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Bachelor's Degree in Mathematics

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# DYNAMICS AND MODELLING OF INFECTIOUS DISEASES

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**Abstract**

We present an introduction to basic epidemiological models for the spread of infectious diseases, emphasising the role of the basic reproductive number $R_0$ and giving a detailed description of the dynamics. The models that we discuss are the so-called Susceptible-Infectious ($SI$), the Susceptible-Infectious-Susceptible ($SIS$), the epidemic and endemic Susceptible-Infectious-Recovered ($SIR$) and the Susceptible-Infectious-Treated-Recovered ($SITR$). At the end of the thesis we present a discussion of some recent extensions of these basic models aimed at describing the evolution of the Covid pandemic in its earlier stages.

# Contents

Remark: throughout this work, we will use the following shorthands and notation:

- "s.t." for "such that";
- $\forall$ for "for all";;
- $\exists$ for "exists";;
- "TFAE"; for "the following are equivalent";;

Furthermore, the symbol "log"; will stand for the natural logarithm (base $e$).

Table 1: Description of state variables of compartmental models of infectious disease dynamics presented in Section 3.

| | |
|---|---|
| $S$ | Susceptible Individuals |
| $I$ | Infectious Individuals |
| $R$ | Recovered Individuals |
| $T$ | Treated Individuals |

Table 2: Description of parameter values in compartmental models of infectious disease dynamics presented in Section 3, with $t$ denoting time.

| **Parameters** | **Interpretation** | **Units** |
|---|---|---|
| $\beta$ | Per-capita contact rate | $t^{-1}$ |
| $\gamma$ | Per-capita recovery rate of the infected individuals | $t^{-1}$ |
| $1/\gamma$ | Duration of the infectious period | $t$ |
| $\mu$ | Per-capita birth/death rate | $t^{-1}$ |
| $1/\mu$ | Mean lifetime | $t$ |
| $\eta$ | Per-capita recovery rate of treated individuals | $t^{-1}$ |
| $\alpha$ | Fraction of infected individuals for unit time selected for treatment | $t^{-1}$ |
| $\delta$ | Fraction at which the treatment reduces infectivity | |

Table 3: Basic reproductive number, $R_0$, for the different compartmental models considered in Section 3.

| **Model** | $R_0$ |
|---|---|
| $SIS$ | $\beta/\gamma$ |
| epidemic $SIR$ | $\beta/\gamma$ |
| endemic $SIR$ | $\beta/(\gamma+\mu)$ |
| $SITR$ | $\frac{\beta}{\alpha+\gamma}\left(1+\frac{\alpha\delta}{\eta}\right)$ |

# 1 Introduction

The recent Covid19 pandemic has led to a revived interest in infectious diseases. As in the past, mathematical models have become again important tools in analysing the spread and control of infectious diseases. The model formulation process clarifies assumptions, variables, and parameters; moreover, models provide conceptual results such as thresholds, basic reproduction numbers, contact numbers, and replacement numbers. The models that we present in this work describe both epidemics, which are sudden outbreaks of a disease, and endemic situations, in which a disease is always present.

Mathematical modelling in epidemiology provides understanding of the underlying mechanisms that influence the spread of the disease and it suggests control strategies. In fact, models often identify behaviours that are unclear in experimental data, often because data are non-reproducible and the number of data points is limited and subject to errors in measurement. For example, one of the fundamental results in mathematical epidemiology is that most mathematical epidemic models usually exhibit "threshold" behaviour, which is to say that if the average number of secondary infections caused by an infectious individual is less than one, a disease will die out, while if it exceeds one there will be an epidemic. This broad principle, consistent with observations and quantified via epidemiological models, has been used routinely to estimate the effectiveness of vaccination policies and the likelihood that a disease may be eliminated or eradicated. Hence, even if it is not possible to verify hypotheses accurately, agreement with hypotheses of a qualitative nature is often valuable [BCC12].

Although a model for smallpox was formulated and solved in 1760 by Daniel Bernoulli (a member of a famous family of mathematicians who had been trained as a physician) in order to evaluate the effectiveness of variolation of healthy people with the smallpox virus, deterministic epidemiology modelling seems to have started in the 20th century. In 1906 W.H. Hamer formulated and analysed a discrete time model in his attempt to understand the recurrence of measles epidemics. His model may have been the first to assume that the incidence (number of new cases per unit time) depends on the product of the densities of the susceptible and infectious individuals. Subsequently, Sir R.A. Ross was interested in the incidence and control of malaria, so he developed differential equation models for malaria as a host-vector disease in 1911. Starting in 1926 W.O. Kermack and A.G. McKendrick published papers on epidemic models and obtained the epidemic threshold result that the density of susceptible individuals must exceed a critical value in order for an epidemic outbreak to occur. Mathematical epidemiology seems to have grown exponentially starting in the middle of the 20th century, so that a tremendous variety of models have now been formulated, mathematically analysed, and applied to infectious diseases [Het00].

Therefore, acknowledged the importance of the matter, the aim of this thesis is to present some basic epidemiological models and to include a couple of extensions of these basic models aiming at describing the evolution of the Covid pandemic.

Aside from some familiarity with general ODE's theory, no previous knowledge of dynamical systems is required. However, since a lot of preliminary results are recalled through the whole dissertation, the entire Section 2 has been devoted to present them in a plain way. Firstly, we will recall the basic definitions and properties of dynamical systems and we will present a result concerning the property of positively invariance of bounded regions with smooth boundary. Afterwards, we will elaborate on equilibrium points and their stability and we will state the theorems that we will need in the following sections.

In Section 3, we introduce and study some classical deterministic compartmental models. In particular, we will study the Susceptible-Infectious ($SI$), the Susceptible-Infectious-Susceptible ($SIS$), the epidemic Susceptible-Infectious-Recovered ($SIR$), the endemic Susceptible-Infectious-Recovered ($SIR$) and the Susceptible-Infectious-Treated-Recovered ($SITR$).

Lastly, in Section 4 we present a discussion of two recent extensions of these basic models aiming at describing the evolution of the Covid pandemic [Gio+20; LR21]. These were published in specialized journals during the early stages of the pandemic. The first article [Gio+20] uses the Susceptible-Infectious-Diagnosed-Healed-Ailing-Recognized-Threatened-Extinct ($SIDARTHE$) model, whereas the second study [LR21] introduces a modified Susceptible-Exposed-Infectious-Recovered ($SEIR$) model.

For this thesis we have relied mainly upon three sources: the book on dynamical systems by Lawrence Perko [Per01], the chapter by Fred Brauer and Carlos Castillo-Chavez about epidemic models [BCC12] and the review paper on the mathematics of infectious diseases by Herbert W. Hethcote [Het00]. In particular, [Per01] has been consulted for Section 2, whereas [BCC12] and [Het00] were our main sources for Section 3.

Finally, we mention what seem to be two minor original contributions in this thesis. The first is identifying regions of parameters for which the stable endemic equilibrium in the endemic $SIR$ model is a focus or a node (Proposition 3.15). The second is to complete the rigorous proof of some results from [BCC12] by taking care of a technical detail that was missing in their analysis (see Remark A.6).

# 2 Preliminaries of continuous time dynamical systems

For the sake of completeness, we include in this section most of the standard definitions and theorems from the theory of ordinary differential equations (ODE's) needed in subsequent sections. Proofs for nearly all of these results can be found in [Per01] and [HSD12].

## 2.1 Basic definitions and properties

**Definition 2.1.** An **(autonomous) ordinary differential equation (ODE)** is a differential equation of the type

$$\dot{x} = f(x),$$

where $x \in \mathbb{R}^n$ and $f : \mathbb{R}^n \to \mathbb{R}^n$ is a vector field of class $C^1$.

The word "autonomous" emphasises the property that $f$ does not depend explicitly on the independent variable $t$.

**Definition 2.2.** Let $\dot{x} = f(x),\ x \in \mathbb{R}^n$, be a differential equation. If $x_0 \in \mathbb{R}^n$ then we denote by $\Phi_t(x_0)$ the unique solution to the initial value problem

$$\begin{cases} \dot{x} = f(x), \\ x(0) = x_0. \end{cases}$$

The function

$$\begin{aligned} \Phi : \mathbb{R} \times \mathbb{R}^n &\to \mathbb{R}^n, \\ (t, x) &\mapsto \Phi_t(x), \end{aligned}$$

is called the **flow** of the differential equation[1].

**Proposition 2.3** (Properties of the flow)**.**

$$\begin{aligned} \Phi_0(x) &= x && \forall x \in \mathbb{R}^n, \\ \Phi_t(\Phi_s(x)) &= \Phi_{t+s}(x) && \forall x \in \mathbb{R}^n, \forall s, t \in \mathbb{R} \end{aligned}$$

Standard theorems of ODE's guarantee that the map $\Phi_t : \mathbb{R}^n \to \mathbb{R}^n$, $x \mapsto \Phi_t(x)$, is smooth and invertible, with smooth inverse for all $t \in \mathbb{R}$.

[1] To simplify the exposition we assume that $\Phi_t(x)$ is defined for all $t \in \mathbb{R}$. Such property might not hold for a general vector field $f$, but one may always reparametrise $t$ such that this property holds, see e.g. [Per01, Theorem 1 in Section 3.1]

**Definition 2.4.** The set $\Omega \subset \mathbb{R}^n$ is said to be **invariant** if $\forall x \in \Omega$ it holds that $\Phi_t(x) \in \Omega \ \forall t \in \mathbb{R}$.

**Definition 2.5.** The set $\Omega \subset \mathbb{R}^n$ is said to be **positively invariant** if $\forall x \in \Omega$ it holds that $\Phi_t(x) \in \Omega \ \forall t \geq 0$.

**Definition 2.6.** A function $h : \mathbb{R}^n \to \mathbb{R}$ is a **first integral** if $h(\Phi_t(x)) = h(x)$ $\forall x \in \mathbb{R}^n$, $\forall t \in \mathbb{R}$.

**Proposition 2.7.** *1. Level sets of first integrals are invariant.*

*2. If $h$ is differentiable, then it is a first integral if and only if $\nabla h(x) \cdot f(x) = 0$ $\forall x \in \mathbb{R}^n$.*

The following result is used at several points of this work and for this reason we present a proof.

**Lemma 2.8.** *Let $\Omega \subset \mathbb{R}^n$ be a bounded region with smooth boundary. Let $n(x)$ denote the outward unit vector of $\Omega$ at $x \in \partial\Omega$. If $f(x) \cdot n(x) \leq 0$, $\forall x \in \partial\Omega$, then $\Omega$ is positively invariant.*

*Proof.* Let's take $x_0 \in \Omega$. Suppose $\Phi_{t_0}(x_0) \notin \Omega$ for some $t_0 > 0$. Then there exists $t_1 > 0$ such that $\Phi_{t_1}(x_0) \in \partial\Omega$ and

$$\left.\frac{d}{dt}\right|_{t=t_1} \Phi_t(x_0) \cdot n(\Phi_t(x_0)) > 0.$$

However

$$\frac{d}{dt}\Phi_t(x_0) \cdot n(\Phi_t(x_0)) = f(\Phi_t(x_0)) \cdot n(\Phi_t(x_0)).$$

Evaluating at $t = t_1$, we find

$$f(\Phi_{t_1}(x_0)) \cdot n(\Phi_{t_1}(x_0)) > 0,$$

which is a contradiction. □

## 2.2 Equilibrium points and their stability

**Definition 2.9.** Let $\dot{x} = f(x)$, $x \in \mathbb{R}^n$ be a differential equation. A point $x_0 \in \mathbb{R}^n$ is said to be an **equilibrium point** for the ODE if $\Phi_t(x_0) = x_0 \ \forall t \in \mathbb{R}$.

**Lemma 2.10.** *A point $x_0 \in \mathbb{R}^n$ is an equilibrium point if and only if $f(x_0) = 0$.*

**Definition 2.11.** The equilibrium point $x_0 \in \mathbb{R}^n$ is said to be

1. **stable** if $\forall \epsilon > 0$ there exists $\delta > 0$ such that if $x \in B_\delta(x_0)$ then $\Phi_t(x) \in B_\epsilon(x_0)$ $\forall t \geq 0$;

2. **unstable** if it is not stable;

3. **asymptotically stable** if $x_0$ is stable and $\exists \epsilon > 0$ such that if $x \in B_\epsilon(x_0)$ then
$$\lim_{t \to \infty} \Phi_t(x) = x_0.$$

**Theorem 2.12** (Lyapunov's Spectral Theorem)**.** *Let $x_0 \in \mathbb{R}^n$ be an equilibrium point and let $A = Df(x_0)$ be the Jacobian matrix of $f$ at $x_0$. Then*

*(i) If all eigenvalues of $A$ have negative real part, then $x_0$ is asymptotically stable;*

*(ii) If $A$ has at least one eigenvalue with positive real part then $x_0$ is unstable.*

**Definition 2.13.** An equilibrium point $x_0$ is called **hyperbolic** if all eigenvalues of the matrix $A = Df(x_0)$ have non-zero real part.

As a consequence of Theorem 2.12, the stability of the hyperbolic equilibrium point $x_0$ can be determined by the linearization matrix $A = Df(x_0)$.

## 2.3 Equilibrium points of two-dimensional systems

We now specialize our discussion to differential equations on $\mathbb{R}^2$.

**Definition 2.14.** An equilibrium point $x_0$ is called:

1. a **node** when both eigenvalues of the matrix $A = Df(x_0)$ are real and of the same sign. The node is asymptotically stable when the eigenvalues are negative and unstable when they are positive.

2. a **focus** when the eigenvalues of the matrix $A = Df(x_0)$ are complex-conjugate with non-zero real part. The focus is asymptotically stable when the eigenvalues have negative real part and unstable when they have positive real part.

