, 0]$. We assume that the reaction of the population to the epidemic is triggered at time $t = 0$. Therefore, the initial condition S_0 is the steady state of the susceptible population for the “non-behavioural” case $M = 0$. The constitutive law (3.6) may be written as

$$F(t) = \int_0^\infty \beta(M(t-\tau))S(t-\tau)F(t-\tau)H(t-\tau)A_\mu(\tau)d\tau,$$

where $H(\cdot)$ is the Heaviside step function. Assuming $\dot{S}(\tau) \approx 0$, for $\tau \leq 0$, one gets

$$F_0(t) = A_\mu(t)(\Lambda - \mu S_0). \quad (4.36)$$

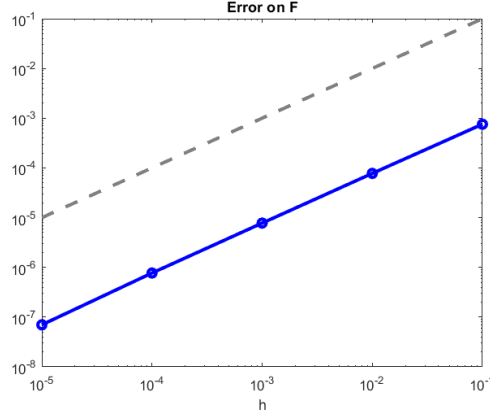


Figure 4.1: Problem (3.9)-(4.37): norm of the errors (solid line) as functions of the step-size, compared to the slope of order one (dashed line).

In our experiments the weight factors $\gamma_1(h)$, $\gamma_2(h)$ and $\gamma_3(h)$ are chosen according to HP2 and HP3. For all the experiments we use

$$g(F) = F, \quad \text{and} \quad \beta(M) = \frac{1}{1 + \alpha M},$$

where $\alpha > 0$ is a decline factor due to voluntary social distancing.

4.6.2 Numerical validation: convergence and positivity test

As first example we integrate problem (4.1) for $t \in [0, 1]$, with

$$\begin{aligned} A_\mu(\tau) &= \beta_0 e^{-(\mu+\nu)\tau}, \quad K(\tau) = a e^{-a\tau}, \quad \beta_0 = R_0 \frac{(\mu + \nu)}{N}, \\ R_0 &= 3.3, \quad \nu = 1/7 \text{ days}^{-1}, \quad \mu = 1/75 \text{ years}^{-1}, \quad a = 1/30 \text{ days}^{-1}, \\ N &= 5 \cdot 10^4, \quad S_0 = 0.8N, \quad \alpha = 10. \end{aligned} \quad (4.37)$$

In order to study the convergence of our numerical scheme, we use the discrete solution computed with step-size $h = 10^{-6}$ as reference solution. The reduction of numerical errors as function of the step-size, shown in Figure 4.1, confirms the linear convergence stated in Theorem 4.2.2.

To compare the performances of the non-standard method (4.1), proposed in this paper, with the ones of other methods, we solve the following problem

$$\begin{aligned} A_\mu(\tau) &= \beta_0 e^{-(\mu+\nu)\tau}, \quad \beta_0 = R_0 \frac{(\mu + \nu)}{N}, \quad R_0 = 20, \\ \nu &= 1/4 \text{ days}^{-1}, \quad \mu = 1/75 \text{ years}^{-1}, \quad N = 5 \cdot 10^7, \quad S_0 = 0.97N. \end{aligned} \quad (4.38)$$

For the sake of simplicity, in this simulation, we don't take into account any be-

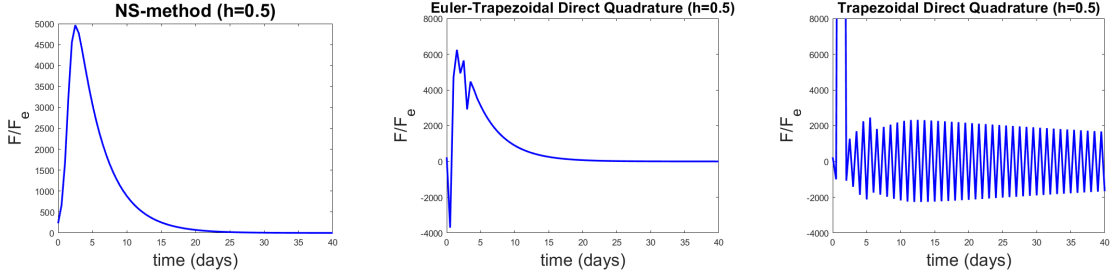


Figure 4.2: Problem (3.9)-(4.38): comparison of numerical solution with $h = 0.5$.

havioural changes within the population. We adapt standard discretization rules to problem (3.9). In particular, we show a first experiment where we couple Euler (see Section 2.3.1) method for $(3.9)_1$ and Trapezoidal direct quadrature (see Section 2.2) for $(3.9)_{2,3}$ thus resulting in an explicit method. In a second experiment we use the implicit Trapezoidal discretization for the whole problem (3.9). Figure 4.2 shows the outcomes of these computations, with step-size $h = 0.5$ (non-standard on the left, Euler-Trapezoidal in the middle and Trapezoidal on the right). It is clear that the two standard methods do not respect the positivity property.

In the next subsections we numerically test model (3.9) by choosing some different infectivity functions taken from studies where specific diseases are considered [1, 24, 34, 59].

4.6.3 Exponential and unimodal IF

We begin with the exponentially distributed IFD already considered Section 4.6.2:

$$A_\mu(\tau) = \beta_0 e^{-(\mu+\nu)\tau}. \quad (4.39)$$

We numerically test the model by using the parameter constellation used in [24]: $R_0 = 20$, $\nu = 1/7 \text{ days}^{-1}$, $\mu = 1/75 \text{ years}^{-1}$, $\alpha = 8 \cdot 10^3$, and $N = 5 \cdot 10^7$, where the values of R_0 and ν refer to measles disease. Taking $K(\tau) = ae^{-a\tau}$ with $a = 1/30 \text{ days}^{-1}$, we observe in Figure 4.3, left panel, an asymptotic convergence to the endemic equilibrium. Since this kernel satisfies the sufficient conditions for local asymptotic stability stated in Theorems 3.4.1 and 3.4.3, the numerical results provide a clear illustration of the theoretical predictions.

In order to show the algorithm's robustness with respect to perturbations of the initial value, we consider three different scenarios: a generic initial value, say $S_0 = 0.20N$; high and low perturbation of the EE, i.e. $S_0 = 0.90S_e$ and $S_0 = 0.99S_e$, respectively. In all of these scenarios, the stability result is confirmed, although

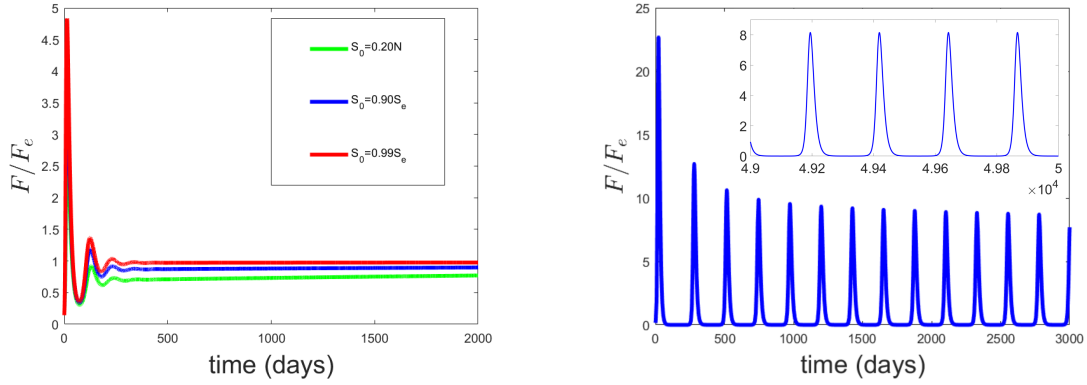


Figure 4.3: Time profile of the ratio F/F_e as predicted by model (3.9) with exponential IFD (4.39). Left panel: case with Erlang memory kernel (3.29). Three different initial conditions are reported: $S_0 = 0.20N$, $S_0 = 0.90S_e$ and $S_0 = 0.99S_e$, where $S_e = 0.26N$. Right panel: case with Erlang memory kernel (3.33), i.e. $K(\tau) = a^2\tau e^{-a\tau}$ (initial condition $S_0 = 0.20N$). Parameter values: see the text.

perturbations far from the endemic equilibrium show a slow convergence (see the green line in Figure 4.3, left panel).

Now, let us assume that the memory kernel has an Erlang distribution with $n = 2$, i.e. $K(\tau) = a^2\tau e^{-a\tau}$. We recall that the sufficient conditions (3.34) guaranteeing the local asymptotic stability of the endemic equilibrium cannot be satisfied in this case. Indeed, oscillations may occur (see Figure 4.3, right panel). After a transient, the oscillations appear to be self-sustained (see Figure 4.3, right panel, top right square). To get more insights into the nature of these oscillations, we consider, in a given interval $[T_1, T_2]$, the amplitudes A_1 and A_2 of the first and last oscillation, respectively. We introduce the percentage *amplitude relative variation* A_{var} as

$$A_{var} = \frac{A_2 - A_1}{A_2} \cdot 100.$$

We get $A_{var} \approx 0.018\%$ in the time interval $[4.9 \cdot 10^4, 5.0 \cdot 10^4]$. Therefore, barring numerical errors this suggests that oscillations could be self-sustained. The integral formulation of the renewal equation (3.9) allows to verify that the occurrence of oscillations can be observed not only when the IFD is given by (4.39), but also in unimodal cases. For example, a similar result may be observed when the IFD is given by:

$$A_\mu(\tau) = \beta_0\tau e^{-(\mu+\nu)\tau}. \quad (4.40)$$

In this case, we still observe oscillations (see Figure 4.4) with $A_{var} \approx 0.017\%$ in the time interval $[4.9 \cdot 10^4, 5.0 \cdot 10^4]$.

