Universidade do Minho
Escola de Engenharia

João Nuno Costa Gonçalves

Epidemiological Models and Optimal Control Theory - Applications to Marketing and Computer Viruses Transmission

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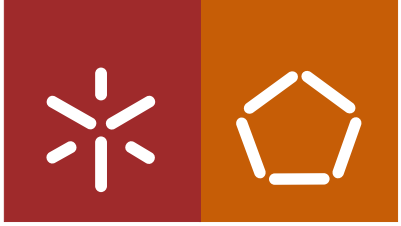

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João Nuno Costa Gonçalves

Epidemiological Models and Optimal Control Theory - Applications to Marketing and Computer Viruses Transmission

Dissertação de Mestrado
Mestrado em Engenharia de Sistemas

Trabalho realizado sob orientação da
Professora Doutora Maria Teresa Torres Monteiro
e da
Professora Doutora Helena Sofia Ferreira Rodrigues

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Para Ti.

Que desejaste que me

tornasse um Homem.

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I see life as a collection of experiences. Therefore, I use this space to express my gratitude to those who made the memory of them infinity.

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Abstract

Epidemiological models and Optimal Control Theory are closely interrelated, inasmuch as the development of control interventions that could help to perceive and minimize the spread of infection diseases is a pressing need. However, the application of these two subjects can be extended to other scientific domains apart from health. In this respect, this dissertation takes advantage of the foundations of Mathematical Epidemiology and Optimal Control Theory to study the dynamics of Viral Marketing within a certain population and, with less detail, Computer Viruses Transmission into network systems.

Due to the fierce marketing competition, not all the marketing campaigns become viral, and formulate strategies that could leverage traditional marketing campaigns is not a trivial task. In this regard, since the process of diffusing Viral Marketing campaigns through social networks can be modeled under concepts of Mathematical Epidemiology and being strategy the keyword, the benefits of Optimal Control Theory on the diffusion of a real viral advertisement are studied and control strategies to maximize the spread of viral messages with low cost are investigated and proposed in optimal time windows. Numerical methods based on the Pontryagin's Maximum Principle (indirect methods) and methods that treat the Optimal Control problem as a nonlinear constrained optimization problem (direct methods) are tested and compared, using different numerical solvers.

In an introductory way, Computer Viruses Transmission and its dynamics are briefly studied by using epidemiological modeling over networks. In addition, an R software package related to the mathematical modeling of infectious diseases is discussed and the propagation of computer viruses within a network system is illustrated.

Keywords: Epidemiological Dynamical Systems; Optimal Control Theory; Pontryagin's Maximum Principle; Mathematical Modeling and Optimization; Viral Marketing; Computer Viruses Transmission.

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Resumo

O desenvolvimento de políticas de controlo é crucial para perceber e minimizar a propagação de doenças infeciosas. Sob este pressuposto, os modelos epidemiológicos e a Teoria do Controlo Ótimo surgem, cada vez mais, intimamente relacionados. Todavia, a aplicação destes domínios científicos pode ser estendida a outras áreas do conhecimento. Neste contexto, a presente dissertação estuda não só as dinâmicas do Marketing Viral no seio de uma população, recorrendo a fundamentos de Epidemiologia Matemática e à Teoria do Controlo Ótimo, mas também, com menor enfoque, a Transmissão de Vírus Informáticos em sistemas em rede.

Atendendo à crescente competitividade dos mercados, nem todas as campanhas de marketing se tornam virais. Para além disso, a conceção de estratégias de marketing que, por sua vez, permitam colmatar as carências das campanhas tradicionais pode revelar-se uma tarefa bastante complexa. Sob evidências científicas de que o processo de difusão de campanhas de Marketing Viral pode ser modelado sob conceitos de Epidemiologia Matemática, são estudadas as potencialidades da Teoria do Controlo Ótimo na disseminação de um campanha real de marketing. Mais ainda, são discutidas e propostas estratégias de controlo para maximizar a difusão de mensagens virais a um baixo custo. São, igualmente, testados e comparados métodos númericos baseados no Princípio do Máximo de Pontryagin (métodos indiretos) e, por outro lado, métodos que tratam um problema de Controlo Ótimo como um problema de otimização não linear com restrições (métodos diretos).

No que concerne à Transmissão de Vírus Informáticos, a sua dinâmica de propagação é analisada, de uma forma puramente introdutória, sob pressupostos relativos à modelação epidemiológica em redes. Para isso, é explorado um pacote do *software* R dedicado à modelação de doenças infeciosas, permitindo ilustrar a propagação de vírus informáticos num dado sistema.

Palavras-chave: Sistemas Dinâmicos Epidemiológicos; Teoria do Controlo Ótimo; Princípio do Máximo de Pontryagin; Modelação Matemática e Otimização; Marketing Viral; Transmissão de Vírus Informáticos.

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List of Acronyms

CVT	Computer Viruses Transmission
DFE	Disease-Free Equilibrium
NLP	Nonlinear Programming Problem
OC	Optimal Control
ODE	Ordinary Differential Equation
PMP	Pontryagin's Maximum Principle
$\mathcal{R}_0$	Basic Reproduction Number
SIS	Susceptible-Infected-Susceptible
SIR	Susceptible-Infected-Recovered
SIRS	Susceptible-Infected-Recovered-Susceptible
VM	Viral Marketing

Introduction

In Mathematics, the art of proposing a question must be held of higher value than solving it.

Georg Cantor

Conventionally, epidemiological models have been widely applied to understand the dynamics of several infectious diseases. Nevertheless, since the dynamics of infections can evolve in an erratic way, research studies have applied Optimal Control Theory to Epidemiology, aiming to provide effective and economic strategies to perceive and curtail the propagation of a virus infection within a certain population. At this point, the relationship between Epidemiology and Optimal Control Theory has proved to be highly correlated [112].

According to H.W. Hethcote in his remarkable article published in 2000, "The Mathematics of Infectious Diseases" [44], the gradual emergence of infectious diseases has awakened the interest of the scientific community to study them. In this regard, the use of mathematical models to describe and control the propagation of viral infections is of utmost importance, in spite of the inherent complexity associated to the formulation of accurate models which reflect the reality of the disease.

Let us focus now on the control measures. Notice that the formulation of strategies that could help to improve knowledge concerning viral outbreaks may really make a difference in saving human lives. This is quite relevant and stimulating, in such a way that the urgency in both contain virus outbreaks and restrict future re-infections triggers research studies in Optimal Control applied to Epidemiology.

However, the relationship between these two areas has been explored beyond its conventional use. In fact, among many others, Viral Marketing (see, e.g., [51, 52, 54]) and Computer Viruses

Transmission processes (see, e.g., [23, 133]) have been increasingly exploited - in the first one, purposing to maximize viral infections (with Viral Marketing messages) within the population and, in the second one, aiming to minimize the propagation of viruses or other malicious programs within computer networks. These processes are the main topics of this dissertation, in which we try to answer to some core questions, related to both subjects, such as:

- How do viruses spread within a population?
- How can Viral Marketing and Computer Viruses Transmission be seen as epidemiological dynamical systems?
- How should marketing companies invest in publicity actions to generate viral campaigns?
- Regarding the Viral Marketing, when and what control measures should be applied to maximize information spreading in an economically sustainable way?
- How do computer viruses spread within a network system?

Objectives and Dissertation Outline

Purposing to tackle the research questions stated before, this dissertation intends to:

- Devise a recent in-depth review of the scientific literature of epidemiological models and Optimal Control Theory.
- Analyze and study the influence of Mathematical Epidemiology and Optimal Control Theory on dynamical systems, particularly on their applications to Viral Marketing and Computer Viruses Transmission.
- Apply and compare indirect and direct numerical optimization techniques on the resolution of the proposed mathematical models.
- Use epidemiological modeling over networks to describe the propagation of computer viruses.

For that, this research project is composed by two main Parts.

The First Part introduces well known concepts related to epidemiological models (Chapter 1) and Optimal Control Theory (Chapter 2), taken from the literature. In the second Chapter, a brief explanation of numerical methods to solve Optimal Control problems is also provided. Throughout the First Part, some scientific literature references and examples are given.

The Second Part uses the theoretical background raised in the First Part to understand the dynamics of Viral Marketing (Chapter 3) and Computer Viruses Transmission (Chapter 4). Specifically, in Chapter 3, we begin to describe Viral Marketing as an epidemiological dynamical system. Then, based on real numerical data from one of the most viral marketing campaigns of all time, epidemiological models with Optimal Control are formulated and studied, aiming to maximize the propagation of a marketing message with a low cost. Here, indirect and direct numerical methods to solve the formulated problems are analyzed and compared, using several numerical solvers. Moreover, Optimal Control strategies related both to the encouragement of susceptible individuals to share the marketing message and the fostering of infected individuals to continue to share it are proposed in optimal time windows.

Finally, in a less detailed perspective, computer virus infections over networks are briefly studied using Network Theory in Chapter 4. In addition, numerical simulations to validate the behavior of epidemic models are performed, using a recent R software package related to the mathematical modeling of infectious diseases.

This dissertation ends with a section devoted to conclusions and future research.

Part I

State of the Art

1 | Epidemiological Models

This Chapter intends to introduce and mathematically describe some of the standard epidemiological models, which divide the host population into mutually-exclusive compartments, namely SIS (Susceptible-Infected-Susceptible), SIR (Susceptible-Infected-Recovered) and SIRS (Susceptible-Infected-Recovered-Susceptible) without births and deaths (vital dynamics). A short overview of some scientific studies related to each model is also provided.

1.1 Introductory Remarks

Throughout this dissertation, epidemiological models should be understood as dynamical systems formed by ordinary differential equations (ODEs), that describe and model the dynamics of an epidemic phenomenon within a certain population.

According to K. Dietz [26], the concept of epidemiology has two possible definitions: the study of the dynamics of infectious diseases within a population or the study of the variables that have influence on the incidence of an infectious disease. Considering only the first definition given by the author, infectious diseases can be seen as clinically identifiable illnesses caused by pathogenic microorganisms, which can be spread among a population, by direct or indirect ways, from one person to another [127]. However, for both definitions, the use of mathematical modeling mechanisms reveals a fruitful tool to better understand infectious processes. At this point, for more details on the application of Mathematics to infectious diseases, the reading of [44] and the references cited therein is highly recommended.

Looking back through history, despite of Daniel Bernoulli had proposed the smallpox model in 1760 (see [8]), the vast majority of the basic theory of Mathematical Epidemiology was only developed between 1900 and 1935. At this time, Mathematical Epidemiology theory was truly

driven by the studies of Kermack and McKendrick in 1927, namely with the paper entitled "A contribution to the mathematical theory of epidemics" [58], in which they proposed a deterministic mathematical model that considers classes of susceptible, infected and recovered individuals. In addition, in 1932 and 1933, these authors also developed new scientific advances on the first paper, cementing the importance of Mathematics in modeling infectious diseases [79].

Since then, more and more models are currently being developed, not only to perceive the dynamics of infectious diseases, but also to formulate control strategies to tackle them, strengthening the role of Mathematical Epidemiology.

From a global and assertive perspective, it is plausible to say that the biggest challenge in Mathematical Epidemiology relates to modeling epidemic phenomena with art, always attempting to formulate models, as simply as possible, capable of tackle the research questions raised [43] (as cited in [107]).

With these motivations, this Chapter intends to introduce and describe some fundamental epidemiological models using Mathematical Epidemiology theory.

The writing of the following sections of this Chapter is mainly based on theory presented in [12, 44, 79]. Nevertheless, more details on mathematical theory of infectious diseases can also be found in, e.g., [11, 14, 20].

1.2 Preliminary Concepts

As a tool to describe dynamical systems, mathematical models can be formulated in order to understand, the best possible, epidemic phenomena. Regarding the process of formulate mathematical models, M. Martcheva describes in [79] a general approach to modeling, which basically consists on an iterative method to tackle a proposed biological question, converting it into a mathematical problem:

1. the model must be analyzed in order to generate critical quantities that play a key role on the dynamics of the solutions;
2. the model should be fitted to given data or used to conduct numerical simulations that, a posteriori, generate experimental data;
3. the parameters of the model should be carefully estimated;

4. the resultant model must be validated through several numerical experiments to attest the reliability of the results, and the dynamics of the model when varying its parameters should be assessed, showing its importance.

Typically, the formulation of epidemic models can follow a stochastic or deterministic approach. In the first one, model variables are characterized by probability distributions. On the other hand, in deterministic epidemic models, the state variables are uniquely determined by both parameters in the model and initial conditions of the variables. These models subdivide the population into mutually-exclusive compartments, where the individuals can move through it according to their disease status, and are usually governed by systems of ODEs with respect to time, subject to given initial conditions. Since in these models the population is subdivided in terms of the disease status, basic terminology for the model compartments, that will be considered in the next sections, is provided as follows.

- **Susceptible** (S): this compartment represents individuals who are susceptible to contract a disease after some kind of contact with an infectious individual.
- **Infected** (I): this compartment represents individuals who are currently infected and can transmit the disease to individuals in the class S who they contact. Infected individuals who can transmit the infectious disease are called infectious. Once infected, these individuals can or not develop immunity against reinfection.
- **Recovered or Removed** (R): this compartment represents individuals who have been infected and have recovered.

In addition, based on [79], basic concepts of epidemiology are also defined.

Definition 1.2.1 (Incidence)**.** *Incidence is the number of individuals who become sick during a specific time interval.*

Definition 1.2.2 (Prevalence)**.** *The concept of prevalence related to an infectious disease can be defined as the number of individuals who have the disease at a particular time. Nevertheless, it is usual to consider this number divided by the total size of the population.*

Definition 1.2.3 (Basic reproduction number)**.** *Denoted by $\mathcal{R}_0$, the basic reproduction number is defined as the average number of secondary infections induced by an infected individual within a susceptible population.*

This number is crucial to understand the dynamics of an epidemic model, as we shall see later.

Based on the explained above, the following sections will describe SIS, SIR and SIRS deterministic models without vital dynamics, under the assumption that the number of individuals in each model compartment is a differentiable function of time t.

1.3 The SIS Model

In the SIS epidemiological model, if an infectious individual comes into successful contact with a susceptible, this one becomes infected and infectious but does not develop immunity against reinfection. Therefore, an individual who get recovered from an infectious disease returns to the class S, becoming once again susceptible to contract the disease. The compartmental diagram for the SIS model, which traduces the arguments given above, is illustrated in Fig. 1.1.

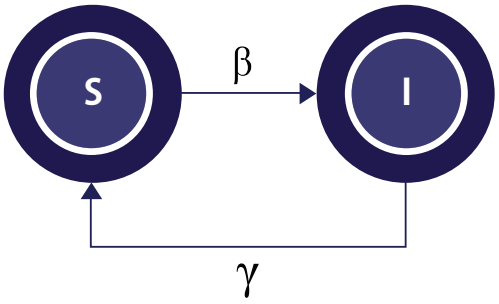


Figure 1.1: Compartmental diagram for the SIS model.

Example 1.3.1. *Gonorrhea, Malaria, Syphilis or Yellow fever, e.g., are good examples of infectious diseases that can be modeled by epidemic models related to the SIS model, since they do not produce an immune response on the individuals who contract them.*

The dynamics of the SIS epidemiological model can be described, at time t, by the following system of ODEs:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\dfrac{\beta S(t)I(t)}{N} + \gamma I(t) \\ \dfrac{dI(t)}{dt} = \dfrac{\beta S(t)I(t)}{N} - \gamma I(t) \ , \end{cases} \tag{1.1}$$

subject to initial conditions $S(0) > 0$ and $I(0) > 0$. The positive parameters β and γ traduce the contact and recovery rate, respectively. The total population size, N, is assumed to be constant over time, i.e.,

$$S(t) + I(t) = N \Leftrightarrow \frac{dN}{dt} = 0 \ , \forall t \in [0, \infty) \ .$$

Remark 1.3.1. *If the recovery rate γ is null in (1.1), the basic SI epidemiological model is obtained. In this model, once an individual is infected remains infected over time.*

If one looks at the system (1.1), the standard incidence $\left(\frac{\beta S(t) I(t)}{N}\right)$ comes from the probability of a susceptible individual contacts with an infected one $\left(\frac{I(t)}{N}\right)$, multiplied by the rate of infections caused by an infected individual ($\beta S(t)$) per unit time t. The $\gamma I(t)$ term relates to the rate of infected individuals who recover per unit time t.

In addition, the length of the infectious period is represented by the fraction $\frac{1}{\gamma}$, and $\mathcal{R}_0 = \frac{\beta}{\gamma}$ is obtained according to the necessary condition for an epidemic, $\frac{dI(t)}{dt} > 0$, considering $S(0) \approx N$.

1.3.1 On the SIS Models

SIS epidemiological models have been the subject of intensive research, notwithstanding its simplicity. In [39], the authors extend the classic deterministic SIS model to a stochastic one and formulate it as a stochastic differential equation for $I(t)$. In fact, the global stability of this model was recently studied in [129], where the author presents a stochastic threshold theorem related to $\mathcal{R}_0$. In this regard, X. Meng et al. [81] developed a method to obtain the threshold of the stochastic SIS model. In addition, other research studies investigate the dynamics of stochastic SIS models with Lévy jumps (see, e.g., [36, 134, 137]), with delay (see, e.g., [2, 53, 69, 87]), or even with nonlinear incidence rate (see, e.g., [66, 118]).

The dynamics and major properties of the SIS model are described next.

1.3.2 Dynamics and Mathematical Properties

To simplify algebraic manipulations, and since $S(t) = N - I(t)$, note that the state system (1.1) can be reduced to the simple ODE (logistic growth equation)

$$\frac{dI(t)}{dt} = I(t)\left(\beta\frac{(N - I(t))}{N} - \gamma\right) . \tag{1.2}$$

To study the dynamics of the SIS model, it is pertinent to determine its equilibrium solutions, that is, solutions I^* that satisfy the condition

$$\frac{dI(t)}{dt} = 0 .$$

Thus, the equilibrium solutions for the SIS model are $E_1 = (N, 0)$, also called Disease-Free Equilibrium (DFE), and $E_2 = \left(\frac{\gamma N}{\beta}, \left(1 - \frac{\gamma}{\beta}\right)N\right)$, called endemic equilibrium. In order to guarantee that the endemic equilibrium is positive in their components, lying in a domain of interest, the recovery rate should be strictly less than the contact rate ($\gamma < \beta$).

Regarding the linear stability of the equilibrium solutions, notice that:

- For E_1, if $\beta > \gamma$ then E_1 is unstable. On the other hand, if $\beta < \gamma$ then E_1 is locally asymptotically stable.
- For E_2, if $\beta > \gamma$ then E_2 is locally asymptotically stable. On the contrary, if $\beta < \gamma$ then E_2 is unstable.

Expressed the other way around, this means that there exists a transcritical bifurcation at $\beta = \gamma$, where $E_1 = E_2$ and the stability of the equilibrium solutions exchanges. For more complete details about stability theory see, e.g., [55].

Remark 1.3.2. *It should be noted that $\beta > \gamma$ and $\beta < \gamma$ correspond to $\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$, respectively.*

Using the analysis above, the following theorem highlights the relevance of $\mathcal{R}_0$ to the dynamics of the SIS model.

Theorem 1.3.1. *Let $(S(t), I(t))$ be a solution pair for the SIS system. Then, the following conditions hold:*

1. *If $\mathcal{R}_0 > 1$, then $\lim_{\mathbb{R}_0^+ \ni t \to \infty} (S(t), I(t)) = \left(\frac{\gamma N}{\beta}, \left(1 - \frac{\gamma}{\beta}\right)N\right)$ (Endemic equilibrium).*
2. *If $\mathcal{R}_0 < 1$, then $\lim_{\mathbb{R}_0^+ \ni t \to \infty} (S(t), I(t)) = (N, 0)$ (Disease-Free equilibrium).*

Proof. Firstly, the ODE (1.2) can be rewritten as

$$\frac{dI}{dt} = AI\left(1 - \frac{I}{B}\right) , \tag{1.3}$$

where $A = \beta - \gamma$ and $B = \dfrac{AN}{\beta}$. Now, excluding the trivial case where $A = 0$, two possible cases must be considered: $A > 0$ and $A < 0$, corresponding to $\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$, respectively.

- (Condition 1) Let $A > 0$. Since the equation (1.3) is a separable non-linear ODE, it follows that

$$\frac{BdI(t)}{I(t)(B - I(t))dt} = A$$

$$\int_0^t \frac{BdI(t)}{I(t)(B - I(t))} = \int_0^t A \; dt$$

$$\int_{I(0)}^{I(t)} \frac{Bdv}{v(B - v)} = A \int_0^t \; dt$$

$$\int_{I(0)}^{I(t)} \frac{dv}{v} + \int_{I(0)}^{I(t)} \frac{dv}{B - v} = A \int_0^t \; dt$$

$$\log(|v|)\Big|_{I(0)}^{I(t)} - \log(|B - v|)\Big|_{I(0)}^{I(t)} = At \; ,$$

since $B - I(t)$ and $B - I(0)$ have the same sign, yields

$$\log\left(\frac{I(t)\big(B - I(0)\big)}{I(0)\big(B - I(t)\big)}\right) = At$$

$$\frac{BI(0)}{\dfrac{B - I(0)}{e^{At}} + I(0)} = I(t) \xrightarrow{t\to\infty} B \; , \tag{1.4}$$

which means that the disease remains in the population, for an indefinite period of time t. As a consequence, $\lim\limits_{\mathbb{R}_0^+ \ni t \to \infty} S(t) = N - \left(1 - \dfrac{\gamma}{\beta}\right)N = \dfrac{\gamma N}{\beta}$, thus proving Condition 1.

- (Condition 2) The proof of Condition 2 is much more simpler. From the hypothesis that $A < 0$ it follows that $B < 0$. Thus,

$$\frac{dI(t)}{dt} \leqslant AI(t) \ . \tag{1.5}$$

This differential inequality leads to $I(t) = I(0)e^{At} \xrightarrow{A<0} 0$. As a consequence

$$\lim_{\mathbb{R}_0^+ \ni t \to \infty} S(t) = N \ , \tag{1.6}$$

which concludes the proof.

□

1.4 The SIR Model

Proposed by Kermack and McKendrick [58], the SIR model is one of the earliest epidemiological models. In detriment of the previous model, SIR model can be applied to infectious diseases that cause an immune response in the body. For a specific disease, if an individual become infected, then it is highly unlikely that a reinfection process occurs, being permanently immune. These arguments can be illustrated by the following compartmental diagram (Fig. 1.2).

Figure 1.2: Compartmental diagram for the SIR model.

Example 1.4.1. *Smallpox, Chickenpox and Rubella, e.g., are infectious diseases that produce an immune response on the individuals who contract them. Hence, they can be modeled under SIR epidemiological models or its derivatives, as long as the immunity feature continues to be met.*

The standard form of this model is given by the following system of ODEs:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\dfrac{\beta S(t) I(t)}{N} \\ \dfrac{dI(t)}{dt} = \dfrac{\beta S(t) I(t)}{N} - \gamma I(t) \\ \dfrac{dR(t)}{dt} = \gamma I(t) \ , \end{cases} \tag{1.7}$$

subject to $S(0) > 0$, $I(0) > 0$ and $R(0) = 0$. The parameters β and γ and the duration of the infection have the same biological meaning as in the SIS model. However, for this epidemic model, in contrast to the previous one, infected individuals move to the class R at a rate γI per unit time t and can not return to the susceptible class S, and the total size of the population is given by $N = S(t) + I(t) + R(t)$.

Similarly to the SIS model, the basic reproduction number, $\mathcal{R}_0$, for the SIR model is $\dfrac{\beta}{\gamma}$, considering that at the beginning of the epidemic almost everyone is susceptible $(S(0) \approx N)$.

Moreover, by setting the right hand side of the system (1.7) to zero, the equilibrium solutions for the SIR model are $(N, 0, 0)$ and $(0, 0, N)$. In the Second Part of this dissertation, the stability analysis of these solutions is carefully studied. Hence, this study is omitted in this section.

1.4.1 On the SIR Models

From an interesting perspective, T. Harko et al. [41] computed, in a parametric form, the exact solution of the SIR model, showing also that the SIR model with vital dynamics can be reduced to an Abel type equation – simplifying the model analysis and the deduction of its properties. Since the concept of incidence is highly important in Mathematical Epidemiology, Y. Chen et al. [24] studied infection age and saturated incidence of an SIR epidemic model, establishing a threshold dynamics. On the other hand, S. Bidari et al. [7] analyzed explicit formulas for the final size distribution for line and star graphs of arbitrary size.

In [67], the introduction of saturated treatment and logistic growth rate into an SIR epidemic model with bilinear incidence is studied. In addition, the authors also establish sufficient conditions for the existence and local stability of the DFE. Regarding the logistic growth in the absence of disease, Q. Liu et al. [76] analyzed the dynamics of a stochastic nonautonomous SIR model, presenting sufficient conditions for the persistence of an epidemic.

Stochastic delayed SIR models with temporary immunity (see, e.g., [71]), vaccination (see, e.g., [72]) or even nonlinear incidence (see, e.g., [75]) are also investigated.

1.4.2 Model Properties and the Maximum Number of Infections

The dynamics of the SIR model can be scrutinized through an assertive analysis on the equations of (1.7). Independently of the initial conditions, $\frac{dS(t)}{dt} \leqslant 0$ and $\frac{dR(t)}{dt} \geqslant 0$, for all t, meaning that the number of susceptible individuals is always decreasing and the number of recovered individuals is always increasing. Hence, since $S(t)$ and $R(t)$ have a monotone behavior and noting that for all t, $0 \leqslant S(t) \leqslant S(0) \leqslant N$ and $0 \leqslant R(0) \leqslant R(t) \leqslant N$, the following limits exist:

$$\lim_{\mathbb{R}_0^+ \ni t \to \infty} S(t) = S_\infty \text{ and } \lim_{\mathbb{R}_0^+ \ni t \to \infty} R(t) = R_\infty \ , \tag{1.8}$$

where S_∞ is called the final size of the epidemic. Moreover, the value of S_∞ can be computed, using the initial conditions, by the equation:

$$\begin{aligned}
\frac{dS(t)}{dt} &= \frac{-\beta S(t) dR(t)}{\gamma N dt} \\
\frac{dS(t)}{S(t)dt} &= \frac{-\beta dR(t)}{\gamma N dt} \\
\int_0^t \frac{dS(t)}{S(t)} &= \frac{-\beta}{\gamma N} \int_0^t dR(t) \\
\log(|S(t)|)\Big|_0^t &= \frac{-\beta}{\gamma N} R(t)\Big|_0^t \\
S(t) &= S(0) e^{\frac{-\beta(R(t)-R(0))}{\gamma N}} \ ,
\end{aligned} \tag{1.9}$$

which allows to conclude not only that $S_\infty > 0$, but also that there are susceptible individuals who never contract the disease.