3. a **center** when the eigenvalues of the matrix $A = Df(x_0)$ are complex-conjugate and have zero real part.

4. a **saddle point** if the eigenvalues of the matrix $A = Df(x_0)$ are real and of opposite signs; the saddle is always unstable.

**Remark 2.15.** Note that nodes, focuses and saddle points are hyperbolic. As a consequence their stability can be determined by their linearization matrix $A = Df(x_0)$. On the other hand, the stability type of a center, or any other equilibrium point that is not hyperbolic, in general depends on the higher order terms in the Taylor expansion of $f$ around $x_0$ and cannot be deduced from the matrix $A = Df(x_0)$.

**Theorem 2.16.** *Let $\Delta$ be the determinant of the matrix $A$ and $\tau$ the trace of $A$, where $A = Df(x_0)$ and $x_0$ is an equilibrium point. The following statements hold:*

*(a) If $\Delta < 0$ then $x_0$ is a saddle point.*

*(b) If $\Delta > 0$ and $\tau^2 - 4\Delta \geq 0$ then $x_0$ is a node; it is asymptotically stable if $\tau < 0$ and unstable if $\tau > 0$.*

*(c) If $\Delta > 0$, $\tau^2 - 4\Delta < 0$ and $\tau \neq 0$, then $x_0$ is a focus; it is asymptotically stable if $\tau < 0$ and unstable if $\tau > 0$.*

*(d) If $\Delta > 0$ and $\tau = 0$ then $x_0$ is a center.*

The results of this theorem are illustrated by Figure 1.

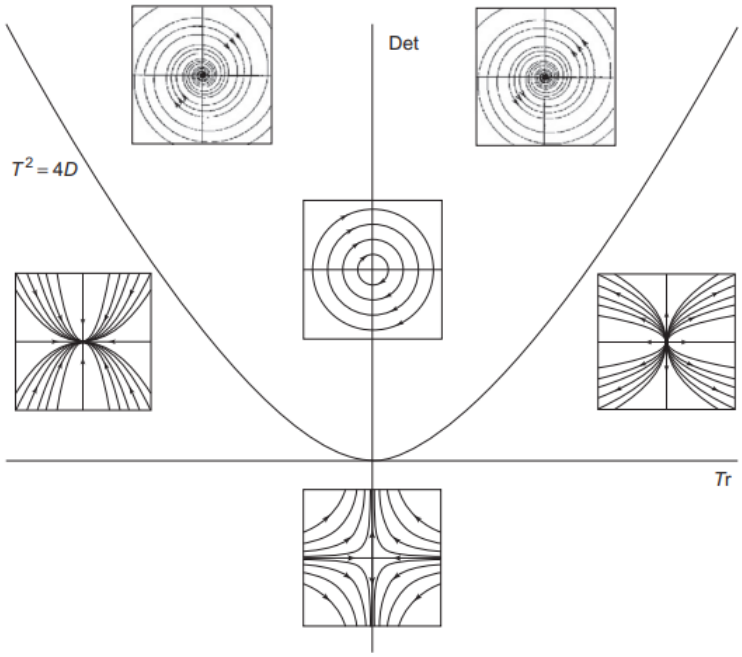



Figure 1: Geometric classification of $2 \times 2$ linear systems in the trace-determinant plane. The figure is taken from [HSD12].

# 3 Deterministic Compartmental Models

We include in this section an introduction to basic epidemiological models for the spread of infectious diseases. Nearly all of these results can be found in [Chi11], [BCC12], [Het00] and [Het89].

Deterministic compartmental models form the simplest models in the mathematical study of infectious disease dynamics. They assume that a population is homogenous, constant and divided in various compartments, where the only distinction is in their disease state. The number of compartments is different from model to model; in the models that we study, we will see four compartments: susceptible individuals $S$, infectious individuals $I$, recovered individuals $R$ and treated individuals $T$. The models that we discuss are the Susceptible-Infectious ($SI$), the Susceptible-Infectious-Susceptible ($SIS$), the epidemic Susceptible-Infectious-Recovered ($SIR$), the endemic Susceptible-Infectious-Recovered ($SIR$) and the Susceptible-Infectious-Treated-Recovered ($SITR$). Despite their simplicity, these models provide notation, concepts, intuition and foundation for considering more refined models.

During the analysis of these models, we will perform the following steps:

1. We introduce the model, describing the phase variables, parameters and hypothesis, and we write the equations describing evolution of the population in the different compartments.

2. We analytically solve the equations (if possible), we classify and determine the stability of equilibrium points and we investigate threshold conditions for the dynamics.

3. We illustrate the phase portrait dynamics indicating any bifurcations.

## 3.1 Susceptible-Infectious ($SI$) Model

The susceptible-infectious ($SI$) model represents diseases where there is no recovery, such as the human immunodeficiency virus (HIV). It therefore divides the population into two compartments: susceptible hosts, denoted by $S$, and infectious hosts, denoted by $I$. Individuals in the first group have not been infected yet and therefore can be infected, while individuals in the second group are already infected. If the total population size is denoted by $N$, i.e. $N = S + I$, we have that the proportion of individuals that is infectious is $I/N$. Introducing a constant $\beta$ to indicate the per-capita contact rate, we can derive the model below, which is schematically represented in Figure 2:

$$\frac{dS}{dt} = -\beta S\frac{I}{N}, \tag{1a}$$
$$\frac{dI}{dt} = \beta S\frac{I}{N}. \tag{1b}$$

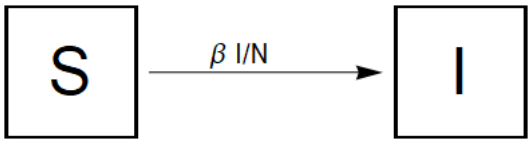



Figure 2: Schematic representation of the $SI$ model.

### 3.1.1 Conservation of total population and dimension reduction of the model

**Proposition 3.1.** *The total number of individuals, $N = S + I$, is a first integral of (1).*

*Proof.* From (1), we can define the vector field $f : \mathbb{R}^2 \to \mathbb{R}^2$ as

$$\begin{pmatrix} S' \\ I' \end{pmatrix} = f\begin{pmatrix} S \\ I \end{pmatrix} = \begin{pmatrix} -\beta S\frac{I}{N} \\ \\ \beta S\frac{I}{N} \end{pmatrix}$$

and the function $h : \mathbb{R}^2 \to \mathbb{R}$ as

$$h\begin{pmatrix} S \\ I \end{pmatrix} = N = I + S.$$

We can evaluate

$$\nabla h\begin{pmatrix} S \\ I \end{pmatrix} = \nabla N = \begin{pmatrix} \dfrac{\partial N}{\partial S} \\ \\ \dfrac{\partial N}{\partial I} \end{pmatrix} = \begin{pmatrix} 1 \\ 1 \end{pmatrix}$$

and observe that

$$\nabla h \cdot \begin{pmatrix} -\beta S\frac{I}{N} \\ \\ \beta S\frac{I}{N} \end{pmatrix} = -\beta S\frac{I}{N} + \beta S\frac{I}{N} = 0.$$

As a consequence, in view of Proposition 2.7, we can assert that $N$ is a first integral. □

Thus we can fix the value of $N$ and reduce the system to one dimension. This corresponds to the substitution $S = N - I$ in (1b) to provide the equation:

$$\frac{dI}{dt} = \beta(N - I)\frac{I}{N}. \tag{2}$$

Since we are interested in solutions for which the number $S$ of susceptible individuals and $I$ of infectious individuals are positive, we consider, for fixed $N \in \mathbb{R}^+$, the domain $\Omega = \{I \in \mathbb{R} : 0 \leq I \leq N\}$.

**Proposition 3.2.** *The domain $\Omega$ is positively invariant under the flow of (2).*

We can give a proof of this result using Lemma 2.8 but in this case we will find the flow explicitly and the proof will follow from there.

### 3.1.2 Analysis of the reduced model

Equation (2) is a separable first-order ODE. For $I \neq 0$ and $I \neq N$ we have

$$\frac{N}{I(N - I)}dI = \beta dt.$$

Integrating gives:

$$\log\left|\frac{N}{I(N - I)}\right| = \beta t + C.$$

Considering the initial condition $I(0) = I_0$, we can solve for $C$ to find:

$$I(t) = \frac{I_0 N}{(N - I_0)e^{-\beta t} + I_0}.$$

Furthermore, we can observe that $I = 0$ and $I = N$ are equilibrium solutions of (2). These are the only equilibrium points of (2), that we will call the disease-free equilibrium point

$$I_{df} = 0,$$

and the endemic equilibrium point

$$I_e = N,$$

in which all the individuals are infected. Therefore the flow of (2) is given by

$$\Phi_t(I) = \begin{cases} \frac{IN}{(N-I)e^{\beta t}+I} & \text{if } I \neq 0, N \\ 0 & \text{if } I = 0 \\ N & \text{if } I = N. \end{cases}$$

In particular, we see that if $I \in [0, N]$ then $\Phi_t(I) \in [0, N]$ for all $t \in \mathbb{R}$, thus proving Proposition 3.2.

We can analyse the stability of the equilibrium points by differentiating

$$g(I) := \beta(N - I)\frac{I}{N}.$$

We have

$$g'(I) = \frac{\beta}{N}(N - 2I).$$

Evaluating $g'$ at $I_{df}$ and $I_e$, we find

$$g'(I_{df}) = \beta > 0, \qquad g'(I_e) = -\beta < 0.$$

It follows from Theorem 2.12 that $I_e$ is unstable, whereas $I_{df}$ is asymptotically stable. These conclusions can also be verified with the explicit expression for $\Phi_t$.

From this analysis we deduce that as long as there is at least one infectious individual, the population will tend towards becoming fully infectious. The dynamics is illustrated in the figures below. Figure 3 illustrates the phase portrait where we observe that the population tends towards becoming fully infectious. On the other hand, Figure 4 shows the plot of $I(t)$ and $S(t)$[2] as functions of time and we also observe that the whole population becomes infected as $t$ grows.

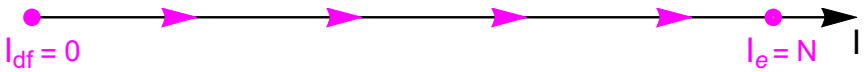



Figure 3: A phase portrait of the reduced $SI$ model (2).

[2]Note that $S(t) = N - I(t)$.

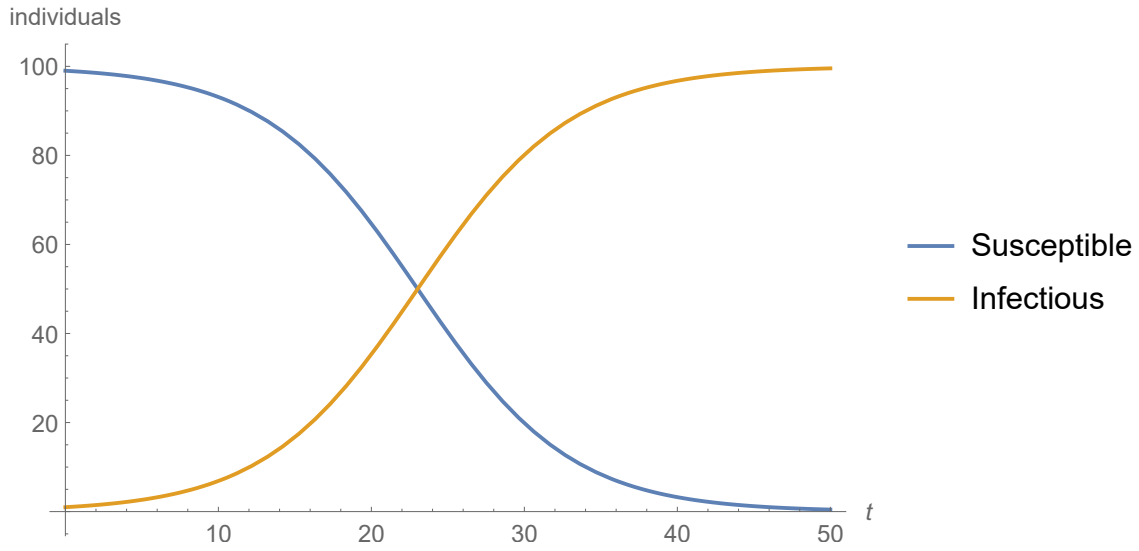



Figure 4: A graph of the explicit solutions $I(t)$ and $S(t)$ of the $SI$ model with $\beta = 1/5$ and initial conditions $I_0 = 1$, $S_0 = 99$.

## 3.2 Susceptible-Infectious-Susceptible ($SIS$) Model

Unlike the $SI$ model, the susceptible-infectious-susceptible ($SIS$) model represents diseases where individuals can recover from the infection and become susceptible again, such as the common cold. Infectious individuals recover at a constant per capita rate, $\gamma$, so $1/\gamma$ is the duration of the infectious period. We can derive the following model:

$$\frac{dS}{dt} = -\beta S\frac{I}{N} + \gamma I, \tag{3a}$$

$$\frac{dI}{dt} = \beta S\frac{I}{N} - \gamma I, \tag{3b}$$

where, as before, $N = S + I$ is the total number of individuals. The model is schematically illustrated in Figure 5.

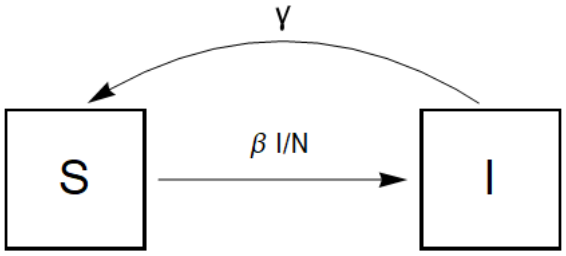



Figure 5: Schematic representation of the $SIS$ model.