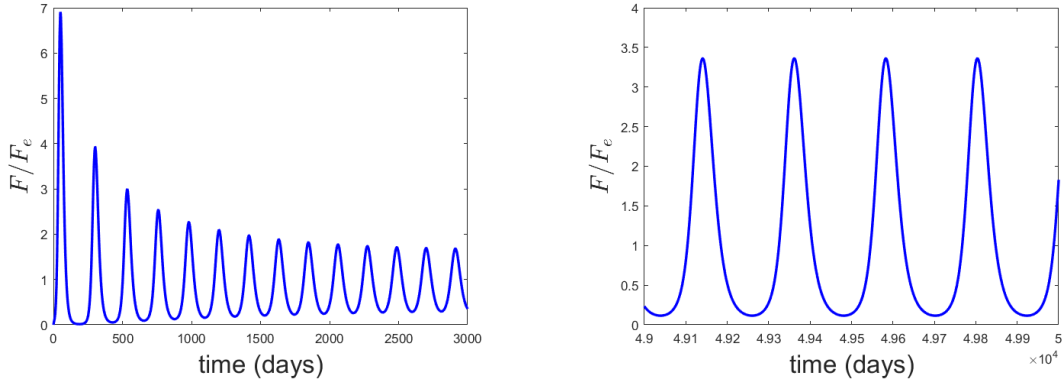


Figure 4.4: Time profile of the ratio F/F_e as predicted by model (3.9). Left panel: case with unimodal IFD (4.40) and unimodal Erlang memory kernel (3.33) (initial condition $S_0 = 0.20N$). Right panel: the same solution shown in the time interval $[4.9 \cdot 10^4, 5.0 \cdot 10^4]$. Parameter values: see the text.

4.6.4 Trapezoidal IF

In 2006, M. G. Roberts proposed an integral equation model for describing the transmission of a generic infectious disease invading a susceptible population [59]. The model was motivated by the global epidemic risk of SARS in 2003. Roberts assumed a trapezoidal distribution for the IF, early used by Aldis and Roberts to assess the effects of a potential smallpox epidemics [1]:

$$A(\tau) = \begin{cases} p_0 \frac{\tau - \tau_a}{(\tau_b - \tau_a)} & \tau_a < \tau < \tau_b \\ p_0 & \tau_b < \tau < \tau_c \\ p_0 \frac{\tau_d - \tau}{(\tau_d - \tau_c)} & \tau_c < \tau < \tau_d \\ 0 & \text{otherwise} \end{cases}. \quad (4.41)$$

In (4.41), the parameter p_0 weights the contacts between the infected and susceptible individuals. It is assumed that all infected individuals have the ability to transmit the infection between time intervals τ_b and τ_c after their own infection, while no transmission occurs before time τ_a and after time τ_d . The transmission probabilities between time intervals τ_a and τ_b , as well as between times τ_c and τ_d , are estimated through linear interpolation.

Here, we employ the IF (4.41) into the integral model (3.9). We also assume that an Erlang distribution with $n = 2$ describes the memory of the population, $K(\tau) = a^2 \tau e^{-a\tau}$. This appears to be somehow coherent with the fact that the available information about SARS was scarce at the time it broke out.

According to [59], we take: $R_0 = 3.3$ and $(\tau_a, \tau_b, \tau_c, \tau_d) = (4, 7, 11, 14)$ days. We also take the information parameter values $\alpha = 5 \cdot 10^4$, $a = 1/30$ and the demographic

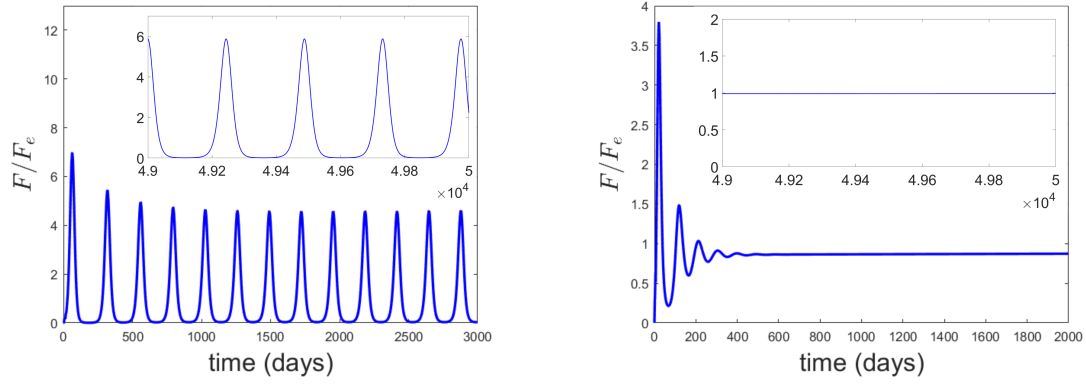


Figure 4.5: Time profile of the ratio F/F_e as predicted by model (3.9). Left panel: case with unimodal Erlang memory kernel (3.33) and IF given by (4.41). Right panel: case with weak Erlang memory kernel (3.29) and IF given by (4.42). The initial condition is $S_0 = 0.70N$. Parameter values: see the text.

parameter values $S_0 = 0.70N$, $\mu = 1/75 \text{ years}^{-1}$, and $N = 5 \cdot 10^7$. The parameter p_0 , which can be obtained from (3.15), is $p_0 = 9.4 \cdot 10^{-9}$.

In Figure 4.5, left panel, it is shown that the interplay between trapezoidal distribution (4.41) and the strong Erlang memory kernel produces oscillations that appear to be self-sustained (see Figure 4.5, left panel, top right square). In the time interval $[4.9 \cdot 10^4, 5.0 \cdot 10^4]$ we get $A_{var} \approx -0.009\%$.

In 2007, Roberts and coworkers [60] studied the transmission of influenza within a population by using the following IF:

$$\tilde{A}(\tau) = \frac{1}{T_I} A(\tau), \quad (4.42)$$

where $A(\tau)$ is given by (4.41) and T_I is the mean infectious period, $T_I = (\tau_d + \tau_c - \tau_a - \tau_b)/2$.

Here, we assume that a weak Erlang distribution describes the memory of the population, $K(\tau) = ae^{-a\tau}$. According to [60], we take: $R_0 = 3$ and $(\tau_a, \tau_b, \tau_c, \tau_d) = (1.2, 2.0, 5.3, 6.1)$ days. All the other parameter values are as in the previous experiment. The parameter p_0 in this case is $p_0 = 6.0 \cdot 10^{-8}$.

In Figure 4.5, right panel, it can be observed that the fraction F/F_e slowly converges after damped oscillations, in agreement with Theorem 3.4.3. Several numerical tests (not displayed here) show that such convergence is faster when the initial condition of the susceptibles is closer to the equilibrium value.

4.6.5 A birth-death process

Glass and Becker studied the influenza transmission within households by assuming that changes in the infectiousness of individuals may be described by a deterministic birth–death process for the growth of the virus population [34]. The death rate applies once the immune system becomes active, T_I days after infection, where T_I is assumed to correspond to the time when symptoms first appear. The proposed infectiousness profile can be obtained, for example, by the following IF:

$$A(\tau) = \begin{cases} p_0 e^{-2} e^{2.5\tau} & \tau \leq T_I \\ p_0 e^5 e^{-\tau} & \tau > T_I \end{cases}, \quad (4.43)$$

We take $T_I = 2$ and $R_0 = 3$. We also take the information parameter values $\alpha = 8 \cdot 10^4$, $a = 1/30$ and the demographic parameter values $S_0 = 0.81N$, $\mu = 1/75$ years⁻¹, and $N = 5 \cdot 10^7$. The parameter p_0 , obtained from (3.15), is $p_0 = 2.14 \cdot 10^{-9}$. In Figure 4.6, it is shown the time profile of the quantity F/F_e : in the left panel we can see the epidemic waves produced by the strong Erlang memory, $K(\tau) = a^2 \tau e^{-a\tau}$. We observe oscillations with very large amplitude (see Figure 4.6, left panel, top right square). In the time interval $[4.9 \cdot 10^4, 5.0 \cdot 10^4]$ it is $A_{var} = 0.5\%$. In the right panel damped oscillations towards a stable equilibrium may be seen in case of weak Erlang memory, $K(\tau) = a e^{-a\tau}$.

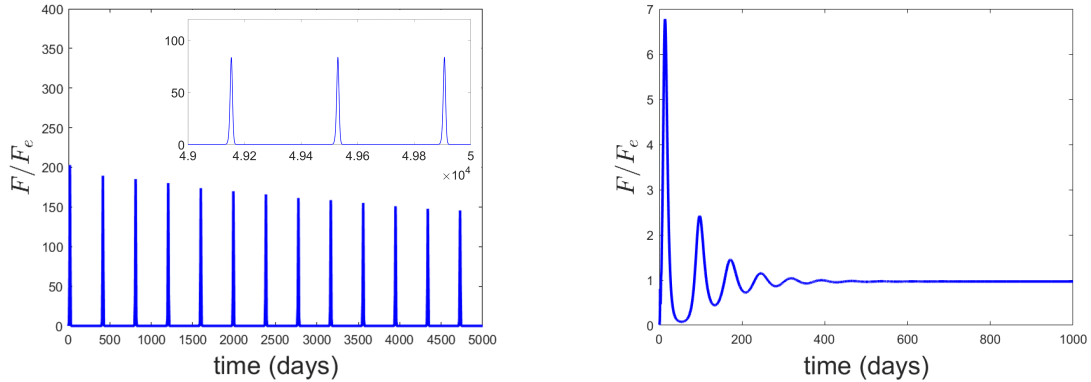


Figure 4.6: Time profile of the Force of infection F/F_e according to model (3.9). Left panel: case with strong Erlang memory kernel, i.e. $K(\tau) = a^2 \tau e^{-a\tau}$ and IF given by (4.43). Right panel: case with weak Erlang memory kernel, i. e. $K(\tau) = a e^{-a\tau}$ and IF given by (4.43). The initial condition is $S_0 = 0.81N$. Parameter values: see the text.