In addition, by (1.8), there exists

$$\lim_{\mathbb{R}_0^+ \ni t \to \infty} I(t) = N - S_\infty - R_\infty = I_\infty \ . \tag{1.10}$$

In fact, it can be shown that if (1.10) holds, the epidemic extinguishes. For that, it is sufficient to integrate the first equation of the system (1.7) with respect to time t:

$$\begin{aligned}\int_0^\infty dS(t) &= -\frac{\beta}{N}\int_0^\infty S(t)I(t)\ dt\ ,\\ S_0 - S_\infty &= \frac{\beta}{N}\int_0^\infty S(t)I(t)\ dt \geqslant \frac{\beta}{N}S_\infty\int_0^\infty I(t)\ dt\ ,\end{aligned} \tag{1.11}$$

which implies that $I(t)$ is integrable on $\mathbb{R}_0^+$. Therefore, $I(t) \xrightarrow{t\to\infty} 0$.

In what concerns to the behavior of $I(t)$, it deeply depends on the parameters β and γ. In fact, the number of infected individuals, over time t, may start to increase until a maximum level be reached, and then, as proved above, decreasing to zero in the asymptotic limit. Mathematically, the prevalence first starts to increase if

$$\left.\frac{dI}{dt}\right|_{t=0} > 0 \overset{I(0)>0}{\Leftrightarrow} \frac{\beta S(0)}{N} - \gamma > 0 \Leftrightarrow \frac{\beta S(0)}{\gamma N} > 1\ . \tag{1.12}$$

This increasing in the prevalence followed by a declining to zero traduces an epidemic. Here, it is possible to determine, explicitly, the maximum number of infected individuals. To this end, first note that since $R(t) = N - S(t) - I(t)$. Then, the state system (1.7) can be reduced to

$$\begin{cases}\dfrac{dS(t)}{dt} = -\dfrac{\beta S(t)I(t)}{N}\\ \dfrac{dI(t)}{dt} = \dfrac{\beta S(t)I(t)}{N} - \gamma I(t)\ .\end{cases} \tag{1.13}$$

Now, dividing the second ODE of the state system (1.13) by the first one, and separating the variables, yields

$$\begin{aligned}\frac{dI(t)}{dt} &= \frac{-dS(t)}{dt} + \frac{\gamma N}{\beta}\left(\frac{dS(t)}{S(t)dt}\right)\\ \int dI(t) &= -\int dS(t) + \frac{\gamma N}{\beta}\int\frac{dS(t)}{S(t)}\\ I(t) &= -S(t) + \frac{\gamma N}{\beta}\log S(t) + C\ ,\ C\in\mathbb{R}\ .\end{aligned} \tag{1.14}$$

Then, is possible to derive an implicit equation for the orbits of the solution of the state system (1.13):

$$C = I(t) + S(t) - \frac{\gamma N}{\beta} \log S(t),\ C \in \mathbb{R}\ . \tag{1.15}$$

Using the initial conditions and observing that the equation (1.15) holds for $(S(0), I(0))$, the arbitrary constant C is given by

$$C = I(0) + S(0) - \frac{\gamma N}{\beta} \log S(0)\ . \tag{1.16}$$

Note also that so far we already proved (1.8). Thus, since the equation (1.15) also holds for $(S_\infty, 0)$, it follows that

$$S_\infty - \frac{\gamma N}{\beta} \log S_\infty = I(0) + S(0) - \frac{\gamma N}{\beta} \log S(0)\ , \tag{1.17}$$

rearranging the terms, yields

$$\begin{aligned} \frac{\gamma N}{\beta}\bigg(\log S(0) - \log S_\infty\bigg) &= S(0) + I(0) - S_\infty \\ \frac{\beta}{\gamma} &= \frac{N \log \dfrac{S(0)}{S_\infty}}{S(0) + I(0) - S_\infty} > 0\ , \end{aligned} \tag{1.18}$$

since $S(0) + I(0) > S_\infty$.

Finally, taking into account that the maximum number of infected individuals is attained when $\dfrac{dI(t)}{dt} = 0$, i.e., when $S(t) = \dfrac{\gamma N}{\beta}$, and using not only both equations (1.15) and (1.16), but also that $I(t) \xrightarrow{t\to\infty} 0$, the maximum number of infected individuals, $I_{\max}$, is given by

$$I_{\max} = I(0) + S(0) + \frac{\gamma N}{\beta}\bigg(\log \frac{\gamma N}{\beta} - \log S(0) - 1\bigg)\ . \tag{1.19}$$

This number is very important, inasmuch as it allows to predict the moment when the number of infections begins to fall down.

Compiling the major properties discussed so far, the dynamics of the SIR model can be described by the following theorems:

Theorem 1.4.1 (Epidemic Threshold Theorem)**.** *Let $S(t)$, $I(t)$ and $R(t)$ be a solution to the model (1.7). Then, the following conditions hold:*

1. *If $\mathcal{R}_0 > 1$, then $I(t)$ starts to increase until $I_{\max}$ has been reached, and then $I(t) \xrightarrow{t\to\infty} 0$ (Epidemic).*
2. *If $\mathcal{R}_0 < 1$, then $I(t) \xrightarrow{t\to\infty} 0$ (DFE).*

Proof. The proof of this theorem can be found in [44]. Therefore, the proof is omitted. □

Theorem 1.4.2 (Kermack and McKendrick, adapted from [13])**.** *The dynamics of the epidemic develops in compliance with (1.7) subject to initial conditions $S(0), I(0)$ and $R(0)$, where $S(0) + I(0) = N$. Hence, the following conditions hold:*

1. *When the diffusion of the viral content stops, there exists a positive number of susceptible individuals that were never infected and $R_\infty = S(0) + I(0) - S_\infty$.*
2. *For $S(0) > \dfrac{1}{\mathcal{R}_0}$, the condition $\left.\dfrac{dI}{dt}\right|_{t=0} > 0$ is necessary and sufficient to the occurrence of a surge.*
3. *If $S(0) > \dfrac{1}{\mathcal{R}_0}$ by a small quantity ε and $I(0) < \varepsilon$, then the final number of susceptible individuals that remain within the population is approximately $\dfrac{1}{\mathcal{R}_0} - \varepsilon$ and $R_\infty \approx 2\varepsilon$.*

Proof. This theorem can be proved using the same technique as in [13]. Hence, the proof is omitted. □

1.5 The SIRS Model

As an intermediate between SIS and SIR epidemiological models, the SIRS model is very known not only in what concerns to the modeling of infectious diseases, but also in terms of developing control actions to minimize the damages caused by them [70].

The biological meaning of the SIRS epidemiological model is almost the same as the standard SIR model, with the exception that this model considers that individuals can lose immunity and

enter to the susceptible state, once again, at a rate δ. Hence, the immune state lasts for a limited time window. The compartmental diagram for this model is illustrated in Fig. 1.3.

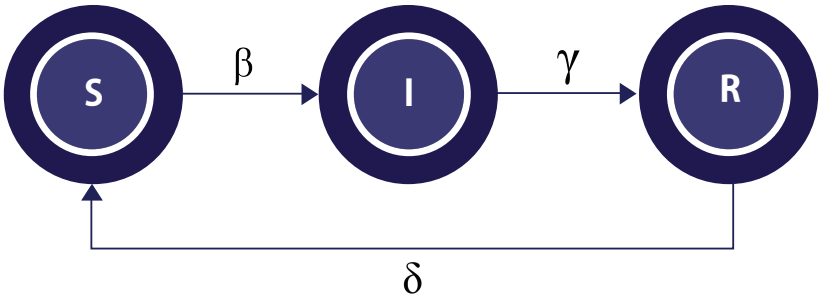


Figure 1.3: Compartmental diagram for the SIRS model.

Example 1.5.1. *Influenza, e.g., is a viral disease that can be modeled by SIRS models.*

Hence, the deterministic SIRS model is formed by the following system of ODEs:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\dfrac{\beta S(t)I(t)}{N} + \delta R(t) \\ \dfrac{dI(t)}{dt} = \dfrac{\beta S(t)I(t)}{N} - \gamma I(t) \\ \dfrac{dR(t)}{dt} = \gamma I(t) - \delta R(t)\ , \end{cases} \tag{1.20}$$

subject to initial conditions $S(0) > 0$, $I(0) > 0$ and $R(0) = 0$.

Once again, the total size of the population, N, is considered constant. Notice that, when infected individuals get recovered, the number of recovered individuals $R(t)$ increases at a rate γ ($\gamma I(t)$). In contrast, this number decreases at a rate δ ($-\delta R(t)$) when individuals lose immunity and become susceptible once again - increasing the number of individuals in the class S ($\delta R(t)$).

Considering $S(t) + I(t) + R(t) = N$, the solutions

$$(S^*, I^*, R^*) = (N, 0, 0) \text{ and } (S^*, I^*, R^*) = \left(\frac{\gamma N}{\beta}, \frac{\delta N(\beta - \gamma)}{\beta(\gamma + \delta)}, \frac{\gamma N(\beta - \gamma)}{\beta(\gamma + \delta)}\right) \tag{1.21}$$

are the equilibrium states of the system (1.20), satisfying the system

$$\begin{cases} \dfrac{dS(t)}{dt} = 0 \\ \dfrac{dI(t)}{dt} = 0 \\ \dfrac{dR(t)}{dt} = 0 \ . \end{cases} \tag{1.22}$$

Note also that the solutions (1.21) correspond to the DFE and endemic state, respectively, and, as before, $\mathcal{R}_0 = \dfrac{\beta}{\gamma}$ considering $S(0) \approx N$, requiring the condition $\mathcal{R}_0 > 1$ to have an epidemic. The calculations related to the linear stability of the steady states are omitted.

1.5.1 On the SIRS Models

Regarding the study of SIRS models, Lyapunov functions are used to prove that the DFE of an SIRS epidemic model with generalized non-linear incidence rate and vaccination is globally asymptotically stable when the $\mathcal{R}_0 \leqslant 1$, and that the endemic equilibrium is globally asymptotically stable for $\mathcal{R}_0 > 1$ [62]. At this point, global stability has also been the subject of intensive research, using Lyapunov functional techniques: Y. Muroya et al. [88] performed a global analysis on an SIRS epidemic model, but with graded cure and incomplete recovery rates, reaching to the same conclusion for $\mathcal{R}_0$ as in [62]; Y. Nakata et al. [91] studied global stability of an endemic equilibrium for an SIRS epidemic model with distributed time delays. In fact, this work was deepened in [30], where sufficient conditions of the rate of immunity loss for the global asymptotic stability of an endemic equilibrium are established. Still on global stability, curious results were derived in [86], where sufficient conditions for the global stability of an endemic equilibrium of a multi-group SIRS epidemic model with varying population sizes were established without a grouping technique by graph theory (frequently used to study multi-group SIR models).

In addition, stochastic fluctuations have already proved to be successful, by providing control interventions to manage the dynamics of an infectious disease (see, e.g., [16, 22]).

1.6 Concluding Remarks

In this Chapter, SIS, SIR and SIRS models were introduced and described. For each one of them, major mathematical properties were recalled and some literature was referenced. These properties will support the arguments given in the Second Part of this dissertation.

It should be mentioned that, in addition to the models previously described, other types of epidemiological models can be found in the current scientific literature (see, e.g., [45, 68, 73, 74, 80, 105, 107], amongst many others).

The importance of $\mathcal{R}_0$ on the model dynamics was mathematically shown. Here, the reading of [79, 107] for the computation and examples of this number using the next generation method is highly recommended.

To sum up, it is possible to assure that mathematical models are increasingly important for the development of Mathematical Epidemiology and, consequently, for the control of disease epidemics. At this point, the conditions to introduce Optimal Control Theory are in place.

2 | Optimal Control Theory

This Chapter introduces the fundamental results on Optimal Control Theory with applications to biological models, for systems with continuous time ODEs. The Pontryagin's Maximum Principle is presented and some numerical methods to solve Optimal Control problems are described. At the end of this Chapter, the relationship between epidemiological models and Optimal Control Theory is studied by revisiting the scientific literature.

2.1 Introduction

With a major importance in several fields, from Biology to Economics, Physics or Engineering, Optimal Control (OC) Theory is an extension of the calculus of variations that seeks to determine optimal strategies to control a given dynamical system, eventually subject to constraints, and optimize (maximize or minimize) a specific performance index [101, 106].

Within biological phenomena, OC provides useful insights into how circumstances involving trade-off situations should be handled [64]. However, OC can be applied to several fields beyond Biology, as will be seen in the Second Part of this dissertation.

Given a dynamical system of ODEs, the behavior of the state system is described by a set of state variables. Moreover, it is assumed that the state variables can be steered using one or more control functions, that will have influence on the dynamics of the state system. Hence, the ultimate goal is to adapt the control function(s) in order to optimize a performance index, hereinafter called objective functional, which in turn balance the objective established a priori. Frequently, this functional represents a trade-off between the intended goal and the cost to achieve it. However, it should be noted that this cost may have other meanings apart from money [64].

Since the main purpose of this dissertation is to interconnect epidemiological models with OC theory, the mathematical theory presented throughout this Chapter was mainly retrieved from [64]. Nonetheless, other good references can be considered (see, e.g., [21, 33, 101]).

2.2 Formulation of an Optimal Control Problem

In this dissertation, the general nonlinear dynamical control system takes the following form:

$$\frac{dx}{dt} = g(t, x(t), u(t)), \quad x(0) = x_0 \ , \quad t \in [0, t_f] \ , \tag{2.1}$$

where the solution $x(t)$ is absolutely continuous and piecewise differentiable such that

$$x(t) = x_0 + \int_0^t g(s, x(s), u(s)) \ ds \ , \tag{2.2}$$

where $g : I \times X \times U \to \mathbb{R}^n$ is a C^1 map, I is an interval of $\mathbb{R}$, X and U are open sets of $\mathbb{R}^n$ and $\mathbb{R}^m$, respectively. The state $x(t) \in \mathbb{R}^n$ is a function of the independent time variable $t \in [0, t_f]$, where t_f is the final time, and $\dfrac{dx}{dt}$ is governed by the function g. Let $U = \{u : I \to \mathbb{R}^m \mid u(\cdot) \text{ is Lebesgue measurable}\}$. The piecewise continuous function $u(t) \in U$ represents the control. The pair $(x(t), u(t))$ is *admissible* when, for a starting point x_0, all trajectories lie in $X \times U$ over $[0, t_f]$. Moreover, control functions that produce admissible trajectories are defined as *admissible control functions*.

Here, the main goal is to find the control function $u(t)$ in (2.1), in order to obtain the best response of the given control system. As the control function is changed, the solution (2.2) will also change. Note that the solution depends on both the control and the initial conditions.

Considering $t \in [0, t_f]$, our basic OC problem relates to find a piecewise continuous control function $u(t)$ and the associated state variable $x(t)$, aiming to maximize an objective functional $J(u)$, that is,

$$\begin{aligned} \max_{u} \quad & J(u) = \int_0^{t_f} f(t, x(t), u(t)) \ dt \\ \text{subject to} \quad & \frac{dx}{dt} = g(t, x(t), u(t)) \\ & x(0) = x_0 \ , \end{aligned} \tag{2.3}$$

$x(t_f)$ could be free or fixed, when the value of $x(t_f)$ is unrestricted or $x(t_f) = x_f,$ respectively.

Henceforth, f, g will always represent continuously differentiable functions in all three arguments. Therefore, the control(s) will always be piecewise continuous and the related states will be piecewise differentiable.

Remark 2.2.1. *An OC problem can be described by three formulations, namely Lagrange, Mayer and Bolza. In addition, it can be proved that these formulations are equivalent (see, e.g., [107] for more detailed information).*

Next, the necessary conditions that an OC and the corresponding state variables must satisfy are introduced.

2.3 Pontryagin's Maximum Principle

Introduced by Lev Pontryagin et al. [101], the Pontryagin's Maximum Principle (PMP) is a fundamental result in OC Theory. These authors introduced the concept of adjoint functions with an analogous purpose as the Lagrange multipliers in multivariate calculus, that affix constraints to the function to be optimized. In this case, adjoint functions are introduced to affix the ODEs to the objective functional.

Before moving on to the PMP, let us define the Hamiltonian function H.

Definition 2.3.1 (Hamiltonian)**.** *Consider the OC problem (2.3). The Hamiltonian H is defined by the equation*

$$H(t, x(t), u(t), \lambda(t)) = f(t, x(t), u(t)) + \lambda(t) g(t, x(t), u(t))$$

Theorem 2.3.1 (Pontryagin's Maximum Principle)**.** *If $u^*(t)$ and $x^*(t)$ are optimal for the OC problem (2.3), then there exists a piecewise differentiable adjoint variable $\lambda(t)$ such that*

$$H(t, x^*(t), u(t), \lambda(t)) \leqslant H(t, x^*(t), u^*(t), \lambda(t)) \ ,$$

for all controls u at each time t, where H is the Hamiltonian and

$$\frac{d\lambda(t)}{dt} = -\frac{\partial H(t, x^*(t), u^*(t), \lambda(t))}{\partial x} \qquad \textit{(adjoint equation)}$$

$$\lambda(t_f) = 0 \qquad \textit{(transversality condition)}$$

Proof. The proof of this theorem can be found in [101]. □

Notice that both the adjoint and Hamiltonian H allow to obtain the *optimality condition*

$$\frac{\partial H}{\partial u} = 0 \ , \quad \text{at } u^* \text{ for each } t \in [0, t_f] \ , \tag{2.4}$$

that states that the Hamiltonian H has a critical point, in the variable u, at u^* for each t.

Remark 2.3.1. *Based on the above, the use of the Hamiltonian is sufficient to derive the necessary conditions that an optimal control and state must satisfy, inasmuch as, considering the PMP, the problem of finding a control that maximizes the objective functional was converted to maximizing the Hamiltonian pointwise with respect to u.*

In addition, the following concavity conditions hold:

- If $\frac{\partial^2 H}{\partial u^2} < 0$ at u^*, for each $t \in [0, t_f]$, then the OC problem is of the maximization type.
- If $\frac{\partial^2 H}{\partial u^2} > 0$ at u^*, for each $t \in [0, t_f]$, then the OC problem is of the minimization type.

Considering that it is possible to solve for the optimal control in terms of $x^*(t)$ and $\lambda(t)$, the formula for $u^*(t)$ is called *characterization of the optimal control*. So, the *optimality system* is defined by the state and adjoint equations together with the characterization of the optimal control and the boundary conditions.

So far, necessary conditions were derived. However, to solve an OC problem, some issues can arise with this method: the necessary conditions could yield multiple solution sets, only some of which are optimal controls, or even they could be solvable when the original OC problem has no solution [64].

Hence, the following results justify the methods used previously.

Theorem 2.3.2. *For the OC problem (2.3), suppose that $f(t, x(t), u(t))$ and $g(t, x(t), u(t))$ are both continuously differentiable functions in their three arguments and concave in x and u. Suppose that $u^*(t)$ is a control, with associated state $x^*(t)$, and $\lambda(t)$ a piecewise differentiable function, such that $u^*(t)$, $x^*(t)$ and $\lambda(t)$ together satisfy, on $[0, t_f]$, the following conditions:*

$$\frac{\partial f}{\partial u} + \lambda(t)\frac{\partial g}{\partial u} = 0 \ ,$$
$$\frac{d\lambda(t)}{dt} = -\left(\frac{\partial f}{\partial x} + \lambda(t)\frac{\partial g}{\partial x}\right) \ ,$$
$$\lambda(t_f) = 0 \ ,$$
$$\lambda(t) \geqslant 0 \ .$$

Then for all controls $u(t)$, yields

$$J(u^*(t)) \geqslant J(u(t)) \ .$$

Proof. See [64] for the proof of this theorem. □

Since the above theorem does not guarantee an optimal control with finite objective functional, the following result gives the existence of $u^*(t)$ with $J(u^*(t))$ finite.

Theorem 2.3.3 (Existence of OC)**.** *Let the set of controls for the problem (2.3) be Lebesgue integrable functions on $[0, t_f]$ with values in $\mathbb{R}$. Suppose that $f(t, x(t), u(t))$ is concave in u, and there exists constants C_4 and $C_1, C_2, C_3 > 0$ and $\beta > 1$ such that*

$$g(t, x(t), u(t)) = \alpha(t, x(t)) + \beta(t, x(t))u(t)$$
$$\big|g(t, x(t), u(t))\big| \leqslant C_1\big(1 + |x(t)| + |u(t)|\big)$$
$$\big|g(t, \widetilde{x}(t), u(t)) - g(t, x(t), u(t))\big| \leqslant C_2\big|\tilde{x}(t) - x(t)\big|(1 + |u(t)|)$$
$$f(t, x(t), u(t)) \leqslant C_4 - C_3|u(t)|^\beta,$$

for all $t \in [0, t_f]$, $x(t)$, $\tilde{x}(t)$, $u(t) \in \mathbb{R}$. Then, there exists an optimal control problem $u^*(t)$ maximizing $J(u(t))$, with $J(u^*(t))$ finite.

Proof. The proof of this theorem can be found in [21, 33]. □

Remark 2.3.2 (Uniqueness)**.** *For a maximization problem, suppose that there is $u^*(t)$ such that $J(u(t)) \leqslant J(u^*(t)) < \infty$ for all control(s) $u(t)$. The optimal control $u^*(t)$ is unique if whenever*

$J(u(t)) = J(u^*(t))$, *then* $u(t) = u^*(t)$ *at all but finitely many points. It should also be emphasized that:*

- *If the functions* $f(t, x(t), u(t))$ *and* $g(t, x(t), u(t))$*, and the right hand side of the adjoint equation are Lipschitz in the state and adjoint variables, then the uniqueness of solutions of the optimality system holds for a small* t_f*.*

- *However, for an autonomous control system, the above argument holds for any* t_f*, and the Hamiltonian* H *is a constant function of time* t *along the optimal path – in the Second Part of the dissertation will only be considered autonomous control systems.*

2.4 Optimal Control with Payoff Terms

OC with payoff terms are often used when it is intended to optimize a function at t_f, in detriment of the entire time interval. An OC problem with payoff terms takes the following form

$$\begin{aligned} \max_u \quad & J(u) = \phi(x(t_f)) + \int_0^{t_f} f(t, x(t), u(t))\ dt \\ \text{subject to} \quad & \frac{dx}{dt} = g(t, x(t), u(t)) \\ & x(0) = x_0\ , \end{aligned} \tag{2.5}$$

where the payoff (or salvage) term $\phi(x(t_f))$ is a goal with respect to the final position or population level $x(t_f)$.

In comparison to the problem (2.3) and regarding the necessary conditions for this problem, the only change is in the transversality condition. The following result establishes the necessary conditions for the problem (2.5).

Proposition 2.4.1. *If* $u^*(t)$ *and* $x^*(t)$ *are optimal for the OC problem (2.5), then there exists a piecewise differentiable adjoint variable* $\lambda(t)$ *such that*

$$H(t, x^*(t), u(t), \lambda(t)) \leqslant H(t, x^*(t), u^*(t), \lambda(t))\ ,$$

for all controls u *at each time* t*, where* H *is the Hamiltonian defined as for (2.3) and*

$$\frac{d\lambda(t)}{dt} = -\frac{\partial H(t, x^*(t), u^*(t), \lambda(t))}{\partial x} \qquad \textit{(adjoint equation)}$$

$$\frac{\partial H}{\partial u} = 0 \qquad \textit{(optimality condition)}$$

$$\lambda(t_f) = \phi'(x^*(t_f)) \qquad \textit{(transversality condition)}$$

Proof. The proof of this proposition can be found in [50] (as cited in [107]). □

2.5 Optimal Control with Bounded Controls

In order to obtain realistic solutions, some OC problems require bounds on the control function $u(t)$. Next, it is considered that the control has a lower bound $u_{\min}$ and an upper bound $u_{\max}$.

So, this type of OC problem takes the form

$$\begin{aligned} \max_{u} \quad & J(u) = \phi(x(t_f)) + \int_0^{t_f} f(t, x(t), u(t))\ dt \\ \text{subject to} \quad & \frac{dx}{dt} = g(t, x(t), u(t)) \\ & x(0) = x_0 \\ & u_{\min} \leqslant u(t) \leqslant u_{\max}\ , \end{aligned} \tag{2.6}$$

where $u_{\min}$ and $u_{\max}$ are fixed and $u_{\min} < u_{\max}$. In addition, the necessary conditions are given as follows.

Proposition 2.5.1. *If $u^*(t)$ and $x^*(t)$ are optimal for the OC problem (2.6), then there exists a piecewise differentiable adjoint variable $\lambda(t)$ such that*

$$H(t, x^*(t), u(t), \lambda(t)) \leqslant H(t, x^*(t), u^*(t), \lambda(t))\ ,$$

for all controls u at each time t, where H is the Hamiltonian defined as for (2.3) and

$$\frac{dx(t)}{dt} = \frac{\partial H}{\partial \lambda}\ , \qquad x(0) = x_0$$

$$\frac{d\lambda(t)}{dt} = -\frac{\partial H}{\partial x} \qquad \textit{(adjoint equation)}$$

$$\frac{\partial H}{\partial u} = 0 \qquad \textit{(optimality condition)}$$

$$\lambda(t_f) = \phi'(x(t_f)) \qquad \textit{(transversality condition)}\ .$$

Hence,

$$\begin{cases} u^*(t) = u_{\min} & \textit{if} \quad \dfrac{\partial H}{\partial u} < 0 \\ u_{\min} \leqslant u^*(t) \leqslant u_{\max} & \textit{if} \quad \dfrac{\partial H}{\partial u} = 0 \\ u^*(t) = u_{\max} & \textit{if} \quad \dfrac{\partial H}{\partial u} > 0\ . \end{cases} \tag{2.7}$$

Proof. The proof of this proposition can be found in [50] (as cited in [107]). □

Apart from the standard examples, the vast majority of the controlled dynamical systems can not be solved algebraically. Thus, the following Section depicts numerical methods to solve OC problems.

2.6 Numerical Methods to Solve Optimal Control Problems

The complexity of general nonlinear programming problems (NLP) with optimal control requires the use of numerical methods in order to optimize a given performance index of a dynamical system subject to constraints [106]. Hence, this Section introduces two types of methods to solve OC problems related to dynamical systems, which are usually described by ODEs: indirect methods and direct methods, that will be described in Sections 2.6.2 and 2.6.3.