### 3.2.1 Conservation of total population and dimension reduction of the model

**Proposition 3.3.** *The total number of individuals, $N = S + I$, is a first integral of (3).*

*Proof.* From (3), we can define the vector field $f : \mathbb{R}^2 \to \mathbb{R}^2$ as

$$\begin{pmatrix} S' \\ I' \end{pmatrix} = f\begin{pmatrix} S \\ I \end{pmatrix} = \begin{pmatrix} -\beta S\frac{I}{N} + \gamma I \\ \\ \beta S\frac{I}{N} - \gamma I \end{pmatrix}$$

and the function $h : \mathbb{R}^2 \to \mathbb{R}$ as

$$h\begin{pmatrix} S \\ I \end{pmatrix} = N = I + S.$$

Simple calculations, similar to the ones in the proof of Proposition 3.1, show that $\nabla h(S, I) \cdot f(S, I) = 0$, so $N$ is a first integral. □

Thus we can fix the value of $N$ and reduce the system to one dimension. This corresponds to the substitution $S = N - I$ in (3b) to provide the equation:

$$\frac{dI}{dt} = \beta(N - I)\frac{I}{N} - \gamma I. \tag{4}$$

Since the total number of individuals remains constant and we are interested in solutions for which the number $S$ of susceptible individuals and $I$ of infectious individuals are positive, we again consider, for fixed $N \in \mathbb{R}^+$, the domain $\Omega = \{I \in \mathbb{R} : 0 \leq I \leq N\}$.

**Proposition 3.4.** *The region $\Omega$ is positively invariant under the flow of (4).*

We can give a proof of this result using Lemma 2.8 but, as for the $SI$ model, we will find the flow explicitly and the proof will follow from there.

### 3.2.2 Analysis of the reduced model

Considering the initial condition $I(0) = I_0$ and proceeding as in Section 3.1.2, we can solve (4) to find:

$$I(t) = \frac{\frac{N}{\beta}(\beta - \gamma)}{\left(\frac{N}{\beta}\frac{(\beta-\gamma)}{I_0} - 1\right)e^{-(\beta-\gamma)t} + 1}.$$

Furthermore, we can observe that $I = 0$ and $I = \frac{N}{\beta}(\beta - \gamma)$ are the only equilibrium solutions of (4). Then (4) has two fixed points, that we will call the disease-free and the endemic equilibrium point, given by

$$I_{df} := 0 \qquad\qquad I_e := \frac{N}{\beta}(\beta - \gamma).$$

Therefore the flow of (4) is given by

$$\Phi_t(I) = \begin{cases} \frac{\frac{N}{\beta}(\beta-\gamma)}{\left(\frac{N}{\beta}\frac{(\beta-\gamma)}{I_0}-1\right)e^{-(\beta-\gamma)t}+1} & \text{if } I \neq 0, \frac{N}{\beta}(\beta - \gamma), \\ 0 & \text{if } I = 0, \\ \frac{N}{\beta}(\beta - \gamma) & \text{if } I = \frac{N}{\beta}(\beta - \gamma). \end{cases}$$

In particular, we see that if $I \in [0, N]$ then $\Phi_t(I) \in [0, N]$ for all $t \in \mathbb{R}$, thus proving Proposition 3.4.

We can analyse the stability of the equilibrium points by differentiating

$$g(I) = \beta(N - I)\frac{I}{N} - \gamma I.$$

We find

$$g'(I_{df}) = \beta - \gamma, \qquad g'(I_e) = -\beta + \gamma.$$

Therefore $I_{df}$ is unstable if $\beta > \gamma$ and asymptotically stable if $\beta < \gamma$. On the other hand, $I_e$ is asymptotically stable if $\beta > \gamma$ and unstable if $\beta < \gamma$.

Our analysis shows that the behaviour of the system is determined by the relative sizes of $\beta$ and $\gamma$. The ratio $\frac{\beta}{\gamma}$ is an essential parameter in epidemiology, called the **basic reproductive number,** $R_0$, and we discuss its interpretation in the following subsection.

### 3.2.3 The basic reproductive number $R_0$

Since $\frac{1}{\gamma}$ is the average duration of the infectious period, the product, $\frac{\beta}{\gamma}$ , is the expected number of new infections from one infected individual in a fully susceptible population through the entire duration of the infectious period. We denote this quantity as the **basic reproductive number,** $R_0$. We may therefore interpret $R_0$ as:

$$R_0 = \begin{pmatrix} \text{Per-capita} \\ \text{contact rate} \end{pmatrix} \begin{pmatrix} \text{Duration of} \\ \text{infection} \end{pmatrix}.$$

From the analysis in Section 3.2.2 we see that if $R_0 < 1$ the disease-free equilibrium point is asymptotically stable and the endemic equilibrium point does not belong to $\Omega$, so the infection in one individual cannot replace itself and the pathogen dies out. On the other hand, if $R_0 > 1$ the disease-free equilibrium point is unstable and the endemic equilibrium is asymptotically stable, so an introduced infectious individual leads to more than one infection, the number of infectious individuals increases and the pathogen spreads in the population until a saturation level is attained at $I = I_e$.

**Remark 3.5.** Actually, one may show that the system (3) undergoes a "transcritical bifurcation" at $R_0 = 1$, illustrated in Figure 6.

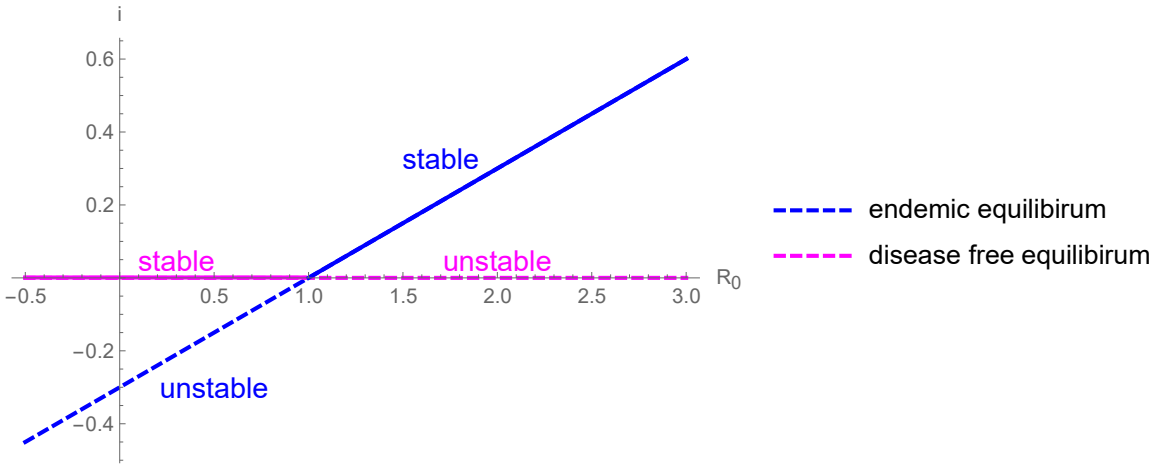
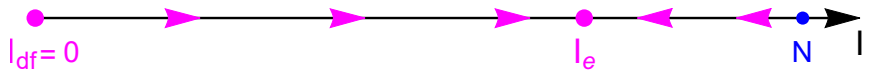
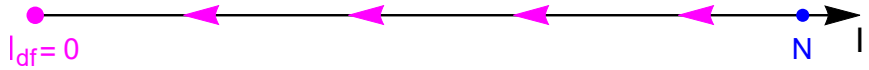



Figure 6: The bifurcation diagram for the endemic $SIS$ model, which shows that the disease-free and endemic equilibria exchange stability when $R_0 = 1$.

The dynamics of the reduced $SIS$ model (4) is illustrated in Figure 7 and of the full $SIS$ mode (3) in Figure 8. Figure 8 shows the plot of $I(t)$ and $S(t)$[3] as functions of time.



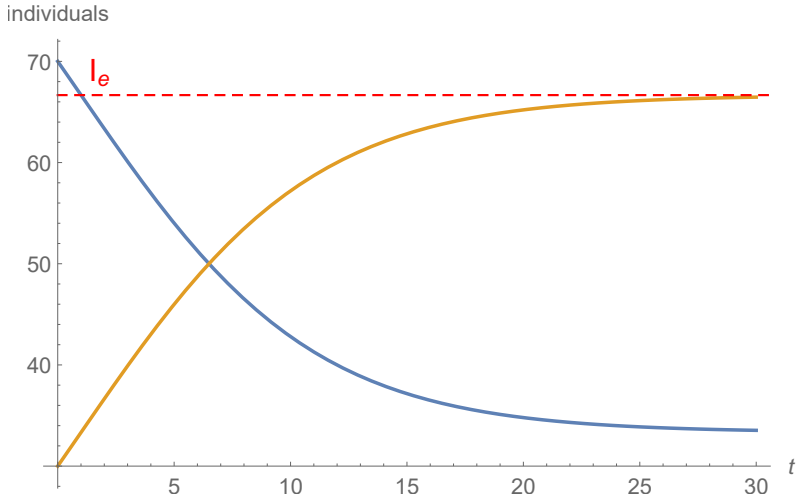

(a) $R_0 > 1$



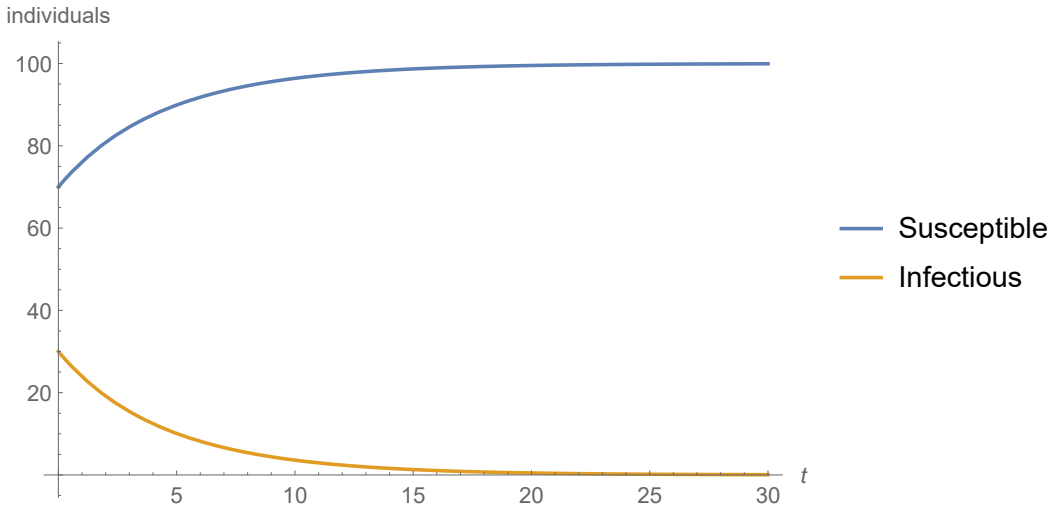

(b) $R_0 < 1$

Figure 7: A phase portrait of the reduced $SIS$ model in the cases $R_0 > 1$ and $R_0 < 1$.

[3] As in the $SI$ model, we have $S(t) = N - I(t)$.




(a) $R_0 > 1$




(b) $R_0 < 1$

Figure 8: Graphs of the explicit solutions $I(t)$ and $S(t)$ of the $SIS$ model, with initial conditions $I_0 = 30$, $S_0 = 70$. In Figure 8a the parameters are $\beta = 0.3$ and $\gamma = 0.1$, so $R_0 > 1$. In this case, the value of $I_e$=66.67. On the other hand, in Figure 8b the parameters are $\beta = 0.1$ and $\gamma = 0.3$, so $R_0 < 1$.

## 3.3 Epidemic Susceptible-Infectious-Recovered ($SIR$) Model

Unlike the $SIS$ model, the susceptible-infectious-recovered ($SIR$) model represents diseases where individuals that recover from the infection have lifelong immunity, such as mumps, measles and rubella. Like in the $SIS$ model, infectious individuals recover at a constant per capita rate, $\gamma$, so $1/\gamma$ is the duration of the infectious period. This time however, the recovered individuals form a new compartment $R$. The model is schematically represented in Figure 9 and the corresponding equations of motion are:

$$\frac{dS}{dt} = -\beta S\frac{I}{N}, \tag{5a}$$

$$\frac{dI}{dt} = \beta S\frac{I}{N} - \gamma I, \tag{5b}$$

$$\frac{dR}{dt} = \gamma I, \tag{5c}$$

where $N = S + I + R$ is the total number of individuals.


Figure 9: Schematic representation of the $SIR$ model.

### 3.3.1 Conservation of total population and dimension reduction of the model

**Proposition 3.6.** *The total number of individuals, $N = S + I + R$, is a first integral of (5).*

The proof is similar to the analogous results for the $SI$ and $SIS$ models presented before.

