UNIVERSITY OF TRENTO

INDUSTRIAL ENGINEERING DEPARTMENT

Master Degree Thesis

Understanding the effect of opinions and behaviours on the spread of infectious diseases

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To my old friend Gojira

Summary

Slow fashion is a movement born recently to contrast the fast fashion trend. It consists in implementing a better and more sustainable supply chain with a positive environmental and social impact. Nevertheless, there is a common bias where people perceive ethical products to be too expensive. This is due partially to the actual high prices of some brands but it is also a consequence of greenwashing. In order to investigate this aspect, an empirical research has been conducted with Atotus. It is both an e-commerce and a physical shop in Trento that sells ethical articles of clothing as a core business and it has built a circular economy network.

A choice based conjoint analysis is performed. The first step is to build two different surveys, which have been submitted to individuals through two channels. Both questionnaires present three product profiles and individuals choose the one they would buy. Each profile is composed by one level of each attribute. These attributes are design, material and price.

Respondents are actual and potential Atotus customers reached through its social networks. Next, the collected data are given as input to the multinomial logit model to identify respondents preferences across attributes. It is the standard model, where only the individual-specific variables are taken into account. The advanced or mixed multinomial logit model includes more detailed aspects, such as heteroscedasticity and random parameters. Moreover, the willingness to pay (WTP) and the market shares of sustainable products are investigated.

The analysis shows that all attributes are equally significant for respondents. This is the case both for the standard model and the mixed one. Moreover, the WTP shows that respondents would pay €38 more in order to buy a t-shirt made of recycled cotton and €34 for a t-shirt made of certified organic cotton. Additionally, the design, p1 and p2 are the preferred ones. As regard the shares prediction, almost 50% of individuals would buy a product made of a sustainable material of design p1 or p2 with a price of 50€.

There is not much literature regarding this specific topic. Indeed, conjoint analysis is performed in many fields, but slow fashion has not been one of them. These results show a starting point of applied data analysis in this specific field. Additionally, it is possible to notice that there is a strong interest among potential and actual customers toward Atotus products and individuals' purchase behavior is coherent with Atotus pricing strategy.

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Part I

Introduction

Chapter 1

Introduction

What has been one of the main dangers humanity has faced throughout its history? The first answers that can be given are war or climate change, but there is another great threat that has severely affected the lives of almost all the population on earth: disease and epidemic. There is not a period neither nowadays nor in the past in which illness has not influenced our lives.

Looking to the past the consequences of the epidemic on the population were worse than today because of the lack of knowledge about medical science and the poor hygienic conditions. During the bubonic plague of the 14th century, for example, 25 million deaths were reported in Europe out of a population of 100 million. Jews were considered responsible for the illness spread and they began to be persecuted. Otherwise, in the course of the Americas' colonization, the disease imported by the Europeans was one of the main causes of the genocide of the local population, causing their defeat against the Spanish conquistadores. In fact, diseases like smallpox and cholera were unknown in these countries and native Americans had no antibodies to contrast them. Other important epidemics, famous for their consequences were Spanish influenza, Smallpox, Typhus, HIV/AIDS, and the more recent Covid-19. It is straightforward to notice the effect that diseases have on our lives.

The development of modern medicine and hygiene can contribute to enhancing the quality of life. An example of this is that only in the last three centuries and especially in the most economical developing countries, a significant increase in life expectancy has been observed [1]. This increase is also happening in the poorest region, like Subsaharian Africa, and even if actually their life expctancy value is lower, newest research predict that they will have the gratest increase in the next thirty years [2]. This study also predict that the phenomena will cause a convergence in the life expectancy between now and 2050.

perchè succede: "Across locations, the burden of disease will continue to shift from communicable, maternal, neonatal, and nutritional diseases to non-communicable diseases." questa è la chiave e lo stesso articolo dice che è dovuto a una diffusione sempre più permeante della sanita e di norme coadiuvate a livello di macro r egione per gestione di malattie e che questo trend va mantenuto. ALLEGA ANCHE LA FIGURA PRESA DALL'ARTICOLO A STO PUNTO.

Even if mortality is decreased, the modification in social patterns and the development of large cities, have had some consequences: a higher density of population has increased

in the 18th and 19th centuries the frequency and magnitude of epidemics.
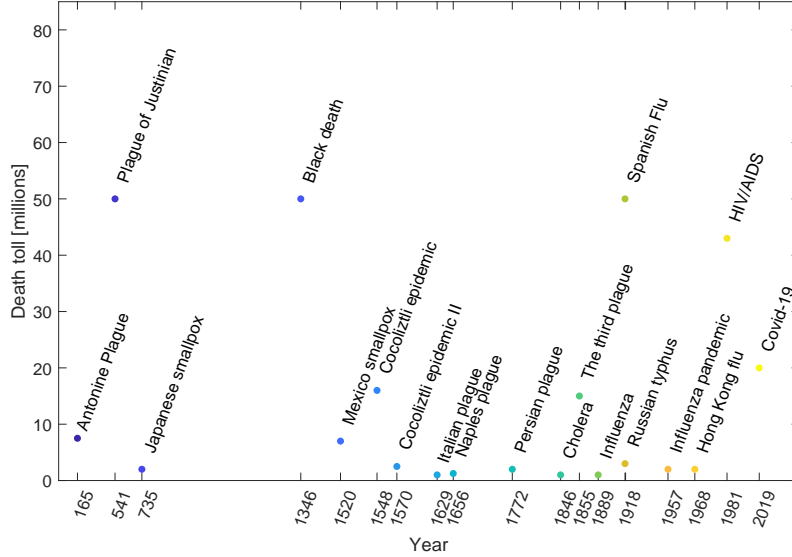

Figure 1.1: A graphical representation of the epidemic distribution over the years and of their associated death toll. It is observable how there are an increment in the number of these event in the last three century.

As it can be seen in 1.1, even if some improvements in health status were achieved, the presence of new disease potentially escalating in an outbreak is a concrete menace that needs attention and consideration on health policies.

However, the health status is not the only aspect concerned with a disease. Being sick causes profound modification in our relationship, work, and social life, for example, causing a deterioration of also the social health. There is also an economic cost associated with the cure necessary to be healed. Only in a few nations worldwide, is treatment covered free of charge by the state. In the majority, being ill can result in having to sustain high costs, causing people to go into debt or not take care. All these effects sum together and influence how populations behave when facing an epidemic. What are the consequences of adopting a certain behavior during a disease outbreak? It's a crucial question and can help to understand how to develop more efficient policies for contrasting epidemics. It is also the question that represents the first objective of study for the present work: can a multidisciplinary-based model be developed, integrating social and epidemiological features and analyzing their mutual relationship during a disease spread?

Taking a step back, it's important to understand why epidemic models are crucial and what this field of research entails. When a new disease emerges, the primary objective is to develop a defense against it. This begins with an epidemiological investigation to understand the disease's origin, the biological mechanisms behind its spread, and its resistance to existing drugs. The goal of this investigation is to gather all available information and understand the unfolding situation. The next critical step is to study the disease's dynamics and create a model to predict its evolution. This requires understanding and

estimating various parameters related to the disease. Key factors include the transmission mechanism within the population, the reproductive rate of the infectious agent in the host, the acquisition and persistence of immunity, and the contagion mechanism. Developing a reliable model is scientifically valuable and serves as a powerful tool for stakeholders. It helps in formulating the best policies to implement during a pandemic emergency. For this purpose, theoretical epidemiology must adapt to provide insights and policy advice. Data acquisition and analysis are fundamental for statistically modeling epidemic coefficients. It is crucial to verify theoretical models against real-world data, as unverified theories cannot be validated. In the end, a model capable of answering stakeholders' questions and making predictions, with or without the implementation of safety regulations, has numerous effects and outcomes on society. It is important to remember that, beyond the economic costs associated with every illness, there are also social costs. Developing instruments to better understand the spread of a disease can help mitigate its impact and lessen the social burden on society also preventing the loss of many lives.
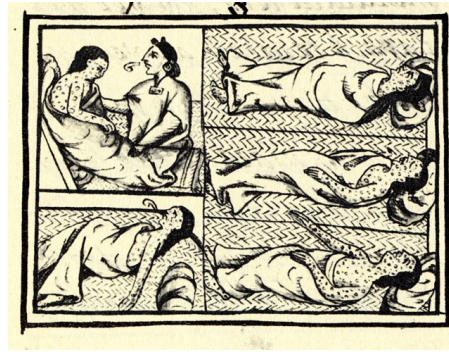
Figure 1.2: Representation of smallpox disease on the Mexican population in the XIV century. Figure from the Florentine Codex [3].

A first straightforward example, of the possible beneficial effects of having an epidemic model, is the possibility of arriving at the synthesis of "simple" parameters, specific for the disease under study, that can respond to questions like:

- is this disease so infective that can cause a pandemic?

- what are the threshold conditions that can cause an outbreak?

At first glance, the problem may appear straightforward. However, the creation of a model capable of evaluating every disease remains an unsolved challenge. Research in this field necessitates a careful balance between simplification and maintaining accuracy.

Over the past century, various aspects of epidemics have been extensively studied. Notable achievements by scientists include:

- Development of epidemiological models using different mathematical tools, such as differential equations and agent-based networks.

- Predictions about the progression of epidemics or reconstructions of the events' dynamics.

- Insights into epidemics, explaining phenomena like the periodicity of re-infection for certain diseases or the seasonal patterns observed in cases such as influenza.

- Understanding the effectiveness of specific strategies against outbreaks, such as vaccines.

Furthermore, by using multilayer networks or systems, more complex analyses can be performed. The objective is to create models capable of simulating the evolution of multiple phenomena simultaneously or to develop a more accurate representation of the real world by constructing more intricate scenarios. Examples of such models include:

- The simultaneous evolution of two different diseases.

- The formation of public opinions during an outbreak.

- The progression of a disease for which a vaccine exists, but where there is public fear of both the disease and potential vaccine side effects.

The work presented in this thesis is part of this type of research. A new model has been developed based on a study of existing multi-system models that integrate epidemic and behavioral or opinion components. Specifically, it is considered interesting to simulate a scenario in which the population is divided into three groups: those who have a positive opinion about the use of safety procedures and follow rules to avoid the transmission of the disease; a second group that opposes following procedures or recommendations, refusing to modify their behavior; and a third group that lacks information about the epidemic situation. This third condition represents the majority of the population when a new disease emerges, as there is limited information available and not yet widely disseminated. In the model, the main mechanism used to change behavior among different social groups is peer pressure. The fatigue from belonging to a certain behavioral spectrum (either being compliant with the rules or against them) is also considered. An epidemiological model capable of tracking both the initial phase of an epidemic and successive waves of contagion is developed, including the possibility of reinfection. A last feature of the work is the verification of its prediction with the use of real data. It is important for a novel multi-system model like the one implemented to have a comparison with reality. When two separate models are coupled together the result is not simply the sum of their previous characteristics, but other different phenomena can arise. Having a study, like the one performed by Meta during the Covid-19 pandemic about people's behavior is an incredible source of data that can permit us to interpret correctly the results given by the model. For this reason, the model's ability to reproduce what happened during the pandemic was initially tested. Then it was verified whether the peer pressure effect alone was sufficient to obtain the observed trends in population behavior or whether the addition of an external, global control factor, such as that represented by special laws implemented by governments, was necessary.

The main result achieved is that the model is functional and demonstrates reliability when compared with COVID-19 data. The study highlights the crucial importance of respecting quarantine measures and avoiding contact when infected, as these actions can significantly reduce the model's reproduction number, thereby limiting the spread of the disease. Altri risultati arriveranno concludendo la tesi.

Chapter 2

Main objectives and summary of the contents

In this chapter, the main objectives pursued with the current work are presented and it is described the composition of the thesis. Starting from an analysis of the theoretical contributions already developed for epidemiology, and in particular focusing on multilayer systems and mean-field models, the following questions are studied:

- how can be modeled in an effective way population behavior? What are the characteristics that must be considered?

- Can people's behavior influence the development of an epidemic?

- Is it sufficient to stop an outbreak by relying on the natural subdivision of the population into compliant and non-compliant groups regarding safety measures, or is the intervention of a central "controller" necessary to set new behavior rules?

- What is the most efficient way to model population collective consciousness about the epidemic's development?

