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A New Model to Capture the Impact of Behaviours on the Spread of Infectious Diseases

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*To my source of
inspiration, Montgomery
Scott*

Summary

The thesis develops and presents a novel multi-system model (i.e., a model that simultaneously captures different coexisting and coupled dynamic phenomena) that couples behavioral and epidemic phenomena by combining a SIR-like epidemiological model with a behavioral model. This behavioral model partitions the population into three compartments:

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

C: Compliant, people that want to avoid becoming infected or infecting others

A: Against, people who not see the epidemic as a risk and do not use protections or change their behavior during the epidemic.

The model's features are not only based on theoretical principal, but also developed considering real empirical datasets regarding both disease and behavior evolution during the recent COVID-19 pandemic. This approach overcomes the limitations of previous works that implement similar models, but use proxies for individual behaviors or rely on the assumption of a direct correlation between opinion and behavior.

The key contributions of the work include:

- **Coupling between behaviors and contagion:** The developed model considers phenomena such as peer pressure, fatigue, non-pharmaceutical interventions (NPIs), self-isolation, and mean-field parameters representing government policies to mitigate the spread of the disease. These elements contribute to modeling the behavioral transitions of the population and realizing the coupling with the disease model.
- **Analysis:** An extensive analysis of the model components is conducted, focusing first on the new behavioral model alone and then on the full model. This allows the development of a foundational framework used for the full multi-system model.
- **Epidemic-reproduction number and simulations:** The epi-behavioral model's reproduction number is computed using the Next-Generation Matrix method. A full set of simulations to test the system behavior for different conditions and parameter sets is also performed.

The flexibility of the developed model and its ability to capture the interplay between individual behaviors and disease spread offer a foundational tool to explore future scenarios in which social dynamics, such as wearing face masks or self-isolating, significantly alter the epidemic evolution.

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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 human populations over time: diseases and epidemics. There were no periods - not in the past, nor nowadays - when illnesses didn't influence human lives.

Looking at the past, the consequences of epidemics on the population were worse than today, mostly 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. The pandemic also triggered social unrest: Jews were considered responsible for the illness spread and they began to be persecuted: there were several attacks and massacres to the Jewish communities in different European cities, like Toulon, Barcelona, Erfurt, Basel, Frankfurt, Strasbourg. The persecution mainly was related to belief that Jews were less affected by the disease, and that were responsible of the poisoning of wells.

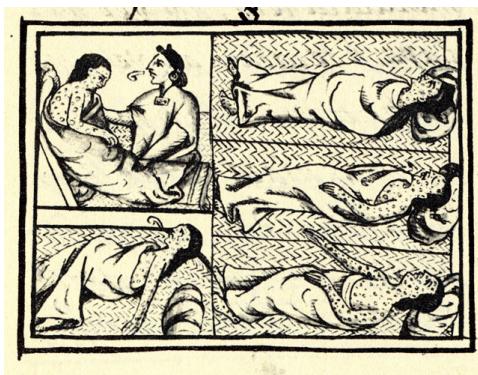


Figure 1.1: Representation of smallpox disease on the Mexican population in the *XIV* century. Figure from the Florentine Codex [1].

Another example is how during the course of the Americas' colonization, the diseases imported by the Europeans were one of the main causes of the genocide of the local population, largely contributing to their defeat against the Spanish conquistadors. 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 the 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 contributed to enhancing the quality of life. An example of this is that only in the last three centuries and especially in the most economically developed countries, a significant increase in life expectancy has been observed [2]. This increase is also happening in poorer regions, such as Sub-Saharan Africa. Although their current life expectancy is lower than that of wealthier countries, recent research [3] predicts a significant rise over the next 30 years. This study also forecasts that this trend will lead to a global convergence in life expectancy between now and 2050. The most plausible explanation for this future prediction is that improvements in healthcare levels lead to changes in the causes of mortality as nations' wealth increases. In poorer regions, the primary causes of death are communicable, maternal, neonatal, and nutritional diseases, whereas in more developed and wealthier countries, non-communicable diseases like cancer and cardiovascular conditions become the main causes of death [4].

However, despite rising life expectancy, epidemics continue to be one of the most significant threats to populations. In fact, there was a notable increase in the frequency and magnitude of reported epidemics during the 19th and 20th centuries [2]. This makes it essential to develop effective policies to control and mitigate their impact, requiring coordinated implementation by countries within the same macro-region.