Since the total number of individuals, $N$, is a first integral, we can fix the value of $N$ and reduce the system to two dimensions. This corresponds to the analysis of (5a) and (5b) independently of (5c), treating $N$ as a constant. We also normalize the variables by the population size, $N$, defining

$$s := \frac{S}{N}, \quad i := \frac{I}{N}, \quad r := 1 - s - i.$$

We obtain the reduced system

$$\frac{ds}{dt} = -\beta si, \tag{6a}$$

$$\frac{di}{dt} = \beta si - \gamma i. \tag{6b}$$

### 3.3.2 Positively invariant phase space

Since we are interested in solutions for which the number $S$ of susceptible individuals, $I$ of infectious individuals and $R$ of recovered individuals are positive, we consider the domain

$$\Omega = \{(s, i) \in \mathbb{R}^+ \times \mathbb{R}^+ : 0 \leq i + s \leq 1\} \subset \mathbb{R}^2.$$

**Proposition 3.7.** *The region $\Omega$ is positively invariant under the flow of (6).*

*Proof.* Note that $\partial\Omega = L_1 \cup L_2 \cup L_3$, where

$$\begin{aligned} L_1 &= \{(s, 0) : 0 \leq s \leq 1\}, \\ L_2 &= \{(s, 1 - s) : 0 \leq s \leq 1\}, \\ L_3 &= \{(0, i) : 0 \leq i \leq 1\}. \end{aligned}$$

The outward normal vector to $\Omega$ along $L_j$ is $n_j$, with

$$n_1 = (0, -1), \quad n_2 = (1, 1), \quad n_3 = (-1, 0).$$

Denote by $f : \mathbb{R}^2 \to \mathbb{R}^2$ the vector field defined by (6), i.e.

$$f(s, i) = \begin{pmatrix} -\beta si \\ \beta si - \gamma i \end{pmatrix}. \tag{7}$$

Then it is straightforward to check that if $(s_0, i_0) \in L_j$, then $f(s_0, i_0) \cdot n_j \leq 0$. We may now apply a generalization of Lemma 2.8 to show that $\Omega$ is positively invariant (see Remark 3.8 below). □

**Remark 3.8.** The proof of Proposition 3.6 requires a slight generalization of Lemma 2.8, since $\partial\Omega$ is not smooth but has "corners" at $L_i \cap L_j$, $i \neq j$. These corner points present no difficulties since if $(s_0, i_0) \in L_i \cap L_j$, then both inequalities $f(s_0, i_0) \cdot n_i \leq 0$ and $f(s_0, i_0) \cdot n_j \leq 0$ hold.

### 3.3.3 Analysis of the reduced model I: equilibrium points and their stability

Unlike the previous models, the $SIR$ model has no explicit analytical solution. However, we present an analysis that will allow us to determine the basic features of the dynamics.

It is easy to see that the only equilibrium points of (6) are of the form $(s, i) = (\xi, 0)$ for any $\xi \in [0, 1]$. They all are disease free equilibria.

The Jacobian matrix of $f$ given by (7) is

$$J(s, i) = \begin{pmatrix} -\beta i & -\beta s \\ \beta i & \beta s - \gamma \end{pmatrix}.$$

Evaluating $J$ at $(s, i) = (\xi, 0)$, we have

$$J^* = \begin{pmatrix} 0 & -\beta\xi \\ 0 & \beta\xi - \gamma \end{pmatrix}.$$

The eigenvalues of $J^*$ are $\lambda_1 = 0$ and $\lambda_2 = \beta\xi - \gamma$. If $\xi > \frac{\gamma}{\beta}$, then Theorem 2.12 guarantees instability. On the other hand, the presence of a 0 eigenvalue implies that the equilibrium point is not hyperbolic and the linear stability analysis is inconclusive (we cannot apply Lyapunov's spectral Theorem 2.12). Nonetheless, using phase space analysis techniques (that we omit), one may show that the equilibrium point $(s, i) = (\xi, 0)$ is stable (but not asymptotically stable) if $\xi < \frac{\gamma}{\beta}$.

### 3.3.4 The basic reproductive number $R_0$ and the replacement number $R_e(t)$

Recall from our discussion in Section 3.2.3 that $R_0 = \frac{\beta}{\gamma}$. Firstly we consider the case $R_0 < 1$: the condition $\xi < \frac{1}{R_0}$ is satisfied for every $\xi \in [0, 1]$ and therefore the equilibrium points $(\xi, 0)$ are stable. On the other hand, if $R_0 > 1$, the equilibrium point $(s, i) = (\xi, 0)$ is stable if $\xi < \frac{1}{R_0}$ and unstable if $\xi > \frac{1}{R_0}$, and hence $R_0$ is again a threshold quantity for this model. Recall that the basic reproductive number $R_0$ is the expected number of new infections from one infected individual in a fully susceptible population through the entire duration of the infectious period. In the case the population is not fully susceptible, it is useful to consider a different quantity called the **replacement number,** $R_e(t)$, which is time dependent and defines the expected number of secondary infections that one infected person would produce through the entire duration of the infectious period. We may therefore interpret $R_e(t)$ as

$$R_e = \begin{pmatrix} \text{Probability of} \\ \text{disease transmission} \\ \text{per unit time} \end{pmatrix} \begin{pmatrix} \text{Duration of} \\ \text{infection} \end{pmatrix} \begin{pmatrix} \text{Proportion of} \\ \text{susceptible} \\ \text{population} \end{pmatrix}.$$

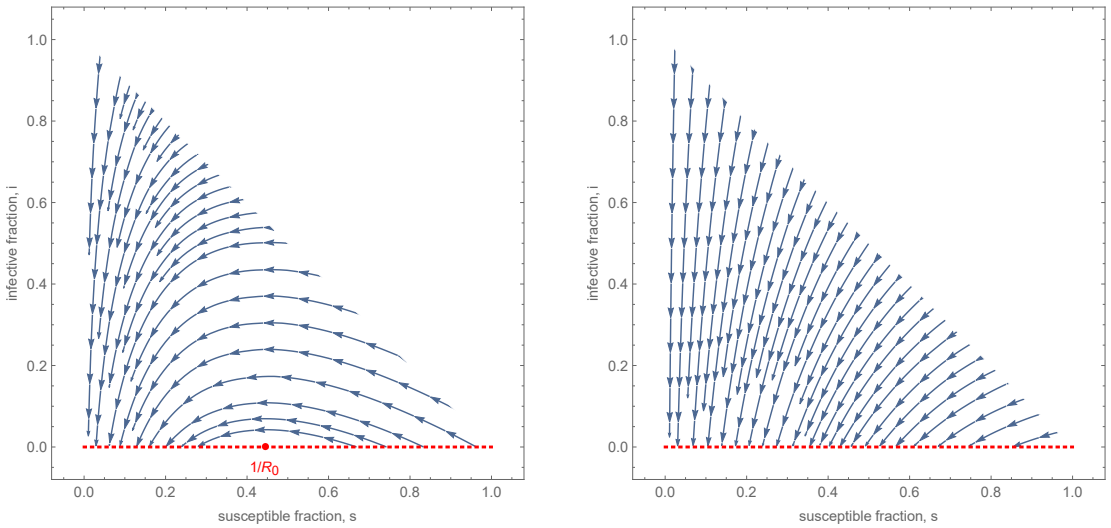
That is to say

$$R_e(t) = \frac{\beta}{\gamma} s(t) = R_0 s(t).$$

Note that (6b) may be rewritten as

$$\frac{di}{dt} = \gamma i(R_e(t) - 1)$$

and therefore if $R_e(t) < 1$ the number of infectious individuals decreases, whereas if $R_e(t) > 1$ the number of infectious individuals increases. The dynamics of the system is illustrated in Figure 10.




(a) $R_0 > 1$ (b) $R_0 < 1$

Figure 10: The phase portrait of the reduced $SIS$ model, in the region of interest $\Omega$. In Figure 10a the parameters are $\beta = 0.9$ and $\gamma = 0.3$, leading to $R_0 > 1$, while in Figure 10b the parameters are $\beta = 0.5$ and $\gamma = 0.6$, $R_0 < 1$. In 10a we see that the equilibrium points where $s < \frac{1}{R_0}$ are stable, the equilibrium points where $s > \frac{1}{R_0}$ are unstable and the maximum proportion of infectious individuals occurs when $s = \frac{1}{R_0}$. On the other hand, we see that $i$ is always decreasing in 10b.

### 3.3.5 Analysis of the reduced model II

**Theorem 3.9.** *Let $(s(t), i(t))$ be a solution of (6), with initial condition $(s_0, i_0) \in \Omega$. The following statements hold:*

*(a) The susceptible fraction $s(t)$ is a decreasing[4] function.*

*(b) If $R_0 s_0 < 1$, then $i(t)$ is decreasing.*

*(c) If $R_0 s_0 > 1$ and $i_0 > 0$, then $i(t)$ increases up to a maximum value*

$$i_{max} = i_0 + s_0 - \frac{1}{R_0} - \frac{\log(R_0 s_0)}{R_0}.$$

*(d) $\lim_{t\to\infty} i(t) = 0$ and $\lim_{t\to\infty} s(t) = s_\infty$ exists.*

*(e) The limiting value $s_\infty$ is characterised as the unique root in $\left(0, \frac{1}{R_0}\right)$ of the equation*

$$i_0 + s_0 - s_\infty + \frac{1}{R_0} \log\left(\frac{s_\infty}{s_0}\right) = 0.$$

Note that the qualitative behaviour predicted by the theorem is illustrated in Figure 10.

*Proof.* (a) Looking at (6a) on $\Omega$, we can observe that

$$\frac{ds}{dt} = -\beta s i \leq 0,$$

so $s(t)$ is decreasing.

(b) If $R_0 s_0 < 1$, then for $t \geq 0$, using (a) we have

$$\frac{di}{dt}(t) = \beta s(t) i(t) - \gamma i(t) \leq i(t)(\beta s_0 - \gamma) = -c i(t),$$

where $c := \gamma(1 - R_0 s_0) > 0$, so $i(t)$ is decreasing.

(c) With some calculations we find that:

$$\frac{di}{ds} = \frac{\frac{di}{dt}}{\frac{ds}{dt}} = \frac{\beta i s - \gamma i}{-\beta i s} = -1 + \frac{\gamma}{\beta s} = -1 + \frac{1}{R_0 s}.$$

[4] We use the concept of decreasing (increasing) with the meaning of non-increasing (non-decreasing) or equivalently weakly decreasing (weakly increasing).

The general solution to this differential equation is

$$i(s) = -s + \frac{1}{R_0}\log(s) + k, \tag{8}$$

where $k$ is a constant. Therefore, the solution $(s(t), i(t))$ is contained on the curve[5] $\gamma_k = \{(s,i) \in \Omega^\circ : i = -s + \frac{1}{R_0}\log(s) + k\}$ for an appropriate value of $k$. Such value may be determined by noting that $(s(t), i(t)) = (s_0, i_0)$ belongs to $\gamma_k$. Hence,

$$k = i_0 + s_0 - \frac{1}{R_0}\log(s_0).$$

The curve that contains the trajectory is thus the graph of the function

$$j(s) = -s + i_0 + s_0 + \frac{1}{R_0}\log\left(\frac{s}{s_0}\right),$$

for $s$ in an appropriate interval satisfying $j(s) \geq 0$. It is straightforward to show that $j$ achieves its maximum at $s = \frac{1}{R_0}$ with maximum value

$$j\left(\frac{1}{R_0}\right) = -\frac{1}{R_0} + i_0 + s_0 + \frac{1}{R_0}\log\left(\frac{1}{R_0 s_0}\right) = i_{max}.$$

(d) Let $g : \mathbb{R}^+ \to \mathbb{R}$ be given by $g(t) = s(t) + i(t)$ for $t \geq 0$. Then we have $g(t) \geq 0$ and

$$g'(t) = s'(t) + i'(t) = -\gamma i \leq 0.$$

These two conditions imply that $\lim_{t\to\infty} g(t)$ exists.

We now show that $g''$ is bounded. We have

$$g''(t) = -\gamma i(t) = -\gamma(\beta s(t) i(t) - \gamma i(t))$$

and hence

$$|g''(t)| \leq \gamma(\beta |s(t)||i(t)| - \gamma |i(t)|).$$

Considering that $(s(t), i(t)) \in \Omega\ \forall t \geq 0$, we have $|s(t)| \leq 1$, $|i(t)| \leq 1$, $\forall t \geq 0$. Therefore

$$|g''(t)| \leq \gamma(\beta + \gamma), \qquad \forall t \geq 0.$$

We may now apply Proposition A.1 from the Appendix to conclude that

$$0 = \lim_{t\to\infty} g'(t) = \lim_{t\to\infty} -\gamma i(t).$$

Therefore $\lim_{t\to\infty} i(t) = 0$ as claimed and, moreover, since $\lim_{t\to\infty} g(t) = \lim_{t\to\infty} s(t) + i(t)$ exists, then necessarily $\lim_{t\to\infty} s(t) =: s_\infty$ exists as well.

[5] In fact, the function $h : \Omega^\circ \to \mathbb{R}$ defined by $h(s,i) = i + s - \frac{1}{R_0}\log(s)$ is a first integral of (6).

(e) Consider the function

$$j(s) = -s + i_0 + s_0 + \frac{1}{R_0} \log\left(\frac{s}{s_0}\right),$$

introduced in (c). We know that $j(s(t)) = i(t)$, $\forall t \geq 0$. Letting $t \to \infty$ gives $j(s_\infty) = 0$ or equivalently

$$-s_\infty + i_0 + s_0 + \frac{1}{R_0} \log\left(\frac{s_\infty}{s_0}\right) = 0,$$

which proves the required condition.

□

As a direct consequence of Theorem 3.9 we have the following corollary that describes the behaviour of the original variables $S, I, R$.