About the last question, consciousness or awareness is a parameter considered useful to gain insight into society's reaction to the disease. Furthermore, there can be differences in behavior when the same conditions occur at different times, such as at the beginning of an outbreak versus several months later. The quantity and quality of information available to the population can make a difference in how deal with difficult situations.

Starting from these questions, the following objectives have been identified:

- Create an original epidemic-behavioral multi-system model capable of tracking the development of a disease and representing behavior modification using a peer pressure mechanism within the same population.

- Add a second control agent to the model, represented by government rules that can modify people's behavior in a centralized way.

- Develop a comprehensive analysis of the epidemiological and behavioral model to understand its mechanisms and correctly interpret the mutual effects arising from the coupling of the social and health systems.

- Conduct a study using available data on population behavior during the COVID-19 pandemic to verify if the developed model can accurately reconstruct events and how people reacted to them.

The work consists of an introductory chapter where the main concepts of social science and epidemiology are presented. This chapter provides all the necessary information to understand the research. It includes a glossary of the most important terms and an overview of the mathematical tools used in epidemiology. Additionally, the different models implemented to simulate an epidemic are shown, with a focus on the properties of the mean fields model, which is the primary model used in the thesis. Furthermore, a historical background of the research field is provided to give a perspective on the principal milestones. In the third chapter, a review of the literature analyzed for the thesis is presented. The articles are categorized into different main topics: epidemiology theories, opinion models, behavior models, and multi-agent and multi-system models. This subdivision highlights the most interesting aspects of each work and identifies the elements that have been considered for inclusion in the thesis. The second part of the thesis is composed of three main chapters. In Chapter Four, the chosen epidemiological and behavioral models are simulated, and their characteristics are studied individually. Chapter Five presents the model resulting from the integration of the two, which becomes a layer of a more complex multi-system model. The main features of this model are analyzed using analytical tools. The study is performed with theoretical values, sensitivity analysis, and the measurement of principal metrics, such as the reproduction rate, to provide a general description of the model and develop an understanding of it. In Chapter Six, the data analysis is presented. The data available from the Meta COVID-19 research are used to test and validate the model, assessing its ability to represent a real scenario. Finally, the last chapter contains the conclusions of the thesis.

Chapter 3

Theoretical background

3.1 Epidemiological theory foundations

Having a clear description of the main concepts in social science and epidemiology is essential for understanding the rest of the work developed. In the first section of the introduction, the theoretical basis and main concepts that will be used in the present work are defined. First, a brief historical review of the milestones in the field of epidemiology is provided, followed by a glossary of the main terms used in this thesis. Finally, the most commonly used mathematical tools are presented.

3.1.1 Epidemiologic research historical background

The research field regarding the development of technique to understand how epidemics can evolve during time has a history starting back in the 20th century. The first important discovery in this field must be attributed to the scientists that find the mechanism used by disease to spread. A first innovative work is the one done by John Snow, that during an epidemic of Cholera in London in 1854 successfully determined the source of the infection, even without knowing its etiological agent. Then advancing in the microbiological research is conducted by Pasteur and Koch. They found the etiological agent of disease, enabling the possibility to treat and prevent people from an infection. Then, Hamer work in 1906 added a first major theoretical contribution. He formulated a theory about the correlation between the course of an epidemic and the interaction, contact ratio, between susceptible and infectious individuals. It is the so called "mass -action" principle. The number of contacts between these two groups determines the spread rate of the disease. This law originally written in discrete time, is then updated in 1908 by Ross, that re-written it in continuous time. For the first time the problem can be studied using a clearly, well defined mathematical theory. Then the contributions of Kermack and McKendrick in 1927 add another fundamental principle to the modern epidemiology. They formulated a threshold theory explaining which condition can generate the development of an epidemic. The theorem affirms that a certain value must be exceed, depending on the proportion of susceptible and infectious individual. Controlling this value permits to understand if the number of infections will increase, until a peak is reached or if the epidemic is a descendent phase [4], [1]. Their contribution with the mass action principle represents the base for the

mean field model theory, that will be presented and analysed in section 3.3.1.

3.1.2   Epidemiological glossary

To permit a better comprehension of the subject analyzed in the present work a list of principal concepts and terms is presented.

Micro and Macro parasite

The first difference when presenting infection is distinguishing the type of origin that can cause it. An etiological organism responsible for a disease can be divided into microparasite and macroparasite. The former live and reproduce within the host, generating an immune response and the infections caused by them usually have two possible outcomes: death or immunity. Infections origins from them are shorter than the life span of an individual, and so have a transient nature.

Types of infectious diseases

Infectious disease is indicated as an illness resulting from the presence of a pathogenic microbial agent. It is possible to distinguish a difference between *transmittable* and *communicable* disease. A transmittable disease can transmitted between persons through unnatural forces. A disease is communicable when the transmission happens directly or indirectly.

Disease transmission

A disease can spread in different ways:

- person to person, for example sexual transmission, involving direct or indirect contact.

- airborne, through inhalation of infected air.

- food or water borne, ingesting contaminated food.

- vector born, the contagion is caused by infected animals.

Furthermore when the diffusion is among the same generations is called horizontal transmission, while vertical transmission is the one developing between different generations, from parents to children. Zoonosis is the phenomenon in which a disease that starts in an animal species mutates and attacks humans. The opposite can also happen and it is called inverse-zoonosis.

Epidemic disease

It occurs when there is a disease with a rapid outbreak, in less than a year there is its development and a peak is reached. The illness is confined to a limited region

Endemic disease

It is a disease that lasts for a long time and requires consideration of its impact on population renewal and in the number of susceptible individuals.

Pandemic disease

It is an epidemic that diffuses across multiple regions, on a global scale. The severity of the disease also makes a distinction in calling a disease a pandemic. For example, a common cold is diffused in the whole world but is not defined as a pandemic by the WHO (World Health Organization).

Incubation, Symptoms, Infected and Infectious

A person having contact with another infectious individual, can or not become infected. The incubation is the period after being infected in which the infection increases in size or number in the host but does not produce the symptoms, that are the effect of the disease.

Outbreak

Is the rapid raise in the number of infected during an epidemic.

Incidence and prevalence

The first term refers to the number of new cases after a certain period (daily or weekly for example), while prevalence is the proportion of the population affected by a disease in a specific time.

Immunity and herd immunity

Immunity is protection from a disease gained after having contracted it. It can last forever or vanish after a certain time. New exposure to the virus does not lead to infection. Herd immunity is instead an important phenomenon in which the defense against a disease is due to the majority of the population being immune, thanks to vaccines or surviving it. This majority protects the rest of the population because slows down the diffusion or stops the spread of the illness.

Virulence and Contagiousness

Virulence is used to describe how aggressive, harmful, and pathogenic is a biological agent in attacking cells. Contagiousness is referred to an individual and it is used to represent the capability to transmit a disease.

Overdispersion and Superspreading

Overdispersion is a term that refers to observing a larger variance than expected from a normal distribution. It is used in statistics to measure superspreading, a circumstance in which there is an anomaly (higher) number of secondary infections.

CFR, IFR and mortality excess

The case fatality rate is the ratio between the number of deaths due to a specific disease and the total number of confirmed positive cases. It is the likelihood for an infected person

to die because of the infection. The infection fatality rate is instead the percentage of people infected with the disease that are expected to die. The two quantities can have a similar value: if every person who contracts the disease and every death attributable to the disease is known and recorded, then the CFR will equal the IFR. The excess in mortality can be calculated by observing the difference between the total death rate (due to any reason) a month per month in a comparison between a year with an epidemic and one without.

Reproduction number R_0

It is the fundamental measure of the infectiousness of a disease. It is the average number of secondary infections caused by one infected person in a fully susceptible population. If it is recalculated during the epidemic progression is called $R(t)$, a time-varying reproduction number. Finally exist also the effective reproduction number, describes the average number of secondary infected people during the epidemic (rescaling the value with the true number of susceptible).

Incubation period and serial interval

The incubation is the time after exposure in which the disease develops and ends when the infected start to show symptoms. The serial interval is instead the time that exists between two transmissions in a chain of infections.

3.2 Opinion/behaviour glossary

To establish a framework suitable for developing and understanding behavioral models, the following key concepts from social science are outlined.

Awareness

It is the knowledge that an individual has on a certain subject or situation. It changes with time and it is developed with information or experience.

Information

The term "information" is often understood as "knowledge communicated." However, due to its central role in contemporary society, there is extensive debate regarding its multiple meanings [5]. Our society can be characterized as an "information society," where the development of information technology has influenced all aspects of life. Currently, the word "information" is associated with two principal meanings. The first, more universal definition, is that information is anything important in answering a question. The second relates to Information Science, the discipline focused on managing this object called information in any form.

Belief

It is the conviction of the truth of a statement or the reality of a being or phenomenon, especially when based on the examination of evidence, but also on matters for which there is no proof.

Studying beliefs change over time and on social networks is an interesting research field. It has the aim to understand why in certain societies new beliefs - such as opinions on climate change or anti-vaccinism spread more quickly than in others. These dynamics can lead to polarization or consensus and occasionally cause backlash effects.

Behaviour

It is how one acts or conducts oneself. It can depend on the response of external stimuli and have effects, especially on others.

Trust

It is the sentiment of confidence associated with the ability, strength, and truth of someone or something.

Perception

It is the mechanism for which something is regarded, understood, or interpreted.

Group decision-making

It is a phenomenon at the intersection of psychology, management, biology, and applied mathematics studying how people in groups interact, exchange information, and realize decisions. The decision made by the group is no longer attributable to any single individual but to the whole group.

Threshold theory

It is a theory formulated by Granovetter in [6] regarding collective behavior. The theory posits that in a society where individuals face two possible alternatives, and their choices involve certain costs and benefits depending on how many others choose each alternative, an individual will decide based on the number of others who have already chosen a particular option when this number exceeds a certain threshold.

Polarization and Consensus

Polarization refers to the variation in beliefs within a population.There are multiple meanings that can be associated to this term. One way to describe it is by measuring the distance between the most extreme beliefs, their spread or at the distribution of opinions in a certain interval.

The use of groups, identifying different concentration can be useful to explain the polarization phenomenon. Groups can be "exogenously" defined where are identified by characterstic like regionality, ethnicity, sex, education level, or other cathegories. To describe

correctly the polarization a clear definition of the groups in consideration is necessary. A first polarization group based is community fracturing, where the population is divided into subgroups[7] .

This contrasts with the term "consensus," which describes a situation where the exchange of opinions, information, or property among individuals leads to general agreement. Both scenarios are studied and modeled using network theory.

Homophily

The tendency to bond and associate with similar others.

3.3   Epidemiological models categorization

Starting from the observation of the real world, the desire to understand better a certain phenomenon is the fundament of mathematical model development. A perfect model does not exist, because it is based on data or on assumptions that are incomplete w.r.t reality. However, a useful model guarantees the possibility of realizing general predictions and can be a powerful instrument for policymakers. For example, an application is the estimation of certain policies' effects on the population during an epidemic. So, the aim is to have meaningful results, under a given set of real-world circumstances. When working on a model the importance of the uncertainty related to claims realized with this instrument must always remembered. This concept is remarked also on the definition of mathematical model present in [8].

> "A Mathematical model is a self-contained collection of one or more variables together with a set of rules (usually formulas and equations) that prescribe the values of those variables. Models serve as an approximate quantitative description of some actual or hypothetical real-world scenario. They are created in the hope that the behavior they predict will capture enough of the features of that scenario to be useful."

There are several different typologies of mathematical models developed. A first classification can be done considering the method used to obtain them: mechanistic, empirical, or conceptual models. The mechanistic model is based on assumptions about reality, or theoretical principles, modeled using a collection of one or more variables together with a self-contained set of rules. These models have an explanatory value on the reality they represent. Empirical models are realized by fitting a set of data. It is a powerful instrument, because data can be modeled quite well, but they lack the explanatory value of the mechanistic model. Finally, with conceptual model, it is meant a verbal description of a real-world scenario.

For the present work, the mechanistic model is the strategy used. This is because, in the epidemiology field, the scopes that conduce to the realization of a model go beyond just fitting data. Examples of possible scopes are:

- following the epidemic evolution

- collect and realize a structure to understand the information related to the disease

- obtaining general insight about control strategies

- realize predictions

Considering the mechanistic category several different types of models have been developed or are adapted to be used in epidemiology modeling. In this section, the principal typologies are now introduced.