Chapter 5

Connections Between Compartmental and Integral Epidemic Models

This chapter provides a detailed analysis of the connection between the integral epidemic model studied in the previous chapters and compartmental epidemic models [15]. In particular, it begins with a theoretical result showing how a system of ODEs can be derived from an integral equation. This is followed by an overview of compartmental epidemic models with information index reported in the literature, which clarifies the foundations on which the integral model was built.

In the second part of the chapter, we introduce and analyze a novel integral epidemic model, emphasizing its connection with the corresponding compartmental formulation. To numerically illustrate the resulting solution profiles, the direct trapezoidal quadrature method (see Section 2.2) is employed. Since the objective of this chapter is to highlight and analyze the connection between integral and compartmental epidemic models, the simulations are intended solely to visualize the qualitative behaviour of the solutions, rather than to provide an in-depth numerical study. For this reason, the trapezoidal rule was employed to obtain more accurate solutions over long time horizons, disregarding specific intrinsic properties of the scheme, such as positivity preservation, that fall outside the scope of this chapter. More broadly, this chapter seeks to establish the integral formulation as a natural extension of classical epidemic models while introducing a novel theoretical contribution that enhances the descriptive capabilities of integral models within socio-epidemiological frameworks.

5.1 From integral to compartmental models

In this section we seek to clarify the mathematical relationship between integral and compartmental epidemic models. For simplicity, we refer to the case without human behaviour and consider the constitutive equation for $\hat{F}(t)$ presented in Section 3.1. The scalar integral equation (3.1) is recalled below in the following form:

$$\hat{F}(t) = \int_{-\infty}^t S(\tau) \hat{F}(\tau) A_{\mu}(t - \tau) d\tau, \quad (5.1)$$

where $A_{\mu}(\tau) = e^{-\mu\tau} A(\tau)$. The definitions of the parameters and functions involved are provided in Tables 3.1–3.2. Following the procedure introduced in [23], we assume that there exist an integer d and matrices $U \in \mathbf{R}^{1 \times d}$, $V \in \mathbf{R}^{d \times 1}$, $\Sigma \in \mathbf{R}^{d \times d}$, such that the IF is given by

$$A(\tau) = U e^{\tau \Sigma} V. \quad (5.2)$$

By deriving with respect to time t , the integral equation (5.1), with the IF given in (5.2), reduces to the following system of ODEs:

$$\dot{\mathbf{Y}} = \Sigma \mathbf{Y} + (\hat{F} S) V, \quad (5.3)$$

where

$$\mathbf{Y}(t) := \int_{-\infty}^t e^{(t-\tau)\Sigma} V \hat{F}(\tau) S(\tau) d\tau,$$

and $\hat{F} = U \mathbf{Y}$. In order to obtain a closed nonlinear system of ordinary differential equations for the $(d+1)$ -vector $(S, \mathbf{Y})$, it is necessary to add a further differential equation for S in (5.3), for example the one provided in (3.2). Within this framework, a wide range of epidemic models involving multiple stages of infectiousness can be derived from system constituted by (3.2)–(5.3).

The proposition below specializes the general result previously discussed to the integral behavioural model presented in Chapter 3.

Proposition 5.1.1. *If the IF is exponentially distributed, then model (3.9) is the behavioural SIR–M model introduced in [24].*

Proof. Let us take $A(\tau) = \beta_0 e^{-\nu\tau}$, where β_0 and ν are two positive constants defined in Table 3.1, respectively. Set

$$I(t) = \int_0^{\infty} e^{-(\mu+\nu)\tau} \beta(M(t-\tau)) F(t-\tau) S(t-\tau) d\tau.$$

Substitution of $A_{\mu}(\tau) = \beta_0 e^{-(\mu+\nu)\tau}$ into the second equation of model (3.9) leads to

$F(t) = \beta_0 I(t)$. It follows:

$$\begin{aligned} \frac{dI(t)}{dt} &= -(\mu + \nu) \int_0^\infty e^{-(\mu+\nu)\tau} \beta(M(t-\tau)) F(t-\tau) S(t-\tau) d\tau + \beta(M(t)) F(t) S(t) \\ &= -(\mu + \nu) I(t) + \beta_0 \beta(M(t)) I(t) S(t). \end{aligned}$$

By denoting $\hat{\beta}(M) = \beta_0 \beta(M)$, we get

$$\dot{I} = -(\mu + \nu) I + \hat{\beta}(M) S I. \quad (5.4)$$

Equations (3.7), (3.8) and (5.4) constitute the extensively studied behavioural SIR–M model introduced by d’Onofrio and Manfredi [24, 25, 26]. $\square$

To visualize the results established in Proposition 5.1.1, we refer to the numerical experiments previously conducted for model (3.9) in Section 4.6.3. These experiments assume an exponentially distributed IFD, as given in (4.39), and the outcomes are summarized in Figure 4.3.

As proved in Proposition 5.1.1, under these conditions model (3.9) reduces to the SIR–M model originally studied by d’Onofrio and Manfredi in 2009 to investigate the sustained oscillations observed in various diseases such as measles [24]. It is well established that the endemic equilibrium of the SIR–M model is locally asymptotically stable when the memory kernel corresponds to an exponentially fading Erlang distribution [24, 69]. It is noteworthy that this result is also recovered through simulations performed directly from the integral formulation (3.9) (see Figure 4.3, left panel).

The second numerical experiment described in Section 4.6.3, assumes that the memory kernel follows an Erlang distribution with parameter $n = 2$, given by $K(\tau) = a^2 \tau e^{-a\tau}$. Under this assumption, it has been shown that when the IFD is given by (4.39), the endemic equilibrium of the corresponding SIR–M model may be destabilized via Hopf bifurcations, resulting in stable recurrent oscillations [24, 26]. As illustrated in Figure 4.3, right panel, this behaviour is likewise reproduced by the integral model, where oscillations emerge and, following an initial transient phase, appear to be self-sustained.

In the following sections, we provide a more detailed overview of the main results on *minimal* ODEs models incorporating information-dependent contact patterns [24, 26], which were only briefly mentioned earlier. By “minimal,” we refer to models of reduced mathematical complexity, in which the epidemic dynamics are represented by slight variations of the basic SIR or SEIR models. While compartmental models

applied to real epidemics often exhibit considerable complexity, they can be regarded as extensions of these minimal models.

5.2 SIR model with prevalence-based social contact pattern

5.2.1 SIR behavioural model

Let us consider the case in which the message function g depends only on the prevalence of the disease, i.e. $g = g(I(t))$. Assume also that the contact rate of individuals depends on the information index defined in (3.3). These assumptions imply that individuals adjust their contact pattern in response to the perceived risks associated with the disease, which, in turn, is “measured” in terms of the disease prevalence. Using a SIR model with demography as the baseline model, one obtains the model introduced in 2009 by A. d’Onofrio and P. Manfredi [24]:

$$\begin{aligned}\dot{S} &= \Lambda - \mu S - \hat{\beta}(M)SI, \\ \dot{I} &= \hat{\beta}(M)SI - (\mu + \nu)I, \\ M(t) &= \int_0^\infty g(I(t - \tau)) H(t - \tau) K(\tau) d\tau.\end{aligned}\tag{5.5}$$

The first and second equations in (5.5) govern the dynamics of the susceptible (S) and infected (I) populations, respectively. In (5.5), the FoI is given by

$$\text{FoI}(M) = \hat{\beta}(M)I.\tag{5.6}$$

The function $\hat{\beta}(M)$ satisfies the following assumptions:

$$\hat{\beta}(0) = \beta_0, \quad \hat{\beta}(M) > 0, \quad \hat{\beta}'(M) < 0,\tag{5.7}$$

where the positive constant β_0 is the baseline transmission rate defined in Table 3.1. All model parameters and functions used in this chapter follow the interpretations and assumptions set forth in Section 3.1. The third equation in system (5.5) describes the evolution of the information index under the stated assumptions. It is easy to show that model (5.5) admits the *disease-free* equilibrium

$$\text{DFE} = \left(\frac{\Lambda}{\mu}, 0, 0 \right),\tag{5.8}$$

and that a classical threshold result can be stated: if $R_0 < 1$, then the DFE is

globally asymptotically stable (GAS); if $R_0 > 1$, then it is unstable, where $R_0 = \beta_0/(\mu + \nu)$, is the basic reproduction number. Furthermore, when $R_0 > 1$, model (5.5) admits a unique *endemic* equilibrium $EE = (S_e, I_e, M_e)$, where

$$S_e = \frac{\mu + \nu}{\hat{\beta}(g(I_e))}, \quad M_e = g(I_e), \quad (5.9)$$

and $I_e > 0$ is the unique solution of the equation:

$$\mu \left(1 - \frac{\mu + \nu}{\hat{\beta}(g(I))} \right) - (\mu + \nu)I = 0. \quad (5.10)$$

In their paper, d'Onofrio and Manfredi show that the stability of EE depends on the shape of the memory kernel K [24]. Therefore, they conclude that, under certain circumstances, human choices may be a key factor behind the occurrence of recurrent epidemics. In the next subsections, we give some details on this matter.