**Corollary 3.10.** *Let $(S(t), I(t), R(t))$ be a solution of (5), with initial condition $S(0), I(0), R(0) \geq 0$ and let $N = S(0) + I(0) + R(0)$. The following statements hold:*

*(a) $S(t)$ is a decreasing function and $R(t)$ is increasing.*

*(b) If $R_0 S(0) < N$, then $I(t)$ is decreasing.*

*(c) If $R_0 S(0) > N$ and $I(0) > 0$, then $I(t)$ increases up to a maximum value*

$$I_{max} = I(0) + S(0) - \frac{N}{R_0}\left(1 - \log\left(\frac{R_0 S(0)}{N}\right)\right).$$

*(d) $\lim_{t\to\infty} I(t) = 0$, $\lim_{t\to\infty} S(t) = S_\infty$ and $\lim_{t\to\infty} R(t) = R_\infty$ exist.*

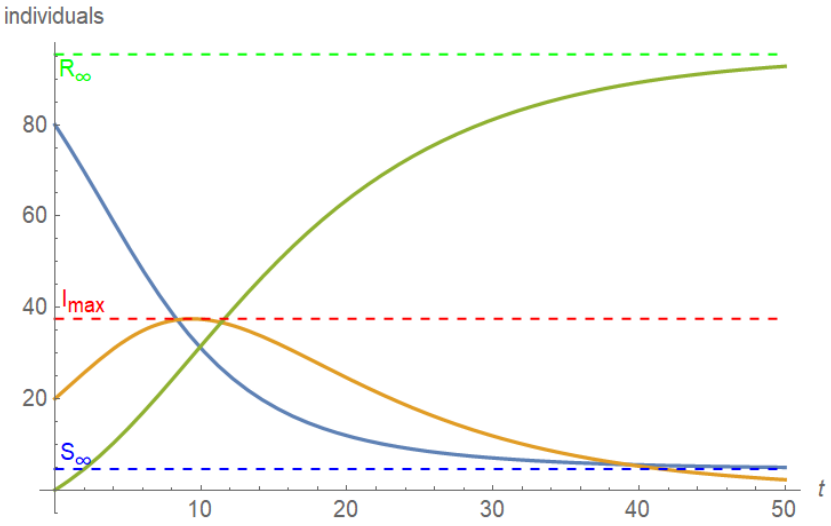
*(e) $S_\infty$ is the unique root in $\left(0, \frac{N}{R_0}\right)$ of*

$$I(0) + S(0) - S_\infty + \frac{N}{R_0} \log\left(\frac{S_\infty}{S(0)}\right) = 0$$

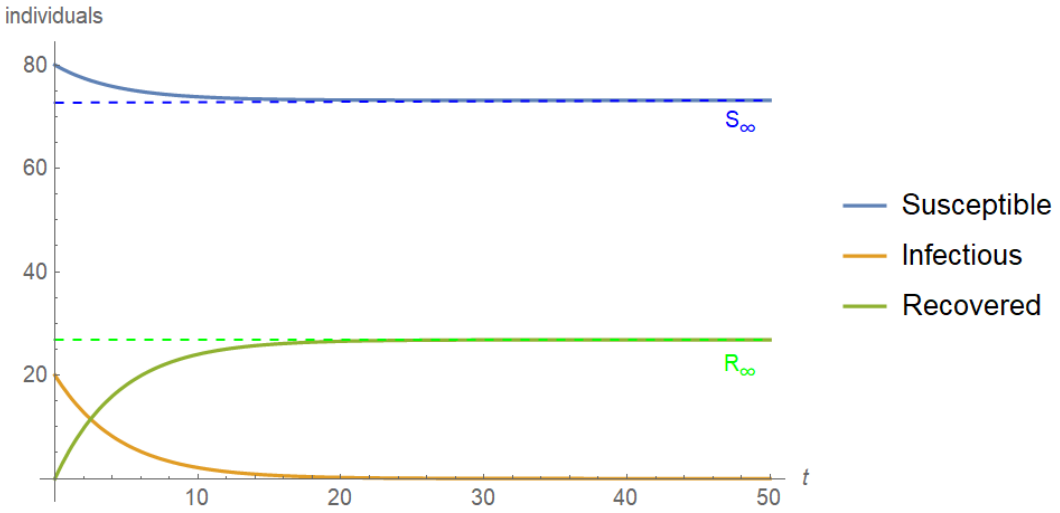
*and $R_\infty = N - S_\infty$.*

The results of this Corollary are illustrated by Figure 17, that presents some numerical solutions of (5).

*Proof.* Note that $S(0) = Ns_0$, $I(0) = Ni_0$, $R(0) = Nr_0$ and $S_\infty = Ns_\infty$. Hence, nearly all of the statements are obvious consequences of Theorem 3.9. It is only left to prove that $R_\infty = N - S_\infty$. We know that $N = S + I + R$ is constant, then $N = \lim_{t\to\infty} S(t) + \lim_{t\to\infty} I(t) + \lim_{t\to\infty} R(t) = S_\infty + 0 + R_\infty$, which proves the result. □




(a) $R_0 > 1$




(b) $R_0 < 1$

Figure 11: Numerical simulation of the $SIR$ model, with initial conditions $I(0) = 20$, $S(0) = 80$ and $R(0) = 0$. In Figure 11a the parameters are $\beta = 0.3$ and $\gamma = 0.1$, so $R_0 > 1$ and the relevant values are computed to be $I_{max} = 37.5$, $S_\infty = 4.57$ and $R_\infty = 95.43$. On the other hand, in Figure 11b the parameters are $\beta = 0.1$ and $\gamma = 0.3$, so $R_0 < 1$ and the limiting values are computed to be $S_\infty = 73.15$ and $R_\infty = 26.85$.

## 3.4 Endemic Susceptible-Infectious-Recovered ($SIR$) Model

The endemic $SIR$ model is an extension of the epidemic $SIR$ model with vital dynamics (births and deaths). We consider a model with a constant birth rate equal to a constant per-capita death rate, $\mu$. In this way, $1/\mu$ is the mean lifetime, which is about 84 years in Italy and about 73 years in the world [Wor]. The model is schematically represented in Figure 12 and leads to the following equations, where, as usual, $N = S + I + R$ is the total number of individuals.

$$\frac{dS}{dt} = \mu N - \mu S - \beta S \frac{I}{N}, \tag{9a}$$

$$\frac{dI}{dt} = \beta S \frac{I}{N} - \gamma I - \mu I, \tag{9b}$$

$$\frac{dR}{dt} = \gamma I - \mu R. \tag{9c}$$

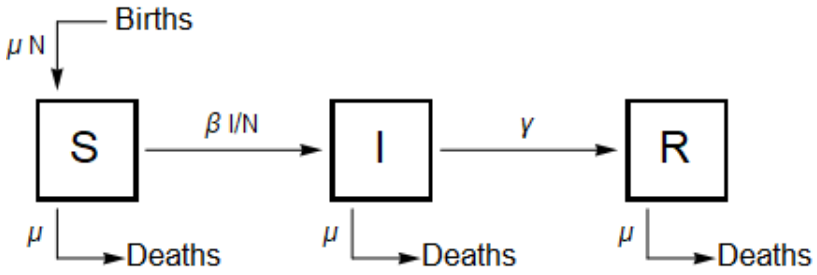



Figure 12: Schematic representation of the endemic $SIR$ model.

### 3.4.1 Conservation of total population and dimension reduction of the model

**Proposition 3.11.** *The total number of individuals, $N = S + I + R$, is a first integral of (9).*

The proof is similar to the analogous results for the $SI$ and $SIS$ models presented before.

The deaths balance the births, so that the population size $N$ is constant. Hence, we can fix the value of $N$ and reduce the system to two dimensions. This corresponds to the analysis of (9a) and (9b) independently of (9c), treating $N$ as a constant. We also normalize the variables by the population size, $N$, defining

$$s = \frac{S}{N}, \qquad i = \frac{I}{N}, \qquad r = 1 - s - i.$$

In this way, we obtain the reduced system

$$\frac{ds}{dt} = -\beta si + \mu - \mu s, \tag{10a}$$

$$\frac{di}{dt} = \beta si - (\gamma + \mu)i. \tag{10b}$$

### 3.4.2 Positively invariant phase space

Since we are interested in solutions for which the number $S$ of susceptible individuals, $I$ of infectious individuals and $R$ of recovered individuals are positive, we consider the domain

$$\Omega = \{(s, i) \in \mathbb{R}^+ \times \mathbb{R}^+ : 0 \leq i + s \leq 1\} \subset \mathbb{R}^2.$$

**Proposition 3.12.** *The region $\Omega$ is positively invariant under the flow of (10).*

The proof is similar to that of Proposition 3.7 and therefore it is omitted.

### 3.4.3 Analysis of the reduced model

Like the epidemic $SIR$ model, the endemic $SIR$ model has no explicit analytical solution. For this model, we only present the classification and the stability analysis of the equilibrium points, indicating their bifurcations, and we illustrate the phase space dynamics and some solutions numerically.

Simple manipulation show that (10) has exactly two equilibrium points, that we call the disease-free, $P_{df}$, and the endemic, $P_e$, equilibrium points:

$$P_{df} = (s_{df}, i_{df}) = (1, 0), \qquad P_e = (s_e, i_e) = \left(\frac{\gamma + \mu}{\beta}, \mu\left(\frac{1}{\gamma + \mu} - \frac{1}{\beta}\right)\right).$$

In order to analyse which of these belongs to $\Omega$ and study their stability, it is convenient to introduce the **basic reproductive number,** $R_0 = \frac{\beta}{\gamma+\mu}$, which can be interpreted as

$$R_0 = \begin{pmatrix} \text{Per-capita} \\ \text{contact rate} \end{pmatrix} \begin{pmatrix} \text{Duration of infection} \\ \text{period adjusted by deaths} \\ \text{of infected individuals} \end{pmatrix}$$

**Theorem 3.13.** *If $R_0 < 1$, then the only equilibrium point in $\Omega$ is the disease free equilibrium $P_{df}$ and it is asymptotically stable. On the other hand, if $R_0 > 1$, then both the disease free $P_{df}$ and the endemic, $P_e$, equilibrium points belong to $\Omega$. Moreover, in this case, $P_{df}$ is an unstable saddle point and $P_e$ is asymptotically stable.*

*Proof.* It is obvious that $P_{df} \in \Omega$ regardless of the value of $R_0$. To analyse $P_e$ we write

$$s_e = \frac{1}{R_0}, \qquad i_e = \frac{\mu}{\beta}(R_0 - 1).$$

It is clear that $s_e > 0$. On the other hand, $i_e \geq 0$ if and only if $R_0 \geq 1$. Moreover, if $R_0 \geq 1$, then

$$s_e + i_e = \frac{1}{R_0}\left(\frac{\gamma + R_0\mu}{\gamma + \mu}\right) \leq \frac{1}{R_0} \leq 1,$$

which shows that $P_e \in \Omega$ if $R_0 \geq 1$.

To analyse stability note that the Jacobian matrix of the vector field $f$ defined by (10) is

$$J(s,i) = \begin{pmatrix} -\beta i - \mu & -\beta s \\ \beta i & \beta s - \gamma - \mu \end{pmatrix}.$$

Evaluating $J$ at $P_{df}$, we obtain

$$J_{df} = \begin{pmatrix} -\mu & -\beta \\ 0 & \beta - \gamma - \mu \end{pmatrix},$$

whose eigenvalues are

$$\lambda_1 = -\mu, \qquad \lambda_2 = \beta - \gamma - \mu = (\gamma + \mu)(R_0 - 1).$$

We have $\lambda_1 < 0$ regardless of the value of $R_0$. On the other hand, $\lambda_2 < 0$ if $R_0 < 1$ and $\lambda_2 > 0$ if $R_0 > 1$. This shows that $P_{df}$ is asymptotically stable if $R_0 < 1$ and a saddle point if $R_0 > 1$.

On the other hand, evaluating $J$ at $P_e$, we have

$$J_e = \begin{pmatrix} -\mu(R_0 - 2) & -\frac{\beta}{R_0} \\ \mu(R_0 - 1) & \frac{\beta}{R_0} - \beta R_0 \end{pmatrix},$$

whose determinant and trace are computed to be

$$\Delta_e = \mu(\gamma + \mu)(R_0 - 1), \qquad \tau_e = -\mu R_0.$$

For $R_0 > 1$ we have $\Delta_e > 0$, $\tau_e < 0$ and it follows from Theorem 2.16 that $P_e$ is asymptotically stable. □

**Remark 3.14.** Actually, one may show that the system (10) when considered as an ODE in $\mathbb{R}^2$ undergoes a "transcritical bifurcation" at $R_0 = 1$. The bifurcation diagram is given below.

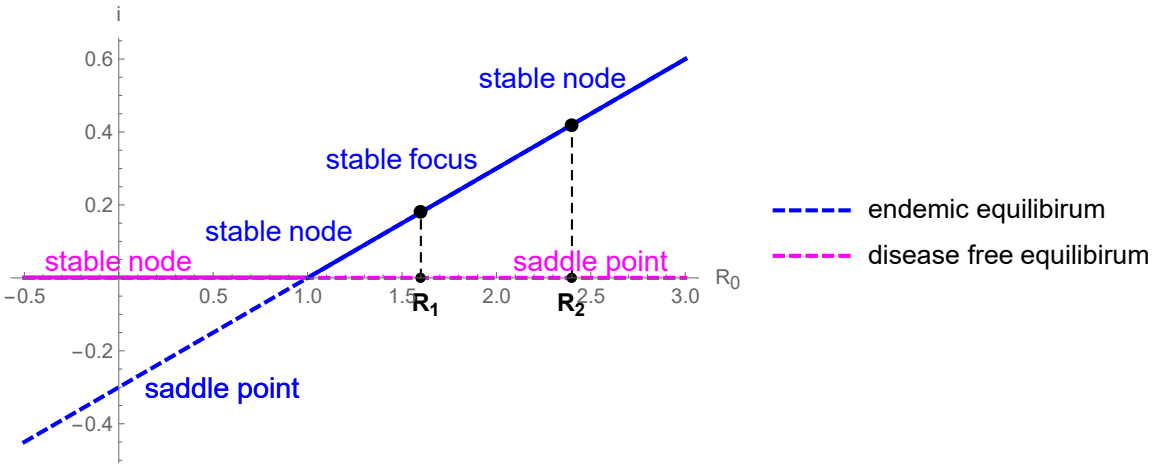



Figure 13: The bifurcation diagram for the endemic $SIR$ model, which shows that the disease-free and endemic equilibria exchange stability when $R_0 = 1$.