There is then a focus, in 3.3.1, because it represents the mathematical base model of the multi-layer system implemented in the present work. It is important to introduce the logic underlying its structure, its main mechanisms, and the first important conclusion that can be derived from it because it is a useful introduction to the approach that will later be employed in the rest of the thesis.

3.3.1 Mean field models

The mean field model, also known as compartmental model is the first developed and most studied type of mathematical model used in epidemiology [9], [10], [1], and [11]. This model assumes that the well-mixed population is divided into several subgroups. Each compartment is a different stage of the disease under consideration. Some possible states are susceptibles, asymptomatic (infected), symptomatic (infected), infected, exposed, vaccinated, quarantined, dead, recovered, and hospitalized. The classes considered in the realized model determine is base structure. The choice to include a certain compartment depends on the disease that is modeled and on the assumptions that are under analysis. Different models can be suitable to analyze the same disease but can be used with different aims. The difference is that a more complex model can emphasize some aspects or effects of the disease, that are not highlighted by a simpler one. In this class of model, the severity of infection usually is not considered: people are infected or not. The transition from one compartment to another is determined by differential equations. There are parameters ruling the transition between groups that are coefficients whose meaning depends on the model's underlying assumptions. The most important metric in this class of models is the so-called "basic reproduction number". It expresses the seriousness of a disease and it is considered the fundamental threshold in epidemiology [12].

SIR model

The model that represents the base for epidemic mathematical studying is the SIR. Here the population or density of individuals is divided into three groups: Susceptible, Infectious, and Recovered. At time t the three groups are identified with the symbols: $S(t)$, $I(t)$, and $R(t)$. The symbols used to indicate the density of each group are s, i, and r, while the capital letters are used to specify both the name of the groups or the absolute number of participants in each one. Usually, the assumption that N remains stable is made. This is possible considering that the term epidemic identifies a disease with a duration much lower than the life of a person. Considering this assumption the number of deaths and births can be neglected. Alternatively, we can consider that the number of births, which is an influx in the S compartment is roughly equal to the number of deaths, which is an outflux. These statements imply $s(t) + i(t) + r(t) = 1$ and $\dot{s} + \dot{i} + \dot{r} = 0$. The disease reproduces horizontal incidence within the population. So, it is the connection with others that causes
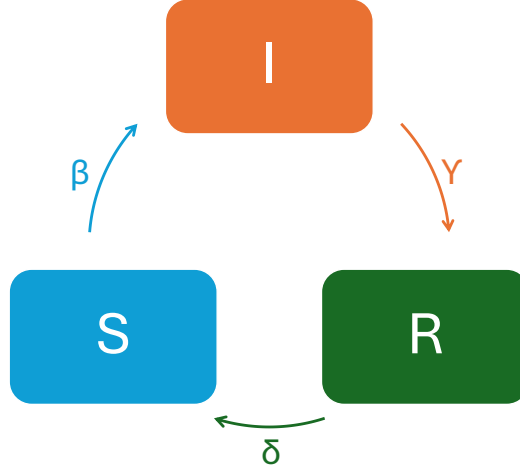
Figure 3.1: An example of the graph structure of a mean field SIRS model. There are three compartments and the flow rate between them is ruled by the coefficients β, γ and δ.

the infection diffusion. β is a coefficient that expresses the number of adequate contacts on average of a person per unit of time. The simplest way to explain the meaning of β is to consider that not every contact between a susceptible and an infected person can generate a contagion. The formula $\beta \frac{I}{N} = \beta i$ is then the average number of contacts with infectives per unit time of one susceptible. Finally, the number of new cases per unit of time due to the $S = Ns$ susceptibles is $\beta \frac{I}{N} S = \beta N i s$. This form of horizontal incidence is called the standard incidence. In this incidence, there is no dependence on population size [13]. This is because the contact of individuals daily is independent of the country dimension in which they live.

The second transition process in the model is that individuals move from the infectious state to the recovered one at a rate γ. So the infection duration lasts an average time of $1/\gamma$ days. The γ parameter represents the healing or recovery rate of the population. A mathematical interpretation of this term is that corresponds to exponentially distributed waiting times in the I compartments. The transfer rate γI corresponds to $P(t) = e^{\gamma t}$. It is the fraction of infected that are still in their class after time t of entering it and with a mean waiting time of $\frac{1}{\gamma}$.

In this initial model, the immunity acquired after recovering from the illness is lifelong. It is equivalent if after being sick a person recovers or dies because it is considered that it will not transmit the infection anymore. This assumption can be modified and there are often diseases in which after a certain period individuals become again susceptible. Another initial simplification is the one of considers the coefficient β and γ constant.

Although the SIR model is quite simple it can predict a very important aspect of an epidemic, the threshold value. This effect was the main novelty studied in the pioneering work of Kermack and McKendrick [9]. They found that in a fully susceptible population if and only if R0 > 1 an infection can start diffusing. Thus the origin of the term "threshold value" in epidemiology.

The dynamic of this system has been extensively studied and analyzed during the years [14], [15], [16], [8], [17], [18], [19], and [20]. The threshold effect distinguishes between two scenarios. The first is when $R_0 < 1$. It is called "free disease equilibrium. In a fully susceptible population, a disease with a R_0 less than one does not spread. The initial small number of infected tends to zero and at equilibrium the whole population has remained in the S group. This state, as proved in [12], is globally asymptotically stable. The second case is when the threshold is larger than one. Here, the number of infected grows until a peak and then decreases to zero. Both this max value of infected and the final number of susceptible can be calculated knowing the system's initial condition (s_0 and i_0) and the value of β and γ, as explained in [13]. This second scenario analyzed with a simple SIR model permits us to immediately understand how a very aggressive infection can spread across a large part of a population. In this situation, if no countermeasures are taken in time, there can be severe consequences on the society, with social and economic damages.

Derivation of I evolution and SIR differential equations presentation

After having theoretically presented the model, it is now explained one method to derive the mathematical form of the infection compartment evolution. The set of differential equations that describe the dynamic of infection is the following:

$$
\begin{cases}
dS(t)/dt = -\beta S(t)I(t) \\
dI(t)/dt = \beta S(t)I(t) - \gamma I(t) \\
dR(t)/dt = \gamma I(t)
\end{cases}
\tag{3.1}
$$

Here $X(t)$ is indicated as the population at time t in the X compartment. Remember that the assumption of constant population size is also done, so $S(t) + I(t) + R(t) = N$ holds.

The number of infected, with an interval Δt, that in a base case can coincide with one day, is given by the equation:

$$
I(t + \Delta t) = I(t) + [\beta S(t)I(t)/N - \gamma I(t)]\Delta t
\tag{3.2}
$$

If the value of N is large, the variables can be considered as continuous, and imposing a time interval close to zero it becomes:

$$
\frac{dI(t)}{dt} = \lim_{\Delta t \to 0} \frac{I(t + \Delta t) - I(t)}{\Delta t} = \beta S(t)I(t) - \gamma I(t)
\tag{3.3}
$$

Consider now the initial state of the system. At the beginning of the disease, considering that there are few infected, the majority of the population is in the susceptible groups, so $S(0) \approx N$. Furthermore, during the initial days of contagion diffusion, this quantity remains stable. Considering this approximation, we have

$$
\frac{dI(t)}{dt} = (\beta S(0) - \gamma)I(t),
\tag{3.4}
$$

which gives now a differential equation with only a variable, $I(t)$, that has a well-known solution:

$$
I(t) = I(0) \exp^{(\beta S(0) - \gamma)t}
\tag{3.5}
$$

27

From the analytic solution of the infectious dynamic equation in 3.5, we can see what happens at the beginning of an epidemic. If the exponential argument has a positive sign it is observed an exponential increase in the number of infected. While, in the opposite case, infected people tend to zero. The value $\frac{\beta}{\gamma}S(0) = 1$ is defined as the epidemic threshold. In the initial phase of the epidemic the relation $\frac{\beta}{\gamma}S(0) = \frac{\beta}{\gamma}N$ holds. This quantity, normalized, is called the basic reproductive rate, and indicated with the symbol R_0.

It measures the intensity of the contagion or the number of secondary infections a sick person can generate. Analysing the equation of susceptibles, with this model we see that it is always decreasing. In the SIR model, if the condition to start the epidemic is satisfied after an increase in the number of Infected, there is a point at which $\frac{\beta}{\gamma}S(0)$ becomes less than one. It is when this happens that the peak of the $I(t)$ curve is reached. Then, the disease begins its falling phase. It is the natural behavior of an epidemic.

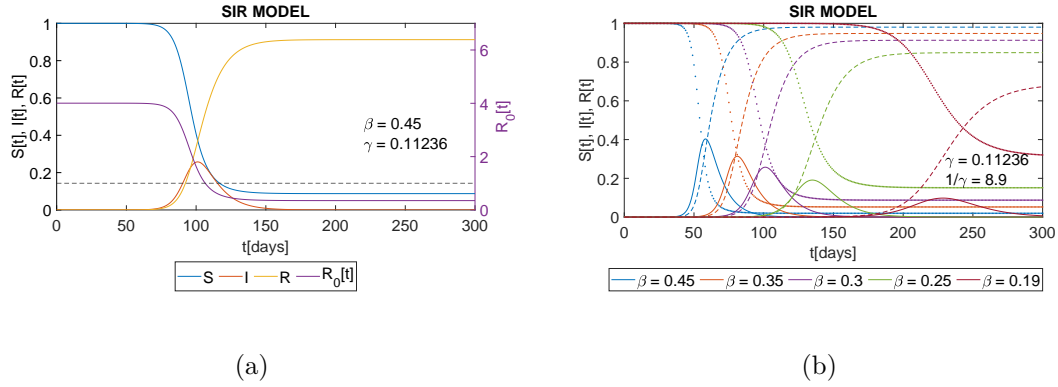(a)   (b)

Figure 3.2: SIR system numerical solutions. Figure a) shows the evolution of compartments in the case of an epidemic. The violet dotted line represents the time-dependent $R_0(t)$. It can be seen that when this parameter is equal to 1, the number of infected reaches its maximum value. In b) are presented different evolutions of the disease varying only the β coefficient. The smaller its value the flattened and the more delayed the infectious curve is.

Other two interesting quantities to consider when a new disease appears are the rate of increase of the infectious and the final size of remaining susceptible at the end of the epidemic. There is a large difference when a population suffers from an epidemic if this ends rapidly because a lot of people get sick or if this number can be controlled, and the infectious curve is flatter. A strategy to flatten the curve can reduce the contact between susceptibles, actuating social distancing or avoiding contact with infected, implementing quarantine measures. These are two simple examples of actions that reduce the value of β. Another countermeasure is represented by vaccination. Its immediate effect on the epidemic is to remove susceptibles people, so the disease can afflict only a small group and be quickly extinguished.

Stochastic models

This is a group of models deriving by the mean-field, but using a different mathematical approach. In this typology, the transition from one state to another is determined using a function of probability. Conceptually are derived using the same framework used with ODE models. They are useful when the disease to study has a lower number of infected or if there is a connection between the epidemic outcome and changes in individual dynamics. This is called demographic variability, and it concerns changes in transmission, births, recovery, or deaths within the population. Using stochastic models with Monte Carlo simulations can be useful to investigate epidemic models on networks [21]. The two most important types of models using this approach consider the time variable as continuous, $t \in [0, \infty)$ and then the state variable is either discrete (Continuous-Time Markov-Chain) or continuous (Stochastic Differential Equations). Referring to the SIR model to make a simple example here the S and I compartments are modeled as random variables. The probability of individuals changing groups depends on infection and recovery, the possible events that can occur. It is called transition probability. In a Markov chain approach the transition probability is discretized, and there is no dependence on the history of the epidemic to know how it will evolve at time $t + \Delta t$. It is necessary to know only the current state of the process at time t. In the Stochastic differential equation, the random variables are continuous.

3.3.2 Networked models

[22], [23],

3.3.3 Agent-based models

VEDI E AGGIUNGI ANCHE [24]

DA WIKI INTEGRA: "An agent-based model (ABM) is a computational model for simulating the actions and interactions of autonomous agents (both individual or collective entities such as organizations or groups) in order to understand the behavior of a system and what governs its outcomes."