Firstly, for the sake of convenience, let us start with a short description of numerical methods to solve dynamical systems of ODEs.

2.6.1 Numerical Methods for Ordinary Differential Equations

When a system of ODEs is to extensive or composed by equations that can not be solved analytically, numerical methods reveal their importance in order to compute a numerical approximation to the solution of an initial-value problem. Among many others, Euler's method and fourth order Runge-Kutta method are here introduced (see, e.g. [104] for numerical examples of these two methods and [96] for other numerical methods to solve ODEs). The following numerical methods assume ODEs in the form

$$\frac{dy(t)}{dt} = f(t, y(t)) \ , \quad y(t_0) = y_0 \ . \tag{2.8}$$

2.6.1.1 Euler's Method

Euler's method approximates the equation (2.8) by

$$y_{n+1} = y_n + hf(t_n, y_n) \ . \tag{2.9}$$

From an equivalent perspective, this method can be described by the following equation:

$$y(t+h) = y(t) + h\frac{dy(t)}{dt} \ , \tag{2.10}$$

where the h is called time step.

This method computes the value at $t+h$ with an error of order $\mathcal{O}(h^2)$. The time step h is crucial to determine the accuracy of the approximation. Here, the smaller the value of h, the greater the accuracy of the approximation. However, the smaller the value of h, the greater the computational effort required. As such, for higher order systems of ODEs, due to its dependency on h, the Euler's method can become unstable and, consequently, does not converge to the solution.

2.6.1.2 Runge-Kutta Method

There are several modifications of the Runge-Kutta method to compute an approximated solution for equations that take the form (2.8). One of these modifications is the fourth order

Runge-Kutta method (RK4), that was developed with the main purpose of producing results with the same accuracy as the methods obtained by Taylor series expansion, but avoiding the computation of several derivatives of f. This method has the following form:

$$y_{n+1} = y_n + \frac{h}{6}\bigg(k_1 + 2k_2 + 2k_3 + k_4\bigg), \tag{2.11}$$

where

$$\begin{aligned} k_1 &= f(t_n, y_n) \\ k_2 &= f(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_1) \\ k_3 &= f(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_2) \\ k_4 &= f(t_n + h, y_n + hk_3)\ . \end{aligned}$$

The parameter h is the time step. Notice not only that y_{n+1} is the approximation of $y(t_n + h)$, but also that the values of $k_i : i = 1, 2, 3, 4$, are computed for each n and used in $n + 1$. In addition, this method has an error of $\mathcal{O}(h^4)$ and it is conditionally stable.

Before moving ahead to the description of indirect and direct numerical methods, the reading of [106] is recommended as a theoretical support for the next sections.

2.6.2 Indirect Numerical Methods

Indirect methods use the PMP to solve OC problems. More precisely, PMP is used to compute the first-order optimality conditions. Thus, the optimal trajectories are derived by solving a multiple-point boundary-value problem. At this point, as an indirect numerical approach, *Forward-Backward Sweep Method* (FBS) is here introduced (see [64] for more details). This method can be used to solve problems with the formulation of (2.3) (minimization problems are obviously included), and generates numerical approximations to the optimal piecewise continuous control $u^*(t)$.

Let us consider $\vec{x} = (x_1, ..., x_{N+1})$ and $\vec{\lambda} = (\lambda_1, ..., \lambda_{N+1})$ as the vector for the state and adjoint, respectively. Hence, this algorithm can be informally understood as follows.

Algorithm 1: Forward-Backward Sweep Method

1. Initial guess for $\vec{u}$ over the time interval ($\vec{u} \equiv 0$ is almost always sufficient).
2. Using $x_1 = x_0$ and the values for $\vec{u}$, solve $\vec{x}$ **forward** in time, in compliance with its differential equation in the optimality system (using, e.g., RK4).
3. Using $\lambda_{N+1} = \lambda(t_f) = 0$ and the values for $\vec{u}$ and $\vec{x}$, solve $\vec{\lambda}$ **backward** in time, in compliance with its differential equation in the optimality system (using, e.g., RK4).
4. Update $\vec{u}$ using the new values for $\vec{x}$ and $\vec{\lambda}$ into the characterization of the OC.
5. Check convergence. If values of the variables in this iteration and the last one are sufficiently close, output the current values as solutions. Otherwise, return to step 2.

Remark 2.6.1. *The convergence speed can be improved when a convex combination between the control values given by the characterization of the optimal control in step 4 and the previous ones is used.*

2.6.3 Direct Numerical Methods

Assertively, direct methods use discretization with respect to time to solve OC problems, in which the objective functional is directly optimized [96] and treat an OC problem as a nonlinear constrained optimization problem (see, e.g., [6] for more details). In these problems, it is common to use nonlinear optimization software packages through platforms such as NEOS server [92] to solve them. Here, among many others, the solvers IPOPT, KNITRO and LOQO are briefly described.

IPOPT [123], for *Interior Point OPTimizer*, is a software package for large-scale nonlinear optimization, written in FORTRAN and C, that implements a primal-dual interior-point method using line search methodologies based on filter methods.

KNITRO [15], for *Nonlinear Interior Point Trust Region Optimization*, is a nonlinear solver for large-scale problems, but also able to support linear, quadratic, and general smooth nonlinear optimization in continuous and integer variables.

LOQO [122], is an optimization solver for smooth constrained problems, supported to linear,

quadratic, nonlinear, convex or nonconvex objectives and constraints in continuous variables. It should be emphasized that for convex problems, this solver finds a globally optimal solution.

Remark 2.6.2. *Some advantages and drawbacks can be pointed out to both indirect and direct methods (see [109]). In this regard, according to E. Trélat [121], direct methods are more robust and less sensitive to the choice of the initial conditions than indirect methods, being more easier to implement. However, reaching to a desirable accuracy highly depends on the chosen NLP solver. In addition, the author states both the possibility of obtain local minima, when the direct discretization of an OC problem is employed, and the necessity of a large amount of memory, which in turn can lead to inefficiencies (when, for instance, the dimension of the problem is too complex). On the other hand, indirect methods provide high levels of numerical accuracy, but its implementation can be quite difficult due to the necessity of computing derivatives and necessary conditions related to the PMP.*

The choice of the best method for a problem deeply depends on the features of the dynamical system. In this regard, in the Second Part of this dissertation, both indirect and direct methods to solve continuous time dynamical systems are compared using different numerical solvers.

Finally, by exploring the current scientific literature, we end this Chapter showing the actual synergies between epidemiological models and OC Theory.

2.7 Problems Mixing Epidemiological Models and Optimal Control Theory

Within an enormous set of applications, some major problems mixing epidemiological models with OC are now highlighted and the relevance and usefulness of this dissertation's topic, regardless of the application, is enhanced.

The application and formulation of mathematical models to describe several epidemic phenomena is gathering further highlight. In addition, to both optimally manage the response of a mathematical system when optimization strategies are added to it, and get the best possible result, OC Theory is often combined with epidemiological models (see [38]).

Conventionally, controlled epidemic models are applied to infectious diseases in an attempt to prevent and control them. From a wide range of topics, epidemiological models with OC have been

applied to study the epidemic spreading based on complex networks, considering virus variation factors [130]; to modeling cholera using quarantine treatments [63], public health interventions [90, 103] or optimal vaccination strategies [18]; to investigate intervention strategies to malaria [17, 31, 89, 93]; to study the transmission dynamics of hepatitis C using control treatments [95, 135]; to modeling HIV transmission [77, 94, 113, 114, 136] or even tuberculosis [10, 85, 115]. In addition, particular attention has been given to Ebola virus, by proposing OC interventions, related to treatment and preventive controls, that could minimize its fatality rate (see, e.g., [4, 9] and the references cited therein).

However, control interventions must be applied using cost-reducing principles. In this regard, P. Giamberardino and D. Iacoviello [37] proposed a controlled SIR epidemiological model with state dependent switching cost index in order to use resources in a rational way, depending on the severity of the disease.

Summarizing, it is now clear that the integration of OC Theory in epidemiological models allows to formulate strategies that optimize a given objective functional, which in turn eventually provides an optimal solution for a certain complex problem.

The fruitful relationship between OC Theory and Epidemiology has also been applied to modeling Viral Marketing (VM) and Computer Viruses Transmission (CVT) processes. Throughout the Second Part of this dissertation, scientific literature related to these subjects is presented.

2.8 Concluding Remarks

This Chapter recalled necessary conditions and an existence result related to standard OC problems. Since the majority of the controlled dynamical systems are too complex to be algebraically solved, indirect and direct numerical methods to solve OC problems were described and discussed. The relationship between epidemiological models and OC Theory was pointed out by briefly revisiting current scientific literature.

Next, in the Second Part of this work, epidemiological models and OC Theory are used to understand the dynamics of VM, aiming to provide control policies that may maximize information spreading with low cost. In addition, CVT is also studied by taking advantage of numerical simulations related to epidemic models over networks.

Part II

Applications: Marketing and Computer Viruses Transmission

List of Publications

The Second Part of the dissertation introduces the work that formed the basis for the development of four scientific research publications, published, in press or submitted to publication. Hence, the full list of publications is as follows.

1. **J.N.C. Gonçalves**, H.S. Rodrigues and M.T.T. Monteiro. Optimal Control Strategies for an Advertisement Viral Diffusion. *In: A. Ismael Vaz, J. Paulo Almeida, J. Fernando Oliveira and A. Adrego Pinto (eds.) XVIII Congress of Operational Research. IO 2017. In Press (Springer Proceedings in Mathematics and Statistics)*, 2017.

2. **J.N.C. Gonçalves**, H.S. Rodrigues and M.T.T. Monteiro. A Contribution of Dynamical Systems Theory and Epidemiological Modeling to a Viral Marketing Campaign. *In: A. Madureira, A. Abraham, D. Gamboa, P. Novais (eds.) Intelligent Systems Design and Applications. ISDA 2016, Advances in Intelligent Systems and Computing*, Springer, 557:974–983, 2017.

3. **J.N.C. Gonçalves**, H.S. Rodrigues and M.T.T. Monteiro. On the Dynamics of a Viral Marketing Model with Optimal Control using Indirect and Direct Methods. *Submitted to Mathematical Methods in the Applied Sciences*, 2017.

4. **J.N.C. Gonçalves**, H.S. Rodrigues and M.T.T. Monteiro. Optimal Control using an Epidemiological Model on a Viral Marketing Campaign. *Submitted to Communications in Mathematics and Statistics*, 2016.

3 | Viral Marketing

Within a marketing context, the transmission of a marketing message to a specific audience can be quite erratic. In this Chapter, a definition and description of the Viral Marketing process is given. Moreover, in order to understand the dynamics of a viral message within a population, a SIR model applied to a real Viral Marketing campaign is introduced and studied. With the main purpose of maximize, in a economically way viable, the impact of a marketing message under different marketing strategies, OC Theory is incorporated into the mathematical models. Furthermore, numerical simulations and methods to solve the models are also tested and compared, aiming to provide guidelines to marketers.

3.1 Understanding the Dynamics of Viral Marketing

Marketing is a valuable tool to orient and increase the performance of an organization [110], in such a way that companies frequently use marketing strategies to enhancing and strengthening a brand. However, over time, to cope with progress in technology, traditional marketing strategies have proved to be ineffective from the perspective of meeting demanding conditions of consumers [102]. Therefore, since the beginning of a successful marketing campaign deeply depends on the motivation of individuals in such a way that they will feel challenged and inspired to spread a marketing message within their social network [1], marketing professionals have been trying to design attractive and viral campaigns, based on strategies that allow to revert the drawbacks of traditional marketing.

One of these strategies is known as VM, which refers to the process that takes advantage of the word-of-mouth to replicate and diffuse a marketing message into a large set of customers [65]. According to N. Adelsarbanlar and B. Khoshtinat [1], VM can be seen as a virus, by using

communication and distribution channels to diffuse a specific information content. Whereas, in traditional marketing, communication strategies are deeply concentrated in the customer, VM creates mechanisms in order to avoid both the implication of the original source, by increasing information exchange among customers and consumers [126], and the necessity to establish a direct contact with targeted individuals [84]. In addition, VM has a snowball effect by replicating the message among the susceptible population, increasing the number of 'infected' individuals [42]. As a statement of VM's effectiveness, A. Dobele et al. [28] consider that this strategy can provide three crucial perks to a marketing company: low investment costs, using network effects; the transmission process of a message is voluntary which in turn should increase the probability of being visited by potential consumers, and induces more effectiveness in targeting individuals (see, e.g., [98] for more detailed information about reasons for forwarding messages). Nevertheless, when planning and implementing VM strategies, marketing professionals must guarantee that characteristics like entertainment, humor, curiosity, surprise, useful information, and relevant content are not neglected, in order to convert a simple message into a viral and attractive one [27].

According to M. Woerndl et al. [126], VM has, however, some drawbacks that must be highlighted: negative word-of-mouth that could lead to boycott and ruin; privacy issues and the fact that consumers can feel exploited, cheated and used. Moreover, these authors stand out the uncontrollable nature of this marketing strategy, namely related to an eventual loss over content and few possibilities to measure success and timing.

Thus, in view of the above, the ultimate goal of VM relates to reach a large audience with a low cost associated to it, by exploiting network effects which in turn maximize the process of spreading the intended information. Note that this goal can be seen as a trade-off and the use of mathematical tools might be a fruitful solution to reach the masses without compromising the financial capacity of a company.

Assertively, the unpredictable dynamics of VM, justified by the difficult in predicting the success of a marketing campaign, is the main motivation for the choice of this application in this dissertation - since it is crucial to find strategies that could control the uncertainty associated to the impact of a specific marketing campaign and, consequently, turn it into a viral, successful and effective message. Taking into account that one of the major difficulties in diffusing a viral message relates to create mechanisms that convince and persuade individuals to spread it on the

best time window, OC Theory is considered. Over time, OC problems applied to information epidemics have been proposed and discussed [51, 52, 54, 60]. More precisely, these research studies focus on study optimal strategies to promote information diffusion in social networks and environments. In this regard, and considering that VM can be modeled by epidemiological models [65, 108, 116, 124], the dynamics and impact of a real VM advertisement, Dove Real Beauty Sketches, are studied using mathematical theory presented in the First Part of this dissertation. Produced in 2013, Dove Real Beauty Sketches is a publicity campaign that focuses on state the definition of beautiful, promoting self-esteem and changing the perception of beauty [29]. This campaign was a huge success across the globe, considered the most viral video advertisement of all time [117], and represents an excellent example of how to build a successful viral marketing campaign.

Thereby, under a mathematical perspective and based on Dove's campaign numerical data, the next sections study the dynamics of VM and optimal policies that might help marketing professionals to increase information diffusion with low cost in future campaigns.

3.1.1 Viral Marketing as an Epidemiological Dynamical System

Motivated by the overwhelming success of Dove's campaign, the mathematical properties and dynamics of the campaign are now studied - from the parameters estimation to the stability of the mathematical model, simulated in MATLAB. Mathematically, using an SIR epidemiological model, it is shown not only that the campaign was a viral epidemic, but also that it can be leveraged and optimized by epidemiological and mathematical modeling, which offer important guidelines to maximize the impact of a viral message and minimize the uncertainty related to the conception and outcome of new marketing campaigns.

3.1.1.1 Can Mathematical Epidemiology and Marketing Work Together?

As mentioned above, taking into consideration that VM can be modeled under standard epidemic models, an SIR epidemiological model is considered to analyze, over time, the dynamics of the Dove Real Beauty Sketches marketing campaign, under initial conditions related to the campaign real data presented in [117]:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta \dfrac{S(t)I(t)}{N} \\ \dfrac{dI(t)}{dt} = \beta \dfrac{S(t)I(t)}{N} - \gamma I(t) \\ \dfrac{dR(t)}{dt} = \gamma I(t)\ , \end{cases} \qquad \begin{cases} S(0) = 10^9 - 30000 \\ I(0) = 30000 \\ R(0) = 0\ . \end{cases} \tag{3.1}$$

In terms of marketing, we define compartment S as the audience that marketers want to reach with the marketing message; I as the individuals that transmit the message or recommend a product to friends or relatives, motivated by monetary interests or just for the attractiveness of the intended message, and R as the population who stop transmitting the message because, for instance, it is no longer appealing. Regarding the parameters in terms of marketing, β can be seen as the predisposition to deal with the message and be motivated to share it, and γ as the interruption of the message transmission by individuals [108].

The total host population N, estimated by 10^9 [111], is considered to be constant in time.

Noting that $R(t)$ derives from $S(t)$ and $I(t)$ and considering the fractions $s = \dfrac{S}{N}$, $i = \dfrac{I}{N}$, $r = \dfrac{R}{N}$, the SIR system (3.1) and its initial conditions can be normalized as follows.

$$\begin{cases} \dfrac{ds(t)}{dt} = -\beta s(t) i(t) \\ \dfrac{di(t)}{dt} = \beta s(t) i(t) - \gamma i(t) \\ r(t) = 1 - s(t) - i(t)\ , \end{cases} \qquad \begin{cases} s(0) = \dfrac{S(0)}{N} \\ i(0) = \dfrac{I(0)}{N} \\ r(0) = 0\ . \end{cases} \tag{3.2}$$

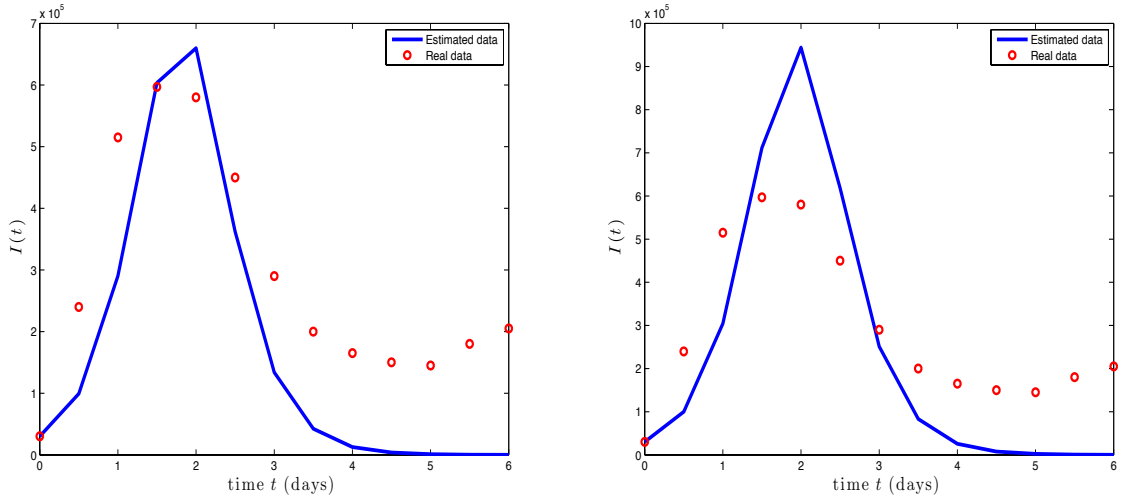
According to the analysis developed in the First Part of this dissertation, the marketing message is widely diffused if $\dfrac{di(t)}{dt} > 0$. Considering $s \approx 1$ in this inequality we get $\dfrac{\beta}{\gamma} > 1$. Here, the basic reproduction number, $\mathcal{R}_0 = \dfrac{\beta}{\gamma}$, represents secondary transmissions sent by a single individual to a specific target audience. Moreover, if $\mathcal{R}_0 < 1$ the message is not widespread. On the other hand, if $\mathcal{R}_0 > 1$ the message is broadly sent among the susceptible population [108].

Note that, by the side of marketers, an accurate estimation of the parameters β and γ deeply helps to monitor the evolution of the campaign among the social network environment. Hence, in the next Section, we estimate β and γ in order to adjust the model to the campaign real data. This estimation has taken into account the first seven days of Dove's campaign, where the

maximum peak of infection is attained.

3.1.1.2 Parameters Estimation using an Optimization Approach

The estimation of β and γ has a significant importance in the model dynamics and adjustment to the campaign real data. However, it is not trivial to find strategies or recipes to estimate model parameters. Moreover, in the marketing field, this difficult increases exponentially, due to its erratic behavior. Another important aspect is linked to the fact that, related to the word-of-mouth and network effects processes, social behavior is not predictable with a desired accuracy. So, find a perfect method to estimate parameters related to stochastic patterns it is quite difficult, and marketers must take these critical aspects into account upon the design of future campaigns, in order to have more control on the evolution of it. Although, there are accurate estimation methods among the literature to fit the model to data from an outbreak. Based on [79] (pp. 125–129), FMINSEARCH function, associated to Nelder-Mead method (see [61]), is used from the MATLAB optimization toolbox. Hence, $\beta \approx 67.5244$ and $\gamma \approx 65.0751$ were obtained as optimal parameters. In the appendix of [19], it is possible to find the sensitivity equations for the SIR model, which constitute an important indicator to study the effect of β and γ variation. In this context, considering the first seven days of the campaign, some simulations to assess and analyze the parameters variation are performed (Fig. 3.1).



(a) $\beta \approx 67.5244$, $\gamma \approx 65.0751$

(b) $\beta \approx 57.5244$, $\gamma \approx 55.0751$

Figure 3.1: The effect of β and γ variation in the adjustment to the campaign real data.

Figure 3.1(a) displays the estimated data with optimal parameters. Figure 3.1(b) presents the estimated data with a smooth variation in β and γ. According to Fig. 3.1(b), it becomes clear that a slight variation on the optimal parameters leads to a misalignment between the estimated and the real data.

3.1.1.3 Qualitative Analysis of the Solutions

This Section performs a stability and dynamical analysis of the model, by linking it to the Dove's campaign, based on standard Mathematical Epidemiology and Dynamical Systems theory. Henceforth, as a starting point to analyze and discuss mathematical properties related to stability and dynamics of the model, it will be considered the system (3.2).

Let $f : [0,1]^2 \to \mathbb{R}^2$ be a C^1 map. It is possible to write (3.2) as

$$\frac{dx}{dt} = f(x(t)),\ \forall t \in [0,\infty)\ ,$$

where

$$x(t) = \begin{bmatrix} s \\ i \end{bmatrix} \text{ and } f(x(t)) = \begin{bmatrix} -\beta s(t)i(t) \\ \beta s(t)i(t) - \gamma i(t) \end{bmatrix}.$$

By solving $f(x(t)) = 0_{\mathbb{R}^2}$, it is concluded that all the points in the form $(s^*, 0)$, where s^* can be any number on $[0,1]$, are equilibrium solutions of the system. However, special focus is devoted to two solutions: $E_1 = (1,0)$, when the levels of infection are null, and $E_2 = (0,0)$, related to the end of the epidemic, when $r \approx 1$. In analyzing equilibrium solutions it is pertinent to obtain the linearized matrix of the system (3.2), in a neighborhood of a critical point (s^*, i^*), for which $i^* = 0$:

$$\begin{aligned} J_f(s^*,0) &= \begin{bmatrix} \dfrac{\partial\left(-\beta s(t)i(t)\right)}{\partial s} & \dfrac{\partial\left(-\beta s(t)i(t)\right)}{\partial i} \\ \dfrac{\partial\left(\beta s(t)i(t) - \gamma i(t)\right)}{\partial s} & \dfrac{\partial\left(\beta s(t)i(t) - \gamma i(t)\right)}{\partial i} \end{bmatrix}\Bigg|_{(s^*,0)} \\ &= \begin{bmatrix} 0 & -\beta s^* \\ 0 & \beta s^* - \gamma \end{bmatrix}. \end{aligned} \tag{3.3}$$

Considering the characteristic equation $\det(J_f(s^*, 0) - \lambda I_2) = 0$, the eigenvalues of the Jacobian matrix $J_f(s^*, 0)$ are $\lambda_1 = 0$ and $\lambda_2 = \beta s^* - \gamma$. In general, by evaluating the eigenvalues at the equilibrium solutions, we can infer its stability. However, for E_2, linear stability analysis may not be sufficient. In this particular case, more advanced mathematical techniques to study the dynamics of the system around E_2 are necessary.

Considering the optimal parameters obtained in the previous section, it follows, for E_1 and E_2, respectively, that

$$\lambda_2 = \beta - \gamma > 0 = \lambda_1 \text{ and } \lambda_2 = -\gamma < 0 = \lambda_1 \ .$$

In relation to E_1, since $\Re(\lambda_2) > 0$, according to basic stability properties of linear systems, E_1 is an unstable solution. Establishing a relationship with marketing, this solution corresponds to the case that the fraction of individuals who recommend the marketing campaign increases faster than it stops being shared (Viral Marketing epidemic). Considering the equilibrium E_2, is not possible to conclude, from the linear stability analysis by itself, whether or not the equilibrium solution E_2 is stable. In this regard, the behavior of the autonomous systems as well as its equilibrium solutions can be described through a phase-space, attending to the equation (1.15). Hence, Fig. 3.2 shows one orbit of the SIR system (3.2) and illustrates the impact, over time, of Dove's campaign.

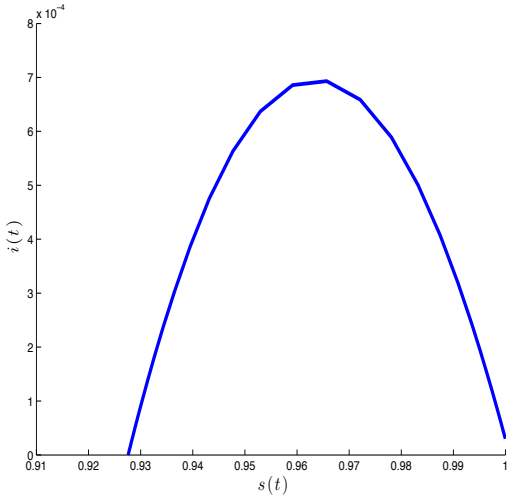


Figure 3.2: Orbit of the nonlinear SIR model.