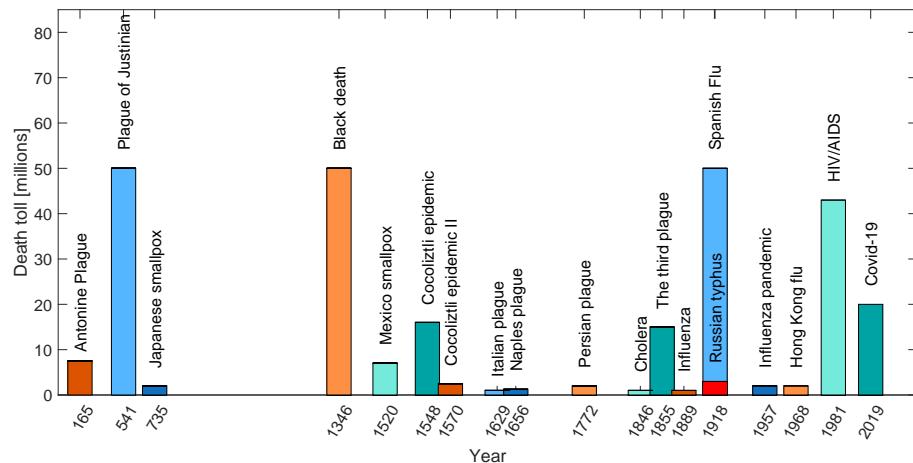


Figure 1.2: A graphical representation of the distribution of known epidemics over the years and of their associated death toll. It is observable how there is an increment in the number of these events in the last three centuries. Data extracted from [5, 6]

Figure 1.2 highlights this trend. Although it becomes increasingly difficult to obtain

reliable information about disease outbreaks the further back in time one goes, epidemics remain a tangible and present threat. This underscores the need for attention and action in shaping health policies that effectively address these dangers. However, health status is not the only factor impacted by diseases. Illness can profoundly alter relationships, work, and social life, leading to a deterioration in overall social well-being as well [7]. There is also an economic cost associated with the cure. Only in a few nations worldwide, is treatment covered free of charge by the state. In the majority of countries, being ill can result in having to sustain high costs, causing people to go into debt or not take care [8, 9]. 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 that 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: How can a multidisciplinary model be developed that integrates elements from both social and epidemiological aspects to provide some insights into their mutual influence during the spread of a disease?

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 crucial step involves studying the dynamics of the disease and developing a predictive model for its evolution. This process requires understanding and estimating various parameters associated with the disease, including the transmission mechanisms within the population, the reproductive rate of the infectious agent, the acquisition and persistence of immunity, and the contagion mechanism. Creating a reliable model is not only scientifically valuable but also serves as a powerful tool for stakeholders, helping to formulate effective policies during a pandemic emergency. Theoretical epidemiology aims to provide insights and policy recommendations in this context. Furthermore, data acquisition and analysis are essential for statistically modeling epidemic coefficients. Ultimately, a model that can address stakeholders' questions and make predictions—whether or not safety regulations are implemented—has significant implications for society. Beyond the economic costs associated with illnesses, there are also substantial social costs. Developing tools to understand better disease transmission can help mitigate its impact and alleviate the social burden, potentially saving numerous lives.

A clear example of the potential benefits of having an epidemic model is the ability to generate synthetic insights that are easy to understand and can be expressed numerically. Such models can provide answers to critical questions like:

- Is the disease so infective that can cause a pandemic?
- What are the threshold conditions that can cause an outbreak?
- What is the expected number of infections over time?

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 epidemic modeling requires striking a balance between simplification and maintaining accuracy. A good model effectively reproduces key phenomena with reasonable sophistication. While creating an overly complex model that attempts to incorporate every detail of a disease might be tempting, it often requires significant effort and data. In many cases, such models don't outperform simpler ones that focus on capturing the most important dynamics of disease spread. By prioritizing essential characteristics, simpler models can provide more practical insights while remaining computationally efficient.