Agent-based models are an alternative technique used to represent disease evolution. This approach is implemented based on the observation of spontaneous connections made by individuals. The evolution of a disease is considered over complex and realistic networks. The focus of this type of model is to understand how the network structure influences the epidemic by observing parameters such as the rate of spread.

In this framework, the nodes of the graph represent individuals (with all the properties that the modeler deems relevant for the study), while the edges represent interactions between people. Nodes can also represent subgroups of people, and using weights on edges makes it possible to characterize the strength of these interactions.

An advantage of this type of model is that it offers a very intuitive approach to epidemic modeling. Using an individual perspective guarantees an immediate interpretation of the model. A precise agent-based model can provide a greater understanding of the illness under consideration and direct information about countermeasures to implement to stop or mitigate its spread. However, to be powerful and capable of performing good analyses

and predictions, a lot of information must be integrated into the model. Only if the agent-based model is highly capable of realistically reconstructing a real network can it be a reliable instrument, and achieving this requires very complex work.

Figure 3.3: Agent based network representation

An example of a possible mathematical implementation of this class of models is now briefly presented. One possible technique to describe peer-to-peer contact in a graph structure is realized through a probabilistic framework. Here, assuming a total of n agents in the model, spread processes can be described as a function of probability. Using Markov processes, each agent has a certain probability of transitioning from one state of the disease to another. To calculate the value of these probabilities, both the information derived from the network structure and parameters related to the disease, such as infectivity and recovery rates, are used. In this way, a stochastic evolution model of the processes is developed.

Considering an SIS model described with a Markov process: it has a dimension of 2^n, while implementing an SIRS model requires a dimension of 3^n. Because the size of models developed in this manner becomes rapidly enormous, a mean-field approximation is employed. It is based on the assumptions of a network composed of a sufficiently large number of agents and on the independence of these nodes. With this technique, by taking expectations, the transition rates of individuals are approximated. Using these approximations, the boundary values of the agent's probability of infection can be determined at each time step [12].

3.3.4 Multilayer systems and networks

AGGIUNGI ANCHE [25]

The complex dynamic of interactions existing in the real world, develops in multiple patterns, with complicated relationships. This connection can change over time, and using the theory of multilayer systems it can improve the comprehension of such complexity. Additional information can be added to the model, for example, different types of interactions, like physical contact or information sharing, time dependency coefficients, or reliance between different parameters in nature, creating cause-effect relationships. It is a more recent development of the research, the traditional network theory was revisited, to create a framework that can include multiple networks, that evolve and influence each other [26] and can be helpful to manipulate complex systems like human relationships. Some interesting results obtained are the possibility that the onset of one disease can depend on the onset of the other one. There can be regimes in which the criticality of the two dynamics

is interdependent and others in which the critical effect is only one-directional [26]. One possible way to develop models with this structure is to imagine that each layer represents a different type of interaction. An epidemiological example is a layer in which the physical contact between people is simulated and another represents social structure, the network of relations that every person has. This instrument provides a natural representation of coupled structure and dynamical processes. It has been presented in multiple works in the past years, for example in CITA. The dynamic realized in multiple systems can be single or coupled. In the first, there is a top layer with its dynamic evolution running on top of a multilayer network. The coupled structure instead is the one in which the phenomena described in each layer evolve with the influence of what is happening in the other. Multilayer networks have multiple dimensions of connectivity, called "aspects" and they have to be considered simultaneously. They can also be considered with two different mathematical structures.
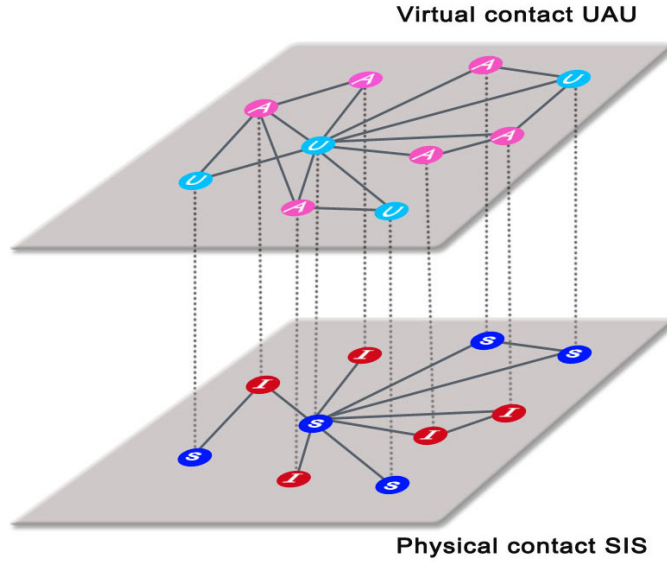

Figure 3.4: Representation of a multiplex structure. The figure is taken by the work of [27] and shows the network implemented in their model. There is an awareness and an epidemic layer. In this case, the node connected with the interlayer connection represents the same individual.

The first uses the same set of compartmental structure and mean-field models presented in the section before 3.3.1. Here, from a mathematical point of view, there is no such difference in the manipulation and analysis of the system. The distinction relies on the meaning of the compartments and parameters created and on the dependence of the coefficients, which can be time and state-dependent.

The second option considers an agent-based structure. Here, considering a graph structure, composed of nodes and links between them, it is possible to classify three types of edges:

- intra-layer edges, the connection of nodes on the same layer.

- inter-layer edges, the connection between a replica of the same node, but lying on a different layer of the structure;

- inter-layer edges, but coupling nodes representing distinct entities.

A possible representation is done using a 4th order tensor or coupling a set of adjacency matrices, called "supra-adjacency matrices". The feature that can be studied is the structural properties of the network, depending on how the various layers are coupled together. The presence of clusters or most central nodes is also important.

Chapter 4

Review of epidemiological behavioural and opinion models in literature

The scientific community's interest in epi-behavior models has existed for several years. Initially, as noted by [28], the behavioral aspect of epidemiology was not given significant attention. Its development has been a gradual process, resulting from years of evolution in research.

In fact, in the initial works [9], the focus of scientists was primarily on describing the evolution of diseases. The resulting models did not account for the effect of behavior; the population was considered homogeneously mixed, leading to random contact between susceptibles and infectives [4, 12]. It was only later, as epidemiological models proved effective and reliable in describing and predicting disease spread, that interest among policymakers grew. Tools capable of integrating real data with epidemiological models emerged, aiding decision-making on matters such as the duration of school closures or travel restrictions, as described in [28].

Furthermore, new categories of models have emerged, such as agent-based models, networked models, and multi-layer/multi-system models. Despite their differing approaches, these models aim to integrate various population characteristics—such as contact structure, age distribution, and movement patterns—to address the limitations of the original homogeneity assumption [10]. This focus on societal composition and behavior naturally stems from the desire to use modeling tools as a reference for decision-making in safety and health. One possible approach to incorporating changes in the structure of models that describe aspects of behavior or population composition is to do so implicitly.

In these models, the behavior of the population is implicitly considered by integrating time-variable parameters that capture changes in societal behavior. This approach represents the classical modeling technique used in the formulation of epidemiological models. Examples of studies that have utilized this methodology for analyzing COVID-19 include [29–31].

Although models developed in this way have proven to be powerful tools for generating insights about disease dynamics and providing recommendations to policymakers, they

fall short in their ability to accurately reconstruct how populations behave during an epidemic outbreak. The desire to explore this aspect and develop a framework capable of simultaneously simulating both behavior and disease diffusion—where each mutually influences the other—has driven the development of a specific research field dedicated to behavioral epidemics.

But how can behavior be integrated into pre-existing epidemiological theory? To better address this question, we follow the classification proposed in [32], which offers a possible subdivision of behavioral literature based on the different approaches that most articles focus on. Three major categories emerge:

- The source of information used to make decisions;

- The type of information used to make decisions;

- The effect of behavioral change on the dynamic described by the model.

4.1 Information's sources

When analyzing the source of information, there is a clear distinction between works [33] that assume governments and populations base their decisions on precise data, such as the number of infected individuals (prevalence) [34, 35], and those that consider more informal sources, such as conversations between people, public opinion, or media representations of the situation [36, 37]. These media sources include both traditional outlets like television and newspapers, as well as newer platforms like social networks. This distinction highlights the diversity in how behavioral factors are integrated into models, reflecting the varying degrees of reliability and influence these sources have on decision-making processes during an epidemic.

Regarding information quality and the negative effect of misinformation spreading within the population, an example is the fear of vaccination [38]. Several works analyze the effect that fear of vaccination has had on the spread of infection [39, 40]. An example of how this phenomenon can arise is the story of an article originally published by a prestigious source. Even though the thesis presented in this work was later proven wrong by the scientific community [41], the negative impact in terms of spreading fear about vaccines has persisted and, in many cases, has become deeply ingrained. In this specific case, it caused a decrease in herd immunity and a resurgence of measles [28].

4.2 Classification of different types of information

After introducing the impact that information quality may have, another interesting aspect is related to the different types of information used in model development. Some articles focus on the effect of media on behavior [34, 42], while others consider peer-to-peer conversations, information exchange, and beliefs among individuals [35]. These are completely different approaches, even though they aim to achieve the same effect: simulating the evolution of people's opinions and behavior. Using media involves hypothesizing that the population is influenced by a few "central" information nodes, so the same news, data,

or future predictions are shared with everyone. In contrast, models that use personal information exchanges can depict a scenario where many different ideas about the disease situation circulate simultaneously. Another concept used in models that simulate a sort of "collective consciousness" is referred to as "awareness" [43]. To model how awareness spreads in the population, it is often treated like a disease [**Granell_2013**, **Granell2014**, 25, 44–46]. Although there are many differences between these two, the main idea is that theories and concepts about a certain topic can spread among people, which can be considered at a higher level as a unified opinion. For example, there may be many different personal positions on how to respond to a health emergency like COVID-19, but it is possible to abstract the various opinions and reconstruct what the majority of people, or macro-groups, ultimately feel. They may either be more cooperative and in favor of following guidelines issued by authorities, or more focused on their well-being and inclined to act independently. This process can be related to opinion formation studies, which aim to understand how people form their ideas [47, 48] and also analyze the possible formation of opinion distributions, such as perfect consensus, consensus, polarization, clustering, or dissensus.

4.3 How integrate behavior in epidemiology

While the type and source of information are crucial for understanding the basic framework and synthesizing key concepts of models that consider population behavior, the final criterion used to categorize works related to epidemiological behavior is how the influence of people's behavior on the model is integrated. This aspect is one of the most interesting and was a key focus of the literature reviewed for this thesis, as it plays a significant role in comparing and selecting relevant works for this research.

There are various ways to describe behavior in response to an epidemic and integrate this aspect into epidemic models [25, 49, 50].

The first approach involves observing and simulating how connected individuals' states are linked to specific behaviors and how this influences the epidemic. This category includes agent-based models. Additionally, the reverse relationship, where disease spread alters individual behavior, has also been considered, as seen in [51].

There is also a broad class of mean-field models that explicitly consider the effects of behavior. In these models, time-varying or state-varying parameters are used, resulting in a non-linear system of equations where the parameters are not constant but change based on information such as disease prevalence [**CITA**].

Another possibility involves modifying the structure or connections in the network used to simulate disease evolution [**CITA**]. In network-based models, data extracted from social network structures [**CITA**] or small-world models [**CITA**] are often used to simulate connections between people more realistically.

In the following paragraphs, several articles are presented using this classification to simplify their categorization. Each article is then discussed in more detail, highlighting its original contribution.

4.3.1 Models in which change the individual' state

Chapter 5

Old review

The development of a epidemiological model, that can capture the evolution of a disease influenced by the behaviour of individuals, begins from a study and review of the most significant works already present in this research topic. These are the different main type of model that have been investigated:

- deterministic/mean field models

- opinion models

- multilayer networks

- opinion-disease models

Now it is presented for each of them, the main aspect and knowledge, useful for the development of my model.