Figure 13 indicates that the endemic equilibrium point passes from a saddle to a stable node, to a stable focus and again to a stable node as $R_0$ increases. This seems to be an original observation of this thesis, which is shown to be true in the following proposition.

**Proposition 3.15.** *Let*

$$1 < R_1 = \frac{2(\gamma+\mu-\sqrt{\gamma^2+\gamma\mu})}{\mu} < R_2 = \frac{2(\gamma+\mu+\sqrt{\gamma^2+\gamma\mu})}{\mu}.$$

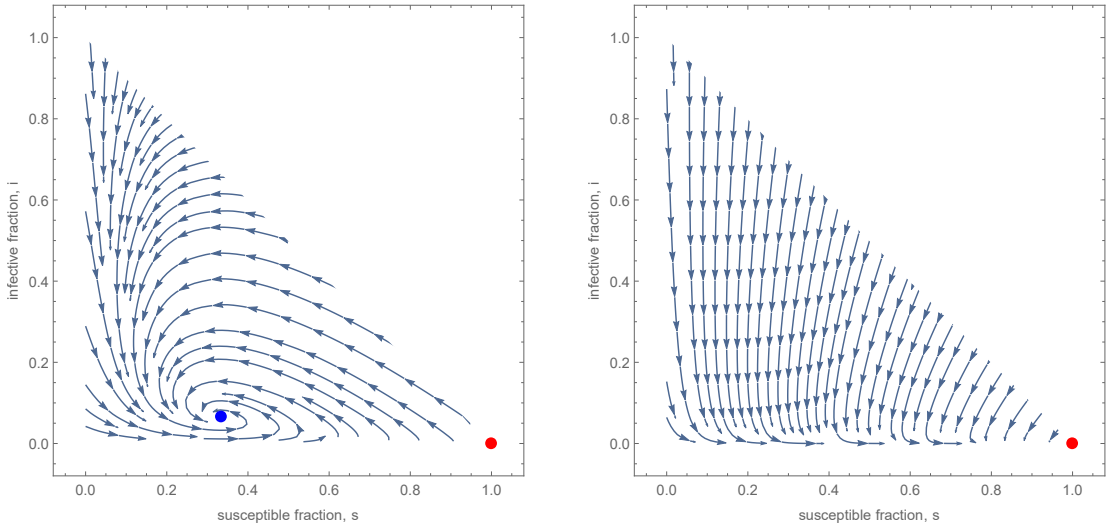
- *If* $1 < R_0 < R_1$ *or* $R_0 > R_2$, *then* $P_e$ *is a stable node* .
- *If* $R_1 < R_0 < R_2$, *then* $P_e$ *is a stable focus* .

*Proof.* After some calculations, we find that $4\Delta_e - \tau_{df}^2 = F_{\gamma,\mu}(R_0)$, where

$$F_{\gamma,\mu}(x) = 4\mu(\gamma+\mu)(x-1) - \mu^2 x^2.$$

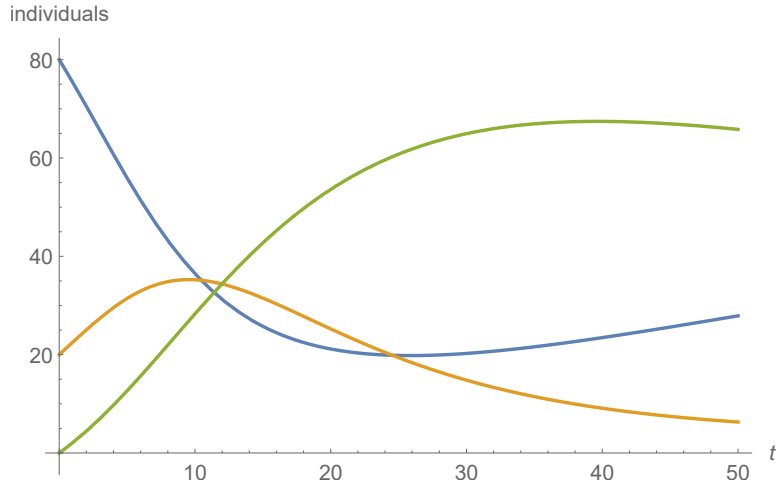
For fixed $\gamma$, $\mu$ the graph of $F_{\gamma,\mu}$ is a parabola that opens downwards and which has two real roots at $x = R_1$ and $x = R_2$. It follows that $F_{\gamma,\mu}(R_0) > 0$ if $R_1 < R_0 < R_2$ and instead $F_{\gamma,\mu}(R_0) < 0$ if $1 < R_0 < R_1$ or $R_0 > R_2$. The result then follows from Theorem 2.16. □

The dynamics of the system is illustrated in Figures 14 and 15.

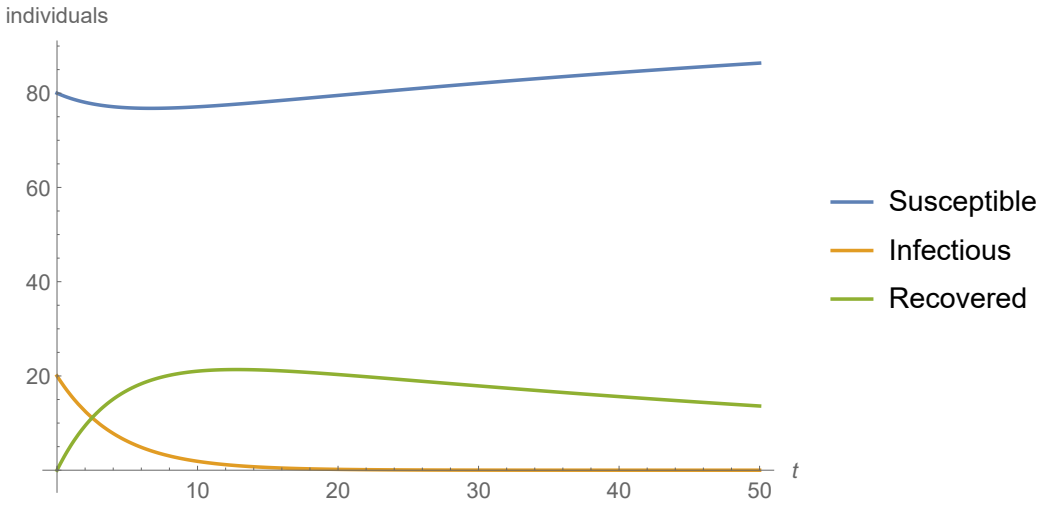



(a) $R_0 > 1$ (b) $R_0 < 1$

Figure 14: A phase portrait of the reduced endemic $SIR$ model. In Figure 14a the parameters are $\beta = 1/2$, $\gamma = 9/60$ and $\mu = 1/60$, so $R_0 > 1$, while in Figure 14b the parameters are $\beta = 1/3$, $\gamma = 39/60$ and $\mu = 1/60$, so $R_0 < 1$. We see the disease free equilibrium point outlined in red and the endemic equilibrium point outlined in blue. The value of $R_0$ in Figure 14a satisfies $R_1 < R_0 < R_2$ which is why we see a stable focus.




(a) $R_0 > 1$




(b) $R_0 < 1$

Figure 15: Numerical simulation of the endemic $SIR$ model, with initial conditions $I(0) = 20$, $S(0) = 80$ and $R(0) = 0$. In Figure 15a the parameters are $\beta = 0.3$ and $\gamma = 0.1$, so $R_0 > 1$, while in Figure 15b the parameters are $\beta = 0.1$ and $\gamma = 0.3$, so $R_0 < 1$.

## 3.5 Susceptible-Infectious-Treated-Recovered ($SITR$) Model

The susceptible-infectious-treated-recovered ($SITR$) model represents diseases for which a treatment has been found. This introduces a new compartment of treated individuals, $T$, and $N = S + I + T + R$ is the total number of individuals. In this model, a fraction $\alpha$ of infected individuals per unit time is selected for treatment and the treatment reduces infectivity by a fraction $\delta$. Afterwards, treated individuals recover at a constant per capita rate $\eta$, whereas non-treated infectious individuals recover at a constant per capita rate $\gamma$. The model is represented in Figure 16 and leads to the system:

$$\frac{dS}{dt} = -\beta \frac{S}{N}(I + \delta T), \tag{11a}$$

$$\frac{dI}{dt} = \beta \frac{S}{N}(I + \delta T) - \gamma I - \alpha I, \tag{11b}$$

$$\frac{dT}{dt} = \alpha I - \eta T, \tag{11c}$$

$$\frac{dR}{dt} = \gamma I + \eta T. \tag{11d}$$

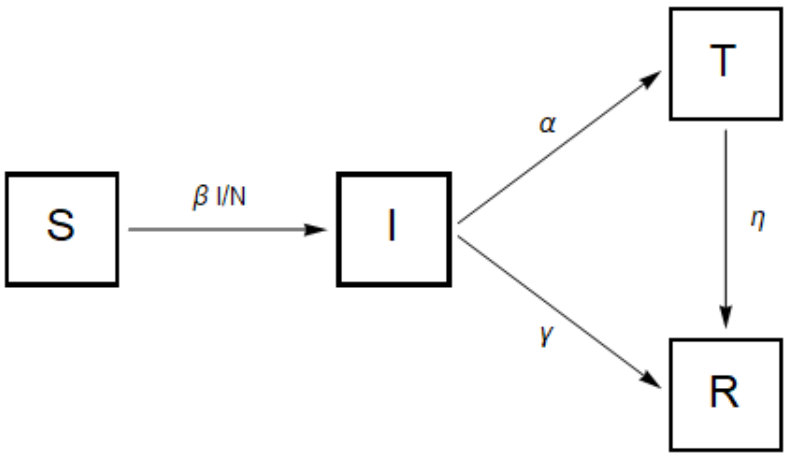



Figure 16: Schematic representation of the $SITR$ model.

### 3.5.1 Conservation of total population and dimension reduction of the model

**Proposition 3.16.** *The total number of individuals, $N = S + I + T + R$, is a first integral of (11).*

The proof is similar to the analogous results for the $SI$ and $SIS$ models presented before.

Since the total number of individuals, $N$, is a first integral, we can fix the value of $N$ and reduce the system to three dimensions. This corresponds to the analysis of (11a), (11b) and (11c) independently of (11d), treating $N$ as a constant. We also normalize the variables by the population size, $N$, defining

$$s := \frac{S}{N}, \qquad i := \frac{I}{N}, \qquad \tau := \frac{T}{N}, \qquad r := 1 - s - i - \tau.$$

We obtain the reduced system

$$\frac{ds}{dt} = -\beta s(i + \delta\tau), \tag{12a}$$
$$\frac{di}{dt} = \beta s(i + \delta\tau) - \gamma i - \alpha i, \tag{12b}$$
$$\frac{d\tau}{dt} = \alpha i - \eta\tau. \tag{12c}$$

### 3.5.2 Positively invariant phase space

Since we are interested in solutions for which the number $S$ of susceptible individuals, $I$ of infectious individuals, $T$ of treated individuals and $R$ of recovered individuals are positive, we consider the domain

$$\Omega = \{(s, i, t) \in \mathbb{R}^+ \times \mathbb{R}^+ \times \mathbb{R}^+ : 0 \leq s + i + \tau \leq 1\} \subset \mathbb{R}^3.$$

**Proposition 3.17.** *The region $\Omega$ is positively invariant under the flow of (12).*

*Proof.* Note that

$$\partial\Omega = L_1 \cup L_2 \cup L_3 \cup L_4,$$

where

$$\begin{aligned}
L_1 &= \{(s, 0, \tau) : 0 \leq s, \tau,\ s + \tau \leq 1\},\\
L_2 &= \{(s, i, 1 - s - i) : 0 \leq s, i,\ s + i \leq 1\},\\
L_3 &= \{(0, i, \tau) : 0 \leq i, \tau,\ i + \tau \leq 1\},\\
L_4 &= \{(s, i, 0) : 0 \leq s, i,\ s + i \leq 1\}.
\end{aligned}$$

The normal outward vector to $\Omega$ along $L_j$ is $n_j$, with

$$n_1 = (0, -1, 0), \qquad n_2 = (1, 1, 1), \qquad n_3 = (-1, 0, 0), \qquad n_4 = (0, 0, -1).$$

Denote by $f : \mathbb{R}^3 \to \mathbb{R}^3$ the vector field defined by (12), i.e.

$$f(s,i,t) = \begin{pmatrix} -\beta s(i+\delta\tau), \\ \beta s(i+\delta\tau) - \gamma i - \alpha i, \\ \alpha i - \eta\tau \end{pmatrix}. \tag{13}$$

Then it is straightforward to check that if $(s_0, i_0, \tau_0) \in L_j$, then $f(s_0, i_0, \tau_0) \cdot n_j \leq 0$. We may now apply Lemma 2.8 to show that $\Omega$ is positively invariant[6]. □

### 3.5.3 Analysis of the reduced model I: equilibrium points and their stability

The $SITR$ model has no explicit analytical solution. It is easy to see that the only equilibrium points of (12) within $\Omega$ are of the form $(s,i,\tau) = (\xi, 0, 0)$ for any $\xi \in [0,1]$. They all are disease free equilibria. For simplicity, in the following analysis we will only consider the case where $\delta = 0$.

The Jacobian matrix of $f$ given by (13) is

$$J(s,i,\tau) = \begin{pmatrix} -\beta i & -\beta s & 0 \\ \beta i & \beta s - \gamma - \alpha & 0 \\ 0 & \alpha & -\eta \end{pmatrix}.$$

Evaluating $J$ at $(s,i,\tau) = (\xi, 0, 0)$, we have

$$J^* = \begin{pmatrix} 0 & -\beta\xi & 0 \\ 0 & \beta\xi - \gamma - \alpha & 0 \\ 0 & \alpha & -\eta \end{pmatrix}.$$

The eigenvalues of $J^*$ are

$$\lambda_1 = 0, \qquad \lambda_2 = -\eta, \qquad \lambda_3 = \beta\xi - \alpha - \gamma.$$

The presence of a zero eigenvalue implies that the equilibrium points are non-hyperbolic.