5.2.2 Prevalence-based SIR model with exponential fading memory

Consider an exponentially fading memory kernel given by the Erlang distribution with $n = 1$, $K(t) = \text{Erl}_{1,a}(t)$, introduced in (3.29).

The model (5.5) with (3.29) can be written as an ODEs model using the linear chain trick:

$$\begin{aligned} \dot{S} &= \Lambda - \mu S - \hat{\beta}(M)SI, \\ \dot{I} &= \hat{\beta}(M)SI - (\mu + \nu)I, \\ \dot{M} &= a(g(I) - M). \end{aligned} \quad (5.11)$$

The linear stability analysis of EE goes through the Jacobian matrix evaluated at EE:

$$J(EE) = \begin{pmatrix} -\mu - \hat{\beta}(M_e)I_e & -\hat{\beta}(M_e)S_e & -\hat{\beta}'(M_e)S_e I_e \\ \hat{\beta}(M_e)I_e & 0 & \hat{\beta}'(M_e)S_e I_e \\ 0 & ag'(I_e) & -a \end{pmatrix}.$$

The characteristic polynomial is given by:

$$p(\lambda) = \lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0,$$

where the coefficients a_0 , a_1 , and a_2 are given by: $a_0 = aI_eS_e(\mu g'(I_e)\hat{\beta}'(M_e) - \hat{\beta}^2(M_e))$, $a_1 = a\mu + a\hat{\beta}(M_e)I_e + \hat{\beta}^2(M_e)S_eI_e - ag'(I_e)\hat{\beta}'(M_e)S_eI_e$, and $a_2 = \mu + a +$

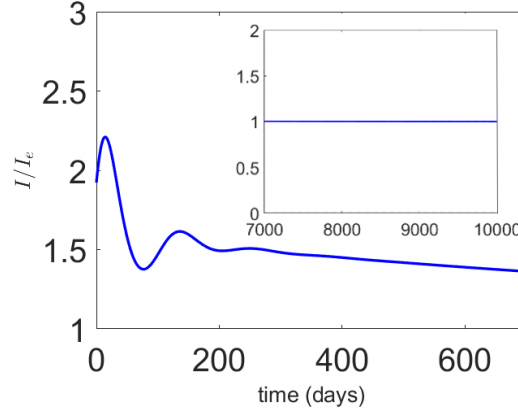


Figure 5.1: Time profile of the ratio I/I_e as predicted by model (5.11) with exponential fading memory kernel (3.29) and $g(I) = kI$, $\hat{\beta}(M) = \beta_0/(1 + \alpha M)$. The parameter values are $R_0 = 20$, $\beta_0 = R_0(\mu + \nu)$, $\nu = 1/7 \text{ days}^{-1}$, $\mu = 1/75 \text{ years}^{-1}$, $N = 6 \cdot 10^7$, $\lambda = \mu N$, $k = 1$, $\alpha = 1$, $a = 1/30 \text{ days}^{-1}$.

$$\hat{\beta}(M_e)I_e.$$

According to the well-known Routh-Hurwitz criterion [48], all roots of the characteristic polynomial $p(\lambda)$ will have negative real parts if and only if the following conditions are satisfied:

$$a_2 > 0, \quad a_0 > 0, \quad a_2 a_1 > a_0.$$

These conditions provide a stability criteria for EE in terms of the model parameters and the functions $\hat{\beta}(M)$ and $g(I)$. In this case, the three Routh-Hurwitz criteria are met due to the properties of g and the assumption given in Table 3.2. Therefore, the equilibrium EE is locally asymptotically stable.

The time profile of the solution of model (5.11) when $g(I) = kI$, $\hat{\beta}(M) = \beta_0/(1 + \alpha M)$ is provided in Figure 5.1. Note that in this formulation, the parameter k is the *information coverage* that represent the phenomenon of under-reporting [14].

5.2.3 Prevalence-based SIR model with unimodal memory

Consider now a memory kernel given by the unimodal Erlang distribution introduced in (3.33).

Model (5.5) with (3.33), using again the linear chain trick, becomes:

$$\begin{aligned}
 \dot{S} &= \Lambda - \mu S - \hat{\beta}(M)SI, \\
 \dot{I} &= \hat{\beta}(M)SI - (\mu + \nu)I, \\
 \dot{M}_1 &= a(g(I) - M_1), \\
 \dot{M} &= a(M_1 - M),
 \end{aligned} \tag{5.12}$$

where M_1 is the auxiliary variable in (3.5). The local asymptotic stability of EE = (S_e, I_e, M_{1e}, M_e) derived in (5.9) and (5.10) with $M_{1e} = M_e$, can be assessed through the following Jacobian matrix:

$$J(\text{EE}) = \begin{pmatrix} -\mu - \hat{\beta}(M_e)I_e & -\hat{\beta}(M_e)S_e & 0 & -\hat{\beta}'(M_e)S_e I_e \\ \hat{\beta}(M_e)I_e & 0 & 0 & \hat{\beta}'(M_e)S_e I_e \\ 0 & ag'(I_e) & -a & 0 \\ 0 & 0 & a & -a \end{pmatrix}.$$

The characteristic polynomial $p(\lambda)$ of J is

$$p(\lambda) = \lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0,$$

where $a_4 = 1$, $a_3 = 2a + \mu + \hat{\beta}(M_e)I_e$, $a_2 = a^2 + 2a(\mu + \hat{\beta}(M_e)I_e) - (\mu + \nu)\hat{\beta}(M_e)S_e$, $a_1 = a^2(\mu + \hat{\beta}(M_e)I_e) - 2a(\mu + \nu - \hat{\beta}(M_e)I_e - a^2(\mu + \nu)\hat{\beta}'(M_e)g'(I_e)I_e/\hat{\beta}(M_e))$, and $a_0 = -a^2(\mu + \hat{\beta}(M_e)I_e) - a^2\mu(\mu + \nu)\hat{\beta}'(M_e)g'(I_e)I_e/\hat{\beta}(M_e)$.

Using the Routh-Hurwitz criterion, all roots of the characteristic polynomial $p(\lambda)$ will have negative real parts if and only if the following conditions are satisfied:

$$a_i > 0, \quad \forall i = 0, \dots, 4; \quad a_2a_1 - a_3a_0 > 0; \quad a_3a_2a_1 - a_4a_1^2 - a_3^2a_0 > 0. \tag{5.13}$$

As shown by d'Onofrio and Manfredi, it is possible, in principle, to analytically characterize the local stability of the endemic state for the four-dimensional system above. However, even for simple choices of the functions $g(I)$ and $\hat{\beta}(M)$, the analysis becomes quite cumbersome. In [24], they prove some partial results: the local asymptotic stability of EE is preserved if the rate parameter a is very small or very large, but for intermediate values of a , sustained oscillations occur. Figure 5.2 illustrates an example of oscillating time profile of the ratio I/I_e .

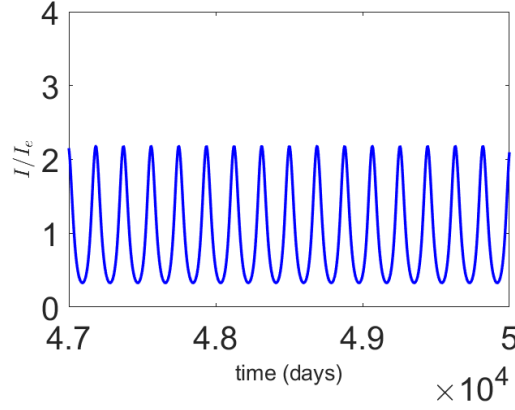


Figure 5.2: Time profile of the ratio I/I_e as predicted by model (5.12) with unimodal memory kernel (3.33) and $g(I) = kI$, $\hat{\beta}(M) = \beta_0/(1 + \alpha M)$. The parameter values are $R_0 = 20$, $\nu = 1/7 \text{ days}^{-1}$, $\mu = 1/75 \text{ years}^{-1}$, $\beta_0 = R_0(\mu + \nu)$, $N = 6 \cdot 10^7$, $\Lambda = \mu N$, $k = 1$, $\alpha = 1$, $a = 1/30 \text{ days}^{-1}$.

5.3 SIR model with incidence-based social contact pattern

5.3.1 SIR behavioural model

Some behavioural epidemiology models associate the response of individuals to the incidence of the disease (i.e. the new cases) rather than prevalence, since in the practice the prevalence often remains an unobservable factor. In the SIR model (5.5) the incidence is given by the quantity $\hat{\beta}(M)SI$. Therefore, assuming that the risk perception is linearly dependent on available information on infection incidence, one gets [26]:

$$\begin{aligned} \dot{S} &= \Lambda - \mu S - \hat{\beta}(M)SI, \\ \dot{I} &= \hat{\beta}(M)SI - (\mu + \nu)I, \\ M(t) &= \int_0^\infty k\hat{\beta}(M(t-\tau))S(t-\tau)I(t-\tau)H(t-\tau)K(\tau)d\tau. \end{aligned} \tag{5.14}$$

In model (5.14) the information coverage $k \in (0, 1]$ modulates the level of incidence perceived by individuals. The algebraic system of equilibria reads:

$$\begin{aligned} 0 &= \Lambda - \mu S_e - \hat{\beta}(M_e)S_e I_e, \\ 0 &= I_e(\hat{\beta}(M_e)S_e - (\mu + \nu)), \\ M_e &= k\hat{\beta}(M_e)S_e I_e. \end{aligned}$$

From this, it can be deduced two possible cases: (i) $I_e = 0$, which corresponds to

the DFE defined in (5.8). It is possible to show that $R_0 < 1$ implies that the DFE is globally asymptotically stable, and $R_0 > 1$ implies that DFE is unstable; (ii) $\hat{\beta}(M_e)S_e = \mu + \nu$, which corresponds to the EE given by

$$S_e = \frac{\mu + \nu}{\hat{\beta}(M_e)}, \quad M_e = k(\mu + \nu)I_e, \quad (5.15)$$

and $I_e > 0$ is the solution of the equation:

$$\Lambda - \frac{\mu(\mu + \nu)}{\hat{\beta}(k(\mu + \nu)I)} - (\mu + \nu)I = 0. \quad (5.16)$$

The EE only exists when $R_0 > 1$.