5.1 Opinion models

In the analysis performed by Wang [25], are presented mechanism implemented to explain co-evolution spreading in complex network. The principal methodologies created over time are threshold model, that can use a linear threshold or a "Watts threshold". Here each node has a random different threshold, based on a certain distribution. Using a threshold means that a node change opinion on the basis of its neighbours' belief. The shape of the network is then fundamental for an opinion to spread. The best scenario is the one in which there is a low degree of randomness, and the network is regular. Also, cluster can have a reinforcement effect, if they are sufficiently connected to the resto of the graph. Their work then report analysis based on competition or cooperation of opinions "contagions". And a SAR model is presented. Similar to a SIR, here the meaning of letter A is "adopted". It means becoming convinced of a certain opinion, but with a probability or rate to then return to the previous behaviour.

Also the work of [52] define and test some different models based on trade-off between the benefit of having connections and the penalty for acquiring infections. It is showed that when the behaviour of people depends on maximizing their net benefit, the individual

risk perception plays an important role in the formulation of a cost function. The models derived with this so called co-evolutionary approach, have an overall dynamic very correlated between the two strati: it is a feedback loop between infection spreading, people behaviour adaptation and consequently structural modification in the network.

5.2 Multilayer network

One work based on feedback between two networks concatenated is the one performed by Peng et all, [53]. Here there is explained a model based on two graphs, where one simulates the evolution of a disease, using a SIR or SIRS dynamic, and another explicit the behaviour of individual in a UPAU network. U means uninformed, P pro-physical distancing and A anti-physical distancing. In this network the people's conduct influence the β coefficient of the epidemic diffusion. They demonstrate the effectiveness of having an opinion in reducing the negative effect of a disease and that lengthening the duration time for which an individual maintains opinion can help suppressing the transmission. Study the effect of competition in a multilayer network is the objective of Teslya et all research [54]. At cause of interpersonal communication individual can change their opinion. They are divided in two main groups, positive or negative w.r.t health conduct. Here is also inserted an influence due to assortatively when contacting with others. Their principal results further than the fact that opinion influence disease, is realizing a model in which the two opinions can coexist at equilibrium. There can be oscillation of prevalence due to increased transmissibility of infection. In SIR model they demonstrate a reverse correlation between the rate of social contact and the peak magnitude of infectious. The causes of oscillations in the disease dynamic are a high infection rate and a pronounced difference in infection rate between individuals with different opinions. The others important factors are the high-rate opinion exchange and high sensitivity of population to prevalence. In the article [55] the opinion about vaccination is taking in consideration, into a SIR+V mean field model. Conversating is the mean used by individual to modify their opinion. With a very positive opinion susceptible individuals can choose to take the vaccine. Interesting they use a r factor to describe the extremism in opinion. Varying this coefficient, they observe that the best scenario for delay the development of an epidemic is the one where the society is neutral. So, when there aren't compromise or persuasion, but the conversation is based on "rational" arguments. Another works analysing two competing opinion is [40]. Here population is sensitive to both fear of vaccine and disease. These two interact and the vaccination grow rate increases only if the fear of the disease is larger than the of vaccine. The infection curve is very influenced by the presented dynamic, and the best scenario is obviously the one in which the fear of vaccine does not exist. However, in the case where the two fears coexist there is an improvement in the number of infected, for multiple infection waves. The work by Auld [56], reflect an observed characteristic in society: pessimistic expectations over the future induce a more risky behaviours. This conclusion derives observing and simulation evolution correlated to the news about a vaccine. This knowledge causes a decrease in infection rate before the vaccine becomes available. Then there is a return to normal behaviour. If there are not information, pessimism cause more risky behaviour. In [**Sontag_2022**] there is another SIR and opinion model, with population that is divided in trusting and distrusting. They add in the model the effect of fading

and a global force, that simulates central interventions. The main interesting conclusion of their work is that strong public intervention have a similar effect to the network to the ones obtained if the population is composed of trusting and compliant individuals. However, higher percentages of distrusting cause the model to pass a phase transition where outbreaks cannot be suppressed. A different approach in using a multilayer network is the one realised in [57], where the social structure of a town is re-created. Every layer describes the places populated by individuals: from house, to work, distinguishing between different type of work, and considering a level for friendship. Each person is present to more than a layer and, in each layer, relates to different agents, based on the social group's provenience. Using this approach, they have found that the level in which is easier for an outbreak to develop is the one associated with friendship. Here the interaction is closer with others, the security level is lower. For this reason, a lower value of transmissibility rate β is sufficient to have an epidemic with many susceptible involved.

5.3   Opinion-disease model

The work done by Funk and its colleagues [32], it is very interesting: they collect and explain systematically the behavioural reaction of population in response to a pandemic. They classify the human behaviour subject to different possible sources of information. An information can be global available or local. This reflects the way it radiates or if develops in social cluster. Another important difference is related to objectivity. Certain information is based on belief and can change with time. This typology can be influenced by the social connections of an individual or by the influence of external agents, like media. Cognitive bias also can have an impact on our opinions: amplification, confirmation, anchoring bias. They then focus on the influence of self-initiated action in the control of disease diffusion. When an individual change its behaviour, form a modelling point of view this can influence: its probability to change state (from S to I for example). The value of β or γ. Modification in the contact network, with a self-isolation or adherence to more cautious conduct. Fear has an important role in how people face epidemic. Due to this emotion, people can decide to get vaccinated for example (or not, if they are more frightened by vaccines). Another phenomenon observed and influenced by fear is saturation. When there is many infectious people tend to decrease their number of contact and this cause a decrement in the I curve. Another multilayer network with two opinion, 0 where individual not take precautions and 1 where the protective measures are used, is presented in [58]. This model is associated to a SIS disease one. The article studies the stability of asymptotically equilibria of the system. Assuming different value of a parameter used to describe risk perception they found a set of final possible states. The most interesting is a stable asymptotical equilibrium in which there is a periodic epidemic outbreak and a consequently population behaviour response, changing behaviour to a safer. An analysis of people choices about vaccinations is done by [**Bauch_2012**], they study the feedback between the positive effect due to vaccination and the fear of being vaccinated. In fact, thanks to vaccines, the disease incidence can become very low, and the perception of risk related to them can seem larger. They implemented an approach based on game theory and using social learning. A possibility to integrate the effect of opinion in the dynamic of an epidemic, is creating different subgroups of susceptible. They are separated according to their level of opinion, and the less they belief

in use of NPI, for example, the higher probability of being infectious they have. This is the approach used in [35]. They also implemented different functions describing the influence between opinion and the possibility to become infected. The influence of media has also been analysed. This is interesting, because it's a communication channel that can be used by government, and so it is an available control measure that can be implemented, to try control the behaviour of population. For example in [34] a parameter depending on I value simulates the effect of media covering the news about the disease. Increasing the number of infectious cause, the creation of news and other media about it. These can have as effect to induce more people practice social distance for example. Study both the effect of media, see as a central node of communication joined with opinion evolution is done in [51]. Nodes co-exist into two layer, one for disease spreading and one for awareness, (unaware-aware-unaware model). In their application the awareness process without media, must reach a certain level on the transmissibility of awareness to influence the onset of epidemic. Instead, with an influence of media, greater than zero, this "metacritical" point disappears. A central broadcast, even with a small communication influence power, as a direct effect on all the network dynamic.

Part II

Behavioral Disease Model

Chapter 6

Epidemic and Behavioral model alone: a presentation

To develop a multi-layer network combining an epidemiological layer with a behavioral one, we first study the dynamics of each layer separately, as presented here.

In this section, we describe the developed SIRS epidemiological model, followed by the Careless, Compliant, Against behavioral model. A sensitivity analysis is also conducted. Understanding the underlying dynamics of each graph is crucial for better comprehending the dynamics emerging from the multi-layer structure.

6.1 SIRS model

β is the transmission rate parameter for person-to-person contact, γ is the recovery rate, δ is the rate at which immunity recedes following recovery, and $R(t)$ is the recovered fraction of the population

To describe the epidemic evolution a SIRS model is implemented. It is an extension of the most famous SIR. Its main addition is the possibility for individuals to become again susceptible after a certain period of time beyond the end of the infection. The choice of a SIR-like model is done because they are well-known as capable to describe disease like the COVID-19 CITA. From an epidemiological point of view, an "Exposed" compartment will be very suitable, to describe better the evolution of the disease. In fact, in this class of infections, after the contact with an infectious there is a certain period of incubation before the development of symptoms and contagiousness. Nevertheless this compartment was not insert in the model, because it was demonstred CITA, that also a more simple SIR can be able to model correctly the disease. In this case for realise a better fit of the real data a delay in the time scale of the system can be added in the model. This delay can be considered as an extra time to ... CITA E VEDI ARTICOLO.

The possibility of become again susceptibles is added in the model, because it is considered an interesting feature in the study of a long range time scenario. Considering the effect of people behaviour on the evolution of a disease, it is hypothesized that two keys moment of this influence can be the initial stages and after the first peak of epidimic. All'inizio il sirs si comporterà come un modello sir normal, perchè non ci saranno abbbastanza tempo

trascorso perchè le persone possano reinfettarsi. Però dopo le persone posso no reinfettarsi e la loro opinione e comportamento diventerà importante. da spiegare meglio

6.2 Behavioural model

The behavioural network alone is composed of three compartments. These are Compliant, *Co*, Careless *Ca*, Against *Ag*. The differential equations describing the model evolution are 6.1:

$$\begin{cases} \dot{Ca} = -k_1 CaCo - k_2 CaAg + \lambda_1 Co + \lambda_2 Ag \\ \dot{Co} = k_1 CaCo - \lambda_1 Co \\ \dot{Ag} = k_2 CaAg - \lambda_2 Ag \end{cases} \tag{6.1}$$

As initial condition the hypothesis is that at the start time of the simulation most of the population is in the Careless compartment. It is considered that if a new infection developed, it is not well known and so population have little information about it. The Careless compartment is composed by people that do not know about the risk associated with becoming infected, or that have not sufficient fear of the infection to modify their normal behaviour. As an example of this possible initial configuration it is considered the covid-19 case in Italy. At the early stage of its development, when the disease was spreading in China it was not considered a menace for most of the population in western countries. It is seen as a disease involving a different and far state. So, when the epidemic arrives in Europe and Italy, both the population and the government did not expect it and there is an initial time delay before the countermeasures were activated and before reliable information about the evolution of the disease are available to the population. There are then two opposite behavioural standings: Compliant and Against. In the Compliant set there are population worried about the disease and that want to reduce their possibilities of becoming infected. Conversely, the Against is formed by a group of individuals that have anti-scientific ideas about the disease. Here are summarised phenomena like:

- vaccine denialism;

- misinformation diffusion;

- refusal about existence of the disease;

- lack of trust on doctors and government policies.

For the study of model evolution different coefficient values has been considered. The rates studied in the models have the following meaning:

- k_1 influence rate between Ca and Co;

- k_2 influence rate between Ca and Ag;

- λ_1 rate of leave compliant behaviour due to fatigue;

- λ_2 rate of leave against behaviour due to fatigue.

44

The behaviour of the model is influenced by the value of each of this parameter. For example if the compliant have strong influence, the equilibrium of the model will be composed by most of the population with Compliant behaviour and an Against groups that tend to zero. On contrary, the opposite group composition will be the result. However, if the fatigue due to being Against is less than the one related with being Compliant the final equilibrium can be favourable w.r.t the Against group, even if the rate of $k_1 \geq k_2$. These effects can be explained looking at the equilibrium for time that goes to infinite. It is found that depends on comparison between the ratios that can be calculated with the formula:

$$R_i = \frac{k_i}{\lambda_i} \tag{6.2}$$

This expression is the reproductive ratio of each behaviour. The behaviour with the grater value has a dominant effect on the final stable value reached by the compartments.