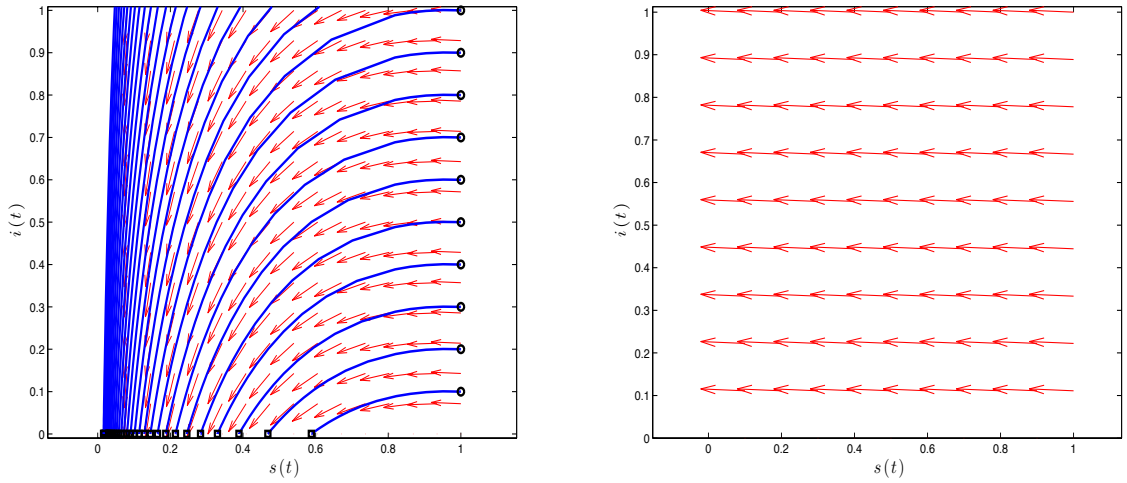
Following an assertive analysis of the Fig. 3.2, $s = 1$ represents the launching of the marketing

campaign, where almost nobody knows about it ($i \approx 0$). Over time, individuals begin to spread the marketing campaign through network effects and the fraction of people who have contact with the marketing message reaches a maximum level, that is

$$\lim_{s \to \frac{1}{\mathcal{R}_0}^+} i(s) = i_{\max} \ .$$

Note that $i_{\max}$ can be obtained through the equation (1.19). Then, when a large fraction of people has already contacted with the campaign, the fraction of infected people tends to decay to zero. It should be noted that the greater the sharing of the message, the greater the fraction of infected individuals. Below, the mathematical results previously analyzed are illustrated.

Figure 3.3(a) refers to the nonlinear model (3.2) and, for each trajectory considered, the initial and final point are marked with a *circle* and a *square*, respectively. Figure 3.3(b) displays the vector field of the linearized model, generated by (3.3) at the equilibrium E_1. From the phase-plane analysis we infer that E_2 is stable, since all trajectories starting in an arbitrary $a \in B(E_2, \delta)$, for $\delta > 0$, move about a finite range of distance, converging to an ending point $b \in \mathbb{R}^2_+$ nearby E_2. In other words, the solutions contract back in the direction of E_2. In what concerns to the vector field showed in Fig. 3.3(a), the vectors behavior is perfectly expectable due to the fact that, by the first equation of (3.2), $\frac{ds}{dt} < 0$, since $s, i > 0$. At this point, $s = \frac{1}{\mathcal{R}_0}$ is crucial to understand the dynamics of the system. For starting points $(a_0, b_0) \in \mathbb{R}^2_+$ in a neighborhood of $(s^*, 0)$, for which $s^* \ll \frac{1}{\mathcal{R}_0}$, the trajectories tend to return to a nearby solution $(s_F, 0) \in B(E_2, \delta)$, for $\delta > 0$. Relating to the marketing context, this means that if a small group of persons has contact with the marketing campaign, then it will not be widespread (no viral epidemic). Concerning to E_1, as calculated previously, this solution is unstable since the trajectories grow away from the equilibrium, and computing simulations confirm this finding (Fig. 3.3(b)). In other words, supposing that starting points $(a_0, b_0) \in \mathbb{R}^2_+$ are nearby $(s^*, 0)$, for which $s^* > \frac{1}{\mathcal{R}_0}$, the trajectories tend to diverge from the equilibrium, causing a viral epidemic. Hence, these equilibrium solutions are unstable. In terms of marketing, this means that the number of infected individuals - who recommend the campaign - tends to, gradually, grow up, until the maximum level of infection be reached. Then, over time, this number tends to fall down to nearby zero. According to Fig. 3.3(b), the instability of E_1 is even more evident through the vector field, which corroborates that Dove's campaign was a viral epidemic.



(a) Nonlinear SIR model

(b) Linearized SIR model at E_1

Figure 3.3: Vector fields of the SIR model.

The next Section applies OC Theory to the mathematical model, aiming to optimize information spreading.

3.2 Optimizing Information Spreading using Optimal Control

In this Section, the process of diffusing a viral marketing message with a low cost is studied using OC Theory and an SIR epidemiological model. In Section (3.2.1), a control intervention related to the encouragement of susceptible individuals to act as spreaders is formulated. Next, another control policy related to the fostering of infected individuals to continue to spread the message is implemented (Section (3.2.2)).

3.2.1 SIR Model with One Control

The standard SIR model characterized by the system (3.2) is now considered. Henceforth, throughout this Chapter, we assume the marketing definitions of the state variables and model parameters described in Section 3.1.1.1 and summarized in Table 3.1.

Hence, under the marketing assumptions of the SIR model described so far, the following Section formulates an OC problem, in order to optimize information spreading with a low cost.

Table 3.1: Summary of variables and parameters of SIR model under a VM context.

Symbol	Description	Estimated value	Reference
β	Predisposition to share the marketing message	67.5244	[79]
γ	Interruption of the message transmission	65.0751	[79]
N	Total population size at the campaign launching	10^9	[111]
$S(0)$	Number of susceptible individuals at the campaign launching	$10^9 - 30000$	[117]
$I(0)$	Number of infected individuals at the campaign launching	30000	[117]
$R(0)$	Number of recovered individuals at the campaign launching	0	[117]

3.2.1.1 Formulation of an Optimal Control Problem

To the system (3.2), one control function $u(\cdot)$ is added. Hence, the resultant controlled SIR epidemiological model is given by the following system of ordinary differential equations:

$$\begin{cases} \dfrac{ds(t)}{dt} = -\beta s(t)i(t) - u(t)s(t) \\ \dfrac{di(t)}{dt} = \beta s(t)i(t) + u(t)s(t) - \gamma i(t) \\ r(t) = 1 - s(t) - i(t)\ , \end{cases} \qquad \begin{cases} s(0) = 1 - 3 \times 10^{-5} \\ i(0) = 3 \times 10^{-5} \\ r(0) = 0\ . \end{cases} \tag{3.4}$$

This system was firstly introduced in [54]. The control function $u(\cdot)$ represents the recruitment of susceptible individuals to act as spreaders within social networks (e.g. through advertisements in mass media [51]) by marketing companies, purposing to maximize the levels of information diffusion.

Let t_f be the considered campaign deadline. The set of admissible control functions is defined by

$$\Omega = \left\{ u(\cdot) \in L^2(0, t_f) \mid 0 \leqslant u(t) \leqslant u_{\max},\ \forall t \in [0, t_f] \right\}. \tag{3.5}$$

For the bounded control $u(t)$, the application of the control policy is maximum if $u = u_{\max}$ and null if $u = 0$. The main goal is to find the optimal value u^* for the control u, in such a way that the state trajectories are the solution of the system (3.4) over $[0, t_f]$, and maximize the objective functional (3.6). As a trade-off, the OC problem consists in maximize the spreading of

the message (maximizing the fraction of individuals who have contacted with it) and minimize the intervention costs related to the implementation of the control strategy u, i.e.,

$$\max_{\Omega} J(u(\cdot)) = i(t_f) + r(t_f) + \int_0^{t_f} -Bu^2(t)\ dt\ , \tag{3.6}$$

subject to (3.4), where the nonnegative constant B represent the weight of the investment costs associated to the control strategy u. At this point, under a marketing perspective and assuming that the costs do not take a linear form, we consider a quadratic structure for the weighted control function instead of a linear form as in [54].

Let $s^*(t), i^*(t), r^*(t)$ and $\lambda_1^*(t), \lambda_2^*(t), \lambda_3^*(t)$ denote the optimal states and adjoint functions, respectively.

Theorem 3.2.1. *There exists an optimal control function $u^*(t) \in \Omega$ and a set of corresponding solutions $(s^*(t), i^*(t))$ that maximize the objective functional (3.6) subject to (3.4).*

Proof. This theorem is proved based on the Theorem 4.1 proposed in [33]. Hence, the following conditions must be satisfied:

1. The set of solutions of the controlled system with initial conditions (3.4) and $u \in \Omega$ is not empty.

2. The set Ω is closed and convex.

3. The right hand side of the state system (3.4) is continuous, bounded above by a sum of the bounded function u and the state variables, and can be written as a linear function in u with state variables as coefficients.

4. The integrand of the objective functional (3.6) is concave on Ω with respect to u and there exists constants $C_0 > 0$, C_1 and $C_2 > 1$ such that

$$-Bu^2(t) \leqslant C_1 - C_0\big|u(t)\big|^{C_2}\ .$$

The Condition 2 is satisfied since Ω is closed and convex by definition. Condition 3 is satisfied according to the following arguments (see, e.g., [32, 130]):
The state system (3.4) can be rewritten as

$$V(x) \doteq \frac{dx}{dt} = Ax + B(x) \ , \tag{3.7}$$

where the matrix A, the state vector x and the nonlinear function $B(x)$ are defined as follows.

$$A = \begin{pmatrix} -u & 0 \\ u & -\gamma \end{pmatrix}, \quad x = \begin{bmatrix} s & i \end{bmatrix}^T \quad \text{and} \quad B(x) = \begin{pmatrix} -\beta si & \beta si \end{pmatrix}^T .$$

In addition, notice that:

$$\begin{aligned} \Big|B(x) - B(\tilde{x})\Big| &= \left| \begin{pmatrix} -\beta\Big[(s-\tilde{s})i + \tilde{s}(i-\tilde{i})\Big] & \beta\Big[(s-\tilde{s})i + \tilde{s}(i-\tilde{i})\Big] \end{pmatrix}^T \right| \\ &\leqslant 2\big|\beta i\big|\big|s-\tilde{s}\big| + 2\big|\beta\tilde{s}\big|\big|i-\tilde{i}\big| \\ &\leqslant C\big(\big|s-\tilde{s}\big| + \big|i-\tilde{i}\big|\big) \ , \end{aligned}$$

where $C = \max\big\{2\big|\beta i\big|, 2\big|\beta\tilde{s}\big|\big\}$. Therefore,

$$\big|V(x) - V(\tilde{x})\big| \leqslant D\big|x - \tilde{x}\big| \ , \tag{3.8}$$

by taking $D = C + \|A\|$, which proves that $V(x)$ is uniformly Lipschitz continuous. Hence, also attending to the definition of Ω and the non-negativity of the state solutions, Condition 1 is satisfied. The state variables $s(t), i(t)$ and the objective functional (3.6) take values in a compact interval $[0,1]$, and $0 \leqslant u(t) \leqslant u_{\max},\ \forall t \in [0, t_f]$. Hence, $V(x)$ is bounded above by a sum of the bounded function u and the state variables. Moreover, it is easy to show that $V(x)$ can be written as a linear function in $u(t)$ with state variables as coefficients, thus Condition 3 is satisfied. Finally, Condition 4 is proved since the integrand of the objective functional (3.6) is concave on Ω and

$$-Bu^2(t) \leqslant C_1 - C_0\big|u(t)\big|^{C_2} \ , \tag{3.9}$$

if $C_0 = B$, $C_1 = 1$, $C_2 = 2$ were chosen. Hence the proof is concluded.

□

According to the Pontryagin's Maximum Principle (PMP) [101] and considering the OC problem (3.6), if $u^*(\cdot)$ is a control that is optimal for the problem, then exists a Lipschitz continuous map called covector $\lambda : [0, t_f] \to \mathbb{R}^2$, $\lambda(t) = (\lambda_1(t), \lambda_2(t))$, such that

$$\frac{ds(t)}{dt} = \frac{\partial H}{\partial \lambda_1}, \quad \frac{di(t)}{dt} = \frac{\partial H}{\partial \lambda_2} \tag{3.10}$$

and

$$\frac{d\lambda_1(t)}{dt} = -\frac{\partial H}{\partial s}, \quad \frac{d\lambda_2(t)}{dt} = -\frac{\partial H}{\partial i} \ , \tag{3.11}$$

where the Hamiltonian function H is defined by

$$\begin{aligned} H\Big(s(t), i(t), u(t), \lambda_1(t), \lambda_2(t)\Big) &= -Bu^2(t) \\ &+ \lambda_1(t)\Big(-\beta s(t)i(t) - u(t)s(t)\Big) \\ &+ \lambda_2(t)\Big(\beta s(t)i(t) + u(t)s(t) - \gamma i(t)\Big) \ . \end{aligned} \tag{3.12}$$

Theorem 3.2.2. *Given the optimal solution $(s^*(t), i^*(t))$ of the state system (3.4) and objective functional (3.6), associated to the optimal control variable $u^*(t) \in \Omega$, there exists adjoint functions $\lambda_1(t)$, $\lambda_2(t)$ such that*

$$\begin{cases} \dfrac{d\lambda_1(t)}{dt} = \lambda_1(t)\Big(\beta i^*(t) + u^*(t)\Big) - \lambda_2(t)\Big(\beta i^*(t) + u^*(t)\Big) \\ \dfrac{d\lambda_2(t)}{dt} = \lambda_1(t)\Big(\beta s^*(t)\Big) - \lambda_2(t)\Big(\beta s^*(t) - \gamma\Big) \ , \end{cases} \tag{3.13}$$

with transversality conditions

$$\lambda_1(t_f) = 0 \text{ and } \lambda_2(t_f) = 1 \ . \tag{3.14}$$

Further, the optimal control u^* is represented by

$$u^*(t) = \min\left\{ \max\left\{ \frac{s^*(t)\Big(\lambda_2(t) - \lambda_1(t)\Big)}{2B}, 0 \right\}, u_{\max} \right\} \ . \tag{3.15}$$

Proof. Following the PMP [101], and setting $s(t) = s^*(t), i(t) = i^*(t)$, $u(t) = u^*(t)$, the adjoint system (3.13) comes from

$$\begin{cases} \dfrac{d\lambda_1(t)}{dt} &= -\dfrac{\partial H\big(s^*(t), i^*(t), u^*(t), \lambda_1(t), \lambda_2(t)\big)}{\partial s} \\ \dfrac{d\lambda_2(t)}{dt} &= -\dfrac{\partial H\big(s^*(t), i^*(t), u^*(t), \lambda_1(t), \lambda_2(t)\big)}{\partial i} \end{cases}. \tag{3.16}$$

Since $i(t_f)$ and $r(t_f)$ appear as payoff terms in the objective functional (3.6), then $\lambda_1(t_f) = 0$ and $\lambda_2(t_f) = 1$.

Moreover, the optimality condition

$$\frac{\partial H\Big(s^*(t), i^*(t), u^*(t), \lambda_1(t), \lambda_2(t)\Big)}{\partial u} = -2Bu^*(t) + \Big(\lambda_2(t) - \lambda_1(t)\Big)s^*(t) = 0 \tag{3.17}$$

holds almost everywhere of $[0, t_f]$. Thus, by (3.17) and considering the boundedness condition of u on Ω, we obtain (3.15).

□

Based on the above, it is possible to derive the optimality system, which consists in the state system (3.4), the adjoint system (3.13) and transversality conditions (3.14) with the characterization (3.15):

$$\begin{cases} \dfrac{ds^*(t)}{dt} = -\beta s^*(t)i^*(t) - u^*(t)s^*(t) \\ \dfrac{di^*(t)}{dt} = \beta s^*(t)i^*(t) + u^*(t)s^*(t) - \gamma i^*(t) \\ \dfrac{d\lambda_1(t)}{dt} = \lambda_1(t)\Big(\beta i^*(t) + u^*(t)\Big) - \lambda_2(t)\Big(\beta i^*(t) + u^*(t)\Big) \\ \dfrac{d\lambda_2(t)}{dt} = \lambda_1(t)\Big(\beta s^*(t)\Big) - \lambda_2(t)\Big(\beta s^*(t) - \gamma\Big) \\ r^*(t) = 1 - s^*(t) - i^*(t) \\ u^*(t) = \min\left\{ \max\left\{ \dfrac{s^*(t)\Big(\lambda_2(t) - \lambda_1(t)\Big)}{2B}, 0 \right\}, u_{\max} \right\}, \end{cases} \tag{3.18}$$

with initial values $s(0) = 1 - 3 \times 10^{-5}$, $i(0) = 3 \times 10^{-5}$, $r(0) = 0$ and transversality conditions $\lambda_1(t_f) = 0$ and $\lambda_2(t_f) = 1$. The optimal control and states can be obtained by solving (3.18)

using numerical methods for OC problems. It should be noted that by the fact that the state and adjoint functions are bounded and preserve the Lipschitz structure, u^* is unique for t_f sufficiently small (see, e.g., [49]).

3.2.1.2 Numerical Implementation and Methods

For the controlled SIR model previously proposed, this Section studies the dynamics of the state and control variables and its impact on the performance index. To meet this goal, the solutions of the associated OC problem are compared using indirect and direct numerical methods.

For that, three distinct weight factors $B = 10^{-1}$, $B = 10$ and $B = 10^2$ are considered to generate numerical simulations. These factors traduce, respectively, a low, moderate and high investment cost scenario in recruiting susceptible population to act as spreaders on the target population, by marketers. The choice of these values is related to the formulation of investment cost scenarios that best described the marketing reality. At this point, as an indirect numerical approach, *Forward-Backward Sweep method* (FBS) is considered. This method was implemented and solved in MATLAB (the complete code is included in the Appendix Section).

To use direct methods, the problem (3.6) must be discretized with respect to time t. For that, a first order Euler's method was considered with $t_n = nh : n \in \{0, 1, ..., M\}$, and a step size $h = \frac{3}{500}$, where the error in each step is proportional to h^2.

Let $F : [0, 1]^2 \to \mathbb{R}^2$ is a C^1 map. According to the Euler's scheme, for

$$\dot{x} = F\big(x(t)\big), \ \forall t \in [0, 6] \ , \tag{3.19}$$

where

$$x(t) = \begin{bmatrix} s(t) \\ i(t) \end{bmatrix} \text{ and } F\big(x(t)\big) = \begin{bmatrix} -\beta s(t)i(t) - u(t)s(t) \\ \beta s(t)i(t) + u(t)s(t) - \gamma i(t) \end{bmatrix},$$

it comes, for $x(t_n) \approx x_n$, that

$$\frac{x_{n+1} - x_n}{h} = f\big(x_n\big) \Rightarrow x(t + h) = x(t) + hF\big(x(t)\big) \ . \tag{3.20}$$

Therefore, the problem (3.6) is discretized into the following nonlinear constrained optimization problem:

$$
\begin{aligned}
&\max_{\Omega} \quad i(M) + r(M) - \frac{3B}{M}\left(u^2(0) + u^2(M) + 2\sum_{k=1}^{M-1} u^2(k)\right) \\
&\textit{s.t.} \quad \begin{bmatrix} s(k+1) \\ i(k+1) \end{bmatrix} = \begin{bmatrix} s(k) \\ i(k) \end{bmatrix} + h\left\{ \begin{bmatrix} -\beta s(k)i(k) - u(k)s(k) \\ \beta s(k)i(k) + u(k)s(k) - \gamma i(k) \end{bmatrix} \right\},
\end{aligned} \tag{3.21}
$$

where $k \in \{0, 1, ..., M-1\}$, and M is the the last index of the Euler's scheme and corresponds to $t_f = 6$.

KNITRO, IPOPT and LOQO were used as software packages to solve the discretized problem above. The problem (3.21) was modeled in the algebraic modeling language AMPL [34] (the complete code is included in the Appendix Section) and numerically solved in NEOS Server platform [92]. It should be noted that the CPU time can vary depending on the machine used in NEOS server platform. Thus, this variable is not taken into account when analyzing the results. Numerical results are presented in the next Section.

3.2.1.3 Numerical Results and Discussion

This Section reports and compares the computacional results not only for the state and control variables of the state system (3.4), but also for the objective functional (3.6) using different numerical solvers related to both indirect and direct numerical methods described so far.

First of all, Figure 3.4 shows the influence of OC application in Dove's marketing strategy. At this point, it is possible to notice that the fraction of infected people, that is, individuals that pass or recommend Dove's campaign, increase significantly when OC Theory is applied, for different values of B. The levels of infection obtained for these values will be discussed later.

Further, each graphic illustrates the solution obtained for both control and state variables, by using the different numerical solvers. In the computacional measurements, all the direct solvers converged and a local optimal solution was attained for all the solvers and weight factor variations. In Table 3.2, the number of iterations and function evaluations spent by each direct solver for each weight factor B is presented. Table 3.3 reports the obtained values for the proposed objective functional.

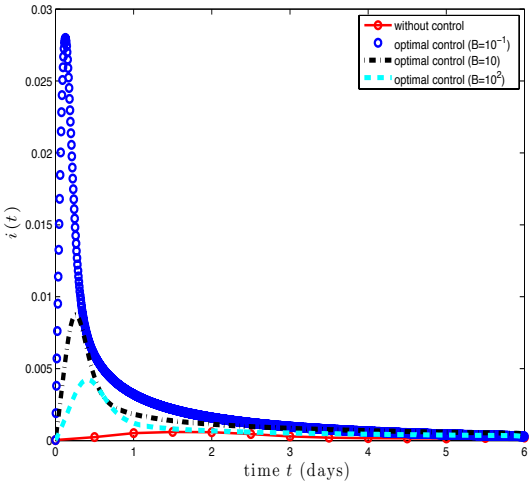


Figure 3.4: The effects of Optimal Control application on $i(t)$.

Table 3.2: Computational performance indicators of direct solvers.

Solver	$B = 10^{-1}$		$B = 10$		$B = 10^2$	
	iterations	evaluations	iterations	evaluations	iterations	evaluations
KNITRO	10	12	11	13	10	12
IPOPT	21	22	26	27	23	24
LOQO	21	21	28	28	26	26

Table 3.3: Objective functional values.

Solver	$B = 10^{-1}$ $\approx J(u(t))$	$B = 10$ $\approx J(u(t))$	$B = 10^2$ $\approx J(u(t))$
FBS	0.8836	0.4643	0.2739
KNITRO	0.8841	0.4643	0.2743
IPOPT	0.8841	0.4643	0.2743
LOQO	0.8689	0.4638	0.2734

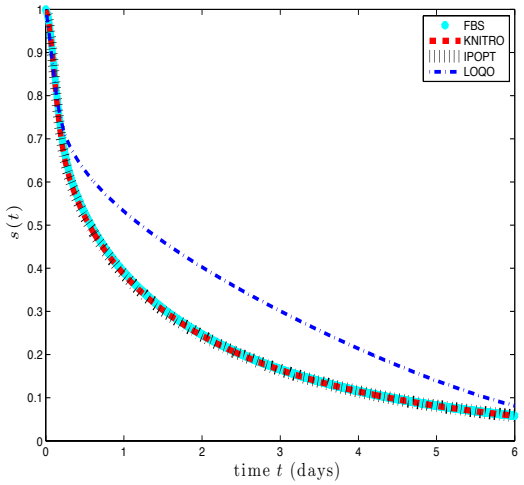
Starting with the weight factor $B = 10^{-1}$ (Fig. 3.5), all the solvers suggest that for a small investment cost in the control u, the fraction of susceptible population decreases over time t,

which means that the control measure adopted is effective on encouraging individuals to spread the marketing message within their social network, inducing, therefore, high levels of infection in a short period of time (see Fig. 3.5(b)). All the methods tested also show that virtually all the target population tends to spread the message, since, for $t = 6$, the fraction of susceptible population is almost null (see Fig. 3.5(a)) and the fraction of individuals tends to fully recover (see Fig. 3.5(c)).

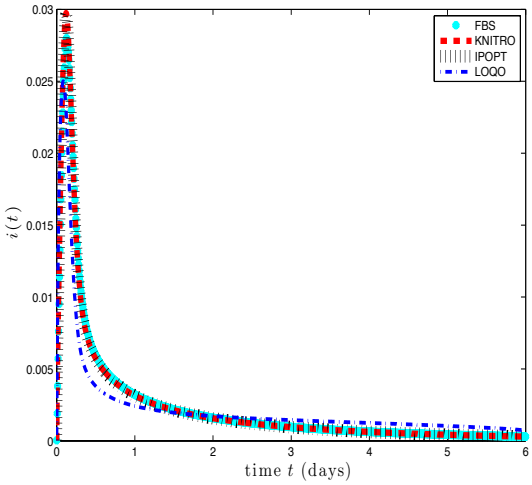
In terms of the application of the control policy (Fig. 3.5(d)), mainly LOQO's curve allows to infer that, since the investment cost is low, the control u should be applied gradually from the beginning of the campaign in order to attain the maximum peak of infection. In fact, the higher levels of cost functional (3.6) are obtained for $B = 10^{-1}$ (see Table 3.3). It should be emphasized that despite of the dynamics of LOQO be different than the obtained with the other solvers for the control signal $u(t)$, its behavior on the state variables as well as on the cost functional is consistent with the dynamics produced by all numerical solvers tested. The differences in the dynamics of the solvers can be explained by the numerical steps that promote global convergence (see, e.g., [100]).

On the other hand, for a reasonable investment cost in implementing u (Fig. 3.6), all the solutions obtained show that the levels of information spreading are lower than the ones obtained for $B = 10^{-1}$ (see, Fig. 3.6(b)). In fact, whereas for $B = 10^{-1}$ the majority of the individuals spreads or has contact with the marketing message, Fig. 3.6(a) reveals that for $B = 10$ about half of the target population is not stimulated to share the intended message, which in turn induces lower levels of recovery (see Fig. 3.6(c)). Moreover, Fig. 3.6(d) suggests that facing a reasonable investment cost in control actions, the control measures should be applied with higher intensity at the beginning of the campaign and not over time as in the case of $B = 10^{-1}$. In terms of the proposed objective functional, for $B = 10$, all the solvers conducted to an approximated solution, which is roughly half than the obtained for $B = 10^{-1}$ (see Table 3.3). This fact is natural, since the adoption of this control weight represents a bigger effort in terms of cost minimization.