5.3.2 Incidence-based SIR model with exponential fading memory

When the memory kernel is given by the first-order Erlang distribution (3.29), model (5.14) becomes:

$$\begin{aligned} \dot{S} &= \Lambda - \mu S - \hat{\beta}(M)SI, \\ \dot{I} &= \hat{\beta}(M)SI - (\mu + \nu)I, \\ \dot{M} &= a(k\hat{\beta}(M)SI - M). \end{aligned}$$

Following the same steps in Subsection 5.2.2, it can be easily shown that the EE given in (5.15) and (5.16) is locally asymptotically stable regardless of the parameter values [26].

5.3.3 Incidence-based SIR model with acquisition-fading memory

d'Onofrio and Manfredi [26] considered the acquisition-fading memory kernel to represent the circumstance that the information acquisition and the information fading are two processes with much different timescales, the former being much faster than the latter. Such a unimodal memory kernel takes the form:

$$K(t) = \frac{1}{T_b - T_a} \left(e^{-t/T_b} - e^{-t/T_a} \right), \quad (5.17)$$

where T_a and T_b represent the two time scales of the two processes. In particular, T_a represents the timescale of the first process, which is the formation and acquisition of information, while T_b corresponds to the timescale of the second process, which

governs the fading of the acquired information. It is straightforward to observe that $K(0) = 0$, that reflects the negligible use of current information. Furthermore, if $T_a \rightarrow 0$, then $K(t) \approx Erl_{1,b}$ and if $T_b \rightarrow T_a$ then $K(t) \approx Erl_{2,a}$. Applying the linear chain trick, the model (5.14) with (5.17) becomes:

$$\begin{aligned}\dot{S} &= \Lambda - \mu S - \hat{\beta}(M)SI, \\ \dot{I} &= \hat{\beta}(M)SF - (\mu + \nu)I, \\ \dot{M}_1 &= a(k\hat{\beta}(M)SI - M_1), \\ \dot{M} &= b(M_1 - M),\end{aligned}\tag{5.18}$$

where, again, M_1 is the auxiliary variable in (3.5). The linear stability of EE defined in (5.15) and (5.16) follows from the analysis of the eigenvalues of the Jacobian matrix:

$$J(\text{EE}) = \begin{pmatrix} -\mu - \hat{\beta}(Me)I_e & -\hat{\beta}(Me)S_e & 0 & -\hat{\beta}'(Me)S_eI_e \\ \hat{\beta}(Me)I_e & 0 & 0 & \hat{\beta}'(Me)S_eI_e \\ ak\hat{\beta}(Me)I_e & ak\hat{\beta}(Me)S_e & a & ak\hat{\beta}'(Me)S_eI_e \\ 0 & 0 & b & -b \end{pmatrix}.$$

The characteristic polynomial is

$$P(\lambda) = \sum_{i=0}^4 q_i(a, b)\lambda^i,$$

where:

$$\begin{aligned}q_0(a, b) &= ab\hat{\beta}(Me)S_e(\hat{\beta}(Me)I_e - \mu k\hat{\beta}'(Me)S_eI_e), \\ q_1(a, b) &= ab\left(\mu + \hat{\beta}(Me)I_e - (\mu - \hat{\beta}(Me)S_e)k\hat{\beta}'(Me)S_eI_e - \hat{\beta}^2(Me)S_eI_e(a + b)\right), \\ q_2(a, b) &= ab(1 - k\hat{\beta}'(Me)S_eI_e) + (\hat{\beta}(Me)I_e + \mu)(a + b) - \hat{\beta}^2(Me)S_eI_e, \\ q_3(a, b) &= a + b + \hat{\beta}(Me)I_e + \mu, \\ q_4(a, b) &= 1.\end{aligned}$$

The third Routh-Hurwitz condition in (5.13) is violated for intermediate values of a and b . Therefore, EE destabilizes and sustained oscillations occur. Figure 5.3 shows the oscillatory dynamics when $g(I) = kI$, $\hat{\beta}(M) = \beta_0(1 - qM)_+$. Note that this piecewise-linear behavioural response, where q is the reactivity factor as in Subsection 5.3.2, has been used in [26] to represent a threshold value of the information index above which the risk of infection is zero. Note also that in case $a = b$ the memory kernel reduces to the second-order Erlang distribution $K(t) =$

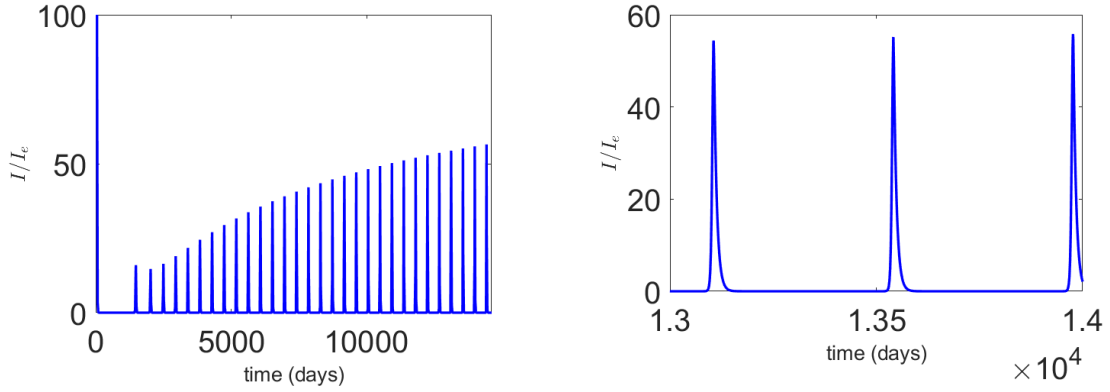


Figure 5.3: Time profile of the ratio I/I_e as predicted by model (5.18) with unimodal memory kernel (5.17) and $g(I) = kI$, $\hat{\beta}(M) = \beta_0(1 - qM)_+$. The parameter values are $R_0 = 15$, $\nu = 1/7$ days $^{-1}$, $\mu = 1/50$ years $^{-1}$, $\beta_0 = R_0(\mu + \nu)$, $N = 6 \cdot 10^7$, $\lambda = \mu N$, $k = 1$, $q = 1.96 \cdot 10^5$, $a = 1/30$ days $^{-1}$, $b = 0.1a$.

$Erl_{2,a}(t)$. In such a case, all the previous results continue to hold.

5.4 A novel integral model with incidence-based social contact pattern

Analogously to the approach adopted for the SIR behavioural model in Section 5.3, we now formulate the integral behavioural model under the assumption that individuals adjust their contact patterns in response to disease incidence rather than prevalence. The integral epidemic model with a prevalence-based contact pattern has been extensively discussed in the previous chapters: introduced and analyzed in Chapter 3, and discretized in Chapter 4.

We now present this new incidence-based integral model, which, as in the case of the prevalence-based model, generalizes compartmental models with incidence-dependent social contact patterns by allowing the inclusion of a generic infectivity function. The formulation of the integral behavioural epidemic model is given by:

$$\begin{aligned} \dot{S}(t) &= \Lambda - \mu S(t) - \beta(M(t))S(t)F(t), \\ F(t) &= \int_0^\infty \beta(M(t-\tau))S(t-\tau)F(t-\tau)A_\mu(\tau) d\tau, \\ M(t) &= \int_0^\infty k\beta(M(t-\tau))S(t-\tau)F(t-\tau)H(t-\tau)K(\tau) d\tau. \end{aligned} \tag{5.19}$$

The difference between model (5.19) and model (3.9) is that now the information index M is the weighted sum of current incidence, given by $k\beta(M)SF$, and past incidence trends. We also assume, from now on, that (4.8) holds.

Let $\mathbf{u}(t) = (S(t), F(t), M(t))$ denote the solution of model (5.19), and let D represent the region given by

$$D = \{(x, y, z) : 0 < x \leq S_{\max}, 0 \leq y \leq F_{\max}, 0 \leq z \leq M_{\max}\}, \quad (5.20)$$

where:

$$S_{\max} = \frac{\Lambda}{\mu}, \quad F_{\max} = \Lambda \hat{A}_{\mu}(0) + \frac{\Lambda}{\mu} \int_0^{\infty} |A'_{\mu}(\tau)| d\tau, \quad M_{\max} = k F_{\max} \frac{\Lambda}{\mu},$$

and $\hat{A}_{\mu}(0)$, defined in Table 3.2, is $\hat{A}_{\mu}(0) = \int_0^{\infty} A_{\mu}(\tau) d\tau$. To study the basic properties of the solution $\mathbf{u}(t)$, we suppose that, over the interval $(-\infty, 0]$, the state variables S , F and M are bounded functions and $0 < S(t) \leq \Lambda/\mu$, $F(t) \geq 0$, $M(t) \geq 0$. The next result shows that these properties hold true for all $t \in \mathbb{R}$.