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, networks or agent-based models [10, 11].
- Predictions about the progression of epidemics or reconstructions of the events' dynamics [12–14].
- Insights into epidemics, explaining phenomena like the periodicity of re-infection for certain diseases or the seasonal patterns observed in cases such as influenza [15].
- Understanding the effectiveness of specific strategies against outbreaks, such as vaccines or quarantines [16].

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 [17].
- The formation of public opinions during an outbreak [18].
- 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 [19].

1.1 Presentation of the work realized

The work presented in this thesis is part of the multidisciplinary field of research. Its focus lies in understanding the mutual influence between human behavior and an epidemic. On the one hand, it examines how the presence of an epidemic affects individuals' behavior; on the other hand, it explores how these behavioral changes influence the progression and dynamics of the epidemic itself.

For this reason, a new model has been developed, drawing from studies of existing multi-system models and from empirical data that integrate both epidemic and behavioral aspects.

The framework involves coupling a SIR-like disease model with a new behavioral model consisting of three compartments: Heedless, Against, and Compliant individuals. These categories are designed to represent different courses of action taken during a disease epidemic.

- The Heedless individuals represent the segment of the population that is either unaware of the disease's spread, particularly in its early stages, or indifferent to the risks, continuing with regular routines that may increase their likelihood of infection.
- The Against group includes skeptics who reject precautionary measures and refuse to follow established guidelines.
- The Compliant group consists of those who actively seek to avoid infection by adhering to health policies and precautions.

The model incorporates mechanisms to account for changes in behavior among social groups, primarily driven by peer pressure, but also considers the intervention of a central global actor. Additionally, it accounts for fatigue associated with adhering to a particular behavioral spectrum (either complying with or opposing rules). The epidemiological model developed can track both the initial phase of an epidemic and successive waves of contagion, including the possibility of reinfection.

A distinguishing aspect of this work is its reliance on empirical data. Unlike other studies that use ad-hoc assumptions or data from sources not directly related to behavior (such as opinions), this thesis leverages research conducted during the COVID-19 pandemic. This research provides insights into people's behavior, including opinions about the disease, trust or distrust in doctors or governments, and actions such as mask-wearing and handwashing.

For a novel multi-system model such as the one developed here, an analysis of its dynamics is performed. The two components alone have been studied, and the social one, which represents a novelty, has been thoroughly analyzed and simulated. Furthermore, an epidemic reproduction number, defined as E_0 in the thesis, is calculated and used to understand which model-free disease scenarios can evolve into an epidemic if perturbed by the entry of infected individuals.

A powerful feature of multi-system models is their ability to reveal phenomena that would not be apparent when examining individual components in isolation. This comprehensive approach enables a deeper understanding of complex interactions and dynamics. Moreover, the developed model is designed as a flexible framework, capable of adapting to a wide range of scenarios, reflecting the multiplicity of reality.

Chapter 2

Main objectives and summary of the contents

In this chapter, the main objectives pursued with the current work are presented and the composition of the thesis is described. 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 population behaviors be effectively included in epidemiological models? 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?

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 mechanism 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.

The work consists of an introductory chapter 3 where the main concepts of social science and epidemiology are presented. This chapter provides all the necessary information

to understand the present research. It includes a glossary 3.1.2 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 in section 3.3, 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 4th 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 II part of the thesis is composed of four main chapters. In chapter 5 the reasons that led to the development of a new model are presented. In Chapter 6, the chosen epidemiological and behavioral models are simulated. The SIRS epidemic model is presented, and its features are described. Conversely, the behavioral model developed for the thesis is introduced, the assumptions made for its dynamics are explained, and a set of simulations and analyses is performed. This analysis is conducted to gain a clear understanding of how the hypothesized social dynamics can evolve and to develop initial insights crucial for understanding the behavior of the fully coupled final model. Chapter 7 presents the model resulting from the integration of the two layers, forming part of a more complex multi-system model. The implementation of this coupling is explained, detailing how the mutual influence between the two components operates. The main features of this model are analyzed using both simulations and insights gained from calculating the epidemic reproduction number. In fact, the threshold effect, which explains whether a disease-free equilibrium can evolve into an epidemic, is determined, and its value is calculated under varying model scenarios. 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. In this chapter, the theoretical basis and main concepts that will be used in the present work are defined.