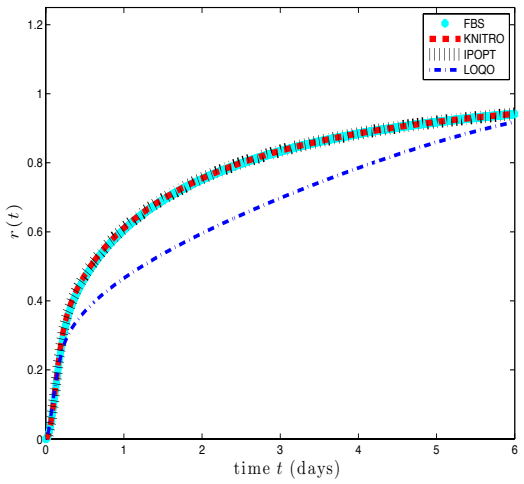
Regarding the factor $B = 10^{2}$, illustrated in Fig. 3.7, the simulations related to the fraction of susceptible and recovered population are omitted in order to avoid redundancy. However, for a high investment cost in u, the recorded levels of infection are the lowest (Fig. 3.7(a)). In terms of the intensity of the application of the control policy, the results are analogous to the obtained for $B = 10$, but the values are smaller (see Figs. 3.6(d) and 3.7(b)).



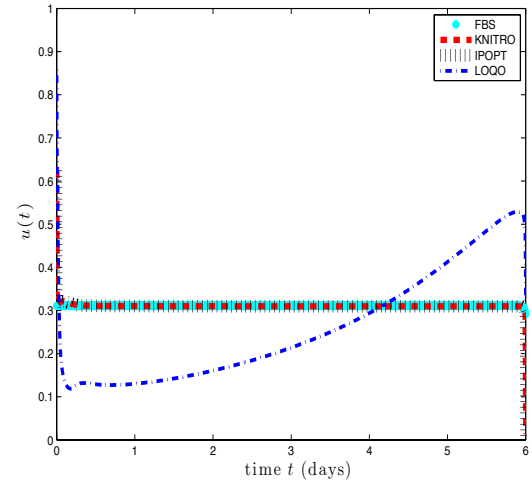
(a) Fraction of susceptible population



(b) Fraction of infected population

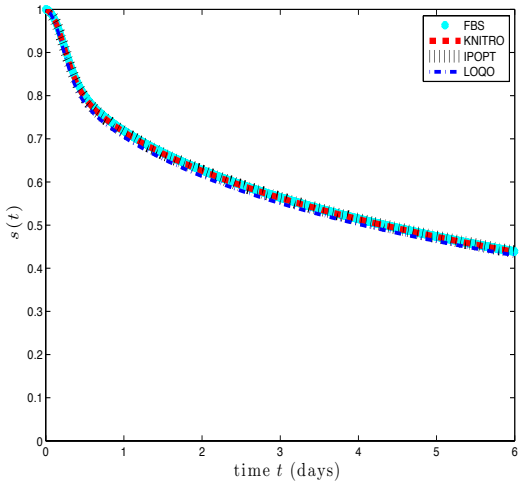


(c) Fraction of recovered population

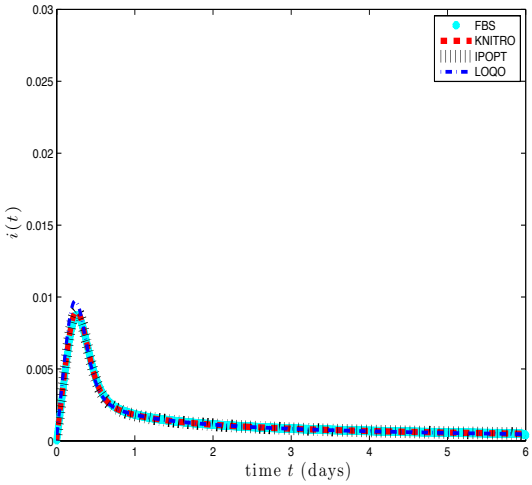


(d) Optimal control

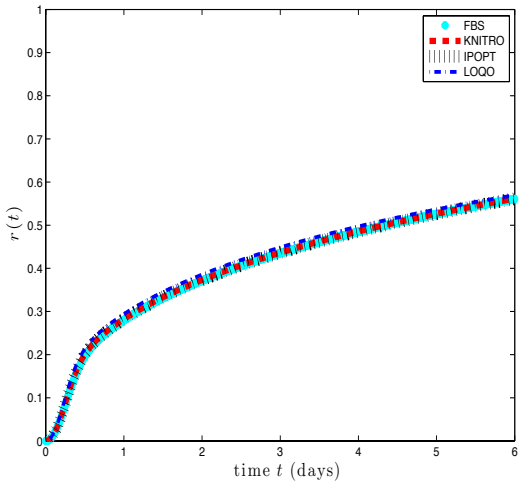
Figure 3.5: Model dynamics for FBS, KNITRO, IPOPT and LOQO under a low investment cost in u. Parameter values: $B = 10^{-1}$ and $u_{\max} = 1$.



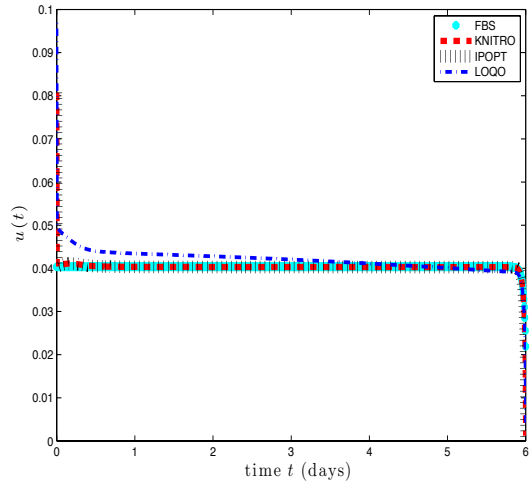
(a) Fraction of susceptible population



(b) Fraction of infected population

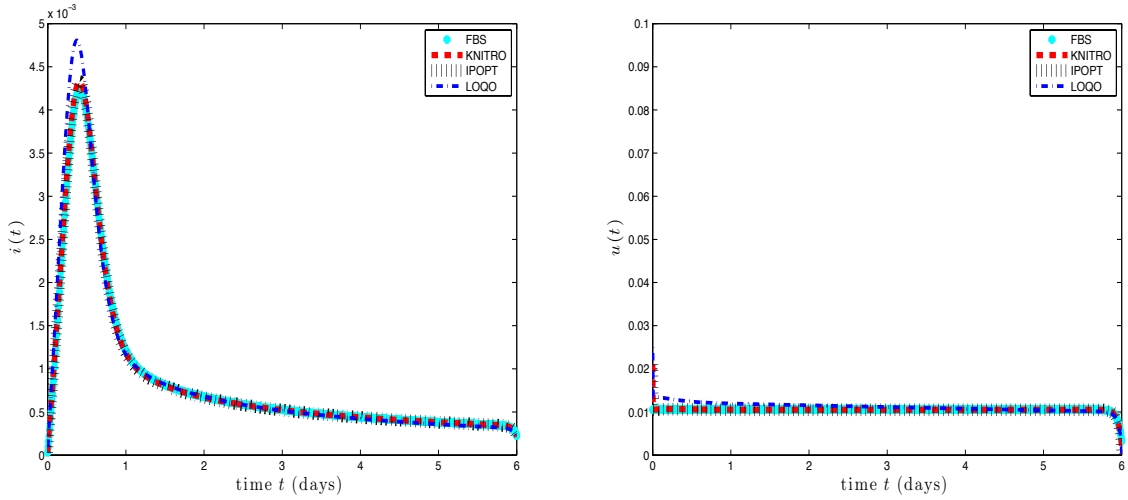


(c) Fraction of recovered population



(d) Optimal control

Figure 3.6: Model dynamics for FBS, KNITRO, IPOPT and LOQO under a reasonable investment cost in u. Parameter values: $B = 10$ and $u_{\max} = 0.1$.



(a) Fraction of infected population

(b) Optimal control

Figure 3.7: Model dynamics for FBS, KNITRO, IPOPT and LOQO under a high investment cost in u. Parameter values: $B = 10^2$ and $u_{\max} = 0.025$.

Transversally, whatever the factor B considered, it is possible to observe that different numerical solvers lead to an analogous dynamics, being exactly the same for KNITRO and IPOPT (red and black curves are always superimposed). However, KNITRO leads to a smaller number of iterations and function evaluations than IPOPT (see Table 3.2).

Our results support the conclusion that both indirect and direct methods lead to analogous behaviors in terms of state variables, confirming the reliability of the obtained results. However, in what concerns to the control variable u, the dynamics vary inasmuch as the investment costs in control policies have a direct influence on the objective functional. At this point, numerical simulations showed that when the investment cost related to the adoption of u decreases, the magnitude of u increases. In contrast when the investment cost associated to the implementation of u increases, the magnitude of u decreases.

Having analyzed the situation of attracting susceptible individuals to spread the message, the next Section studies the case in which the infected individuals are also stimulated to continue to spread it.

3.2.2 SIR Model with Two Controls

In Section 3.2.1, an OC strategy to encourage susceptible individuals to spread the marketing message was tested. However, it is also important to fostering infected individuals to continue to spread the marketing message in order to maintain high levels of information spreading. Motivated by this purpose, another control function, $u_2(t),\ \forall t \in [0,6]$, is added to the system (3.4), resulting in the following system of ordinary differential equations:

$$\begin{cases} \dfrac{ds(t)}{dt} = -\big(\beta + u_2(t)\big)s(t)i(t) - u_1(t)s(t) \\ \dfrac{di(t)}{dt} = \big(\beta + u_2(t)\big)s(t)i(t) + u_1(t)s(t) - \gamma i(t) \\ r(t) = 1 - s(t) - i(t)\ , \end{cases} \qquad \begin{cases} s(0) = 1 - 3 \times 10^{-5} \\ i(0) = 3 \times 10^{-5} \\ r(0) = 0\ . \end{cases} \tag{3.22}$$

This system was adapted from [51]. The new control function $u_2(t)$ expresses the fostering of infected individuals to continue to spread the marketing message into the social network (e.g., through vouchers, rewards, monetary stimuli) [51]. Moreover, the set of admissible control functions becomes

$$\Omega = \left\{ (u_1(\cdot), u_2(\cdot)) \in (L^2(0,t_f))^2 \mid 0 \leqslant u_1(t) \leqslant u_{1\max} \ \wedge\ 0 \leqslant u_2(t) \leqslant u_{2\max},\ \forall t \in [0,t_f] \right\}.$$

Similarly to the OC problem formulated in the previous Section, the objective functional is now defined as

$$\max_{\Omega} J(u_1(\cdot), u_2(\cdot)) = i(t_f) + r(t_f) + \int_0^{t_f} -\Big[Bu_1^2(t) + Cu_2^2(t)\Big]\ dt\ , \tag{3.23}$$

subject to (3.22), where the non-negative constants B and C represent the weights of the investment costs associated to the control signals u_1 and u_2, respectively. The existence of an optimal solution can also be derived, recalling [33] for existence results of optimal solutions.

Let $\lambda_i(t) : i = 1,2.$ be the adjoint functions, $s^*(t), i^*(t)$ the optimal state variables and $u_i^*(t) : i = 1,2.$ the optimal controls. For the sake of simplicity, the necessary conditions, for the proposed OC problem, that the state and control variables must satisfy are derived as follows.

Hamiltonian:

$$\begin{aligned} H(s(t), i(t), u_1(t), u_2(t), \lambda_1(t), \lambda_2(t)) = &-(Bu_1^2(t) + Cu_2^2(t)) \\ &+ \lambda_1(t)\left[-\big(\beta + u_2(t)\big)s(t)i(t) - u_1(t)s(t)\right] \\ &+ \lambda_2(t)\left[\big(\beta + u_2(t)\big)s(t)i(t) + u_1(t)s(t) - \gamma i(t)\right] . \end{aligned}$$

Adjoint system:

$$\begin{cases} \dfrac{d\lambda_1}{dt} = \lambda_1(t)\left[\big(\beta + u_2^*(t)\big)i^*(t) + u_1^*(t)\right] - \lambda_2(t)\left[\big(\beta + u_2^*(t)\big)i^*(t) + u_1^*(t)\right] \\ \dfrac{d\lambda_2}{dt} = \lambda_1(t)\left[\big(\beta + u_2^*(t)\big)s^*(t)\right] - \lambda_2(t)\left[\big(\beta + u_2^*(t)\big)s^*(t) - \gamma\right] , \end{cases} \tag{3.24}$$

with transversality conditions $\lambda_1(t_f) = 0$ and $\lambda_2(t_f) = 1$.

In addition, using the Hamiltonian maximizing condition at the interior points, the optimal controls $u_1^*(t)$ and $u_2^*(t)$ are characterized by:

$$u_1^*(t) = \min\left\{ \max\left\{ \frac{s^*(t)\Big(\lambda_2(t) - \lambda_1(t)\Big)}{2B}, 0 \right\}, u_{1\max} \right\} , \tag{3.25}$$

and

$$u_2^*(t) = \min\left\{ \max\left\{ \frac{s^*(t)i^*(t)\Big(\lambda_2(t) - \lambda_1(t)\Big)}{2C}, 0 \right\}, u_{2\max} \right\} . \tag{3.26}$$

The optimality system consists in the state system (3.22), the adjoint system (3.24) and transversality conditions with the characterizations (3.25) and (3.26).

Theorem 3.2.3 (Uniqueness of Optimality System)**.** *Given the initial value problem (3.22) and the objective functional (3.23), the optimal solution* $(s^*(t), i^*(t))$*, with associated optimal control functions* $u_1^*(t)$*,* $u_2^*(t)$*, and the adjoint functions* $\lambda_1(t)$*,* $\lambda_2(t)$ *are unique for* t_f *sufficiently small.*

Proof. Let $(s, i, \lambda_1, \lambda_2)$ and $(\overline{s}, \overline{i}, \overline{\lambda_1}, \overline{\lambda_2})$ be two solutions of the optimality system. Consider $s = e^{\phi t}a_1$, $i = e^{\phi t}a_2$, $\lambda_1 = e^{-\phi t}b_1$, $\lambda_2 = e^{-\phi t}b_2$. Analogously, consider $\overline{s} = e^{\phi t}\overline{a_1}$, $\overline{i} = e^{\phi t}\overline{a_2}$, $\overline{\lambda_1} = e^{-\phi t}\overline{b_1}$, $\overline{\lambda_2} = e^{-\phi t}\overline{b_2}$, where ϕ is a constant.

Let

$$u_1(t) = \min\left\{ \max\left\{ \frac{a_1(b_2 - b_1)}{2B}, 0 \right\}, u_{1\max} \right\},$$

$$u_2(t) = \min\left\{ \max\left\{ \frac{e^{\phi t} a_1 a_2 (b_2 - b_1)}{2C}, 0 \right\}, u_{2\max} \right\} ,$$

and

$$\overline{u_1}(t) = \min\left\{ \max\left\{ \frac{\overline{a_1}(\overline{b_2} - \overline{b_1})}{2B}, 0 \right\}, u_{1\max} \right\} ,$$

$$\overline{u_2}(t) = \min\left\{ \max\left\{ \frac{e^{\phi t}\overline{a_1 a_2}(\overline{b_2} - \overline{b_1})}{2C}, 0 \right\}, u_{2\max} \right\} .$$

Henceforth, for the sake of simplicity, it will be considered u_1, $\overline{u_1}$, u_2, $\overline{u_2}$ instead of their specific characterizations. Using the assumptions above, the first state equation of the optimality system becomes

$$e^{\phi t}\frac{da_1}{dt} + \phi e^{\phi t} a_1 = -(\beta + u_2) e^{2\phi t} a_1 a_2 - u_1 e^{\phi t} a_1 \ . \tag{3.27}$$

Similarly, for $\lambda_1 = e^{-\phi t} b_1$, yields

$$e^{-\phi t}\frac{db_1}{dt} - \phi e^{-\phi t} b_1 = e^{-\phi t}(b_1 - b_2)\Big[(\beta + u_2) e^{\phi t} a_2 + u_1\Big] \ . \tag{3.28}$$

Now, the equations for s and $\overline{s}$, i and $\overline{i}$, λ_1 and $\overline{\lambda_1}$, λ_2 and $\overline{\lambda_2}$ are subtracted. Then, each of these equations is multiplied by an appropriate difference of functions and integrated from 0 to t_f. Hence, the four integral equations resultant from these operations are listed below.

$$
\begin{aligned}
&\frac{1}{2}\big(a_1(t_f)-\overline{a_1}(t_f)\big)^2+\phi\int_0^{t_f}(a_1-\overline{a_1})^2\ dt\\
&=-\int_0^{t_f}e^{\phi t}\Big[(\beta+u_2)a_1a_2-(\beta+\overline{u_2})\overline{a_1a_2}\Big](a_1-\overline{a_1})\ dt-\int_0^{t_f}(u_1a_1-\overline{u_1a_1})(a_1-\overline{a_1})\ dt\\
&=-\int_0^{t_f}e^{\phi t}\Big[\beta\Big((a_1-\overline{a_1})a_2+\overline{a_1}(a_2-\overline{a_2})\Big)+\overline{u_2}(a_1-\overline{a_1})a_2+\overline{u_2a_1}(a_2-\overline{a_2})\Big](a_1-\overline{a_1})\ dt\\
&\quad-\int_0^{t_f}e^{\phi t}\Big[(u_2-\overline{u_2})a_1a_2\Big](a_1-\overline{a_1})\ dt-\int_0^{t_f}\Big[(u_1-\overline{u_1})a_1+\overline{u_1}(a_1-\overline{a_1})\Big](a_1-\overline{a_1})\ dt\ ,
\end{aligned}
\tag{3.29}
$$

$$
\begin{aligned}
&\frac{1}{2}\big(a_2(t_f)-\overline{a_2}(t_f)\big)^2+\phi\int_0^{t_f}(a_2-\overline{a_2})^2\ dt\\
&=\int_0^{t_f}e^{\phi t}\Big[(\beta+u_2)a_1a_2-(\beta+\overline{u_2})\overline{a_1a_2}\Big](a_2-\overline{a_2})\ dt+\int_0^{t_f}(u_1a_1-\overline{u_1a_1})(a_2-\overline{a_2})\ dt\\
&\quad-\gamma\int_0^{t_f}(a_2-\overline{a_2})^2\ dt\\
&=\int_0^{t_f}e^{\phi t}\Big[\beta\Big((a_1-\overline{a_1})a_2+\overline{a_1}(a_2-\overline{a_2})\Big)+\overline{u_2}(a_1-\overline{a_1})a_2+\overline{u_2a_1}(a_2-\overline{a_2})\Big](a_2-\overline{a_2})\ dt\\
&\quad+\int_0^{t_f}e^{\phi t}\Big[(u_2-\overline{u_2})a_1a_2\Big](a_2-\overline{a_2})\ dt+\int_0^{t_f}\Big[(u_1-\overline{u_1})a_1+\overline{u_1}(a_1-\overline{a_1})\Big](a_2-\overline{a_2})\ dt\\
&\quad-\gamma\int_0^{t_f}(a_2-\overline{a_2})^2\ dt\ ,
\end{aligned}
\tag{3.30}
$$

$$
\begin{aligned}
&\frac{1}{2}\big(b_1(0)-\overline{b_1}(0)\big)^2+\phi\int_0^{t_f}(b_1-\overline{b_1})^2\ dt\\
&=-\int_0^{t_f}\Big[(b_1-b_2)\Big[(\beta+u_2)e^{\phi t}a_2+u_1\Big]-(\overline{b_1}-\overline{b_2})\Big[(\beta+\overline{u_2})e^{\phi t}\overline{a_2}+\overline{u_1}\Big]\Big](b_1-\overline{b_1})\ dt\\
&=-\int_0^{t_f}e^{\phi t}\Big[\beta\Big(\overline{a_2}\big[(b_1-\overline{b_1})-(b_2-\overline{b_2})\big]+(a_2-\overline{a_2})(b_1-b_2)\Big)+\overline{u_2a_2}\big[(b_1-\overline{b_1})-(b_2-\overline{b_2})\big]\\
&\quad+\overline{u_2}(b_1-b_2)(a_2-\overline{a_2})\Big](b_1-\overline{b_1})\ dt-\int_0^{t_f}e^{\phi t}(b_1-b_2)(u_2-\overline{u_2})a_2(b_1-\overline{b_1})\ dt\\
&\quad-\int_0^{t_f}\Big[\overline{u_1}\Big((b_1-\overline{b_1})-(b_2-\overline{b_2})\Big)+(u_1-\overline{u_1})(b_1-b_2)\Big](b_1-\overline{b_1})\ dt\ ,
\end{aligned}
\tag{3.31}
$$

$$
\begin{aligned}
&\frac{1}{2}\big(b_2(0)-\overline{b_2}(0)\big)^2+\phi\int_0^{t_f}(b_2-\overline{b_2})^2\ dt\\
&=-\int_0^{t_f}e^{\phi t}\bigg[(b_1-b_2)(\beta+u_2)a_1-(\overline{b_1}-\overline{b_2})(\beta+\overline{u_2})\overline{a_1}\bigg](b_2-\overline{b_2})\ dt-\gamma\int_0^{t_f}(b_2-\overline{b_2})^2\ dt\\
&=-\int_0^{t_f}e^{\phi t}\bigg[\beta\Big[\overline{a_1}\Big((b_1-\overline{b_1})-(b_2-\overline{b_2})\Big)+(a_1-\overline{a_1})(b_1-b_2)\Big]+\overline{u_2a_1}\Big((b_1-\overline{b_1})-(b_2-\overline{b_2})\Big)\\
&\quad+(b_1-b_2)(a_1-\overline{a_1})\overline{u_2}\bigg](b_2-\overline{b_2})\ dt-\int_0^{t_f}e^{\phi t}(b_1-b_2)(u_2-\overline{u_2})a_1(b_2-\overline{b_2})\ dt\\
&\quad-\gamma\int_0^{t_f}(b_2-\overline{b_2})^2\ dt\ .
\end{aligned}
\tag{3.32}
$$

Next, in these four integral equations, some terms are estimated. Firstly, notice that

$$
\int_0^{t_f}(u_1-\overline{u_1})^2\ dt\leqslant\frac{C_1}{4B^2}\int_0^{t_f}(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ ,
\tag{3.33}
$$

and

$$
\int_0^{t_f}(u_2-\overline{u_2})^2\ dt\leqslant\frac{C_2e^{2\phi t_f}}{4C^2}\int_0^{t_f}(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ .
\tag{3.34}
$$

Therefore, using these estimates, the following inequalities are obtained:

$$
\begin{aligned}
&\frac{1}{2}\big(a_1(t_f)-\overline{a_1}(t_f)\big)^2+\phi\int_0^{t_f}(a_1-\overline{a_1})^2\ dt\\
&\leqslant D_1\int_0^{t_f}(a_1-\overline{a_1})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt+E_1e^{\phi t_f}\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2\ dt\\
&\quad+F_1e^{3\phi t_f}\int_0^{t_f}(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ ,
\end{aligned}
\tag{3.35}
$$

$$
\begin{aligned}
&\frac{1}{2}\big(a_2(t_f)-\overline{a_2}(t_f)\big)^2+\phi\int_0^{t_f}(a_2-\overline{a_2})^2\ dt\\
&\leqslant D_2\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt+E_2e^{\phi t_f}\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2\ dt\\
&\quad+F_2e^{3\phi t_f}\int_0^{t_f}(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ ,
\end{aligned}
\tag{3.36}
$$

$$
\begin{aligned}
&\frac{1}{2}\big(b_1(0)-\overline{b_1}(0)\big)^2+\phi\int_0^{t_f}(b_1-\overline{b_1})^2\ dt\\
&\leqslant D_3\int_0^{t_f}(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt+E_3e^{\phi t_f}\int_0^{t_f}(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\\
&\quad+F_3e^{3\phi t_f}\int_0^{t_f}(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ ,
\end{aligned}
\tag{3.37}
$$

$$
\begin{aligned}
&\frac{1}{2}\big(b_2(0)-\overline{b_2}(0)\big)^2+\phi\int_0^{t_f}(b_2-\overline{b_2})^2\ dt\\
&\leqslant D_4\int_0^{t_f}(b_2-\overline{b_2})^2\ dt+E_4e^{\phi t_f}\int_0^{t_f}(a_1-\overline{a_1})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\\
&\quad+F_4e^{3\phi t_f}\int_0^{t_f}(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ .
\end{aligned}
\tag{3.38}
$$

By adding all of these estimations, and noting that $e^{\phi t_f}\leqslant e^{3\phi t_f}$, it follows that

$$
\begin{aligned}
&\frac{1}{2}\Big[\big(a_1(t_f)-\overline{a_1}(t_f)\big)^2+\big(a_2(t_f)-\overline{a_2}(t_f)\big)^2+\big(b_1(0)-\overline{b_1}(0)\big)^2+\big(b_2(0)-\overline{b_2}(0)\big)^2\Big]\\
&\quad+\phi\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\\
&\leqslant\tilde{D}\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\\
&\quad+\tilde{F}e^{3\phi t_f}\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ .
\end{aligned}
\tag{3.39}
$$

Rearranging the terms,

$$
\begin{aligned}
&\frac{1}{2}\Big[\big(a_1(t_f)-\overline{a_1}(t_f)\big)^2+\big(a_2(t_f)-\overline{a_2}(t_f)\big)^2+\big(b_1(0)-\overline{b_1}(0)\big)^2+\big(b_2(0)-\overline{b_2}(0)\big)^2\Big]\\
&\leqslant(\tilde{D}+\tilde{F}e^{3\phi t_f}-\phi)\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\ dt\ ,
\end{aligned}
\tag{3.40}
$$

where $\tilde{D}$ and $\tilde{F}$ depend on the coefficients and the bounds of a_1, a_2, b_1, b_2.

By choosing $\phi>\tilde{D}+\tilde{F}$ and $t_f<\dfrac{1}{3\phi}\log\left(\dfrac{\phi-\tilde{D}}{\tilde{F}}\right)$ yields

$$
\begin{aligned}
0 &\leqslant \frac{1}{2}\Big[\big(a_1(t_f)-\overline{a_1}(t_f)\big)^2+\big(a_2(t_f)-\overline{a_2}(t_f)\big)^2+\big(b_1(0)-\overline{b_1}(0)\big)^2+\big(b_2(0)-\overline{b_2}(0)\big)^2\Big] \\
&\leqslant (\tilde{D}+\tilde{F}e^{3\phi t_f}-\phi)\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\;dt \qquad (3.41)\\
&\leqslant 0\ ,
\end{aligned}
$$

which implies that

$$
(\tilde{D}+\tilde{F}e^{3\phi t_f}-\phi)\int_0^{t_f}(a_1-\overline{a_1})^2+(a_2-\overline{a_2})^2+(b_1-\overline{b_1})^2+(b_2-\overline{b_2})^2\;dt=0\ . \qquad (3.42)
$$

Thus, knowing that $(\tilde{D}+\tilde{F}e^{3\phi t_f}-\phi)<0$, we have $a_1=\overline{a_1}, a_2=\overline{a_2}, b_1=\overline{b_1}, b_2=\overline{b_2}$ and $(s,i,\lambda_1,\lambda_2)=(\overline{s},\overline{i},\overline{\lambda_1},\overline{\lambda_2})$, which concludes the proof. □

3.2.2.1 Numerical Results and Discussion

With the inclusion of the new control function u_2, the main goal is to provide insights related to when and which control strategies should be applied to maximize the spreading of information and minimize costs. Using MATLAB software to solve the optimality system, numerical results are obtained by using FBS method.