Proposition 5.4.1. *Assume that $\lim_{\tau \rightarrow \infty} A(\tau) = 0$, then the region D defined in (5.20) is positively invariant for model (3.9).*

Proof. The positivity and the bounds for S and F can be obtained by direct integration of the first equation in (3.9) and comparison arguments, as in Theorems 3.2.1 and 3.2.2. The bounds for M are easily obtained from the fact that $\beta(\cdot)$ is a positive decreasing function and that $\beta(0) = 1$. $\square$

Model (3.9) admits the *Disease-Free* Equilibrium (DFE) given by

$$\text{DFE} = (\bar{S}_0, 0, 0), \quad (5.21)$$

where $\bar{S}_0 = \Lambda/\mu$. An Endemic Equilibrium (EE) is given by a triple of constants

$$\text{EE} = (S_e, F_e, M_e),$$

obtained from equalities:

$$\Lambda - \mu S_e - \beta(M_e) S_e F_e = 0, \quad \beta(M_e) S_e \hat{A}_{\mu}(0) = 1, \quad M_e = k \beta(M_e) S_e F_e. \quad (5.22)$$

The existence and uniqueness of EE may be written in terms of the basic reproduction number (3.15), as stated in the following Proposition.

Proposition 5.4.2. *If $R_0 < 1$, then the model (3.9) has no endemic equilibria in D ; if $R_0 > 1$, then model (3.9) has a unique endemic equilibrium in D .*

Proof. Let us introduce the function:

$$G(x) = r_1(x) - r_2(x),$$

where

$$r_1(x) = \frac{\mu}{\mu R_0 - x}, \quad r_2(x) = \beta \left(\frac{\Lambda k x}{\mu R_0} \right).$$

Using (5.22) and (3.15) one gets:

$$M_e = \frac{\Lambda k F_e}{\mu R_0}, \quad S_e = \frac{\Lambda}{\mu R_0 \beta(M_e)},$$

and $F_e \in (0, F_{max}]$ such that $G(F_e) = 0$.

Since

$$r'_1(x) > 0, \quad r_1(0) = 1/R_0, \quad r'_2(x) < 0, \quad r_2(0) = 1,$$

it follows that if $R_0 > 1$, then $r_2(0) > r_1(0)$ and $G(x)$ has a unique zero in $[0, F_{max}]$. Finally, it is straightforward to check that $G(x)$ has no zeros in $[0, F_{max}]$ when $R_0 < 1$. $\square$

We now briefly discuss the stability of equilibria through the application of the linearisation theory for Volterra integral equations [55] introduced in Section 2.1. The following proposition provides a sufficient condition for the local asymptotic stability of equilibria.

Proposition 5.4.3. *Any equilibrium $\tilde{E} = (\tilde{S}, \tilde{F}, \tilde{M})$ of model (3.9) is locally asymptotically stable if the characteristic equation*

$$\beta(\tilde{M})\tilde{S}\hat{A}_\mu(w) + k\beta'(\tilde{M})\tilde{F}\tilde{S}\hat{K}(w) = 1 + \frac{\beta(\tilde{M})\tilde{F}}{w + \mu}, \quad (5.23)$$

has no solution for $Re(w) \geq 0$.

Proof. (Sketch) We limit ourselves to observe that equation (3.23) is equivalent to the characteristic equation (3.22). The statement may be proven through direct application of the Paley-Wiener Theorem [44]. $\square$

In the following, we apply Proposition 3.3.1 to some special cases of interest. In our analysis, we assume that $Re(w) > 0$. All results can be easily extended to $Re(w) \geq 0$, by passing to the limit as $\tilde{w} \rightarrow w$, with $Re(\tilde{w}) > 0$.

At the DFE (3.12) the characteristic equation (3.23) simply reduces to $\bar{S}_0\hat{A}_\mu(w) = 1$ and $Re(w) > 0$ implies

$$\left| \frac{\Lambda}{\mu} \hat{A}_\mu(w) \right| \leq \frac{\Lambda}{\mu} \hat{A}_\mu(0).$$

Therefore, from Proposition 3.3.1 and (3.15), if $R_0 < 1$ the DFE is locally asymptotically stable. For the DFE we also prove the following global result.

Proposition 5.4.4. *If the IF satisfy $\lim_{\tau \rightarrow \infty} A(\tau) = 0$, and $R_0 < 1$, then the DFE is Globally Asymptotically Stable (GAS).*

Proof. Since for $R_0 < 1$ the DFE is locally asymptotically stable, we need to prove its attractiveness. Consider equation (5.19)₂ in the form

$$F(t) = F_0(t) + \int_0^t \beta(M(t-\tau))S(t-\tau)F(t-\tau)A_\mu(\tau)d\tau,$$

where

$$F_0(t) = \int_t^\infty \beta(M(t-\tau))S(t-\tau)F(t-\tau)A_\mu(\tau)d\tau.$$

Since $\beta(\cdot) \leq 1$ and $S(\cdot) \leq \Lambda/\mu$, then

$$F(t) \leq F_0(t) + \frac{\Lambda}{\mu} \int_0^t F(\tau)A_\mu(t-\tau)d\tau.$$

Consider $\bar{t} < t$, we have

$$F(t) \leq F_0(t) + \frac{\Lambda}{\mu} \int_0^{\bar{t}} F(\tau)A_\mu(t-\tau)d\tau + \frac{\Lambda}{\mu} \sup_{t \geq \bar{t}} F(t) \int_0^{t-\bar{t}} A_\mu(\tau)d\tau,$$

and, by applying the limit superior to both sides, we obtain

$$\limsup_{t \rightarrow \infty} F(t) \leq \frac{\Lambda}{\mu} \hat{A}_\mu(0) \sup_{t \geq \bar{t}} F(t).$$

Passing to the limit as $\bar{t} \rightarrow \infty$, in the previous expression, it is

$$(1 - R_0) \limsup_{t \rightarrow \infty} F(t) \leq 0,$$

and the result comes straightforward considering $R_0 < 1$. $\square$

As for the stability of the endemic equilibrium EE, the next proposition gives a sufficient condition for its local asymptotic stability provided that the memory kernel K has one of the following two forms: Dirac delta and exponentially fading memory.

Proposition 5.4.5. *If $K(t) = \delta(t)$ or $K(t) = \text{Erl}_{1,a}(t)$, then EE is LAS for any choice of the functions β and A_μ .*

Proof. Let be $\text{Re}(w) > 0$. First of all observe that:

$$|\beta(M_e)S_e\hat{A}_\mu(w)| \leq 1, \quad \text{Re}\left(1 + \frac{\beta(M_e)F_e}{w + \mu}\right) > 1, \quad (5.24)$$

where the first inequality comes from condition (5.22)₂ and the second one is a direct consequence of $\text{Re}(w) > 0$.

When $K(t) = \delta(t)$, the characteristic equation (5.23) becomes:

$$\beta(M_e)S_e\hat{A}_\mu(w) = 1 + \frac{\beta(M_e)F_e}{w + \mu} - k\beta'(M_e)F_eS_e. \quad (5.25)$$

In view of (5.24), since $\beta'(\cdot) < 0$, the real part of the l.h.s. of (5.25) is less than 1, while the real part of the r.h.s. is strictly greater than 1. Therefore, equation (5.25) cannot be satisfied.

If $K(t) = ae^{-at}$, then $\hat{K}(w) = a/(a + w)$ and from (5.23) one gets:

$$\beta(M_e)S_e\hat{A}_\mu(w) = 1 + \frac{\beta(M_e)F_e}{w + \mu} - k\beta'(M_e)F_eS_e\frac{a}{a + w},$$

where, analogously to the previous case, the real part of the r.h.s. is greater than one. $\square$

5.4.1 Numerical simulations

In this section, we perform numerical simulations to show the dynamics of model (5.19) when specific infectivity functions are selected. Let us first write the model (5.19) as:

$$\begin{aligned} S(t) &= S_0 + \int_0^t (\Lambda - \mu S(t - \tau) - \beta(M(t - \tau))S(t - \tau)F(t - \tau))d\tau, \\ F(t) &= F_0(t) + \int_0^t \beta(M(t - \tau))S(t - \tau)F(t - \tau)A_\mu(\tau)d\tau, \\ M(t) &= M_0(t) + k \int_0^t \beta(M(t - \tau))S(t - \tau)F(t - \tau)K(\tau)d\tau, \end{aligned} \quad (5.26)$$

where $S_0 > 0$ is the initial size of susceptible population and $F_0(t)$ and $M_0(t)$ are known initial functions. Following the approach outlined in Section 4.6.1 for defining the initial conditions, we also set: $F_0(t) = (\Lambda - \mu S_0)A_\mu(t)$, and, consequently, $M_0(t) = (\Lambda - \mu S_0)K(t)$. The simulations are obtained by discretizing model (5.26)

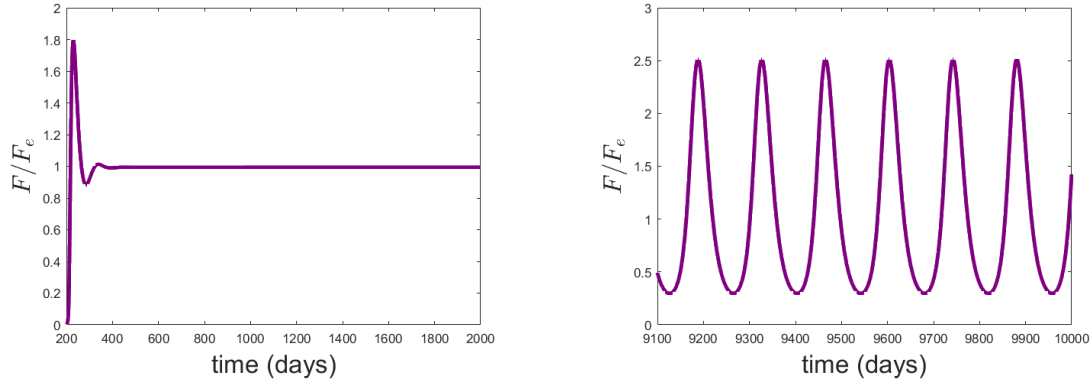


Figure 5.4: Time profile of the ratio F/F_e as predicted by model (5.19) with $\beta(M) = 1/(1 + \alpha M)$ and IFD given by (4.39). Left panel: case with $K(t) = \text{Erl}_{1,a}(t)$. Right panel: case with $K(t) = \text{Erl}_{2,a}(t)$. The parameter values are: $R_0 = 20$, $\nu = 1/7 \text{ days}^{-1}$, $N = 6 \cdot 10^7$, $\mu = 1/75 \text{ years}^{-1}$, $\Lambda = \mu N$, $\beta_0 = R_0(\mu + \nu)/N$, $a = 1/30 \text{ days}^{-1}$, $k = 1$, and $\alpha = 5 \cdot 10^3$.

via trapezoidal Direct Quadrature method [13], described in Section 2.2.