First, a brief historical review of the emergence of the epidemiology field is provided, focusing on the explanation of its genesis. Indeed, these key findings laid the foundation of modern epidemiology. The following section presents a glossary of the key terms used throughout this thesis. This glossary ensures clear communication of the core concepts that will be referenced later. Initially, terms related to epidemiology are explained, followed by definitions of behavior-related concepts.

Subsequently, the most commonly used mathematical tools are introduced, including an overview of various modeling techniques. Special attention is given to the theoretical background of the mean-field model.

3.1.1 Epidemiological research historical background

The research field regarding the development of techniques 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 found the mechanism used by disease to spread. A first innovative work was the one did 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, advances in the microbiological research were 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's 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, or contact ratio, between susceptible and infectious individuals. It was 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, was then updated in 1908 by

Ross, that re-written it in continuous time. For the first time the problem could be studied using a clearly, well defined mathematical theory. Then the contributions of Kermack and McKendrick in 1927 added 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 -called reproduction number- 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 [2, 20]. 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.

CFR, IFR and mortality excess

The case fatality rate, CFR, is the ratio between the number of deaths due to a specific disease and the total number of confirmed positive cases detected by testing. The infection fatality rate, IFR, 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) in a month per month in a comparison between a time period with an epidemic and one without.

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 or water.
- Vector born: the contagion is mediated 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 infects humans. The opposite can also happen and it is called inverse-zoonosis.

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.

Epidemic disease

An increase in disease prevalence typically manifests as a rapid outbreak. This type of illness is confined to a limited geographical region, unlike a pandemic which affects a much larger area.

Immunity and herd immunity

Immunity refers to the protection from a disease gained after contracting it or after the vaccination. This immunity can be lifelong or diminish over time. When a person is immune, re-exposure to the virus does not result in infection, or there is only a reduced chance of being infected, known as partial immunity.

Herd immunity is a phenomenon where a large portion of the population becomes immune, either through vaccination or surviving the disease. This majority limits the spread of the illness, indirectly protecting those who are not immune by slowing or halting disease transmission.

Incidence and prevalence

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

Incubation, Symptoms, Infected and Infectious

When a person comes into contact with an infectious individual, they may or may not become infected. The incubation period refers to the time after infection when the disease grows within the host without producing symptoms.

Symptoms refer to the physical signs of illness caused by a disease in the affected individual.

A person is described as infectious when they carry the disease and can transmit it to others, while infected refers to someone who has been exposed to the infection and has become ill.

Incubation period and serial interval

The incubation is the time after exposure in which the infection develops in the host 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.

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 lives and reproduces 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. Most viral and bacterial parasite, are into the microparasitic category.

Instead, macroparasite may be described by those having no direct reproduction within their host. Arthropods and helminths are in this category. They are larger and have a much longer generation times than microparasites, with a life span that can be a considerable fraction of host life span.

Outbreak

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

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 brought about by low numbers of spreaders.

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).

Reproduction number \mathcal{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 $\mathcal{R}(t)$, a time-varying reproduction number. Finally exist also the effective reproduction number, that is obtained rescaling the Reproduction number value with the true number of susceptible.

Types of infectious diseases

An infectious disease is indicated as an illness resulting from the presence of a pathogenic microbial agent such as bacteria, viruses, parasites or other microorganism.

It is possible to distinguish between *transmittable* and *communicable* disease. A transmittable disease can be transmitted between persons through unnatural routes. A communicable disease is one that spreads from one person or animal to another or from a surface to a person.

Virulence and Contagiousness

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

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.

Behaviour

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

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.

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.

Homophily

The tendency to bond and associate with similar others.

Information

The term "information" is commonly understood as "knowledge communicated." However, given its crucial role in modern society, there is considerable debate about its various meanings [21]. Today's world is often described as an "information society," where the advancement of information technology has impacted nearly every aspect of life.

Currently, the term "information" carries two key meanings. The first, more general, definition refers to anything that is valuable in answering a question. The second definition pertains to Information Science, the discipline that manages information in all its forms. In this context, information is something with the capacity to inform. On a fundamental level, anything that is not entirely random can be considered to convey some degree of information.

Perception

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

Polarization and Consensus

Polarization refers to the divergence of beliefs within a population. There are several mathematical methods available to measure the degree of polarization. For instance, one can collect data on opinions, beliefs, or behaviors within a group and then measure the distance between the most extreme views, or analyze their distribution across a defined range.