In what follows, two approaches are considered. Firstly, simulations of the control weights are performed using both optimal controls u_1^* and u_2^*, in order to assess which control weights induce a higher cost functional. Secondly, these pairs are used to model and compare scenarios related to high, low and equal investments costs in publicity actions.

- **Control Weights Simulation using u_1^* and u_2^***

Firstly, using both optimal controls u_1^* and u_2^*, the variation of the control weights on both infected individuals $(i(t))$ and control signals is studied over $[0,6]$. The simulations of the control weights are performed under three strategies described in Table 3.4.

The choice of the values for B and C was based on several experimental simulations that aimed at obtaining the results that best described the reality of each strategy. For the sake of consistency, since at the beginning of a viral epidemic the main goal is to attract susceptible

Table 3.4: Marketing strategies using Optimal Control.

# Strategy	Marketing context
Strategy 1: $B = 1$ and $C \in \{x \mid x = 10^{-i} : i = 1, ..., 3\}$	Low investment costs in encouraging infected individuals to continue to spread the marketing message (e.g., exploiting social networks such as Facebook and Twitter).
Strategy 2: $B = 1$ and $C \in \{x \mid x = 10^{i} : i = 1, ..., 3\}$	High investment costs in fostering infected individuals to continue to spread the marketing message (e.g., monetary rewards and stimuli, expensive promotional gifts and international trips).
Strategy 3: $B \in \{x \mid x = 10^{i} : i = 0, ..., 3\}$ and $C = 1$	Increasing the investment costs in recruiting susceptible individuals to act as spreaders.

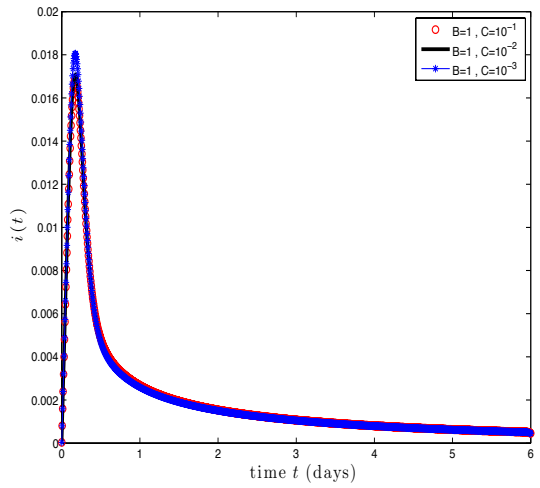
individuals, it is conceivable to start with an intermediate investment cost in detriment of a lower one. For this reason, the above strategies do not consider $B < 1$. The simulations regarding the three strategies are illustrated in Fig. 3.8.

Figure 3.8(a) shows that around the first day, the fraction of infected individuals is higher when the investment costs in fostering people who had already been in contact with the marketing message are low, namely for $B = 1$ and $C = 10^{-3}$.

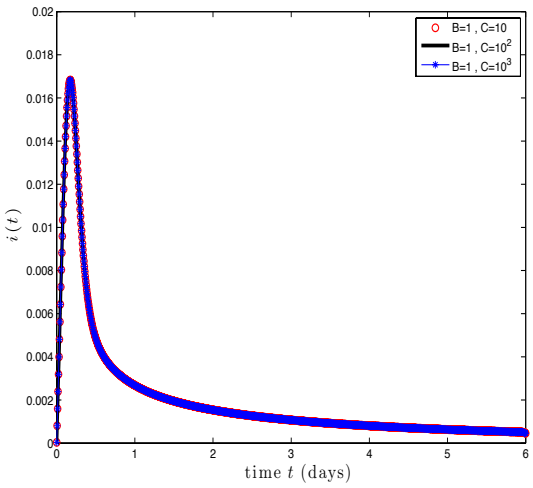
Regarding the Strategy 2 (Fig. 3.8(b)), by fixing $B = 1$, as the control weight C increases, infection levels do not vary. Thus, it is plausible to conclude that higher costs in implement further publicity strategies to fostering infected individuals do not result in higher levels of spreading.

In what concerns Strategy 3, Fig. 3.8(c) reports that as the control weight B increases, the fraction of people who have contact with the intended message is diminishing all the time and the maximum peak of infection is attained increasingly late.

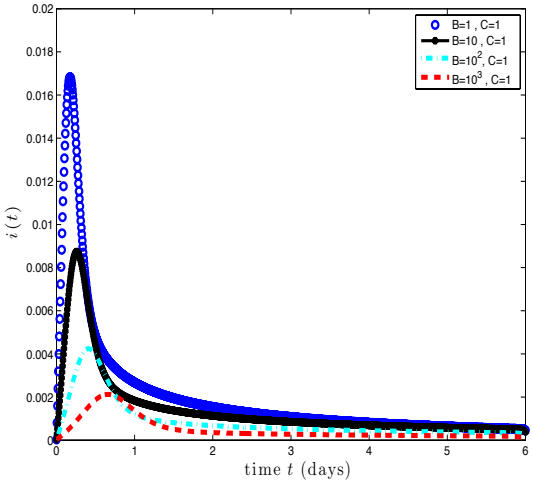
Transversally, whatever the strategy considered, the smaller the investment costs neither in fostering infected individuals to continue to spread the message, nor in recruit susceptible individuals to act as spreaders, the greater the levels of information spreading.



(a) Fraction of infected individuals for Strategy 1



(b) Fraction of infected individuals for Strategy 2



(c) Fraction of infected individuals for Strategy 3

Figure 3.8: Variation of the control weights on $i(t)$ for the different marketing strategies.

Overall, Table 3.5 presents the values for objective functional (3.23), by varying the control weights for each strategy. Here, the pairs of control weights that induce a higher cost functional are highlighted in *bold*, for each strategy. The choice of the highlighted pairs was based on the aim of portray low, high and equal investment cost scenarios, respectively.

Table 3.5: Summary of optimal cost functionals varying control weights (B, C).

Strategy 1		Strategy 2		Strategy 3	
(B, C)	$\approx J(u_1^*, u_2^*)$	(B, C)	$\approx J(u_1^*, u_2^*)$	(B, C)	$\approx J(u_1^*, u_2^*)$
$\mathbf{(1, 10^{-3})}$	$\mathbf{0.699766}$	$\mathbf{(1, 10)}$	$\mathbf{0.698692}$	$\mathbf{(1, 1)}$	$\mathbf{0.698693}$
$(1, 10^{-2})$	0.698815	$(1, 10^2)$	0.698691	$(10, 1)$	0.463743
$(1, 10^{-1})$	0.698704	$(1, 10^3)$	0.698691	$(10^2, 1)$	0.273901
–	–	–	–	$(10^3, 1)$	0.159022

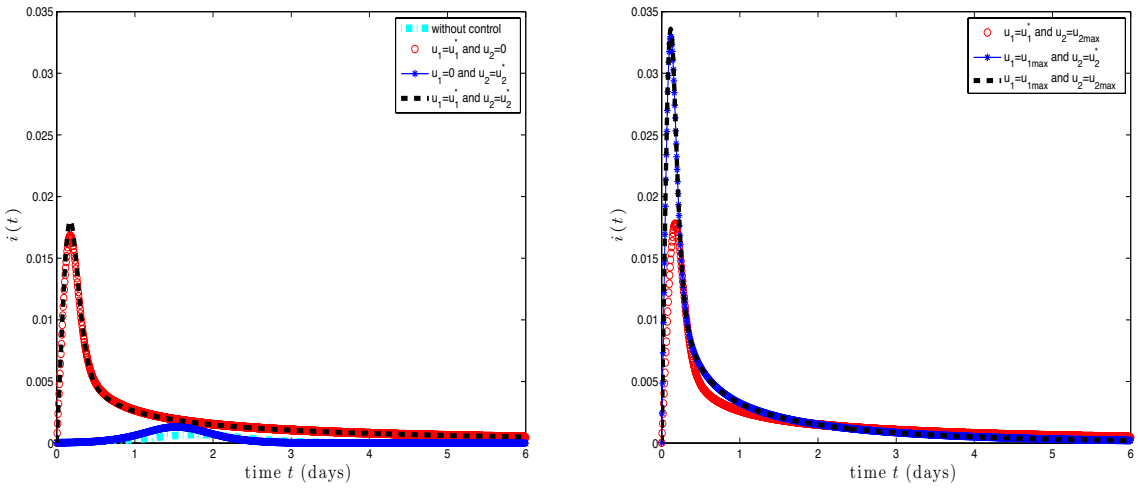
- **Control Experiments for Different Investment Cost Scenarios**

The scenarios based on the control weights highlighted in Table 3.5 are now simulated, varying the control functions $u_1(t)$ and $u_2(t)$, $\forall t \in [0, 6]$. If the company has monetary funds to invest in extra publicity actions either on susceptible or already infected individuals, upper control policies are tested. The upper control is here defined as the maximum control value for which the cost functional (3.23) is positive, and it represents the maximum application of a control measure. Henceforth, let $u_{1\max}$ and $u_{2\max}$ be the upper controls related to the control functions u_1 and u_2, respectively.

1) Low Investment Cost Scenario using Control Weights $(B, C) = (1, 10^{-3})$

In Fig. 3.9(a) it is possible to note that the fraction of infected individuals is significantly higher whenever optimal control is applied. In this regard, the implementation effect of the controls u_1 and u_2 is assessed over $[0, 6]$, see Fig. 3.10.

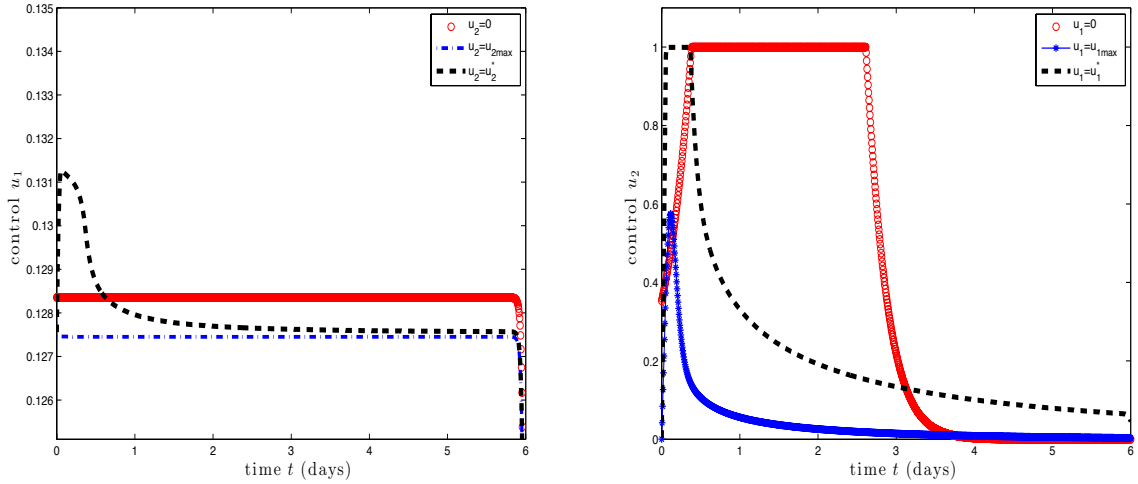
Observing Figs. 3.10(a) and 3.10(b) at the launch of the advertisement, the best policy is to implement the optimal control combination (u_1^*,u_2^*), in order to rapidly attain the maximum peak of infection at the end of the first day of the Dove's campaign (see Fig. 3.9(a)). Then, during the next 2 days, the control u_2 is at the upper bound (see, Fig. 3.10(b)), suggesting in this time window that the best policy is to apply $(0, u_2^*)$ in such a way as to encourage infected individuals to continue to spread the message. Hence, at the end of $t = 2$, when the levels of recovery begin to increase, the pair $(u_1^*, 0)$ should be implemented in order to minimize the rate of recovered individuals by attracting new susceptible individuals to diffuse the intended message,



(a) Optimal controls

(b) Mixing upper and optimal controls

Figure 3.9: Infected individuals for $B = 1$, $C = 10^{-3}$, $u_{1\max} = 0.4$, $u_{2\max} = 1$.



(a) Optimal control u_1 varying u_2

(b) Optimal control u_2 varying u_1

Figure 3.10: Optimal controls u_1 and u_2 for $B = 1$, $C = 10^{-3}$, $u_{1\max} = 0.4$, $u_{2\max} = 1$.

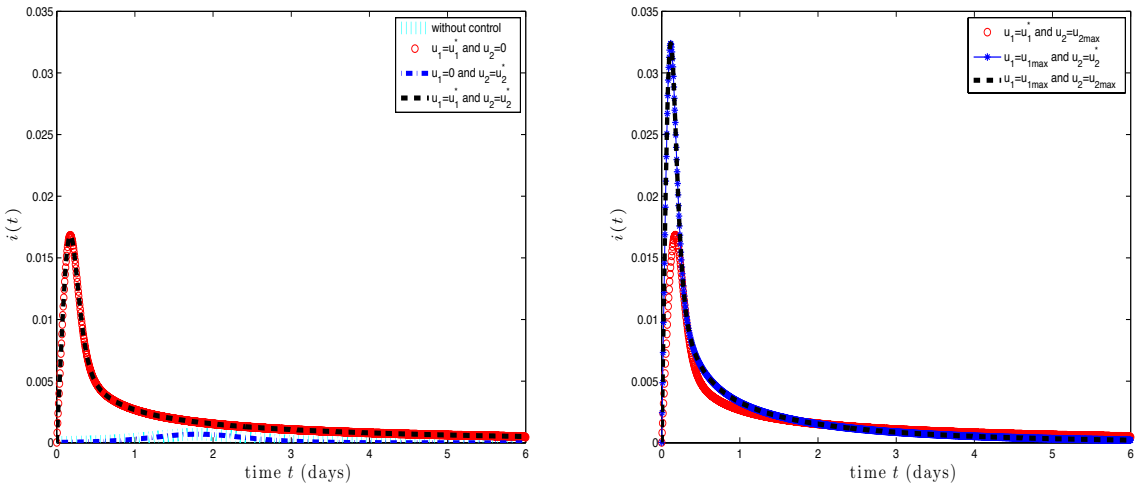
see Fig. 3.10(a). In terms of cost functional, despite of infection levels attain a maximum level using $(u_{1\max}, u_{2\max})$, $J(u_{1\max}, u_{2\max}) \approx 0.002095$, which means that a double investment in both control policies compromises the objective of minimize costs.

In contrast, based on Table 3.5, $J(u_1^*, u_2^*) \approx 0.699766$, that is, the simultaneous use of the

control functions u_1^* and u_2^* fulfills the proposed trade-off, notwithstanding by applying $(u_1^*, 0)$ an excellent marketing performance is obtained with less costs ($J(u_1^*, 0) \approx 0.698678$). This is underpinned by the fact that, for the control intervention $(u_1^*, u_{2\max})$, the cost functional (3.23) is almost the same as the obtained when the optimal controls u_1^* and u_2^* are applied ($J(u_1^*, u_{2\max}) \approx 0.695835$). These arguments show the importance of the control u_1 to attain the maximum peak of infection at the beginning of the campaign.

2) High Investment Cost Scenario using Control Weights $(B, C) = (1, 10)$

In this scenario, analogously to the previous one, the fraction of infected individuals at the beginning of the campaign is higher with the implementation of control policies than without it, see Fig. 3.11(a).

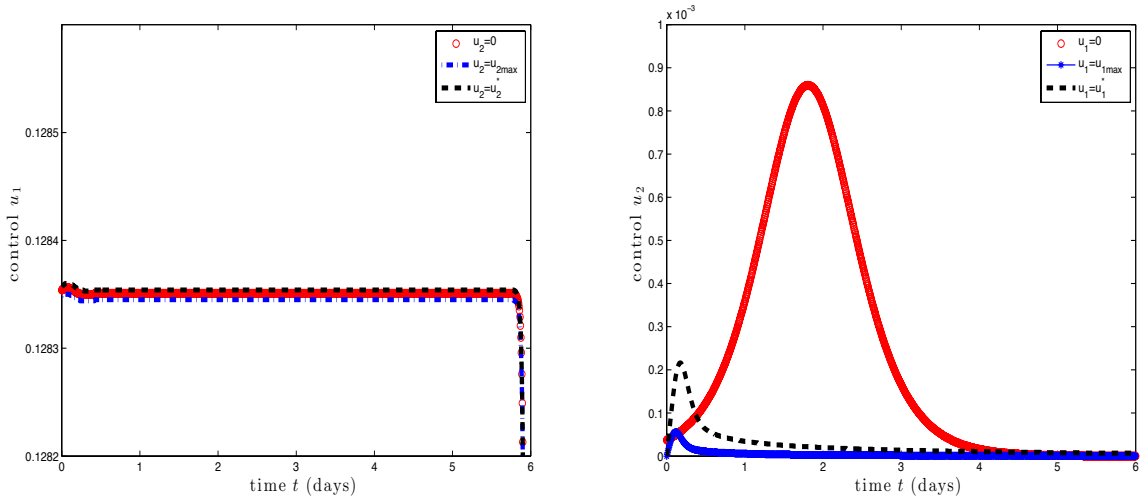


(a) Optimal controls

(b) Mixing upper and optimal controls

Figure 3.11: Infected individuals for $B = 1$, $C = 10$, $u_{1\max} = 0.4$, $u_{2\max} = 0.01$.

However, Fig. 3.12(b) illustrates that the magnitude of the control u_2^* is significantly lower when compared to the magnitude of u_1^* (Fig. 3.12(a)), due to the high investment costs imposed. By linking this finding with Fig. 3.11(a), it is possible to infer not only that the optimal control measure u_2^* applied by itself has no direct influence on the information dissemination (*dark blue curve*), but also that by applying $(u_1^*, 0)$, the levels of information diffusion are satisfactory.



(a) Optimal control u_1 varying u_2

(b) Optimal control u_2 varying u_1

Figure 3.12: Optimal controls u_1 and u_2 for $B = 1$, $C = 10$, $u_{1\,\max} = 0.4$, $u_{2\,\max} = 0.01$.

When upper control measures are applied, the infection levels increase substantially, see Fig. 3.11(b). Nonetheless, similarly to the previously scenario, the use of $(u_{1\,\max}, u_{2\,\max})$ results in a residual cost functional $(J(u_{1\,\max}, u_{2\,\max}) \approx 0.001591)$.

At this point, upper control policies for u_2 are disadvantageous, inasmuch as the adoption $(u_1^*, u_{2\,\max})$ leads to the same infection levels as the obtained by using both optimal controls, see Figs. 3.11(a) and 3.11(b). Furthermore, $J(u_1^*, 0) \approx 0.698678$, which means that the application of the control u_1^* is a sufficient condition to achieve the proposed trade-off.

Linking with the previous scenario, it also can be notice that the control u_1 has much more influence on the information spreading than u_2, as one would have expected.

In order to avoid redundancy, the control simulations using $B = C = 1$ are omitted. However, in this case, the importance of the control intervention u_1 overlaps u_2.

3.3 Discussion

This Chapter introduced VM as an epidemiological dynamical system. In addition, based one of the most viral video advertisements of all time - Dove Real Beauty Sketches, OC Theory was used with the aim of maximize information spreading with a low cost associated to it. In this regard, two controlled systems of ODEs were formulated and several numerical solvers were used

to ensure the reliability of the results. Moreover, in order to improve the timing of the information diffusion, optimal time windows to apply the control policies u_1 and u_2 were recommended, for each scenario.

In terms of guidelines to marketing professionals, our results showed that when the investment cost related to the adoption of u decreases, the magnitude of u increases. In contrast when the investment cost associated to the implementation of u increases, the magnitude of u decreases. Moreover, it was possible to infer that the control u_1 is a crucial variable to meet the proposed objective.

On the other hand, we observed that the numerical estimation of β and γ may give rise to difficulties, inasmuch as the attainment of a quasiperfect fitting to the real data with a residual margin of error is not always feasible. At this point, as future research, the estimation method used should be improved in order to guarantee the total reliability of the estimation results.

To sum up, OC Theory plays a key role on the effective diffusion of Viral Marketing campaigns, by providing not only higher levels of infection than the obtained without using it, but also by speeding up the transmission process within the target audience.

4 | Computer Viruses Transmission

This Chapter begins to define preliminary concepts and present some state of the art related to the process of computer viruses transmission. Then, the dynamics of a virus within a system is explained and illustrated under network and epidemiology theory.

4.1 Understanding the Dynamics of Computer Viruses Transmission

A computer virus can be defined as a computer program capable of replicating itself and propagate from one computer to another, within a certain network [133], and its behavior is similar to the propagation of a biological virus [40].

According to the report referenced in [78], the origin of computer viruses dates back to 1949, discussed in the paper entitled "Theory and Organization of Complicated Automata", by John von Neumann. In this report, the author groups destructive software, or malicious objects, into four main categories: trojan horses, logic bombs, worms and computer viruses, and describe the four main phases that portray the general behavior of computer viruses within a computer system:

1. Numbness phase, where the user is unaware of the computer virus that is present in his system.

2. Propagation phase, where the virus attempts to attach itself to other files present in the system. Consequently, other machines in the network can become infected.

3. Triggering phase, referring to the moment when the virus is detected by the user.

4. Damaging phase, where, as the name suggests, the virus causes damages along the system network.

4.1.1 Can Mathematical Epidemiology and Computer Viruses Transmission Work Together?

Following up the continuous advancement of the internet and social networks, computer viruses became more efective and harder to detect and remove [120] (as cited in [99]), raising the potential for damage, loss and destruction of data within companies and individuals [25].

Over time, in order to try to understand the dynamics of CVT and minimize its propagation, epidemiological models began being intensively explored since the initial study proposed by J. Kephart and S. White in [57]. In this regard, C. Zhang and H. Huang [133] refer that the current studies on virus epidemiology have been mainly focused on two major subjects, namely viruses spreading on fully-connected networks, under the assumption that every machine on the network is equally likely to be accessed by other one, and viruses spreading on complex networks, related to network topology (see, e.g., [132]).

Still on the subject of study the dynamics of CVT, R. Thommes and M. Coates [119] studied the propagation of computer viruses, but the length of latency was not assessed. Moreover, according to [82], P. Yan and S. Liu [131] proposed a SEIR (Susceptible–Exposed–Infected–Recovered) model that assumes, with a given mathematical probability, a permanent immunization period related to the recovered hosts, which simply bears no relation to reality. In this regard, to overcome these obstacles, B. Mishra and D. Saini [83] proposed a SEIRS model with latent and temporary immune periods, which, in fact, can confirm the propagation of malicious objects. The transmission of worms in computer networks was also studied in [82], e.g., where the authors formulated an e-epidemic SIRS model for the fuzzy transmission of worms, assessing low, medium and high dynamics of infection, in order to better understand the worms dynamics within a computer network.

Regarding the computer viruses whose life period is associated to latent and disruptive phases (disruptive viruses), Y. Wu et al. [128] proposed an heterogeneous epidemic model to assess the prevalence of disruptive computer viruses in the case that every node in a network has its own virus-related attributes. Besides, some measures of containing the prevalence of malicious objects are provided by these authors.

Aiming to minimize the damages caused by computer viruses, C. Gan et al. [35] explored the influence of vaccination probability on the diffusion of computer viruses using a novel SIRS model

with generalized nonlinear incidence, providing also effective strategies to minimize the prevalence of viruses. From a related perspective, I. Ahn et al. [3] introduced an C-SEIRA (Susceptible-Exposed-Infectious-Removed-Antidotal) epidemic model with optimal control to both minimize the rate of virus infections and the cost of isolating infectious computers from the network, showing that OC Theory has a positive effect on the control the virus epidemic. For the proposed model, this work also studied stability conditions of the equilibrium solutions.

The information above allows to infer that the process of CVT is being increasingly understood, as well as it shows that Mathematical Epidemiology and OC Theory reveal a key tool to model complex problems.

Established some introductory remarks on CVT, Network Theory to describe the dynamics of computer viruses is now presented.

4.1.2 Virus Epidemic over Networks

According to [56], network and epidemiology theory are intrinsically related. In [125], G. Witten and G. Poulter discussed the spread of infectious diseases on networks, and described several common network models, namely Random, Watts-Strogatz, Lattice, Barabasí–Albert and Scale-free networks. According to these authors, each one of these models has some parallels and disparities from those expected of typical human contact networks.

Epidemiological models can be adapted to networks. Thus, based on [5], the next Section introduces the SIS and SIR epidemiological models over homogeneous networks. Heterogenous models can also be defined. However, for the sake of convenience, only homogeneous models are considered.

Notice that, throughout the following sections, the dynamics of the models are presented associated to an universe of computer network systems rather than individuals.

4.1.2.1 Homogeneous Network Models

In this type of network models, all nodes have degree very close to $\langle k \rangle$. Thus, by considering that computer systems have, approximately, $\langle k \rangle$ chances of contagion from neighbours, we can rewrite the standard SIS and SIR systems as follows.

Homogeneous SIS Epidemiological Model:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\dfrac{\beta\langle k\rangle S(t)I(t)}{N} + \gamma I(t) \\ \dfrac{dI(t)}{dt} = \dfrac{\beta\langle k\rangle S(t)I(t)}{N} - \gamma I(t)\ , \end{cases} \qquad \begin{cases} S(0) = S_0 > 0 \\ I(0) = I_0 > 0\ . \end{cases} \tag{4.1}$$

The classes S and I represent the number of computer systems which are susceptible to become infected by a malicious object and the number of machines which are infected, respectively. Regarding the model parameters, susceptible machines move to the class I at a rate β, becoming infected. Then, after the implementation of some kind of anti-infection intervention, an infected machine becomes susceptible again at a rate γ.

Considering $S(0) \approx N$, we define $\mathcal{R}_0 = \dfrac{\beta}{\gamma}$ as the number of secondary infections produced by a single infected machine within a computer network system. At this point, it is easy to prove that if $\mathcal{R}_0 < \dfrac{1}{\langle k\rangle}$, then an outbreak does not occur. On the contrary, if $\mathcal{R}_0 > \dfrac{1}{\langle k\rangle}$, an epidemic occurs. The model dynamics is the same as for the standard non-networked SIS model described in Chapter 1.