Equilibrium and stability analysis

There are different final equilibrium value of the system depending on the values of the parameters. In particular, the four coefficients are combined, obtaining two reproduction rates R_1, R_2. First the nullclines lines are calculated and plotted. To do this, the system can be reduced to two equations assuming the mass conservation and that the following relation holds: $N = Ca + Co + Ag$ Then the first two equations are rewritten, rescaling also Ca,Co,Ag with N, the humans population. Using mass conservation condition the Ag term can be substituted in the first equation, resulting in a system of two equations with two unknowns. The nullclines lines are calculated and varying the R1 and R2 values the different scenarios are evaluated.

$$\begin{cases} \dot{Ca} = -k_1 Co Ca - k_2(N - Co - Ca)Ca + \lambda_1 Co + \lambda_2(N - Co - Ca) \\ \dot{Co} = k_1 Co Ca - \lambda_1 Co \end{cases}$$

The equations become

$$\begin{cases} \dot{x} = -k_1 yx - k_2(1 - y - x)x + \lambda_1 y + \lambda_2(1 - y - x) \\ \dot{y} = k_1 yx - \lambda_1 y \end{cases}$$

The nullclines lines can be calculated imposing $\dot{x} = 0$ and $\dot{y} = 0$. Solving the system with this condition applied gives the following two equations. For the first nullcline, with $\dot{x} = 0$:

$$y = \frac{x(k_2 - k_2 x + \lambda_2)\lambda_2}{x(k_2 - k_1) + \lambda_1 - \lambda_2} \tag{6.3}$$

and for the second with $\dot{y} = 0$

$$x = \frac{\lambda_i}{k_i} = 1/R_i$$

The choice of the right R_i to use for the second nullcline depends on the comparison between the two reproductive ration values. The larger is the one that must be used. Now are presented four main possibilities of the system evolution, and the stability of the found equilibria are studied.
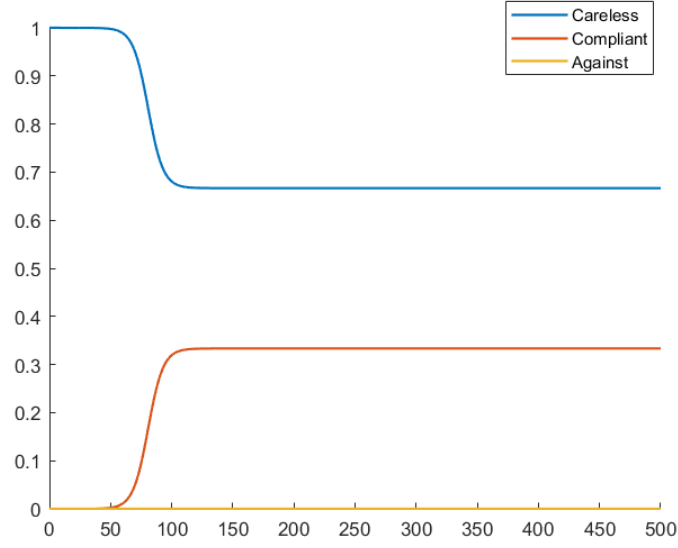
Figure 6.1: The behavioural system dynamic with $R1 > R2$ and $R1 > 1$.

I case: $R_1 > 1$ and $R_1 > R_2$

The plots of the system evolution in this case is:

In this first scenario, as visible in figure 6.1, the Against compartment tend to zero, so the equilibrium point can be calculated as $Ca = \lambda_1/k_1$ and $Co = 1 - \lambda_1/k_1$. The nullcline resulting plot is visible in 6.2.
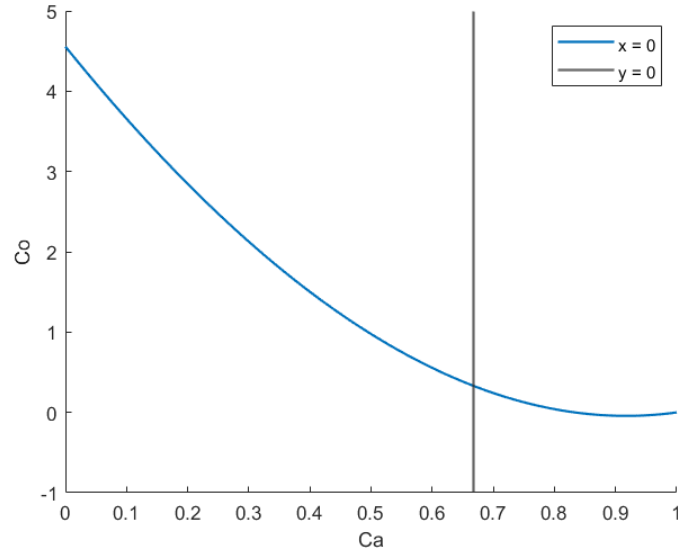

Figure 6.2: The behavioural system nullcline lines with $R1 > R2$ and $R1 > 1$.

The equilibrium found as intersection of the two lines correspond to the one calculated with the numerical equation. With the Routh-Hurwitz criteria the stability of this point is verified. To evaluate if the criteria is satisfied the Jacobian matrix of the system is calculated. Then the equilibrium is used to evaluate the trace and determinant of the system in this value. To see if the equilibrium satisfies Routh-Hurwitz condition it must holds:

- trace(J) < 0

- det(J) > 0

Both condition holds and the solution is asymptotically stable and does not depends on the initial condition.

II case: $R_2 > 1$ and $R_2 > R_1$

The plots of the system evolution has an opposite behaviour w.r.t the first case. So here, is the Compliant compartment that tend to zero at equilibrium.
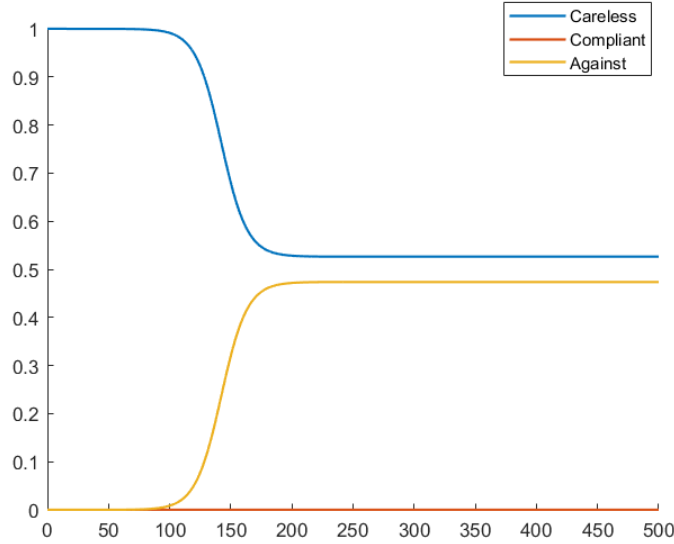
Figure 6.3: The behavioural system dynamic with $R2 > R1$ and $R2 > 1$.

The equilibrium point can be calculated as $Ca = \lambda_2/k_2$ and $Co = 1 - \lambda_2/k_2$. The nullcline resulting plot is visible in 6.4. The equilibrium is asymptotically stable.

III case: $R_1 < 1$ and $R_2 < 1$

If both the "reproduction rates" have a value lower than one, the stable equilibrium is the one in which both Compliant and Against goes to zero.

From the nullcline plot **??**, it can be seen that there is not an intersection. The plot of the second nullcline result in a vertical line with a value grater than one. In this condition the only equilibrium is the one for which $Ca = 1$ and both Ag and Co are equal to zero. The equilibrium is asymptotically stable.

IV case: $R_1 = R_2$

This final situation is the most difficult to analyse. In fact, due to the equal value of the
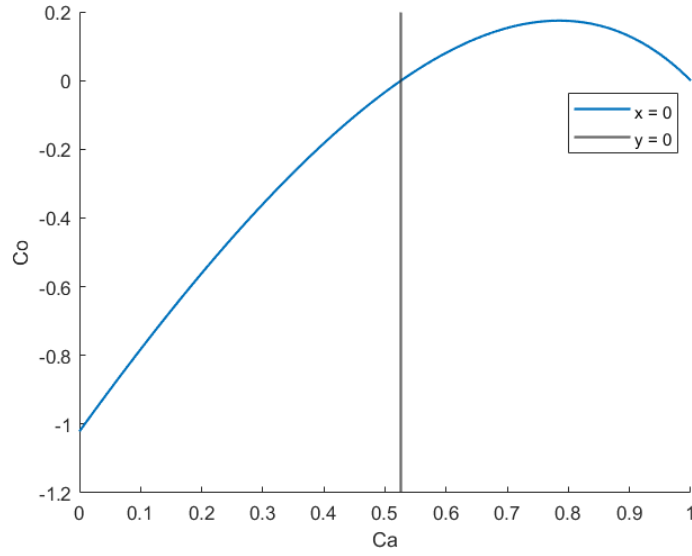
Figure 6.4: The behavioural system nullcline lines with $R2 > R1$ and $R2 > 1$.
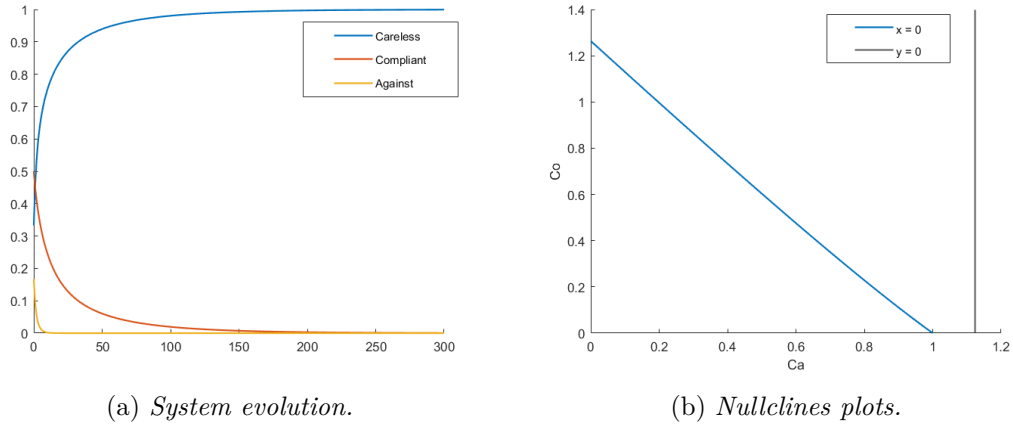

(a) *System evolution.*

(b) *Nullclines plots.*

Figure 6.5: Behavioural system dynamics and nullcline in the case of with $R_1 < 1$ and $R_2 < 1$.

two influence processes, the final equilibrium of the compartments cannot be calculated using only the previous relations, but depends also on the initial condition. The Careless compartment can be calculated using the same equation of previous cases, and the same value is found using both $Ca = \lambda_1/k_1$ and $Ca = \lambda_2/k_2$. As it can be seen from the system evolution and nullcline plots 6.6,at the equilibrium the Against and Compliant groups are formed by a subdivision of the $1 - Ca$ part. The initial condition have an influence on how this subdivision is composed. Using the Routh-Hurwitz criterium nothing can be said on this equilibrium because the determinant of the Jacobian have a null value.

(a) *System evolution.*

(b) *Nullclines plots.*
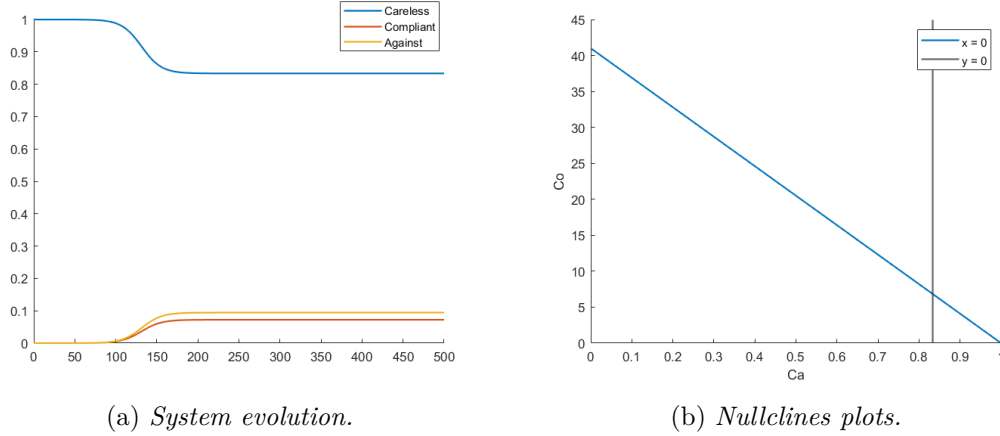
Figure 6.6: Behavioural system dynamics and nullcline in the case of $R_1 = R_2$.