This contrasts with the concept of "consensus," where the exchange of opinions, information, or resources among individuals leads to widespread agreement. Both polarization and consensus can be studied and modeled using network theory [60].

Threshold theory

It is a theory formulated by Granovetter in [22] 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.

Trust

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

3.3 Epidemiological models categorization

Starting from the observation of the real world, the desire to better understand 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 researchers and policymakers. For example, an application is the estimation of certain policies' effects on the population during an epidemic: in this case, the aim is to produce 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 be remembered. This concept is remarked also on the definition of mathematical model present in [14].

"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 types of mathematical models. A first classification can be done considering the method used to obtain them: we thus have mechanistic, empirical, phenomenological or conceptual models. Mechanistic models are 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 set of data. They are a powerful instruments, because data can be modeled quite well, but they lack the explanatory value of the mechanistic models. A phenomenological model describes the empirical relationships between phenomena in a way that aligns with fundamental theory, but it is not directly derived from first principles. These models define the relationships between variables and provide insights into the phenomenon under study. Finally, with conceptual models, it is meant a verbal description of a real-world scenario.

For the present work, a mechanistic/phenomenological model is 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:

- follow the epidemic evolution;
- realize a framework capable of understanding the information related to the disease, as incidence and prevalence for example;
- obtain general insight about control strategies;
- realize predictions.

Considering the mechanistic-phenomenological 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. A focus on mean field model and its basic theoretical concepts is presented in section 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

Mean field model, also known as compartmental model, is the first developed and most studied type of mathematical model used in epidemiology [2, 13, 23, 24]. It assumes that a well-mixed population is divided into several subgroups (or compartments). Each one groups people in a different stage of the disease under consideration. Some possible states are susceptibles, asymptomatic (infected), symptomatic (infected), infected (if in the model no distinction between symptomatic or asymptomatic is done), exposed, vaccinated, quarantined, dead, recovered, and hospitalized. The classes considered in the realized model determine its 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.

For example, both a SIR (Susceptible- Infectious-Recovered) [25] and a SPQEIR (Susceptible -Protected-Quarantined- Exposed- Infectious- Removed) [26] can be used to model COVID-19, but the second model considers explicitly quarantine, exposed and use of protections to avoid infection - elements that cannot be observed or considered with a simpler model like a SIR.

In the mean-field class of models, the severity of infection is typically not considered; individuals are either infected or not. The primary focus is to describe the spread of the disease rather than its biological impact on health states. The transitions between compartments (such as Susceptible, Infected, and Recovered) are governed by differential equations. The parameters that control these transitions are coefficients whose interpretation depends on the underlying assumptions of the model. For example the γ parameter is usually interpreted as the inverse of the time an individual spend in the infectious compartment. Mathematically, these parameters determine the rate of flow between compartments.

The most critical metric in this model category is the "Basic Reproduction Number" (often denoted as R_0), which represents the average number of secondary infections caused by a single infected individual in a fully susceptible population. It is considered a fundamental threshold in epidemiology, indicating the potential severity of an outbreak [10]. By observing the value of R_0 , it is possible to immediately determine whether a newly developed disease can spread within the susceptible population and cause an outbreak leading to an epidemic.

SIR model

The foundational model for studying epidemics mathematically is the SI model. In this model, the population is divided into two compartments: Susceptible (S) and Infected (I). Individuals transition from being susceptible to infected, but there is no recovery, meaning once infected, individuals remain infectious indefinitely.

An extension of this is the SIR model, which adds a third compartment, Recovered (R). This additional compartment represents individuals who have either gained immunity or have died, removing them from the cycle of infection. After spending a certain period in

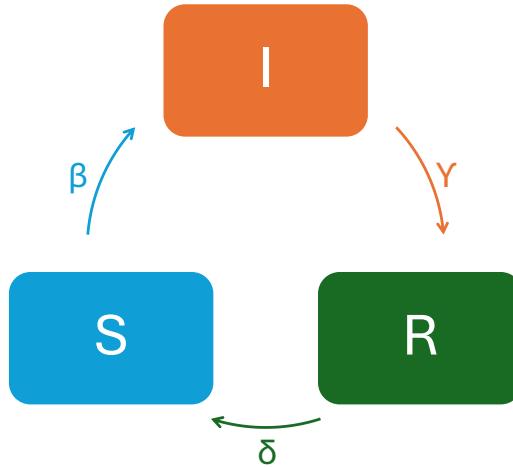


Figure 3.1: An example of the graph structure of a mean field SIRS model. There are three compartments and the flow rate between them is ruled by the coefficients β , γ and δ .

the infected state, individuals transition to the recovered state, making them no longer susceptible to the disease. Now to begin introduce the SIR model structure, first its compartments subdivision is presented. The population or density of individuals is subdivided 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 instead 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.