Homogeneous SIR Epidemiological Model:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\dfrac{\beta\langle k\rangle S(t)I(t)}{N} \\ \dfrac{dI(t)}{dt} = \dfrac{\beta\langle k\rangle S(t)I(t)}{N} - \gamma I(t) \\ \dfrac{dR(t)}{dt} = \gamma I(t)\ , \end{cases} \qquad \begin{cases} S(0) = S_0 > 0 \\ I(0) = I_0 > 0 \\ R(0) = 0\ . \end{cases} \tag{4.2}$$

The biological meaning of the state variables and model parameters is almost the same as for the SIS system (4.1), apart from the fact that, under the SIR model philosophy, after the implementation of some kind of anti-malware policies, an infected machine becomes immune, at a rate γ, and can not become infected again.

Remark 4.1.1. *In this context, the assumption that a machine can become completely immune to reinfections does not capture the real essence of virus dynamics. However, it is plausible to consider that if a computer system is equipped with fully effective anti-malware programs, then the probability of certain malicious objects compromise the whole system is substantially lower than*

without further anti-malware measures. In this regard, the better the fully effective anti-malware programs used, the smaller the probability of a computer system become infected. See [59] for a careful analysis on epidemic models applied to CVT.

Nevertheless, on the basis of the above arguments, and just to briefly illustrate the dynamics of a virus within a network computer system, SIS and SIR epidemic models are considered and simulated in the next Section.

4.1.2.2 SIS and SIR Epidemiological Models over Networks

Recalling that the dynamics of a virus infection in humans is similar to the propagation of virus in computer systems, this Section focuses on Network Theory to model the progression of a virus infection within a given set of computer systems, by both using SIS and SIR epidemiological models and exploring an R package for Mathematical Modeling of Infectious Disease over Networks (EpiModel) [48]. Moreover, the networks are considered to be temporal exponential random graph models (ERGMs), which allow the epidemic modeling to be firmly grounded in empirical data on the contacts and persistent partnerships that can spread infection (see [47] and the references cited therein for more detailed information). In ERGMs, the probability distribution is given by

$$\Pr(Y = y \mid \theta) = \frac{\exp\{\theta^T g(y)\}}{\sum_{y' \in Y} \exp\{\theta g(y')\}} \tag{4.3}$$

where,

- y is the observed network;
- θ is a vector of model coefficients, representing the prevalence of the node-edge structure;
- $g(y)$ is a vector of network statistics;
- $\sum_{y' \in Y} \exp\{\theta g(y')\}$ is a normalizing constant that represents the set of all possible node-edge structures of the network with the size and nodal composition of y.

In EpiModel, both the estimation and simulation of the dynamic networks are implemented using the Markov Chain Monte Carlo algorithm functions from the statnet package [46].

Hereinafter, each individual in the network (represented by a node) can develop relationships/-partnerships (represented by edges) with other individuals in its neighborhood in such a way that virus spreads-itself from node to node. Throughout this Section, the edges of the graphs have no

orientation (undirected graphs), which means that if an individual I_1 has a relationship with an individual I_2, then the symmetric relationship is also valid. Moreover, the graphs are considered to be complete, meaning that every pair of distinct nodes is connected by a unique edge.

The generation of networks in EpiModel requires the use of functions to estimate the generative model for the dynamic partnership networks (*netest*); to simulate replications of the network over time from the model fit in *netest* (*netdx*); to run the stochastic epidemic processes over a network simulated from *netest* (*netsim*).

In Listing 4.1, a source code for a network SIS model is given. Lines 1 and 2 specify the number of nodes and the undirected feature of the edges, in order to create the empty network. The command *offset(edges)* establishes an homogeneous dissolution model in which the edge duration is the same for all partnerships (line 3). In this regard, line 4 introduces a sufficient high duration value in order to guarantee that the connections between the nodes are not dissolved over time. Next, the network dissolution (line 5) and formation (line 6) formulas specify, respectively, how machines in the system dissolve and form partnerships over time, where *edges* and *concurrent* represent the number of partnerships in the network, and the number of nodes which have more than one partnership at any time step, respectively. At this point, since we are considering complete graphs, the maximum number of edges of an undirected graph G with n nodes obeys to the formula

$$G(n, 2) = \frac{n(n-1)}{2} \ . \tag{4.4}$$

Hence, the target statistics vector (line 7) is related to the expected values of the network statistics for each cross-sectional slice of the dynamic network time series. In the listing below, the maximum number of edges for a graph with 20 nodes is considered, as well as the maximum number of nodes with two or more ties - the choice of these values is based on the assumption that machines are connected to one another, in a real computer system networks.

In addition, to simulate the standard SIS and SIR epidemic models, three parameters are required:

- The probability of transmission per act (*inf.prob*);
- The rate of acts that occur within each active partnership during each time step (*act.rate*);

- The probability of an infected node recovers at a given time step (*rec.rate*), with immunity (not permanent) (SIR model) or re-susceptibility (SIS model).

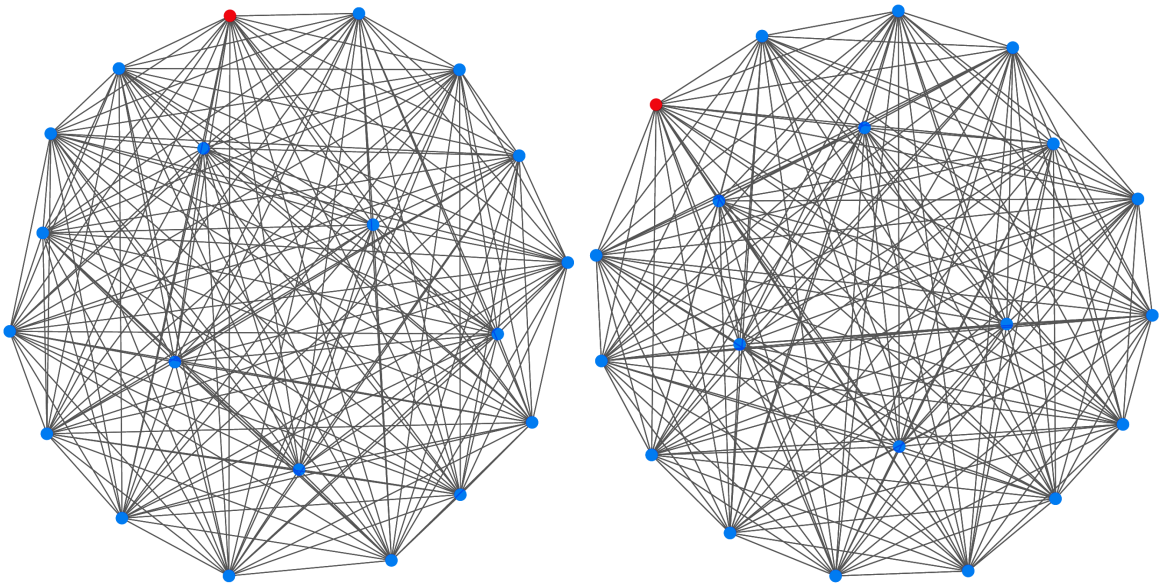
Listing 4.1: Source code for the SIS model - EpiModel.

```
1  size <- 20
2  nw <- network.initialize(size, directed = FALSE)
3  dissolution <- ~ offset(edges)
4  duration <- 10^7
5  coef.diss <- dissolution_coefs(dissolution, duration)
6  formation <- ~ edges + concurrent
7  target.stats <- c(190,20)
8  est <- netest(nw,formation,target.stats,coef.diss,verbose = FALSE)
9  dx <- netdx(est, nsims = 5, nsteps = 10)
10 param <- param.net(inf.prob = 0.4, act.rate = 0.4, rec.rate = 0.2)
11 init <- init.net(i.num = 1, r.num = 0 )
12 control <- control.net(type = "SIS", nsims = 5, nsteps = 10,verbose.int = 0)
13 sim <- netsim(est, param, init, control)
14 plot(sim, type = "network", at = 10, sims = 1,col.status = TRUE)
```

Notice that the listing 4.1 can be adapted to the SIR model, by changing the *type* command accordingly in line 12. In the next simulations, one infected individual at the beginning of the epidemic and a mean infectious period of 5 ($rec.rate^{-1}$) are assumed. In terms of notation, the *blue*, *red* and *green* nodes traduce, respectively, susceptible, infected and recovered computer systems. The epidemic behavior on the network is studied by considering different number of time steps to solve the model over (1, 3 and 10) and 5 simulations to run.

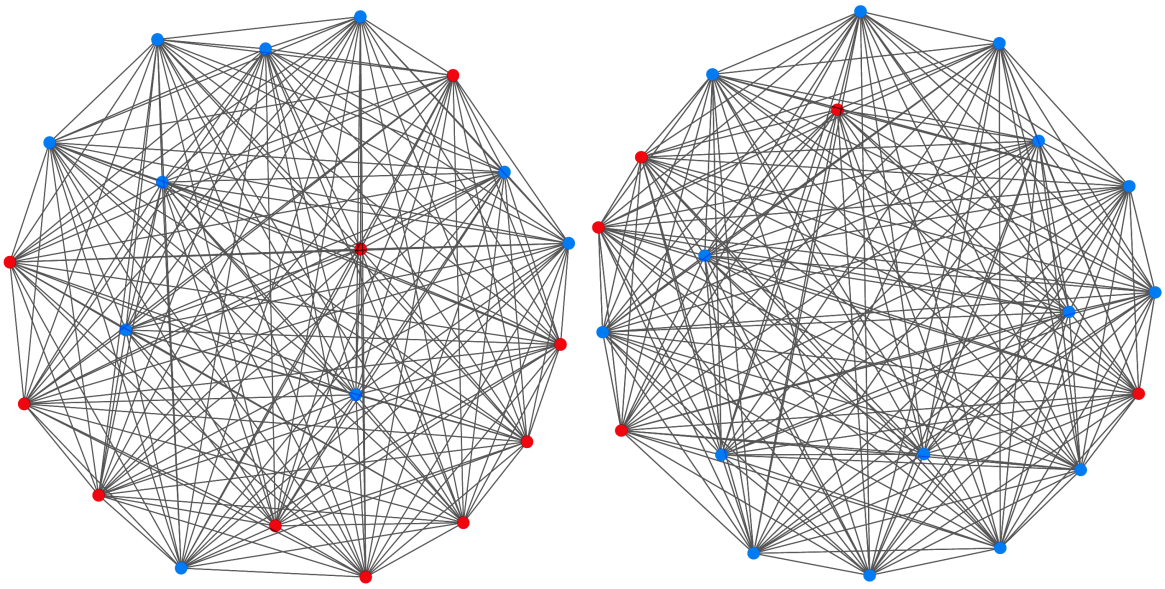
So, figure 4.1 illustrates the beginning of the epidemic, where only a single computer system is infected. At $t = 3$ (Fig. 4.2), attending to the fact that the mean infectious period is 5, the number of infected systems increases in both models. Note that due to the full connections in the computer network, a single infected computer can infect many others.

Now, if anti-malware measures are only taken in the short term, then the number of infected nodes decreases and the infected machines recover from the infection but with re-susceptibility (Fig. 4.3(a)). If, though, anti-malware measures are taken in the long term, then the number of infected nodes decreases and the infected machines recover from the infection but with immunity (Fig. 4.3(b)) (with the important caveat that this immunity should not be seen as permanent. In other words, the machine has only less probability to become infected, as highlighted in the Remark 4.1.1).



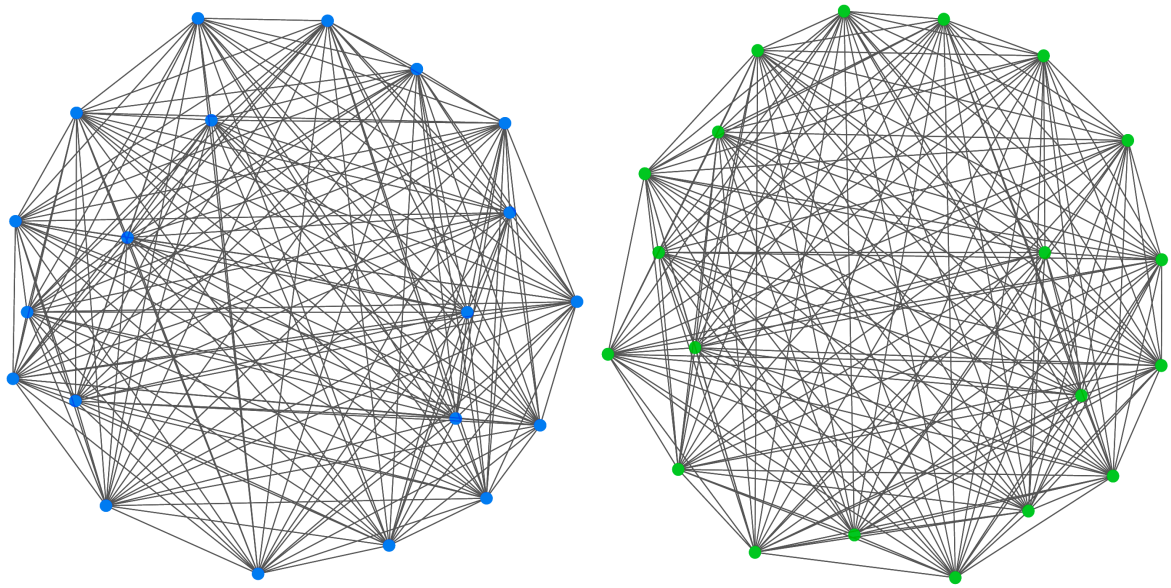
(a) SIS model (b) SIR model

Figure 4.1: Prevalence on SIS and SIR models at $t = 1$: *blue* – susceptible; *red* – infected.



(a) SIS model (b) SIR model

Figure 4.2: Prevalence on SIS and SIR models at $t = 3$: *blue* – susceptible; *red* – infected.



(a) SIS model

(b) SIR model

Figure 4.3: Prevalence on SIS and SIR models at $t = 10$: *blue* – susceptible; *green* – recovered.

Remark 4.1.2. *Although network epidemic models are essential to study the propagation of virus infections, some obstacles arise when high dimensional networks are considered. In the context of challenges for network epidemic models, see [97].*

4.2 Discussion

The propagation of viruses within computer networks is becoming an increasingly urgent issue. This Chapter began to propose a scientific literature review on the subject. Then, in a very superficial way, the infection propagation within computer networks was modeled using Network Theory from a very recent R package for Mathematical Modeling of Infectious Disease over Networks (EpiModel). It should be strengthened that the high dimension of the real networks might be a problem to the use of some softwares to model epidemic phenomena, both in terms of computacional performance and accuracy. In addition, it is appropriate to reinforce the necessity of more realistic models to capture, the best possible, the real dynamics of CVT.

Conclusions and Future Research

The major aim of this dissertation was to understand and explore both Mathematical Epidemiology and Optimal Control Theory. More particularly, these domains were used to understand the dynamics of two different environments - Viral Marketing and Computer Viruses Transmission.

With this purpose, we started by introducing some basic theory related to epidemiological models (Chapter 1) and Optimal Control Theory (Chapter 2). Then, in the Second Part of this dissertation, the dynamics of Viral Marketing and Computer Viruses Transmission was analyzed using the mathematical tools described in the first two chapters. In this process, some major contributions were derived.

Major Contributions

The main perceived remarks on Viral Marketing and Computer Viruses Transmission are described as follows.

In Chapter 3, Viral Marketing is presented as an epidemiological dynamical system. Next, an Optimal Control problem related to the trade-off between the maximization of information spreading and the minimization of the costs associated to it was formulated and studied using not only indirect and direct numerical techniques to solve the problem, but also real numerical data from one of the most viral advertisements of all time - Dove Real Beauty Sketches. At this point, we studied the impact of encouraging susceptible individuals to share the marketing message and concluded that when the investment costs in publicity policies increase, the fraction of individuals who have contact with the marketing message decreases. Additionally, a second control policy related to the fostering of infected individuals to continue to spread the marketing messages was assessed, allowing to provide Optimal Control strategies in optimal time windows.

In Chapter 4, a recent R package for Mathematical Modeling of Infectious Disease over Networks (EpiModel) was used to analyze the dynamics of SIS and SIR models over networks, aiming

to illustrate, in an introductory way, Computer Viruses Transmission. These models were described under an homogeneous network environment and the results correlated positively with the well-known dynamics of these epidemic models.

Future Research

As future work, it would be interesting to test other solvers with additional optimal control measures and perform a sensitivity analysis on the parameters. In addition, a cost-effectiveness analysis to compare the application of control policies separately or in combination is also a major goal to be achieved in Viral Marketing.

Considering that, in marketing campaigns, there are individuals who discourage others to share the marketing message, it is pertinent to consider the inclusion of variables that assess the pejorative impact of anti-propagation campaigns.

On the other hand, since Viral Marketing and Computer Viruses Transmission dynamics depend also on the past, in terms of time, our intention relates to formulate Optimal Control problems with delay differential equations, to give even more realism to the dynamical process of these two phenomena. Moreover, several types of delay, multi-objective optimization and Bang-Bang controls are also pointed as future research.

Blessed are the Mathematicians who breathe, explore, develop and expand the most beautiful language on earth.

João N.C. Gonçalves

Appendix: Source Codes

Forward-Backward Sweep Method Algorithm in Matlab

Listing 2: Forward-Backward Sweep Method in MATLAB.

```
function y = code_FBSMethod_NORM
test = -1;
T=6;
delta = 0.001;
M = 1000;
t=linspace(0,T,M+1);
h=T/M;
h2 = h/2;
N=10^9;
beta=67.524423215969549;
gamma=65.075135004128185;

S=zeros(1,M+1);
I=zeros(1,M+1);
R=zeros(1,M+1);
S(1)=(N-30000)/N;          % initial conditions assigned to x(0)
I(1)=30000/N;
R(1)=0;
u=zeros(1,M+1);
lambda1=zeros(1,M+1);
lambda2=zeros(1,M+1);
lambda3=zeros(1,M+1);
lambda1(M+1)=0;
lambda2(M+1)=1;
lambda3(M+1)=1;
```

```
26
27 J=zeros(1,M+1);
28 while(test < 0)          % while tolerance is attained, repeat the process
29
30     oldu = u;
31     oldS = S;
32     oldI = I;
33     oldR = R;
34     oldlambda1 = lambda1;
35     oldlambda2 = lambda2;
36     oldlambda3 = lambda3;
37
38 for i = 1:M          % forward
39
40 m11 = (-beta*S(i)*I(i))-u(i)*S(i);
41 m12 = (beta*S(i)*I(i))-gamma*I(i)+(u(i)*S(i));
42 m13 = gamma*I(i);
43
44 m21 = (-beta*(S(i)+h2*m11))*(I(i)+h2*m12)-0.5*(u(i)+u(i+1))*(S(i)+h2*m11);
45 m22 = (beta*(S(i)+h2*m11))*(I(i)+h2*m12)-gamma*(I(i)+h2*m12)
46 +0.5*(u(i)+u(i+1))*(S(i)+h2*m11);
47 m23 = gamma*(I(i)+h2*m12);
48
49 m31 = (-beta*(S(i)+h2*m21))*(I(i)+h2*m22)-0.5*(u(i)+u(i+1))*(S(i)+h2*m21);
50 m32 = (beta*(S(i)+h2*m21))*(I(i)+h2*m22)-gamma*(I(i)+h2*m22)
51 +0.5*(u(i)+u(i+1))*(S(i)+h2*m21);
52 m33 = gamma*(I(i)+h2*m22);
53
54 m41 = (-beta*(S(i)+h*m31))*(I(i)+h*m32)-u(i+1)*(S(i)+h*m31);
55 m42 = (beta*(S(i)+h*m31))*(I(i)+h*m32)-gamma*(I(i)+h*m32)
56 +u(i+1)*(S(i)+h*m31);
57 m43 = gamma*(I(i)+h*m32);
58
59     S(i+1) = S(i) + (h/6)*(m11 + 2*m21 + 2*m31 + m41);
60     I(i+1) = I(i) + (h/6)*(m12 + 2*m22 + 2*m32 + m42);
61     R(i+1) = R(i) + (h/6)*(m13 + 2*m23 + 2*m33 + m43);
62 end
63     for i = 1:M          % backward
```

```
64          j = M + 2 - i;
65
66  % ADJOINT FUNCTIONS
67
68  n11 = lambda1(j)*(beta*I(j)+u(j))-lambda2(j)*(beta*I(j)+u(j));
69  n12 = lambda1(j)*(beta*S(j))-lambda2(j)*(beta*S(j)-gamma)-lambda3(j)*gamma;
70  n13 = 0;
71
72  n21 = (lambda1(j)-h2*n11)*(beta*(0.5*(I(j)+I(j-1)))+(0.5*(u(j)+u(j-1))))
73  -(lambda2(j)-h2*n12)*(beta*(0.5*(I(j)+I(j-1)))+(0.5*(u(j)+u(j-1))));
74  n22 = (lambda1(j)-h2*n11)*(beta*(0.5*(S(j)+S(j-1))))-(lambda2(j)-h2*n12)*(
        beta*(0.5*(S(j)+S(j-1)))-gamma)-(lambda3(j)-h2*n13)*gamma;
75  n23 = 0;
76
77  n31 = (lambda1(j)-h2*n21)*(beta*(0.5*(I(j)+I(j-1)))+(0.5*(u(j)+u(j-1))))
78  -(lambda2(j)-h2*n22)*(beta*(0.5*(I(j)+I(j-1)))+(0.5*(u(j)+u(j-1))));
79  n32 = (lambda1(j)-h2*n21)*(beta*(0.5*(S(j)+S(j-1))))-(lambda2(j)-h2*n22)*(
        beta*(0.5*(S(j)+S(j-1)))-gamma)-(lambda3(j)-h2*n23)*gamma;
80  n33= 0;
81
82  n41 = (lambda1(j)-h*n31)*(beta*I(j-1)+u(j-1))-(lambda2(j)-h*n32)*
83  (beta*I(j-1)+u(j-1));
84  n42 = (lambda1(j)-h*n31)*(beta*S(j-1))-(lambda2(j)-h*n32)*
85  (beta*S(j-1)-gamma)-(lambda3(j)-h*n33)*gamma;
86  n43 = 0;
87
88        lambda1(j-1) = lambda1(j) - (h/6)*(n11 + 2*n21 + 2*n31 + n41);
89        lambda2(j-1) = lambda2(j) - (h/6)*(n12 + 2*n22 + 2*n32 + n42);
90        lambda3(j-1) = lambda3(j) - (h/6)*(n13 + 2*n23 + 2*n33 + n43);
91  end
92
93      b=10^2;             % for example
94      u1=min(0.025,max((S.*(lambda2-lambda1))/(2*b),0));
95      u=0.5*(u1 + oldu);
96      J= I(M+1)+R(M+1)-trapz(t,b*(u.^2))
97      temp1 = delta*sum(abs(u)) - sum(abs(oldu - u));
98      temp2 = delta*sum(abs(S)) - sum(abs(oldS - S));
99      temp3 = delta*sum(abs(I)) - sum(abs(oldI - I));
```

```
100       temp4 = delta*sum(abs(R)) - sum(abs(oldR - R));
101       temp5 = delta*sum(abs(lambda1)) - sum(abs(oldlambda1 - lambda1));
102       temp6 = delta*sum(abs(lambda2)) - sum(abs(oldlambda2 - lambda2));
103       temp7 = delta*sum(abs(lambda3)) - sum(abs(oldlambda3 - lambda3));
104       test = min(temp1, min(temp2, min(temp3, min(temp4, min(temp5, min(temp6
              ,temp7))))));
105   end
```

AMPL formulation for the discretized controlled system (3.21) using KNITRO

Listing 3: AMPL formulation for the discretized controlled system (3.21).

```
1  #### PARAMETERS ###
2  param N=1000;
3  param beta=67.524423215969549;
4  param gamma=65.075135004128185;
5  param B=0.1;  # for example
6  param x1_0=(10^9-30000)/(10^9);
7  param x2_0=(30000)/(10^9);
8  param x3_0=0;
9  param tf=6;
10
11 #### VARIABLES ###
12 var x1 {0..N}, >=0 <=1, default x1_0;   # susceptible individuals
13 var x2 {0..N}, >=0 <=1, default x2_0;   # infected individuals
14 var x3 {0..N}, >=0 <=1, default x3_0;   # recovered individuals
15 var u {0..N}, >=0 <=1;        # control variable (recruitment of
       susceptible individuals to act as spreaders) / the upper bound of  "var
        u" must change according to the value of B.
16
17 #### OBJECTIVE FUNCTION ###
18 maximize
19 cost: x3[N]+x2[N]-B*(tf/(2*N))*(u[0]^2+u[N]^2+2*(sum{i in 1..N-1} u[i]^2));
20
21 #### CONSTRAINTS ###
22 subject to
23
```

```
24 i1: x1[0] = x1_0;
25 i2: x2[0] = x2_0;
26 i3: x3[0] = x3_0;
27 f1 {i in 0..N-1}: x1[i+1] = x1[i] + (tf/N)*(-beta*x1[i]*x2[i]-u[i]*x1[i]);
28 f2 {i in 0..N-1}: x2[i+1] = x2[i] + (tf/N)*(beta*x1[i]*x2[i]+u[i]*x1[i]-
       gamma*x2[i]);
29 f3 {i in 0..N-1}: x3[i+1] = x3[i] + (tf/N)*(gamma*x2[i]);
30
31 ### SOLVE ###
32 option solver knitro;                          # case where KNITRO is used.
33 solve;
34
35 ### OUTPUT SCREEN ###
36 display _total_solve_time;
37 printf: " # cost = %24.16e\n", cost;
38 printf: " # N = %d\n", N;
39 printf{i in 0..N}: "%d %24.16e %24.16e %24.16e %24.16e %24.16e\n",
40 i, i*tf/N, x1[i], x2[i], x3[i], u[i];
41 end;
```

Bibliography

[1] N. Adelsarbanlar and B. Khoshtinat. Critical factors and advantage factors influencing the implementation of viral marketing by considering the mediating role of Islamic marketing; a conceptual approach. *Procedia Economics and Finance*, 36:433–440, 2016.

[2] G. Agaba, Y. Kyrychko and K. Blyuss. Time-delayed SIS epidemic model with population awareness. *Ecological Complexity*, 31:50–56, 2017.