The first case we examine is the exponential IFD (4.39). Note that in this case model (5.19) becomes the SIR–M model studied by d’Onofrio and Manfredi [26]. The time profile of the solution of model (5.19) is shown in Figure 5.4. In the left panel, we see the convergence to the locally asymptotically stable endemic equilibrium when $K(t) = \text{Erl}_{1,a}(t)$, in agreement with Proposition 5.4.5. In the right panel, we see sustained oscillations in the case of memory kernel given by $K(t) = \text{Erl}_{2,a}(t)$.

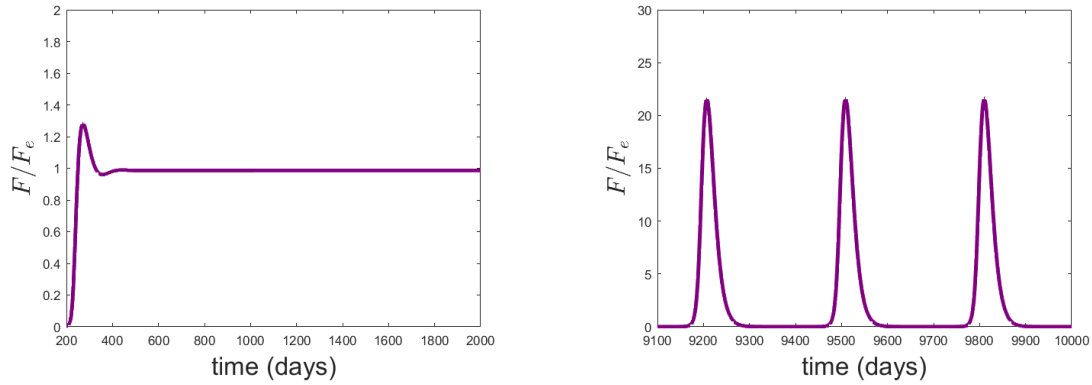


Figure 5.5: Time profile of the ratio F/F_e as predicted by model (5.19) with $\beta(M) = 1/(1 + \alpha M)$ and IFD given by (5.27). Left panel: case with $K(t) = \text{Erl}_{1,a}(t)$. Right panel: case with $K(t) = \text{Erl}_{3,a}(t)$. The parameter values are: $R_0 = 20$, $\nu = 1/7 \text{ days}^{-1}$, $N = 6 \cdot 10^7$, $\mu = 1/75 \text{ years}^{-1}$, $\Lambda = \mu N$, $\beta_0 = R_0/N$, $a = 1/30 \text{ days}^{-1}$, $k = 1$, and $\alpha = 5 \cdot 10^3$.

As a second case, we consider a unimodal IFD:

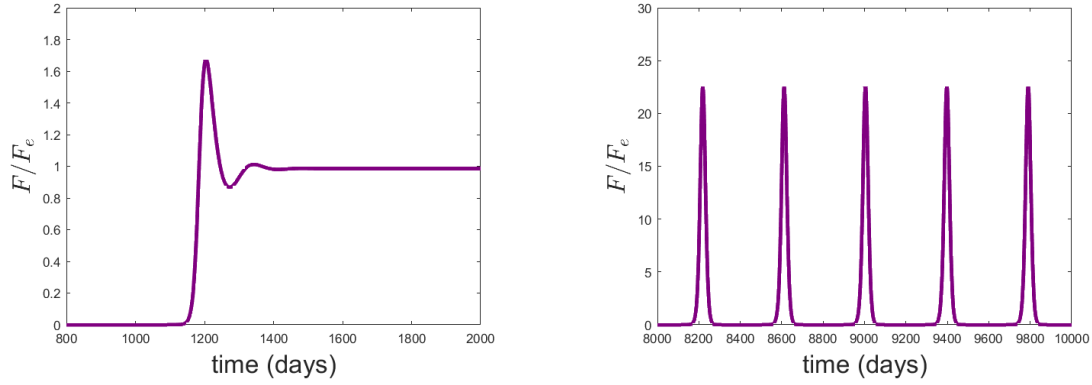


Figure 5.6: Time profile of the ratio F/F_e as predicted by model (5.19) with $\beta(M) = 1/(1 + \alpha M)$ and IF given by (4.41). Left panel: case with $K(t) = \text{Erl}_{1,a}(t)$. Right panel: case with $K(t) = \text{Erl}_{3,a}(t)$. The parameter values are: $R_0 = 3.3$, $\nu = 1/7 \text{ days}^{-1}$, $N = 6 \cdot 10^7$, $\mu = 1/75 \text{ years}^{-1}$, $\Lambda = \mu N$, $a = 1/30 \text{ days}^{-1}$, $k = 1$, $\alpha = 5 \cdot 10^4$, $p_0 = 7.9 \cdot 10^{-9}$ and $(\tau_a, \tau_b, \tau_c, \tau_d) = (4, 7, 11, 14)$.

$$A_\mu(t) = \beta_0(\mu + \nu)^2 t e^{-(\mu + \nu)t} = \beta_0 \text{Erl}_{2, \mu + \nu}(t). \quad (5.27)$$

If the memory kernel is given by $K(t) = \text{Erl}_{1,a}(t)$, then the endemic equilibrium is stable according to Proposition 5.4.5 (see Figure 5.5, left panel). When $K(t) = \text{Erl}_{2,a}(t)$ we still observe damped oscillations towards the endemic equilibrium (see Figure 5.7, left panel). However, when $K(t) = \text{Erl}_{3,a}(t)$ sustained oscillations appear to occur (see Figure 5.5, right panel).

It is worth remarking that, as previously mentioned, the case of Erlang distributions, like (5.27) and $K(t) = \text{Erl}_{3,a}(t)$, could be studied through a compartmental model with an inflated number of differential equations that can be obtained by using the linear chain trick.

As final case, we consider the IF introduced in (4.41). The results are similar to the previous case, since we observe stability of the endemic equilibrium when $K(t) = \text{Erl}_{1,a}(t)$ (Figure 5.6, left panel), damped oscillations when $K(t) = \text{Erl}_{2,a}(t)$ (Figure 5.7, right panel) and possible sustained oscillations when $K(t) = \text{Erl}_{3,a}(t)$ (Figure 5.6, right panel).

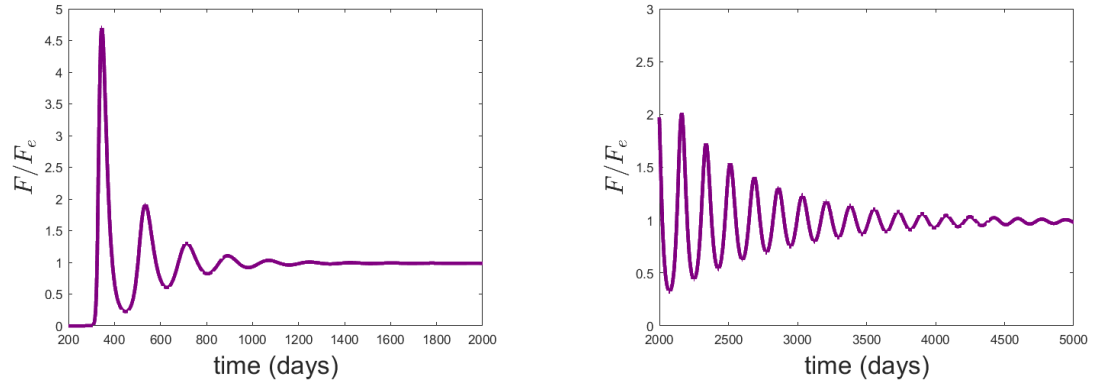


Figure 5.7: Time profile of the ratio F/F_e as predicted by model (5.19). Left panel: case with $K(t) = Erl_{2,a}(t)$, IF given by (5.27) and parameter values as in Figure 5.5. Right panel: case with $K(t) = Erl_{2,a}(t)$, IF given by (4.41) and parameter values as in Figure 5.6.

Chapter 6

Conclusions

6.1 Summary of thesis achievements

The study of epidemic models in integral form, where the infectivity function is expressed in a general manner and is characterised by a generic distribution of the infectious period, is crucial because these models provide a broader framework compared to traditional compartmental models. Notably, the celebrated papers by Kermack and McKendrick in 1927, which laid the foundations for modern mathematical epidemiology, introduced the SIR-type models through an integral constitutive equation. Only for specific forms of the infectivity function do the integral models reduce to the compartmental models ruled by ODEs.

In recent years, research in epidemiological modelling has increasingly focused on sociological factors, which can deeply influence the dynamics of an epidemic. In fact, behavioural effects can often serve as the primary determinant in shaping the progression of an epidemic. In particular, how the population keeps memory of information and rumours regarding the disease may influence the solution of the epidemic models, thereby inducing, for example, recurrent oscillations which, in turn, may be interpreted as multiple epidemic waves.