Introducing the value $R_0 = \frac{\beta}{\gamma+\alpha}$, we can rewrite $\lambda_3 = -(\gamma+\alpha)(1 - R_0\xi)$. Therefore, $\lambda_3 < 0$ if $\xi < \frac{1}{R_0}$ and $\lambda_3 > 0$ if $\xi > \frac{1}{R_0}$.

Our analysis shows that the behaviour of the system depends on whether $R_0$ is larger or smaller than 1. For this model $R_0$ is the basic reproductive number that will be discussed in detail in the next section.

[6] We also require a slight generalization of Lemma 2.8 in the spirit of Remark 3.8, due to the non-smoothness of $\Omega$ at the "corners" $L_i \cap L_j$, $i \neq j$.

If $\xi > \frac{1}{R_0}$, then Theorem 2.12 guarantees instability. On the other hand, the linear stability analysis is inconclusive (we cannot apply Lyapunov's spectral Theorem 2.12). Nonetheless, using phase space analysis techniques (that we omit), one may show that the equilibrium point $(s, i, \tau) = (\xi, 0, 0)$ is stable if $\xi < \frac{1}{R_0}$. In particular, if $R_0 < 1$, all the equilibrium points within $\Omega$ are stable.

### 3.5.4 The basic reproductive number $R_0$ and the replacement number $R_e(t)$

As anticipated above, the basic reproductive number for this model is $R_0 = \frac{\beta}{\gamma+\alpha}$, which allows the following interpretation:

$$R_0 = \begin{pmatrix} \text{Per-capita} \\ \text{contact rate} \end{pmatrix} \begin{pmatrix} \text{Duration of infection period} \\ \text{adjusted by removal of infected} \\ \text{individuals selected for treatment} \end{pmatrix}.$$

As seen above, if $R_0 < 1$ all equilibrium points within $\Omega$ are stable. On the other hand, if $R_0 > 1$, then equilibrium points $(s, i, \tau) = (\xi, 0, 0)$ with $\frac{1}{R_0} < \xi < 1$ are unstable. Hence, $R_0$ is again a threshold quantity for this model.

As in the epidemic $SIR$ model, it is useful to introduce the replacement number, $R_e(t) = R_0 s(t)$. Note that, if we set $\delta = 0$, equation (12b) may be rewritten as

$$\frac{di}{dt} = i(\gamma + \alpha)(R_e(t) - 1)$$

and therefore if $R_e(t) < 1$ the number of infectious individuals decreases, whereas if $R_e(t) > 1$ the number of infectious individuals increases.

### 3.5.5 Analysis of the reduced model II

**Theorem 3.18.** *Let $(s(t), i(t), \tau(t))$ be a solution of (12), with initial condition $(s_0, i_0, \tau_0) \in \Omega$. The following statements hold:*

*(a) The infectious fraction $i(t)$ converges to zero as $t \to \infty$.*

*(b) The susceptible fraction $s(t)$ is a decreasing function and converges to a limiting value $s_\infty > 0$ as $t \to \infty$.*

*(c) The treated fraction $\tau(t)$ converges to zero as $t \to \infty$.*

*Proof.* Let $g : \mathbb{R} \to \mathbb{R}$, $g(t) = (s + i)(t) = s(t) + i(t)$ be a function, for $t \geq 0$. Proceeding as in the proof of Theorem 3.9, one can show that $g(t) \geq 0$, $g'(t) \leq 0$ and $g''(t)$ is bounded for $t \geq 0$.

Therefore, by Proposition A.1 of the Appendix, $\lim_{t\to\infty} g'(t) = 0$. So, since $g'(t) = -(\alpha+\gamma)i(t)$, we have $\lim_{t\to\infty} i(t) = 0$, that proves (a). This shows that, since $\lim_{t\to\infty} g(t)$ exists, then $\lim_{t\to\infty} s(t) =: s_\infty$ exists, that proves (b).

Let $h : \mathbb{R} \to \mathbb{R}$, $h(t) = (s+i+\tau)(t) = s(t)+i(t)+\tau(t)$ be a function, for $t \geq 0$. We proceed as in (a) and we have $h(t) \geq 0$,

$$h'(t) = (s+i+\tau)'(t) = -\gamma i(t) - \eta\tau(t) \leq 0.$$

and

$$|h''(t)| \leq \gamma(\beta+\alpha+\gamma) + \eta(\alpha+\eta), \qquad \forall t \geq 0.$$

Therefore, by Proposition A.1 of the Appendix, $\lim_{t\to\infty} h'(t) = 0$. So, since $h'(t) = -\gamma i(t) - \eta\tau(t)$ and $\lim_{t\to\infty} i(t) = 0$, we have $\lim_{t\to\infty} \tau(t) = 0$. □

**Theorem 3.19.** *Let $(s(t), i(t), \tau(t))$ be a solution of (12), with initial condition $(s_0, i_0, \tau_0) \in \Omega$. The limit value $s_\infty$ of the susceptible fraction $s$ satisfies*

$$\log\left(\frac{s_0}{s_\infty}\right) = R_0\left(i_0 + s_0 - s_\infty\right).$$

*Proof.* Recalling that $(s+i)'(t) = -(\alpha+\gamma)i(t)$ and integrating both parts from $t = 0$ to $t = \infty$ yields

$$\int_0^\infty (s+i)'(t)dt = -(\alpha+\gamma)\int_0^\infty i(t)dt.$$

Thanks to Theorem 3.18, we have $\lim_{t\to\infty} i(t) = 0$ and $\lim_{t\to\infty} s(t) =: s_\infty$, so the left hand side equals

$$s(t) + i(t)\big|_0^\infty = s_\infty - s_0 - i_0.$$

And hence

$$\int_0^\infty i(t)dt = \frac{s_0 + i_0 - s_\infty}{\alpha+\gamma} \tag{14}$$

On the other hand, dividing $s' = -\beta si$ by $s$ and integrating from 0 to $\infty$, gives:

$$\int_0^\infty \frac{s'(t)}{s(t)}dt = -\beta\int_0^\infty i(t)dt$$

However we have

$$\int_0^\infty \frac{s'(t)}{s(t)}dt = \log|s(t)|\big|_0^\infty = \log(s_\infty) - \log(s_0),$$

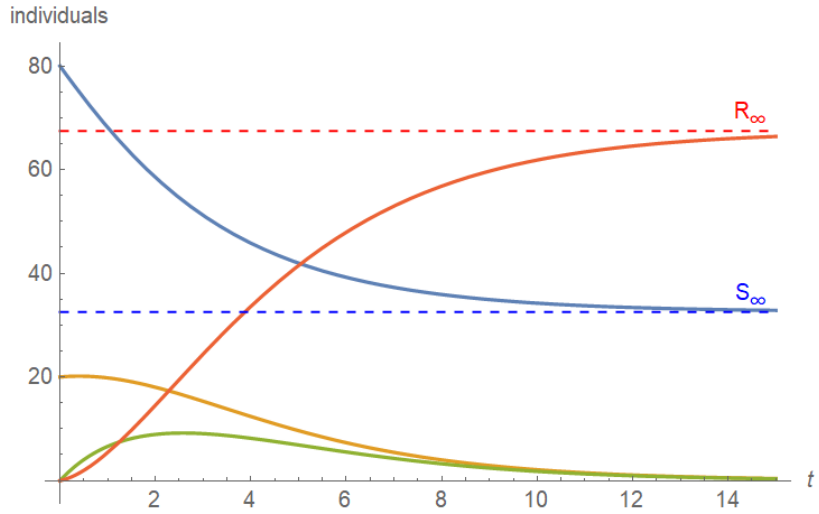
which implies

$$\int_0^\infty i(t)dt = \frac{1}{\beta}\log\left(\frac{s_0}{s_\infty}\right).$$

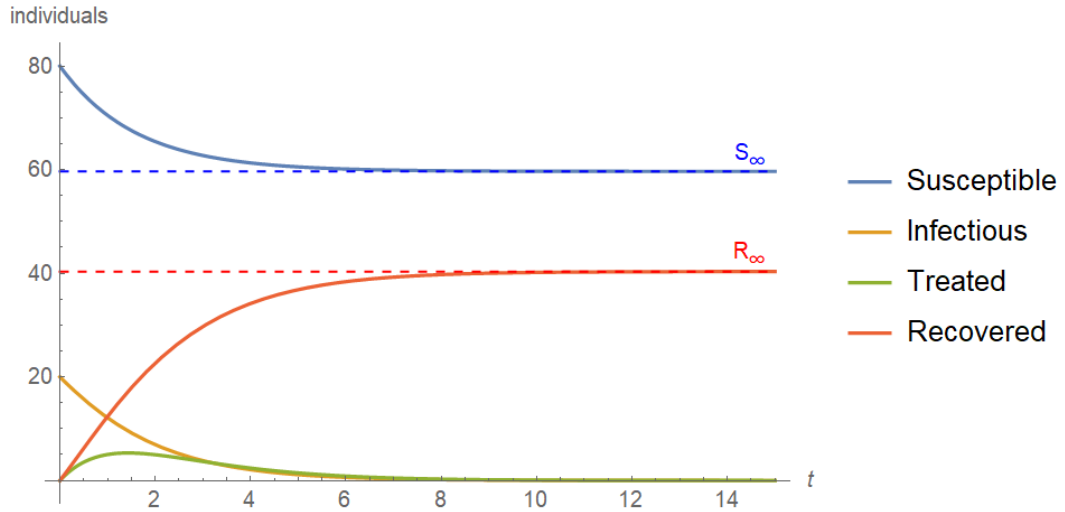
Combining the above expression with (14) proves the result.

□

**Remark 3.20.** In analogy to Corollary 3.10, it is possible to show that $\lim_{t\to\infty} S(t) = S_\infty$ where $S_\infty$ satisfies $I(0)+S(0)-S_\infty+\frac{N}{R_0}\log\left(\frac{S_\infty}{S(0)}\right)=0$. Also $R_\infty = N - S_\infty$.




(a) $R_0 > 1$




(b) $R_0 < 1$

Figure 17: Numerical simulation of the $SITR$ model, with initial conditions $I(0) = 20$, $S(0) = 80$, $T(0) = 0$ and $R(0) = 0$ and parameters $\beta = 0.8$, $\alpha = 0.5$ and $\eta = 0.9$. In Figure 17a $\gamma = 0.1$ so $R_0 > 1$ and the limiting value are computed to be $S_\infty = 32.55$ and $R_\infty = 67.45$. On the other hand, in Figure 17b $\gamma = 0.6$ so $R_0 < 1$ and the limiting value are computed to be $S_\infty = 59.66$ and $R_\infty = 40.34$.

# 4 Models describing the evolution of the Covid pandemic

In this final section we describe two recent extensions of the basic epidemiological models seen before aiming at describing the evolution of the Covid19 pandemic, contained in publications [LR21] and [Gio+20]. We limit ourselves to the description of the compartment classes, the description of the parameters of the models, their schematic representation and the corresponding equations of motion but we do not make any type of analysis or numerical simulations. Our discussion does not enter in the relevant and difficult task of determining the model parameters to fit real data.

## 4.1 The $SIDARTHE$ model

Firstly, we describe the model in the paper by Giordano at al [Gio+20], for the COVID-19 epidemic and the implementation of population-wide interventions in Italy. The authors compare simulation results with real data and model possible scenarios of implementation of countermeasures. To do that, they introduce the $SIDHARTE$ model, an elaborate extension of the $SIR$ model, where the following compartments of individuals are used:

| Symbol | Compartment | Characteristics |
|---|---|---|
| $S$ | Susceptible Individuals | Uninfected |
| $I$ | Infected Individuals | Asymptomatic, infected, undetected |
| $D$ | Diagnosed Individuals | Asymptomatic, infected, detected |
| $H$ | Healed Individuals | Recovered |
| $A$ | Ailing Individuals | Symptomatic, infected, undetected |
| $R$ | Recognized Individuals | Symptomatic, infected, detected |
| $T$ | Threatened Individuals | Acutely symptomatic, infected, detected |
| $E$ | Extinct Individuals | Dead |

The $SIDARTHE$ model discriminates between infected individuals depending on whether they have been diagnosed and on the severity of their symptoms. The distinction between diagnosed and non-diagnosed individuals is important because the former are typically isolated and hence less likely to spread the infection. In the model, they omit the probability rate of becoming susceptible again after having already recovered from the infection, because, contrary to what we now know, reinfection appeared to be negligible at the time of the study (June 2020). Also new parameters are introduced, which are illustrated in the following table.

| Parameters | Interpretation |
|---|---|
| $\alpha$ | Transmission rate from infected |
| $\beta$ | Transmission rate from diagnosed |
| $\gamma$ | Transmission rate from ailing |
| $\delta$ | Transmission rate from recognized |
| $\epsilon$ | Rate of detention for asymptomatic cases |
| $\theta$ | Rate of detention for symptomatic cases |
| $\zeta$ | Probability of developing symptoms in infected |
| $\eta$ | Probability of developing symptoms in diagnosed |
| $\mu$ | Probability of developing life-threatening symptoms in ailing |
| $\nu$ | Probability of developing life-threatening symptoms in recognized |
| $\lambda$ | Recovery rate for infected |
| $\rho$ | Recovery rate for diagnosed |
| $\kappa$ | Recovery rate for ailing |
| $\xi$ | Recovery rate for recognized |
| $\sigma$ | Recovery rate for threatened |
| $\tau$ | Mortality rate |

**Remark 4.1.**

- Typically, $\alpha$ is larger than $\gamma$ (assuming that people tend to avoid contacts with subjects showing symptoms, even though diagnosis has not been made yet), which in turn is larger than $\beta$ and $\delta$ (assuming that subjects who have been diagnosed are properly isolated). These parameters can be modified by social-distancing policies (for example, closing schools, remote working, lockdown). The risk of contagion due to threatened subjects, treated in proper ICUs, is assumed negligible.