[3] I. Ahn, H. Oh and J. Park. Investigation of the C-SEIRA model for controlling malicious code infection in computer networks. *Applied Mathematical Modelling*, 39(14):4121–4133, 2015.

[4] I. Area, F. Ndaïrou, J. Nieto, C.J. Silva and D.F.M. Torres. Ebola model and optimal control with vaccination constraints. *Accepted for publication in Journal of Industrial and Management Optimization*, `https://arxiv.org/abs/1703.01368`, 2017.

[5] A. Arratia and R. Ferrer-i-Cancho. Epidemic models over networks. `https://www.cs.upc.edu/~CSN/slides/11epidemic.pdf`. Accessed on May 2017.

[6] M. Bazaraa, H. Sherali and C. Shetty. Nonlinear Programming: Theory and Algorithms. *Wiley-Interscience (3rd edn.)*, 2006.

[7] S. Bidari, X. Chen, D. Peters, D. Pittman and P. Simon. Solvability of implicit final size equations for SIR epidemic models. *Mathematical Biosciences*, 282:181–190, 2016.

[8] S. Blower and D. Bernoulli. An attempt at a new analysis of the mortality caused by smallpox and of the advantages of inoculation to prevent it. *Reviews in Medical Virology*, 14(5):275–288, 2004.

[9] E. Bonyah, K. Badu and S.K. Asiedu-Addo. Optimal control application to an Ebola model. *Asian Pacific Journal of Tropical Biomedicine*, 6(4):283–289, 2016.

[10] S. Bowong and A.M. Alaoui. Optimal intervention strategies for tuberculosis. *Communications in Nonlinear Science and Numerical Simulation*, 18(6):1441–1453, 2013.

[11] F. Brauer and C. Castillo–Chavez. Mathematical Models in Population Biology and Epidemiology. *Springer New York Dordrecht Heidelberg London (2nd edn.)*, 2012.

[12] F. Brauer, P. Van den Driessche and J. Wu. Mathematical Epidemiology. *Springer-Verlag, Berlin Heidelberg*, 2008.

[13] L. Bubniaková. The Mathematics of Infectious Diseases. *M.Sc. Thesis, Comenius University, Faculty of Mathematics, Physics and Informatics, Bratislava*, 2007.

[14] K. Burnham and D. Anderson. Model Selection and Multi-model Inference: A Practical Information-Theoretic Approach. *Springer, Berlin Heidelberg New York*, 2002.

[15] R. Byrd, J. Nocedal and R. Waltz. KNITRO: An Integrated Package for Nonlinear Optimization. *Large Scale Nonlinear Optimization. Nonconvex Optimization and Its Applications, vol. 83. Springer-Verlag*, 2006.

[16] Y. Cai, Y. Kang and W. Wang. A stochastic SIRS epidemic model with nonlinear incidence rate. *Applied Mathematics and Computation*, 305:221–240, 2017.

[17] L. Cai, X. Li, N. Tuncer, M. Martcheva and A. Lashari. Optimal control of a malaria model with asymptomatic class and superinfection. *Mathematical Biosciences*, 288:94–108, 2017.

[18] L. Cai, C. Modnak and J. Wang. An age-structured model for cholera control with vaccination. *Applied Mathematics and Computation*, 299:127–140, 2017.

[19] A. Capaldi, S. Behrend, B. Berman, J. Smith, J. Wright and A. Lloyd. Parameter estimation and uncertainty quantification for an epidemic model. Math. Biosci. Eng., 9(3):553–576, 2012.

[20] T.J. Case. An Illustrated guide to Theoretical Biology. *Oxford University Press, Oxford*, 2000.

[21] L. Cesari. Optimization - theory and applications, Problems with Ordinary Differential Equations. *Applications of Mathematics, Vol. 17, Springer-Verlag, New-York*, 1983.

[22] Z. Chang, X. Meng and X. Lu. Analysis of a novel stochastic SIRS epidemic model with two different saturated incidence rates. *Physica A: Statistical Mechanics and its Applications*, 472:103–116, 2017.

[23] L. Chen, K. Hattaf and J. Sun. Optimal control of a delayed SLBS computer virus model. *Physica A: Statistical Mechanics and its Applications*, 427:244–250, 2015.

[24] Y. Chen, S. Zou and J. Yang. Global analysis of an SIR epidemic model with infection age and saturated incidence. *Nonlinear Analysis: Real World Applications*, 30:16–31, 2016.

[25] F. Cohen. A short course of computer viruses. *Computer and Security 8*, 149–160, 1990.

[26] K. Dietz. Epidemics and Rumours: A Survey. *Journal of the Royal Statistical Society, Series A*, 130(4):505–528, 1967.

[27] A. Dobele, A. Lindgreen, M. Beverland, J. Vanhamme and R. Van Wijk. Why pass on viral messages? Because they connect emotionally. *Business Horizons*, 50(4):291–304, 2007.

[28] A. Dobele, D. Toleman and M. Beverland. Controlled infection! Spreading the brand message through viral marketing. *Business Horizons*, 48(2):143–149, 2005.

[29] Dove's - Real Beauty Sketches, webpage: `http://www.dove.com/us/en/stories/campaigns/real-beauty-sketches.html`. Accessed on September 2016.

[30] Y. Enatsu, Y. Nakata and Y. Muroya. Lyapunov functional techniques for the global stability analysis of a delayed SIRS epidemic model. *Nonlinear Analysis: Real World Applications*, 13(5):2120–2133, 2012.

[31] Fatmawati and H. Tasman. An optimal control strategy to reduce the spread of malaria resistance. *Mathematical Biosciences*, 262:73–79, 2015.

[32] K. Fister, S. Lenhart and J. McNally. Optimizing chemotherapy in an HIV model. *Electronic Journal of Differential Equations*, 32:1–12, 1998.

[33] W. Fleming and R. Rishel. Deterministic and Stochastic Optimal Control. *Springer-Verlag, New York*, 1975.

[34] R. Fourer, D. Gay and B. Kernighan. AMPL: A Modeling Language for Mathematical Programming. *Duxbury Press, Brooks/Cole Publishing Company*, 2002.

[35] C. Gan, X. Yang, W. Liu, Q. Zhu and X. Zhang. An epidemic model of computer viruses with vaccination and generalized nonlinear incidence rate. *Applied Mathematics and Computation*, 222:265–274, 2013.

[36] Q. Ge, G. Ji, J. Xu and X. Fan. Extinction and persistence of a stochastic nonlinear SIS epidemic model with jumps. *Physica A: Statistical Mechanics and its Applications*, 462:1120–1127, 2016.

[37] P. Giamberardino and D. Iacoviello. Optimal control of SIR epidemic model with state dependent switching cost index. *Biomedical Signal Processing and Control*, 31:377–380, 2017.

[38] T. Götz, R. Rockenfeller and K. Wijaya. Optimization problems in epidemiology, biomechanics & medicine. *International Journal of Advances in Engineering Sciences and Applied Mathematics*, 7(1–2):25–32, 2015.

[39] A. Gray, D. Greenhalgh, L. Hu, X. Mao and J. Pan. A Stochastic Differential Equation SIS Epidemic Model. *SIAM J. Appl. Math.*, 71(3):876–902, 2011.

[40] X. Han and Q. Tan. Dynamical behavior of computer virus on Internet. *Applied Mathematics and Computation*, 217(6):2520–2526, 2010.

[41] T. Harko, F. Lobo and M. Mak. Exact analytical solutions of the Susceptible-Infected-Recovered (SIR) epidemic model and of the SIR model with equal death and birth rates. *Applied Mathematics and Computation*, 236:184–194, 2014.

[42] S. Haryani and B. Motwani. Discriminant model for online viral marketing influencing consumers behavioural intention. *Pacific Science Review B: Humanities and Social Sciences*, 1(1):49–56, 2015.

[43] H.W. Hethcote. Mathematical Understanding of Infectious Disease Dynamics, *Lecture Notes Series Institute for Mathematical Sciences, vol. 16. Chapter 1, National University of Singapore*, pp. 1–61, 2008.

[44] H.W. Hethcote. The Mathematics of Infectious Diseases. *SIAM Review*, 42(4):599–653, 2000.

[45] Q. Hou and T. Wang. Global stability and a comparison of SVEIP and delayed SVIP epidemic models with indirect transmission. *Commun. Nonlinear Sci. Numer. Simulat.*, 43:271–281, 2017.

[46] D. Hunter, M. Handcock, C. Butts, S. Goodreau and M. Morris. ergm: A Package to Fit, Simulate and Diagnose Exponential-Family Models for Networks. *Journal of Statistical Software*, 24(3):1–29, 2008.

[47] S. Jenness, S. Goodreau and M. Morris. EpiModel: An R Package for Mathematical Modeling of Infectious Disease over Networks. *Tutorial report*, 2017.

[48] S. Jenness, S. Goodreau and M. Morris. EpiModel: Mathematical Modeling of Infectious Disease. R Package Version 1.3.0. `http://epimodel.org/`, 2017.

[49] E. Jung, S. Lenhart and Z. Feng. Optimal Control of treatments in a two-strain tuberculosis model. *Discrete and Continuous Dynamical Systems-Series B*, 2(4):473–482, 2002.

[50] M. I. Kamien and N. L. Schwartz. Dynamic Optimization: The Calculus of Variations and Optimal Control in Economics and Management, *North-Holland, New-York*, 1991.

[51] K. Kandhway and J. Kuri. How to run a campaign: Optimal control of SIS and SIR information epidemics. *Applied Mathematics and Computation*, 231:79–92, 2014.

[52] K. Kandhway and J. Kuri. Optimal control of information epidemics modeled as Maki Thompson rumors. *Commun. Nonlinear Sci. Numer. Simulat.*, 19(12):4135–4147, 2014.

[53] H. Kang and X. Fu. Epidemic spreading and global stability of an SIS model with an infective vector on complex networks. *Commun. Nonlinear Sci. Numer. Simulat.*, 27(1–3):30–39, 2015.

[54] A. Karnik and P. Dayama. Optimal Control of Information Epidemics. *Proc. IEEE Commun. System. Networks Conf.*, 1–7, 2012.

[55] A. Katok and B. Hasselblatt. Introduction to the Modern Theory of Dynamical Systems. *Cambridge University Press*, 1997.

[56] M. Keeling and K. Eames. Networks and epidemic models. *J. R. Soc. Interface*, 2(4):295–307, 2005.

[57] J. Kephart and S. White. Directed-graph epidemiological models of computer viruses. *Proceedings of the 1991 IEEE Computer Society Symposium on Research in Security and Privacy, Oakland, CA*, pp. 343–359, 1991.

[58] W. Kermack and A. McKendrick. A contribution to the mathematical theory of epidemics. *Proc. Royal Soc. London, B*, 115(772):700–721, 1927.

[59] M.S. Khan. A computer virus propagation model using delay differential equations with probabilistic contagion and immunity. *International Journal of Computer Networks & Communications*, 6(5):111-128, 2014.

[60] R. Kheirandish, A. Krishen and P. Kachroo. Application of Optimal Control Theory in marketing: What is the optimal number of choices on a shopping website?. *International Journal of Computer Applications in Technology*, 34(3):207–215, 2009.

[61] J. Lagarias, J. Reeds, M. Wright and P. Wright. Convergence Properties of the Nelder-Mead Simplex Method in Low Dimensions. *SIAM Journal of Optimization*, 9(1):112–147, 1998.

[62] A. Lahrouz, L. Omari, D. Kiouach and A. Belmaâti. Complete global stability for an SIRS epidemic model with generalized non-linear incidence and vaccination. *Applied Mathematics and Computation*, 218(11):6519–6525, 2012.

[63] A. Lemos-Paião, C.J. Silva and D.F.M. Torres. An epidemic model for cholera with optimal control treatment. *Journal of Computational and Applied Mathematics*, 318:168–180, 2017.

[64] S. Lenhart and J. Workman. Optimal control applied to biological models. *Chapman & Hall/CRC, Boca Raton, FL*, 2007.

[65] J. Leskovec, L.A. Adamic and B.A. Huberman. The dynamics of viral marketing. *ACM Trans. Web (TWEB)*, 1(1):1–39, 2007.

[66] C. Li, J. Li, Z. Ma and H. Zhu. Canard phenomenon for an SIS epidemic model with nonlinear incidence. *Journal of Mathematical Analysis and Applications*, 420(2):987–1004, 2014.

[67] J. Li, Z. Teng, G. Wang, L. Zhang and C. Hu. Stability and bifurcation analysis of an SIR epidemic model with logistic growth and saturated treatment. *Chaos, Solitons and Fractals*, 99:63–71, 2017.

[68] T. Li, Y. Wang and Z. Guan. Spreading dynamics of a SIQRS epidemic model on scale-free networks. *Commun. Nonlinear Sci. Numer. Simulat.*, 19(3):686–692, 2014.

[69] B. Liu. Convergence of an SIS epidemic model with a constant delay. *Applied Mathematics Letters*, 49:113–118, 2015.

[70] Q. Liu and Q. Chen. Analysis of the deterministic and stochastic SIRS epidemic models with nonlinear incidence. *Physica A: Statistical Mechanics and its Applications*, 428:140–153, 2015.

[71] Q. Liu, Q. Chen and D. Jiang. The threshold of a stochastic delayed SIR epidemic model with temporary immunity. *Physica A: Statistical Mechanics and its Applications*, 450:115–125, 2016.

[72] Q. Liu and D. Jiang. The threshold of a stochastic delayed SIR epidemic model with vaccination. *Physica A: Statistical Mechanics and its Applications*, 461:140–147, 2016.

[73] Q. Liu, D. Jiang, N. Shi, T. Hayat and B. Ahmad. Stationary distribution and extinction of a stochastic SEIR epidemic model with standard incidence. *Physica A: Statistical Mechanics and its Applications*, 476:58–69, 2017.

[74] Q. Liu, D. Jiang, N. Shi, T. Hayat and A. Alsaedi. Asymptotic behavior of a stochastic delayed SEIR epidemic model with nonlinear incidence. *Physica A: Statistical Mechanics and its Applications*, 462:870–882, 2016.

[75] Q. Liu, D. Jiang, N. Shi, T. Hayat and A. Alsaedi. Asymptotic behaviors of a stochastic delayed SIR epidemic model with nonlinear incidence. *Communications in Nonlinear Science and Numerical Simulation*, 40:89–99, 2016.

[76] Q. Liu, D. Jiang, N. Shi, T. Hayat and A. Alsaedi. Periodic solution for a stochastic nonautonomous SIR epidemic model with logistic growth. *Physica A: Statistical Mechanics and its Applications*, 462:816–826, 2016.

[77] A. Mallela, S. Lenhart and N.K. Vaidya. HIV–TB co-infection treatment: Modeling and Optimal Control Theory perspectives. *Journal of Computational and Applied Mathematics*, 307:143–161, 2016.

[78] M. Marietta. An Introduction to Computer Viruses. *Technical report, Energy Systems, Inc., Oak Ridge National Laboratory, U.S. Department of Energy*, 1992.

[79] M. Martcheva. An Introduction to Mathematical Epidemiology. *Springer, New York Heidelberg Dordrecht London*, 2015.

[80] J. Mateus and C. Silva. HIV–TB co-infection treatment: Existence of periodic solutions of a periodic SEIRS model with general incidence. *Nonlinear Analysis: Real World Applications*, 34:379–402, 2017.

[81] X. Meng, S. Zhao, T. Feng and T. Zhang. Dynamics of a novel nonlinear stochastic SIS epidemic model with double epidemic hypothesis. *Journal of Mathematical Analysis and Applications*, 433(1):227–242, 2016.

[82] B. Mishra and S. Pandey. Fuzzy epidemic model for the transmission of worms in computer network. *Nonlinear Analysis: Real World Applications*, 11(5):4335–4341, 2010.

[83] B. Mishra and D. Saini. SEIRS epidemic model with delay for transmission of malicious objects in computer network. *Appl. Math. Comput.*, 188(2):1476–1482, 2007.

[84] A. Mochalova and A. Nanopoulos. A targeted approach to viral marketing. *Electronic Commerce Research and Applications*, 13(4):283–294, 2014.

[85] D.P. Moualeu, M. Weiser, R. Ehrig and P. Deuflhard. Optimal control for a tuberculosis model with undetected cases in Cameroon. *Communications in Nonlinear Science and Numerical Simulation*, 20(3):986–1003, 2015.

[86] Y. Muroya, Y. Enatsu and T. Kuniya. Global stability for a multi-group SIRS epidemic model with varying population sizes. *Nonlinear Analysis: Real World Applications*, 14(3):1693–1704, 2013.

[87] Y. Muroya, T. Kuniya and J. Wang. Stability analysis of a delayed multi-group SIS epidemic model with nonlinear incidence rates and patch structure. *Journal of Mathematical Analysis and Applications*, 425(1):415–439, 2015.

[88] Y. Muroya, H. Li and T. Kuniya. Complete global analysis of an SIRS epidemic model with graded cure and incomplete recovery rates. *Journal of Mathematical Analysis and Applications*, 410(2):719–732, 2014.

[89] G. Mwanga, H. Haario and V. Capasso. Optimal control problems of epidemic systems with parameter uncertainties: Application to a malaria two-age-classes transmission model with asymptomatic carriers. *Mathematical Biosciences*, 261:1–12, 2015.

[90] A. Mwasa and J. M. Tchuenche. Mathematical analysis of a cholera model with public health interventions. *Biosystems*, 105(3):190–200, 2011.

[91] Y. Nakata, Y. Enatsu and Y. Muroya. On the global stability on an SIRS epidemic model with distributed delays. *Discrete and Continuous Dynamical Systems*, Supplement: 410:1119–1128, 2011.

[92] NEOS, webpage: http://www-neos.mcs.anl.gov/neos/. Accessed on March 2017.

[93] K.O. Okosun and O.D. Makinde. A co-infection model of malaria and cholera diseases with optimal control. *Mathematical Biosciences*, 258:19–32, 2014.

[94] K.O. Okosun and O.D. Makinde and I. Takaidza. Impact of optimal control on the treatment of HIV/AIDS and screening of unaware infectives. *Applied Mathematical Modelling*, 37(6):3802–3820, 2013.

[95] G. Pachpute and S.P. Chakrabarty. Dynamics of hepatitis C under optimal therapy and sampling based analysis. *Communications in Nonlinear Science and Numerical Simulation*, 18(8):2202–2212, 2013.

[96] B. Passenberg. Theory and Algorithms for Indirect Methods in Optimal Control of Hybrid Systems. *PhD thesis, Technische Universität München*, 2012.

[97] L. Pellis, F. Ball, S. Bansal, K. Eames, T. House, V. Isham and P. Trapman. Eight challenges for network epidemic models. *Epidemics*, 10:58–62, 2015.

[98] J.E. Phelps, R. Lewis, L. Mobilio, D. Perry and N. Raman. Viral marketing or electronic word-of-mouth advertising: examining consumer responses and motivations to pass along email. *Journal of Advertising Research*, 44(4):333–348, 2004.

[99] J. Piqueira and V. Araujo. A modified epidemiological model for computer viruses. *Applied Mathematics and Computation*. 213(2):355–360, 2009.

[100] M. Poku, L. Biegler and J. Kelly. Nonlinear Optimization with Many Degrees of Freedom in Process Engineering. *Ind. Eng. Chem. Res.*, 43:6803–6812, 2004.

[101] L. Pontryagin, V. Boltyanskii, R. Gamkrelidze and E. Mishchenko. The Mathematical Theory of Optimal Processes, *Pergamon Press, Oxford*, 1964.

[102] L. Porter and G. Golan. From Subservient Chickens to Brawny Men: A Comparison of Viral Advertising to Television Advertising. *Journal of Interactive Advertising*, 6(2):30–38, 2006.

[103] D. Posny, J. Wang, Z. Mukandavire and C. Modnak. Analyzing transmission dynamics of cholera with public health interventions. *Mathematical Biosciences*, 264:38–53, 2015.

[104] Purdue's College of Science, `https://www.math.purdue.edu/files/academic/courses/2010spring/MA26200/1_10.pdf`. Accessed on July 2017.

[105] L. Qi and J. Cui. The stability of an SEIRS model with nonlinear incidence, vertical transmission and time delay. *Applied Mathematics and Computation*, 221:360–366, 2013.

[106] A. Rao. A survey of numerical methods for optimal control. *Technical Report AAS 09-334. Dep. of Mechanical and Aerospace Engineering, University of Florida*, 2009.

[107] H.S. Rodrigues. Optimal Control and Numerical Optimization Applied to Epidemiological Models. *Ph.D. Thesis, University of Aveiro, Department of Mathematics, Portugal*, 2012.

[108] H.S. Rodrigues and M.J. Fonseca. Can information be spread as a virus? Viral marketing as epidemiological model. *Math. Methods Appl. Sci.*, 39(16):4780–4786, 2016.

[109] H.S. Rodrigues, M.T.T. Monteiro and D.F.M. Torres. Optimal control and numerical software: an overview. Systems Theory: Perspectives. *Applications and Developments*, 93–110, 2014.

[110] R.T. Rust, T. Ambler, G.S. Carpenter, K. Kumar and R.K. Srivastava. Measuring Marketing Productivity: Current Knowledge and Future Directions. *Journal of Marketing*, 68(4):76–89, 2004.

[111] S. Schroeder. YouTube Now Has One Billion Monthly Users, webpage: `http://mashable.com/2013/03/21/youtube-one-billion/#Wv1EUzqZGkqx`. Accessed on September 2016.

[112] O. Sharomi and T. Malik. Optimal control in epidemiology. *Annals of Operations Research*, 251(1–2):55–71, 2015.

[113] C.J. Silva and D.F.M. Torres. Global Stability for a HIV/AIDS model. *Commun. Fac. Sci. Univ. Ank. Series A1*, 67(1):93–101, 2017.

[114] C.J. Silva and D.F.M. Torres. Modeling and optimal control of HIV/AIDS prevention through PrEP. accepted for publication in *Discrete and Continuous Dynamical Systems – Series S' (DCDS-S)*. `https://arxiv.org/abs/1703.06446`, 2017.

[115] C.J. Silva and D.F.M. Torres. Optimal control for a tuberculosis model with reinfection and post-exposure interventions. *Mathematical Biosciences*, 244(2):154–164, 2013.

[116] K. Sohn. J.T. Gardner and J.L. Weaver. Viral Marketing - more than a Buzzword. *Journal of Applied Business and Economics*, 14(1):21–42, 2013.

[117] L. Stampler. How Dove's 'Real Beauty Sketches' Became The Most Viral Video Ad Of All Time, webpage: `http://static4.businessinsider.com/image/519cd1336bb3f76344000015-960/screen%20shot%202013-05-22%20at%209.48.32%20am.png`. Accessed on September 2016.

[118] Z. Teng and L. Wang. Persistence and extinction for a class of stochastic SIS epidemic models with nonlinear incidence rate. *Physica A: Statistical Mechanics and its Applications*, 451:507–518, 2016.

[119] R. Thommes and M. Coates. Modeling virus propagation in peer-to-peer networks. *IEEE International Conference on Information, Communication and Signal Processing, ICICS*, pp. 981–985, 2005.

[120] P. Tippet. The Kinetics of computer viruses replication: A theory and preliminary survey. *Safe Computing: Proceedings of the Fourth Anual Computer Viruses and Security Conference, New York*. pp. 66–87, 1991.

[121] E. Trélat. Optimal Control and Applications to Aerospace: Some Results and Challenges. *Journal of Optimization Theory and Applications*, 154(3):713–758, 2012.

[122] R. Vanderbei and D. Shanno. An Interior-Point Algorithm for Nonconvex Nonlinear Programming. *Computational Optimization and Applications*, 13(1–3):231–252, 1999.

[123] A. Wächter and L. Biegler. On the implementation of an interior-point filter line-search algorithm for large-scale nonlinear programming. *Mathematical Programming Series A and B*, 106(1):25–57, 2006.

[124] L. Wang and B. Wood. An epidemiological approach to model the viral propagation of memes. *Applied Mathematical Modelling*, 35(11):5442–5447, 2011.

[125] G. Witten and G. Poulter. Simulations of infectious diseases on networks. *Computers in Biology and Medicine*, 37(2):195–205, 2007.

[126] M. Woerndl, S. Papagiannidis, M. Bourlakis and F. Li. Internet-induced marketing techniques: Critical factors in viral marketing campaigns. *Int. Journal of Business Science and Applied Management*, 3(1):33–45, 2008.

[127] World Health Organization (WHO), webpage: `http://www.who.int/topics/infectious_diseases/en/`. Accessed on April 2017.

[128] Y. Wu, P. Li, L. Yang, X. Yang and Y. Tang. A theoretical method for assessing disruptive computer viruses. *Physica A: Statistical Mechanics and its Applications*, 482:325–336, 2017.

[129] C. Xu. Global threshold dynamics of a stochastic differential equation SIS model. *Journal of Mathematical Analysis and Applications*, 447(2):736–757, 2017.

[130] D. Xu, X. Xu, Y. Xie and C. Yang. Optimal control of an SIVRS epidemic spreading model with virus variation based on complex networks. *Commun. Nonlinear Sci. Numer. Simulat.*, 48:200–210, 2017.

[131] P. Yan and S. Liu. SEIR epidemic model with delay. *J. Aust. Math. Soc. Ser. B*, 48(1):119–134, 2006.

[132] L. Yang, X. Yang, J. Liu, Q. Zhu and C. Gan. Epidemics of computer viruses: A complex-network approach. *Applied Mathematics and Computation*, 219(16):8705–8717, 2013.

[133] C. Zhang and H. Huang. Optimal control strategy for a novel computer virus propagation model on scale-free networks. *Physica A: Statistical Mechanics and its Applications*, 451:251–265, 2016.

[134] X. Zhang, D. Jiang, T. Hayat and B. Ahmad. Dynamics of a stochastic SIS model with double epidemic diseases driven by Lévy jumps. *Physica A: Statistical Mechanics and its Applications*, 471:767–777, 2017.

[135] S. Zhang and X. Xu. Dynamic analysis and optimal control for a model of hepatitis C with treatment. *Commun. Nonlinear Sci. Numer. Simulat.*, 46:14–25, 2017.

[136] Y. Zhou, Y. Liang and J. Wu. An optimal strategy for HIV multitherapy. *Journal of Computational and Applied Mathematics*, 263:326–337, 2014.

[137] Y. Zhou, S. Yuan and D. Zhao. Threshold behavior of a stochastic SIS model with Lévy jumps. *Applied Mathematics and Computation*, 275:255–267, 2016.