Behavioural model experiment

To better comprehend all the possible scenarios that can emerge with the behavioural model a simulation is performed. Four vectors are defined, one for each parameter of the model. A different simulation for each combination of the coefficient is then roll out. In this case the value of the parameters is kept constant during each simulation. The range of variation of each parameter is the following:

- k_1 between 0.1 and 0.99

- k_2 between 0.1 and 0.99

- λ_1 between 1/5 and 1/30 d^{-1}

- k_1 between 1/5 and 1/30 d^{-1}

We observe the evolution of the dynamics of all the states, and to present a summary of the effects we collect for each simulation data such as the final value of the compartment, the max peak value and the corresponding time in which the peak occur. Also here, for the sensitivity plots realization, the reproduction rates deriving from the combination of coefficients, equation (6.2), are used.

The first plots 6.7 are heat maps about the final value reached by various compartments, varying R_1 and R_2.

In these pictures is clearly visible the threshold effect observed in the stability analysis performed earlier. While one of the reproduction ratios becomes larger than the other, the system equilibrium is composed by the dominant group and a portion of Careless individuals.The greater is the ratio, the smaller is the size at equilibrium of the Careless.

Another figure in which this threshold effect can be observed is 6.8.

The plots show how, for a fixed values of λ_1 and k_2, change the size at equilibrium of the system, varying the k_1 coefficient. To highlight the threshold effect due to the comparison of reproduction rates, on the x-axis is plotted the R_1 coefficient, that can be calculated knowing the value of λ_1 and k_1. For the same reason, different R_2 situations are represented.

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(a) *Final Compliant compartment*

(b) *Final Against compartment*
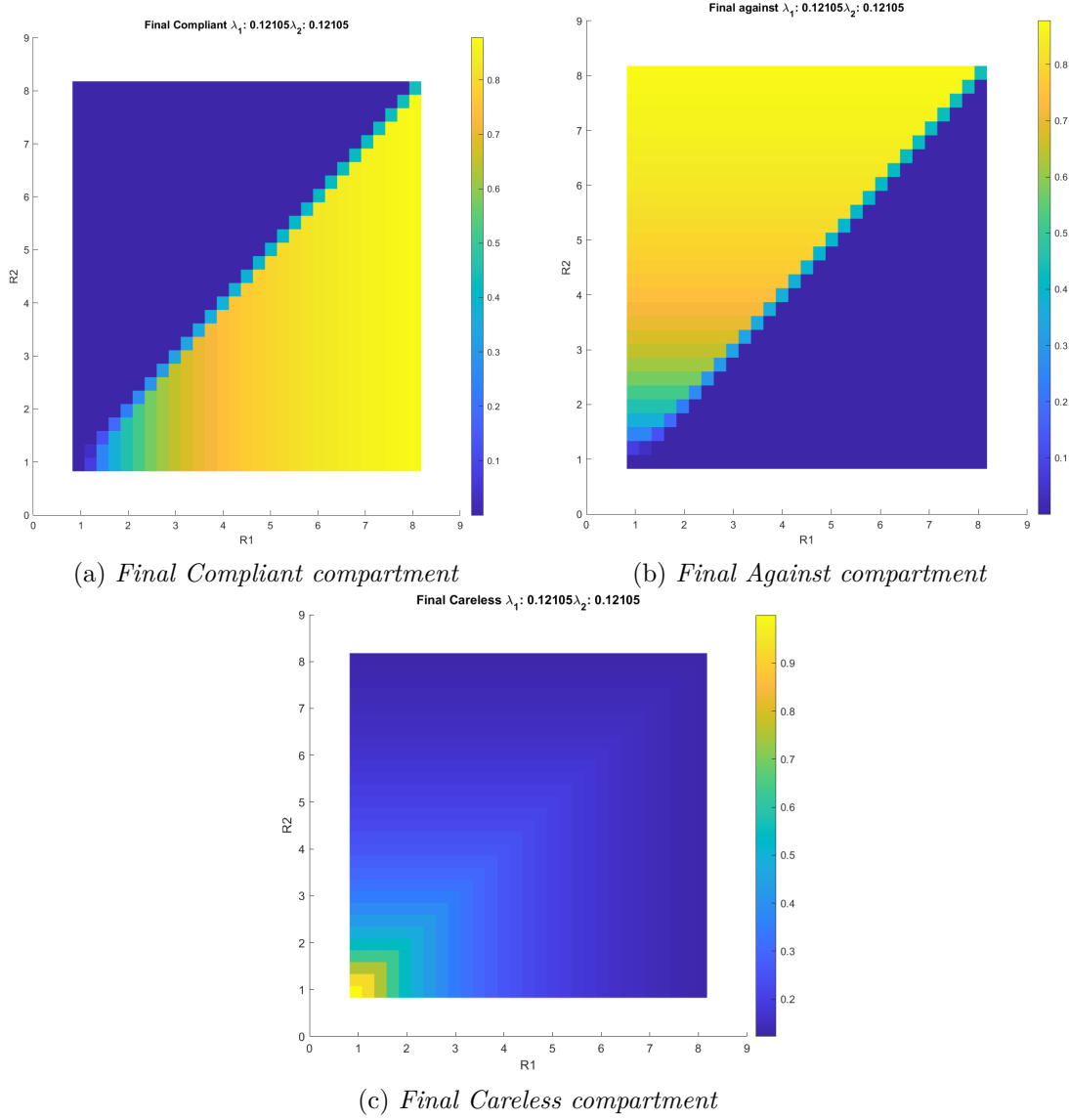

(c) *Final Careless compartment*

Figure 6.7: The final value reached at equilibrium by every compartment in the behavioural model.

The threshold effect is clearly visible also here. Looking at the Compliant and Against final values it can also be seen that after the R_1 reproductive coefficient is became dominant, the increase is the final size observed in the Compliant plot is due to the decrease in the Careless compartment.
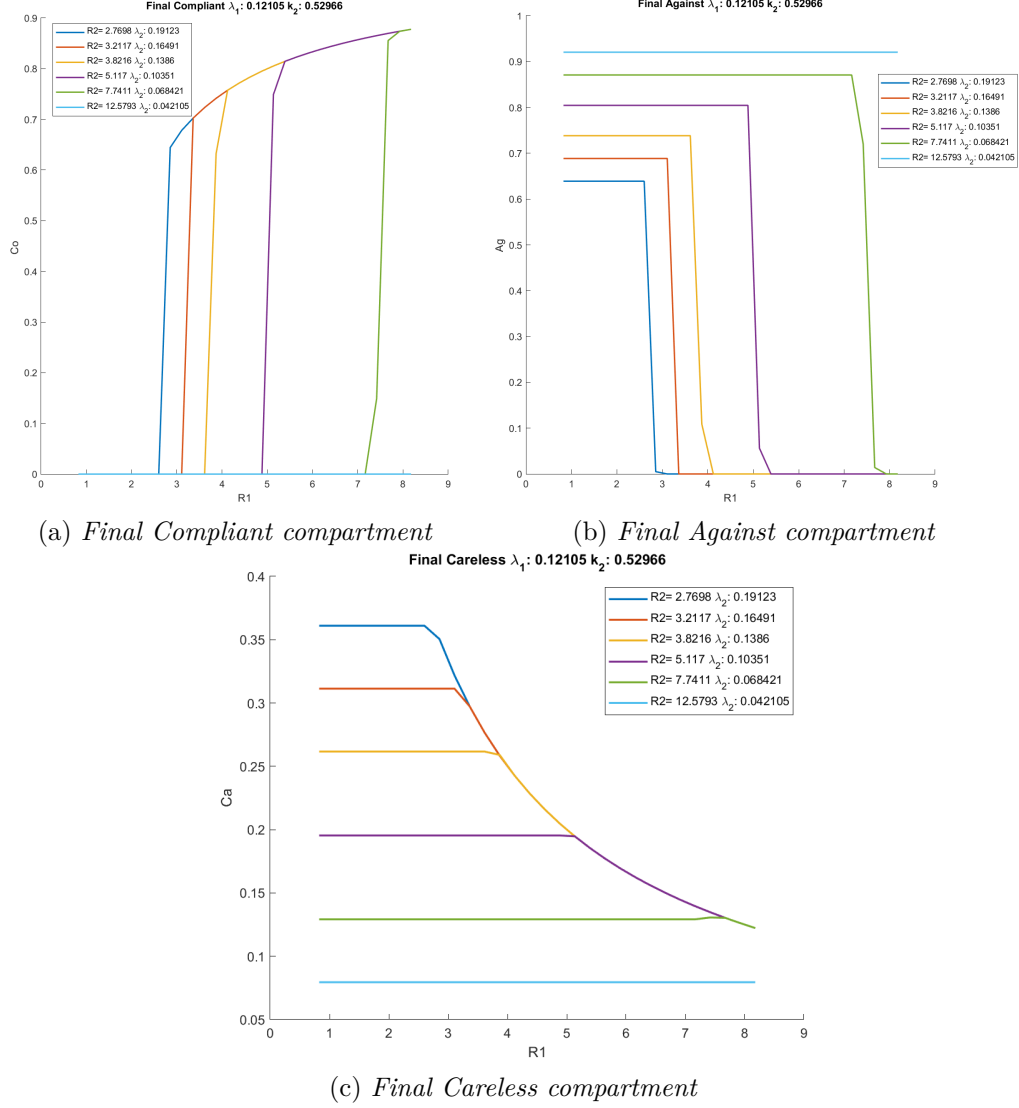
(a) *Final Compliant compartment*

(b) *Final Against compartment*

(c) *Final Careless compartment*

Figure 6.8: The final value reached at equilibrium by every compartment in the behavioural model varying the R_1 coefficient w.r.t different R_2 values.

Chapter 7

Models description and analysis

7.1 Behavioral epidemic model

The model is composed of two layers coupled together: a disease layer, describing the evolution of an epidemic, and a behaviour layer describing the transition among different behaviours during the epidemic development. The behavioral layer has three possible compartments:

H: Heedless, people careless of the risk associated to the infection;

C: Compliant, composed by person that want avoid to become infected or infect others

A: Against, who not consider a new infection developing as a risk for its safety and not use protection or change its behaviour during the epidemic.

This behaviour structure is coupled with a SIRS epidemic model, giving rise to the mutually exclusive compartments.

SH: Susceptible Heedless, the group where there is the majority of the population at the beginning of an epidemic. There is not much information about disease-associated risk and the people in this compartment have no fear of becoming infected and do not modify their behaviors.

SC: Susceptible Compliant, is a group composed of those who want to avoid of becomes infected. People who use non-pharmaceutical interventions to limit the possibility of getting sick.

SA: Susceptible Against, the people that refuse the information spread by media or authority. They do not consider the threat represented by the disease and do not respect the safety rules or recommended behavior to avoid getting sick or infecting others.

IC: Infected Compliant, people infected by the virus. In this group go both the SC and SH compartments because it is considered that even those who have a "neutral"

opinion about the risk associated with the infection, change their minds when become infected. The principal behavior associated with this group is that it is respected the quarantine and so a certain part of the infected avoid contact while they are sick.

IA: Infected Against, compartment composed by the against became sick. They do not respect self-isolation, and diffuse the disease.

RC: Recovered Compliant, people that are healed from the infection. They cannot be infected, but contribute to raising awareness about the risk associated with the disease.

RA: Recovered Against, the part of the recovered formed by against healed from the infection. The most radicalized can be in this group. They are protected by immunity from a disease in which they don't believe.

The resulting system is described by the following system of differential equations:

$$\begin{cases} \dot{SH} = -\phi k_1 SH \cdot C - k_2 SH \cdot A + \lambda_1 SC + \lambda_2 SA + \delta(1 - \phi_n)RC - \beta SH \cdot I \\ \dot{SC} = \phi k_1 SH \cdot C + \delta \phi_n RC - \lambda_1 SC - \beta \rho SC \cdot I \\ \dot{SA} = k_2 SH \cdot A - \lambda_2 SA - \beta SA \cdot I + \delta RA \\ \dot{IC} = \beta \rho SC \cdot I + \beta SH \cdot I + \phi k_3 IA \cdot C - \lambda_3 IC - k_4 IC \cdot A + \lambda_4 IA - \gamma IC \\ \dot{IA} = \beta SA \cdot I - \phi k_3 IA \cdot C + \lambda_3 IC + k_4 IC \cdot A - \lambda_4 IA - \gamma IA \\ \dot{RC} = \gamma IC - k_6 RC \cdot A + \lambda_6 RA + \phi k_5 RA \cdot C - \lambda_5 RC - \delta RC \\ \dot{RA} = \gamma IA + k_6 RC \cdot A - \lambda_6 RA - \phi k_5 RA \cdot C + \lambda_5 RC - \delta RA \end{cases} \quad (7.1)$$

where

- $A = SA + IA + RA$ is the total fraction of Against individuals.