- $\theta$ is typically larger than $\epsilon$, as a symptomatic individual is more likely to be tested.

- $\zeta$ and $\eta$ are comparable in the absence of specific treatment.

- $\mu$ and $\nu$ are comparable if there is no known specific treatment that is effective against the disease, otherwise $\mu$ may be larger. Conversely, $\nu$ may be larger because infected individuals with more acute symptoms, who have a higher risk of worsening, are more likely to have been diagnosed. These parameters can be reduced by means of improved therapies and acquisition of immunity against the virus.

- $\tau$ can be reduced by means of improved therapies.

- $\lambda$, $\rho$, $\kappa$, $\xi$ and $\sigma$ may differ significantly if an appropriate treatment for the disease is known and adopted for diagnosed patients, but are probably comparable otherwise. These parameters can be increased thanks to improved treatments and acquisition of immunity against the virus.
- The authors implemented simulations of the model for which the model parameters were based on the official data (source: Protezione Civile and Ministero della Salute) about the evolution of the epidemic in Italy from 20 February 2020 through 5 April 2020.

The model leads to the following system of eight differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= -S(\alpha I + \beta D + \gamma A + \delta R),\\
\frac{dI}{dt} &= S(\alpha I + \beta D + \gamma A + \delta R) - (\epsilon + \zeta + \lambda)I,\\
\frac{dD}{dt} &= \epsilon I - (\eta + \rho)D,\\
\frac{dA}{dt} &= \zeta I - (\theta + \mu + \kappa)A,\\
\frac{dR}{dt} &= \eta D + \theta A - (\nu + \xi)R,\\
\frac{dT}{dt} &= \mu A + \nu R - (\sigma + \tau)T,\\
\frac{dH}{dt} &= \lambda I + \rho D + \kappa A + \xi R + \sigma T,\\
\frac{dE}{dt} &= \tau T.
\end{aligned} \tag{15}$$

The schematic representation of the model, taken from [Gio+20], is presented in Figure 18.

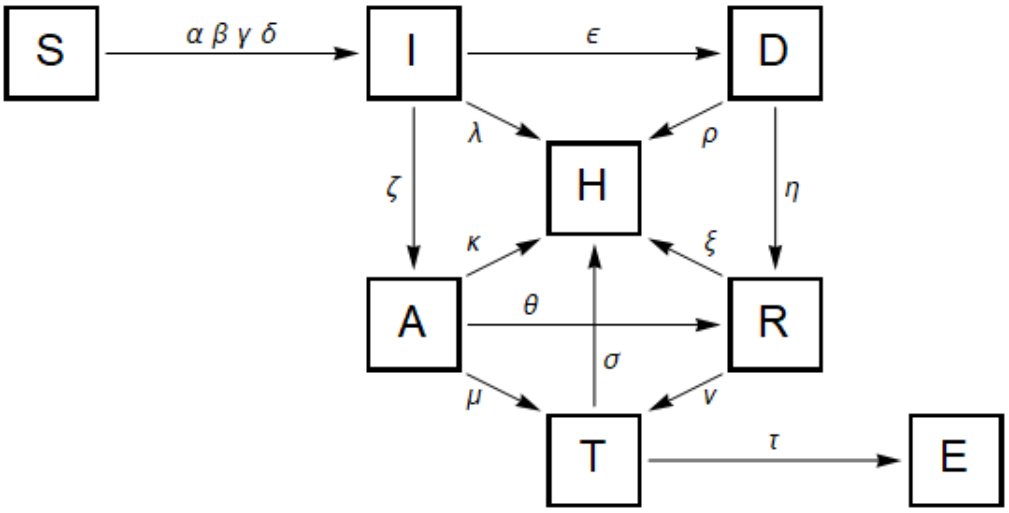



Figure 18: Schematic representation of the $SIDHARTE$ model from [Gio+20].

### 4.1.1 Main features of the model

It is straightforward to prove that the total number of individuals $N = S + I + D + A + R + T + H + E$ is a first integral of (15). In their article [Gio+20], the authors fix $N = 1$ and they proceed to a system reduction. They found that all the possible equilibria are given by $(S, I, D, A, R, T, H, E) = (\bar{S}, 0, 0, 0, 0, 0, \bar{H}, \bar{E})$, with $\bar{S} + \bar{H} + \bar{E} = 1$ and $\bar{S}, \bar{H}, \bar{E} \geq 0$. The paper shows that the basic reproductive number is

$$R_0 = \frac{\alpha + \frac{\beta\epsilon}{\eta+\rho} + \frac{\gamma\zeta}{\theta+\mu+\kappa} + \frac{\delta\eta\epsilon}{(\eta+\rho)(\nu+\xi)} + \frac{\delta\zeta\theta}{(\theta+\mu+\kappa)(\nu+\xi)}}{\epsilon + \zeta + \lambda}$$

and that the stability of the equilibrium occurs for $\bar{S}R_0 < 1$. Further aspects of the analysis can be found in the article.

## 4.2 A modified $SEIR$ model

Finally, we describe some aspects of the article by Lopez and Rodò [LR21]. The researchers introduced a modified $SEIR$ model to predict the COVID-19 outbreak in Spain and Italy. We have not studied the $SEIR$ model throughout this thesis, which is an extension of the the $SIR$ model that introduces the compartment of the exposed individuals $E$, which have contracted the disease but are not contagious yet. In the article, the authors introduce three additional compartments of individuals: quarantined individuals $Q$, deceased because of the virus $D$ and protected individuals $C$[7]. Also new parameters are introduced, which are illustrated in the following table.

| **Parameters** | **Interpretation** |
|---|---|
| $\alpha$ | Protection rate |
| $\beta$ | Infection rate |
| $\gamma$ | Incubation rate |
| $\delta$ | Quarantine rate |
| $\lambda$ | Recovery rate |
| $k$ | Mortality rate of the virus |
| $\mu$ | Natural birth and death rate |
| $\tau$ | Lenght of the protection |

The model parameters $\gamma$ and $k$ are not taken as constant but as functions of time, with an explicit function form

$$k(t) = k_0 \exp(-k_1 t), \qquad \lambda(t) = \lambda_0(1 - \exp(-\lambda_1 t)).$$

The authors attempt to fit real data by performing numerical simulations and adjusting the value of $k_0$, $k_1$, $\lambda_0$ and $\lambda_1$.

Their model leads to the following non-autonomous system of seven differential

[7]It is assumed also that the protected population does not have contact with the infected individuals and therefore cannot be infected.

equations:[8]

$$
\begin{aligned}
\frac{dS}{dt} &= \mu N - \alpha S + \tau C - \beta S \frac{I}{N} - \mu S, \\
\frac{dE}{dt} &= \beta S \frac{I}{N} - \gamma E - \mu E, \\
\frac{dI}{dt} &= \gamma E - \delta I - \mu I, \\
\frac{dQ}{dt} &= \delta I - \lambda(t) Q - k(t) Q - \mu Q, \\
\frac{dR}{dt} &= \lambda(t) Q - \mu R, \\
\frac{dD}{dt} &= k(t) Q, \\
\frac{dC}{dt} &= \alpha S - \tau C - \mu C.
\end{aligned}
$$

where $N = S + E + I + Q + R + D + C$ is the total number of individuals. The population is assumed constant due to the rapid disease spread, i.e. the births and natural death have the same value.

We represent a schematic representation of the model, which is missing in [LR21], in Figure 19.

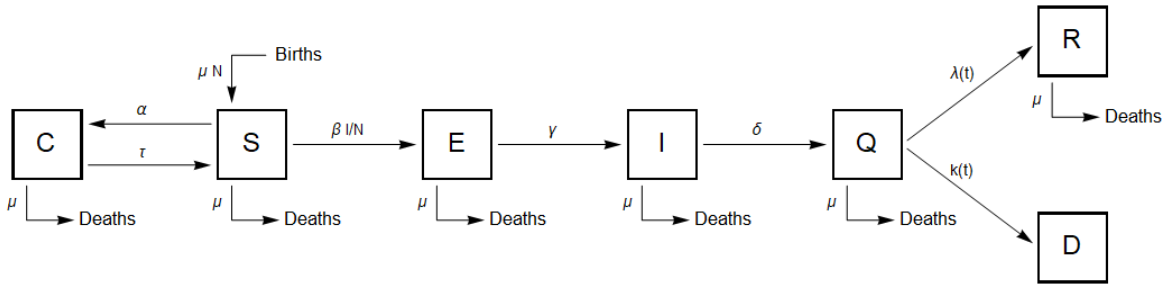



Figure 19: Schematic representation of the model.

[8]The article states that $\frac{dI}{dt} = \gamma E - \delta I - \mu S - \mu I$, but we think that presence of the term "$-\mu S$ " is a typo.

# A Review of useful results from analysis

For the sake of completeness, we include some basic results that are used in the proofs of Theorems 3.9, 3.18 and 3.19.

**Proposition A.1.** *Let $g : \mathbb{R}^+ \to \mathbb{R}$ be of class $C^2$ and assume that*

- $g \geq 0$,
- $g' \leq 0$,
- $g''$ *is bounded.*

*Then*

$$\lim_{t\to\infty} g'(t) = 0.$$

Before giving the proof we recall the following:

**Definition A.2.** The function $g : \mathbb{R}^+ \to \mathbb{R}$ is called **uniformly continuous** if given $\varepsilon > 0$ there exists $\delta > 0$ such that

$$|x - y| < \delta \implies |g(x) - g(y)| < \varepsilon \qquad \forall x, y \in \mathbb{R}^+.$$

**Definition A.3.** The function $g : \mathbb{R}^+ \to \mathbb{R}$ is called **Lipschitz** if there exists $K > 0$ such that

$$|g(x) - g(y)| < K|x - y| \qquad \forall x, y \in \mathbb{R}^+.$$

**Proposition A.4.** *If $g : \mathbb{R}^+ \to \mathbb{R}$ is a Lipschitz function, then it is uniformly continuous.*

*Proof.* Given the value of $\varepsilon > 0$, we take $\delta = \frac{\varepsilon}{K}$. We have

$$|g(x) - g(y)| < K|x - y| < K\delta = \varepsilon \qquad \forall x, y \in \mathbb{R}^+.$$

□

**Proposition A.5.** *If $g : \mathbb{R}^+ \to \mathbb{R}$ is $C^1$ and $g'$ is bounded, then $g$ is Lipschitz.*

*Proof.* Suppose that $|g'(x)| \leq K$, $\forall x \in \mathbb{R}^+$. By the Mean Value Theorem we have

$$|g(y) - g(x)| = |g'(\xi)(y - x)| \leq |g'(\xi)||y - x| = K|y - x|.$$

□

We are ready now to give a proof of Proposition A.1.

*Proof of Proposition A.1.* Since $g''$ is bounded, by Proposition A.5, we know that $g'$ is uniformly continuous. We proceed by contradiction. We suppose that

$$\lim_{t\to\infty} g'(t) \neq 0.$$

Therefore exists $\varepsilon > 0$ such that $\forall N > 0\ \exists t_0 > N$ such that $|g'(t_0)| > \varepsilon$, i.e. $g'(t_0) < -\varepsilon$. Because $g'$ is uniformly continuous, there exists $\delta$ such that

$$|g'(u) - g'(v)| < \frac{\varepsilon}{2},$$

whenever $|u - v| < \delta$.

Let $N = 1$, then $\exists t_1$ such that $g'(t_1) < -\varepsilon$. Let $t \in \left[t_1, t_1 + \frac{\delta}{2}\right]$. Then, $|t - t_1| \leq \frac{\delta}{2} < \delta$. Hence, $|g'(t) - g'(t_1)| < \frac{\varepsilon}{2}$. As a consequence,

$$-\frac{\varepsilon}{2} < g'(t) - g'(t_1) < \frac{\varepsilon}{2}$$

and

$$g'(t) < \frac{\varepsilon}{2} + g'(t_1) < \frac{\varepsilon}{2} - \varepsilon = -\frac{\varepsilon}{2}.$$

By the mean value Theorem

$$g\left(t_1 + \frac{\delta}{2}\right) = g(t_1) + g'(\xi)\frac{\delta}{2} < g(t_1) - \frac{\varepsilon\delta}{4}.$$

Let $N = t_1 + \frac{\delta}{2}$. Repeating the argument given above we get

$$g\left(t_2 + \frac{\delta}{2}\right) < g(t_2) - \frac{\varepsilon\delta}{4} \leq g\left(t_1 + \frac{\delta}{2}\right) - \frac{\varepsilon\delta}{4} < g(t_1) - 2\frac{\varepsilon\delta}{4}.$$

Repeating the whole argument $m$ times we prove the existence of $t_m$ such that

$$g\left(t_m + \frac{\delta}{2}\right) < g(t_1) - m\frac{\varepsilon\delta}{4}.$$

But this contradicts $g \geq 0$, since $m$ is arbitrary. □

**Remark A.6.** We note that Proposition A.1 does not hold without the additional assumption that $g''$ is bounded. For instance, we consider the function

$$\phi(x) = \sum_{n=1}^{\infty} \frac{1}{n^2} f_n(x - n)$$

where $f_n(x) = \frac{2}{\pi}\arctan(n^3 x)$. The function satisfies $\phi \geq 0$, $\phi' \leq 0$ but $\lim_{x\to\infty} \phi'(x)$ does not exists. This detail is overlooked in the analysis of the $SIR$ model in [BCC12, Pag. 354].

# Index

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