In Chapter 3 we introduce an integro-differential model describing an epidemic of an infectious disease taking into account human behavioural feedback. We first focus on the basic properties of the solution, and we prove positivity, boundedness and existence of equilibria. Then, we provide a qualitative analysis of the model based on stability theory described in Section 3.3. The main results consist in sufficient conditions, expressed in terms of the information parameters and functions, guaranteeing the local stability of the endemic equilibrium of the model. In particular we prove that the model admits a stable endemic equilibrium when the memory kernel is a weak Erlang kernel.

Numerical methods play a central role in this study. In Chapter 2, we focus on Volterra integral equations, which constitute a powerful framework for the formulation and analysis of epidemic models with memory effects. We present an overview of classical and modern approaches to their numerical resolution, giving particular attention to equations of convolution type, and we discuss the notions of consistency, stability, and convergence.

Although Volterra-type epidemic models offer strong theoretical insights, the development of appropriate numerical methods is still relatively limited. Conventional schemes often struggle to preserve essential properties of the solutions, such as positivity, boundedness, and the asymptotic behaviour of the continuous model, which restricts their practical usefulness. For this reason, we propose a numerical method based on a weighted non-standard technique, specifically designed for the model under consideration. We prove that our numerical method preserve the positivity and boundedness of the solution and the asymptotic behaviour of the continuous model, without any restriction on the discretization step-size. For this purpose, for any fixed value of the step-size $h > 0$, we regard the numerical method as a discrete-time model and obtained results on the stability of numerical equilibria which replicate the ones described in Chapter 3 for the continuous model. Since it is natural to ask whether the equilibria of the numerical method are close to the equilibria of the integro-differential model, we also study their approximation properties proving the convergence as $h \rightarrow 0$. These properties make the numerical method we propose efficient and reliable in long-time simulations, which are essential to gain a deep understanding in equilibria of the system and their stability properties related to control parameters, especially when theoretical results are not available. Numerical experiments confirm this view. In fact, in Figure 4.2, where our non-standard method is compared with discretizations from the classical literature, it can be seen that, at the same discretization step-size, the former exhibits non-physical oscillations. Moreover, we consider some different infectivity functions used in the literature to describe infectious disease like Influenza and SARS. Still, in such cases, we show that a weak Erlang memory leads to convergence to an endemic equilibrium, while a unimodal memory can determine the appearance of oscillations.

Finally, in Chapter 5, we recall the historical development of papers concerning the basic compartmental models in which the feedback produced by changes in the behaviour of individuals affects the contact pattern. This overview is useful to better understand the connection between integral and compartmental epidemic models. In particular, we show that by selecting an exponential infectivity function,

the model (3.9) reduces to the behavioural SIR-M model investigated by d’Onofrio and Manfredi in [27] for the various forms of the memory kernel. Moreover, the results we obtain are in full agreement with their findings. In the second part of Chapter 5, we propose and analyze a new formulation of an integral epidemic model with an incidence-based contact pattern. This allows us to confirm once again that integral models can be seen as a generalization of compartmental models, and that the results of the latter can be recovered from the integral models for specific choices of the infectivity function.

6.2 Future perspectives

In both models (3.9) and (5.19), one may assume that the infectivity function A_μ has compact support. This assumption reflects the biological fact that infected individuals remain contagious only for a limited period, beyond which their ability to transmit the disease declines sharply or vanishes. This permits assuming that $A_\mu(\tau) = 0$ for all $\tau > \bar{\tau}$. In the simplified case $M(t) = 0$, i.e., when behavioural feedback is neglected, both systems (3.9) and (5.19) reduce to a system of two integral equations with both a pointwise delay $\bar{\tau}$ and a distributed delay. For such problems, a linear test equation was considered:

$$z(t) = az(t - \bar{\tau}) + \int_0^{\bar{\tau}} K(\tau)z(t - \tau)d\tau, \quad (6.1)$$

whose asymptotic properties were studied to assess the numerical stability of the methods applied. Equation (6.1) also finds applications in other physical and biological fields, such as traffic control strategies.

In equation (6.1), we assume $0 < a < 1$, $b \in \mathbb{R}$, and a kernel function $k : [0, \bar{\tau}] \rightarrow \mathbb{R}$, with $\bar{\tau} < \infty$, that is continuous and positive. The parameter $\bar{\tau} > 0$ represents the known constant delay. The initial condition of (6.1) is described by the continuous function $\varphi(t)$ for $t \in [-\bar{\tau}, 0]$. Existence and uniqueness results for the solution to (6.1) in $C[0, +\infty)$ can be found in [2], provided that the initial function satisfies

$$\varphi(0) = a\varphi(-\bar{\tau}) + b \int_0^{\bar{\tau}} k(\tau)\varphi(-\tau)d\tau. \quad (6.2)$$

Furthermore, by a straightforward application of the method of steps it is possible to prove that, if the given functions k and φ possess continuous derivatives of order $m \geq 1$ on their respective domains, the unique solution y belongs to $C^m(j\bar{\tau}, (j+1)\bar{\tau}]$, and it is bounded on $j\bar{\tau}$, for each $j = 0, 1, \dots$.

Let $\Pi_n = \{t_n = nh, n = 0, 1, \dots\}$, where $h > 0$ is the step-size of the discretiza-

tion, which we assume to be constrained by $\bar{r}h = \bar{\tau}$, $\bar{r}$ positive and integer. The discretization of problem (6.2) by a direct quadrature method yields

$$y_n - ay_{n-\bar{r}} = hb \sum_{j=0}^{\bar{r}} \omega_j k(t_j) y_{n-j}, \quad (6.3)$$

where $y_n \approx y(t_n)$, $n = 0, 1, \dots$, and $y_j = \varphi(t_j)$, $j = -\bar{r}, \dots, -1$, being φ the known initial function satisfying (6.2). The coefficients ω_j , $j = 0, \dots, \bar{r}$, are the weights of a quadrature formula. By applying the classical theory on convergence described in Section (4.2), the following result can be proven [50].

Theorem 6.2.1. *Let N be a fixed positive integer. Assume that $k \in C^p[0, \bar{\tau}]$ and $\varphi \in C^p[-\bar{\tau}, 0]$, where p is the order of the underlying quadrature formula. Then, for all sufficiently small step-sizes h satisfying the constrained mesh condition, the direct quadrature solution y_n of equation (6.3) satisfies $\max_{n=0, \dots, N} |y(t_n) - y_n| \leq Ch^p$, for some finite constant C not depending on h .*

As a direction for future work, we plan to discretize the problem using the class of Gregory quadrature formulas and to investigate the associated stability properties, with the aim of deriving conditions under which the discrete system preserves the stability features of the continuous problem. In this context, we also intend to apply and further explore classical direct quadrature methods to this simpler setting, since, as demonstrated in Section 5, they provide more accurate solutions than non-standard approaches, thereby making a deeper analysis of their properties particularly meaningful.

From a modelling perspective, another promising direction for future research is to explore a mixed modelling framework that integrates both constant and distributed delays in the transmission dynamics of infectious diseases. The goal of this approach is to provide a description of epidemic processes by combining centralized policy interventions with heterogeneous individual behavioural responses. To this end, we consider a reduced SIR setting in which the susceptible population remains approximately constant, yielding the following equation for the infected individuals:

$$\dot{I}(t) = \beta(t) I(t) - \gamma I(t). \quad (6.4)$$

The effective contact rate β governing the transmission of the disease is not constant but varies over time. We assume:

$$\beta(t) = f\left((1-b)\beta(t-\tau_1)I(t-\tau_1) + b \int_0^\infty K(\tau)\beta(t-\tau)I(t-\tau)d\tau\right), \quad (6.5)$$

where $f : \mathbb{R}_{\geq 0} \rightarrow \mathbb{R}_{\geq 0}$ is a nonincreasing function with $f(0) = \beta_0$ (the maximal contact rate) and $f'(x) \leq 0$ for $x \geq 0$. The parameter $\tau_1 > 0$ models fixed delays associated with centralized interventions, while the integral term with kernel $K(\tau)$ represents a distributed delay capturing variability in individual response times or disease progression. In particular, when $b = 0$, it reduces to the model proposed in [72], which describes the dependence of the transmission rate on incidence using a constant time lag in the renewal equation. If $b = 1$, only the distributed delay term remains in equation (6.5). Under this assumption, the model can be regarded as a particular variant of the one studied in Section 5.4. Our future aim will be to characterise stability and bifurcation structures associated with the coupled system (6.4)–(6.5), identifying parameter regimes where delayed behavioural feedback induces sustained oscillations or ensures convergence to an equilibrium.

Other lines of future research will concern the extension to more complex models, including scenarios with non-mandatory pharmaceutical interventions (such as vaccination and treatments), whose adoption is typically influenced by opinion-driven changes in human behaviour. Moreover, the impact of host heterogeneity on epidemic dynamics has long been acknowledged [8, 22]. Recent literature introduces general Kermack and McKendrick’s epidemic models that consider heterogeneity, for a population characterized by different traits, with different activity levels or disease parameters [5, 6, 10]. The provided formula expresses the FoI, delineating contributions from individuals infected at a specific time and with a particular trait. This suggests, as a natural extension, a generalization of the constitutive law for the FoI with an information index aimed at exploring how heterogeneity influences epidemic aspects in a model that incorporates human behaviour and broadens the analysis presented in this paper.

Finally, further investigations will address the numerical resolution of the more general epidemic models introduced above, with particular attention to the construction of suitable methods that preserves all the properties of the continuous solution.

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