- $C = SC + IC + RC$ is the total fraction of Compliant individuals.

- $I = \epsilon \cdot IC + IA$ is the fraction of infected people participating into the infection process.

- ϕ is awareness, a parameter that accounts for the current state of the epidemic and influences the behavior persuasion between groups. It can be modeled in different ways.

- ϕ_n it is the awareness normalized, used to split the population while re entering in the susceptible class in the Heedless or Compliant group.

- ρ is the protection factor of Compliant people that reduces their risk of becoming infected.

- β is the infectivity rate associated with the disease.

- γ is the recovery rate.

- δ is the rate at which immunity waves (so that recovered people become susceptible again).

- ϵ specifies the fraction of compliant infected that participate to the infection process.

Figure 7.1: Figure of the epidemic behavior model with its compartment and influxes and out-fluxes.
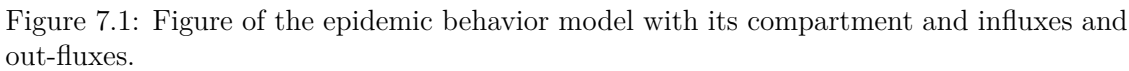
7.1.1 Basic reproduction number calculation

The first analysis that can be performed on the behavioral disease model is to estimate its basic reproduction number. It is defined as the spectral radius of the next-generation matrix. Using the method outlined in [59] and now briefly described this quantity is calculated. Consider a simple disease model in which $i \in R^n$ represents the set of infected compartments, $s \in R^n$ the set of susceptibles compartments, and $r \in R^k$ the set of compartments removed to the infection due to recovery.

To describe the evolution of the system the following notation using matrices is adopted:

- D is a $m \times m$ diagonal matrix; the diagonal elements are the relative susceptibilities of the corresponding susceptible class.

- Π is a $n \times m$ matrix in which the (k, j) element is the fraction of susceptible in the jth compartment that goes into the kth infective compartment.

- b is an n-dimensional row vector of relative horizontal transmissions.

- $\beta'(s, i, r)$ multiplies the b vector and is a scalar factor representing infectivity. It can be constant or a function of other parameters, such as incidence for example.

- V is a $n \times n$ matrix describing the transition between the infected state through death and recovery.

- $g(s, i, r)$ is a continuous function representing the inlet of uninfected through birth or immigration.

- $h(s, i, r)$ is also a function but used for the flow into and out of the recovered compartment because of natural immunity or vaccination.

The resulting disease model is represented by the system:

$$\begin{cases} s' = g(s, i, r) - Ds\beta'(s, i, r)bi \\ i' = \Pi Ds\beta'(s, i, r)bi - Vi \\ r' = h(s, i, r) + Wi \end{cases} \qquad (7.2)$$

Using the defined matrices, according to [59] the basic reproduction number R_0 for the model (7.2) at a disease free equilibrium $(s_0, 0, r_0)$ is given by:

$$R_0 = \beta'(s, i, r)bV^{-1}\Pi Ds_0 \qquad (7.3)$$

The disease-free equilibrium is defined as a locally stable equilibrium of the system in which there is no disease and in the sense that a solution starting close to $(s_0, 0, r_0)$ remains close to this state.

Considering the system dynamic presented in (7.1), it can be rewritten in the matrix form expressed in (7.2) as:

$$\Pi = \begin{bmatrix} \rho & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \qquad D = \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \qquad s = \begin{bmatrix} SC \\ SH \\ SA \end{bmatrix}$$

$$b = \begin{bmatrix} \epsilon & 1 \end{bmatrix} \qquad V = \begin{bmatrix} k_4 A + \lambda_3 + \gamma & -\phi k_3 C - \lambda_4 \\ -k_4 A - \lambda_3 & \phi k_3 C + \gamma + \lambda_4 \end{bmatrix} \qquad i = \begin{bmatrix} IC \\ IA \end{bmatrix}$$

The resulting R_0 find with this method is:

$$SA_0 \left(\frac{\beta (\gamma + \lambda_3 + A k_4)}{\sigma_1} + \frac{\beta \varepsilon (\lambda_4 + C k_3 \phi)}{\sigma_1} \right) + SH_0 \left(\frac{\beta (\lambda_3 + A k_4)}{\sigma_1} + \frac{\beta \varepsilon (\gamma + \lambda_4 + C k_3 \phi)}{\sigma_1} \right) + SC_0 \rho \left(\frac{\beta (\lambda_3 + A k_4)}{\sigma_1} + \frac{\beta \varepsilon (\gamma + \lambda_4 + C k_3 \phi)}{\sigma_1} \right)$$

where

$$\sigma_1 = \gamma \lambda_3 + \gamma \lambda_4 + \gamma^2 + A \gamma k_4 + C \gamma k_3 \phi$$

Figure 7.2: Basic reproduction number of the epidemic behaviour model.

I case: R_0 first evaluation

A first evaluation of the R_0 is done using equation (7.3) and assuming that at the beginning of an epidemic, the majority of the population is in the SH compartment, while SC, SA, and also the A and C groups are close to zero. An initial tentative calculation for the value is done using a set of values assigned to the rest of the coefficients in the matrices. For reference, the following values are used:

$$\beta = 0.40 \qquad \gamma = 0.35 \qquad \rho = 0.65 \qquad \epsilon = 0.15$$
$$k_3 = 0.49 \qquad k_4 = 0.243 \qquad \lambda_3 = 0.143 \qquad \lambda_4 = 0.143$$
$$SC_0 = 50/60e6 \qquad SA_0 = 50/60e6 \qquad C = A = SC_0 \qquad SH_0 = 1 - SC_0 - SA_0$$

With these coefficient values, considering separately the epidemic and behavioral layers the basic reproduction values are:

$$R_0^{SIR} = \frac{\beta}{\gamma} = 1.1429$$

$$R_3^{behav} = \frac{k_3}{\lambda_3} = 3.3566$$

$$R_4^{behav} = \frac{k_4}{\lambda_4} = 1.6993$$

In this situation, an epidemic can spread, in fact $R_0^{SIR} > 1$, and looking at the behavior of people the complaints, associated with R_3^{behav}, have a larger influence than the one of the "against" group.

The epidemic-behaviour reproduction number found using the formula and assuming an initial awareness equal to 0.5 is

$$R_0^{epi-behav} = 0.3898$$

It is immediately noticed that the reproduction number is way below the threshold value of 1. According to the theorem presented in [59] there cannot be the spread of disease if the initial R_0 is under this value. A simulation of the system dynamic confirms this situation. Observing figure 7.3 can be seen that the number of infected tends to decrease and goes to zero. Then the majority of the population remains in the Susceptible compartment, splitting into compliant and heedless subgroups. This is due to the strongest attractivity of compliant behavior simulated in this scenario.
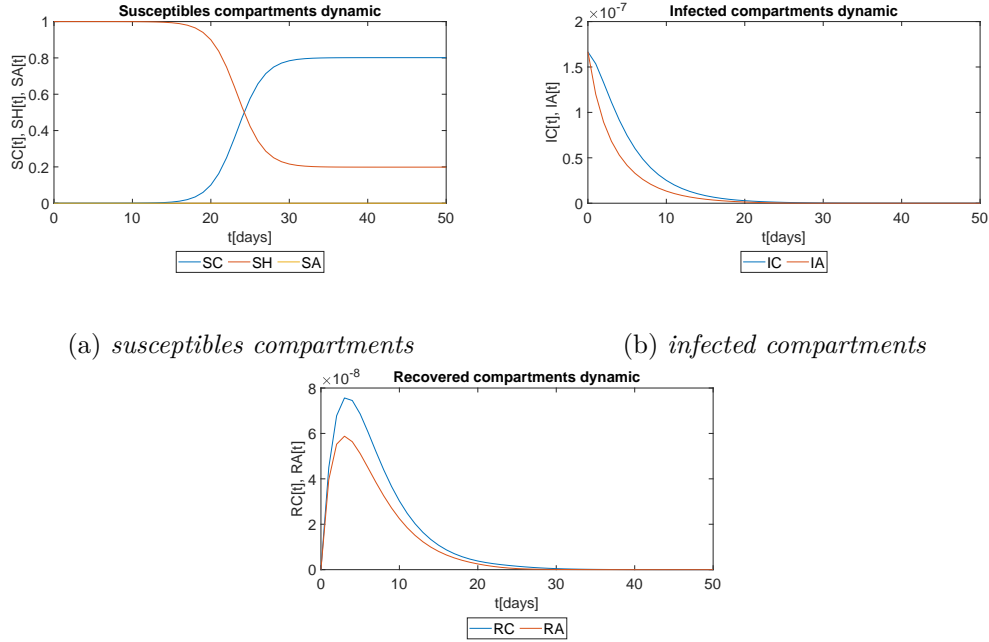
(a) *susceptibles compartments*   (b) *infected compartments*

(c) *recovered compartment*

Figure 7.3: Evolution of the behavioral epidemic model, with fixed parameters for the coefficients and an initial reproduction rate less than 1. It is observed that there aren't conditions for an epidemic to spread.

### *II* case: how parameters influence the R_0 value

A second test performed on the find $R_0^{epi-behav}$ wants to understand what are the parameters that influence more its value. The aim is to find which combination of coefficients can

have a critical impact in the first phase of the disease and cause an overshoot of the threshold value. The value of the reproductive rate is then calculated varying the coefficients and the results are now presented in 7.4.
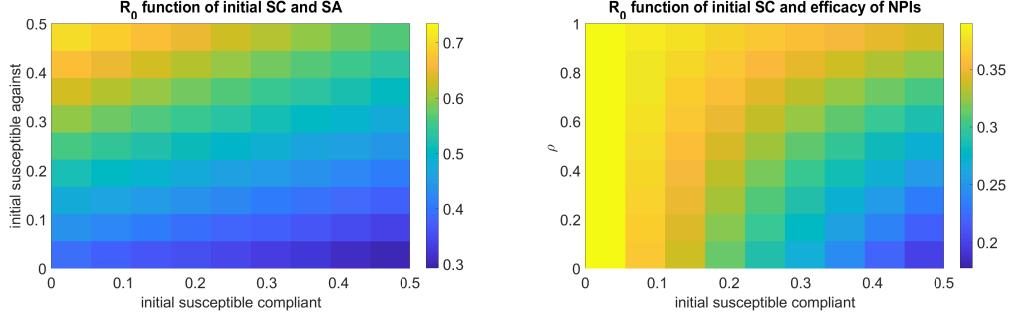
(a) R_0 *varying the initial population value in susceptible compliant and susceptible against group. The more the population is against the higher the reproductive ratio.*

(b) R_0 *varying the number of initial compliant susceptible and the parameter ruling the efficacy of adopting a safe behavior and avoiding contracting the disease. The lower is ρ the higher is the probability of not becoming infected.*
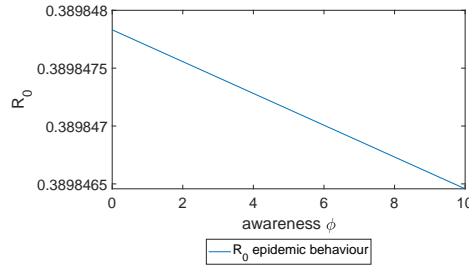
(c) *The influence of the awareness parameter on the R_0.*

Figure 7.4: Value of $R_0^{epi-behav}$ varying some parameters and maintaining constant all the other initial conditions.

The case shown in figure 7.4 is an example of how changes the reproductive rate value. The most interesting observation is that the value of awareness practically does not modify the $R_0^{epi-behav}$. Instead, it can be observed a threshold in the number of initial susceptible compliant for which the reproductive rate becomes smaller, depending on the efficacy of the protective behavior. If there are less than 5% of the population in the compliant group, for any value of ρ the reproductive rate does not change.

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