The SIR model, which will be described in the following sections, is built upon several assumptions that shape the mathematical framework. These guiding principles are introduced in the following paragraphs. Furthermore, the main properties of the model will also be introduced.

Mean-field approximation

The approximation that gives the name to this model class is based on a method that permits the analytical analysis of complex systems. With mean-field, individual-level interactions are averaged, focusing not on individual behavior but on the collective behavior of the population. Using this method, equations can be derived that describe disease evolution in terms of average rates rather than tracking individual agents.

Homogeneous mixing

It is the assumption that all individuals in the population mix homogeneously, meaning that the probability of interacting with any individual is the same. It is a strong assumption because it implies ignoring any spatial or social structure.

3.3.2 Average rates of changes

In the model, differential equations are used to represent the transition of individuals between different compartments. These transitions can depend on the state of the system but, in the simplest form, are based on average rates. The three rates used in the SIRS mean-field model are:

- β : transmission rate;
- γ : recovery rate;
- δ : waning immunity rate.

The flow of individuals in the system is shown in Figure 3.1.

Population being constant

The total population size, represented by the letter N , is the sum of individuals in the three compartments (e.g., Susceptible, Infected, and Recovered). It is often assumed to remain constant during the course of an epidemic. This assumption is based on the idea that epidemics typically last much shorter than the average human lifespan, making the effects of births and deaths negligible. In more complex models, where demographic effects are considered, such as in models of longer-lasting epidemics, the population size can still be assumed constant by balancing the number of births (modeled as an influx into the Susceptible compartment) with the number of deaths (modeled as an outflux). This approach assumes that births and deaths occur at approximately the same rate, keeping the total population size stable over time. The result of this assumption implies that:

- Population density compartments sum up to one.

$$s(t) + i(t) + r(t) = 1$$

- The sum of the derived functions w.r.t. time is equal to zero.

$$\dot{s}(t) + \dot{i}(t) + \dot{r}(t) = 0$$

Recover transition

The first fundamental dynamic process of the model assumes that the infected compartment decreases in size, with a rate of decrease proportional solely to the current number of infected individuals. This gives the following relation:

$$\frac{di}{dt} = -\gamma i, \quad i(0) = i_0,$$

which models a continuous process. Specifically, it assumes that individuals transition from the infectious state to the recovered state at a constant rate, denoted by γ . The physiological meaning of this parameter is that it represents the inverse of the infection's duration, meaning that the average infection lasts $1/\gamma$ days. Thus, γ is the recovery rate, reflecting the speed at which the population recovers from the disease.

Person-to-person disease transmission

To model this quantity fractional terms are used, assuming a population composed of susceptible and infected individuals. Each infectious individual encounters a fraction c of the population per day. If all encounters are equally likely, a fraction s of these encounters will be with susceptibles. Therefore, each infectious individual has $c \cdot s$ encounters with susceptibles. The probability that an encounter leads to transmission is p , meaning the average number of transmissions per day is $p \cdot c \cdot s$. Considering the fraction of infected individuals i , and combining p and c into a single parameter β , the transmission rate becomes:

$$\text{transmission rate} = \beta s i.$$

Here, β represents the transmission coefficient.

Immunity

In SIR model, once individuals recover from the disease, there are no further transitions. Two possible model scenarios can cause this:

- Lifelong immunity acquired after recovery, in this case the meaning of the compartments is $R = \text{recovered}$.
- Death of infected individuals, here the compartments can be intended as $R = \text{removed}$.

Both cases assume that, once the illness period ends, the disease can no longer be transmitted. If immunity acquired from an infection wanes after a certain period, individuals can become susceptible again, leading to an SIRS model. The δ rate is used to express the outflow from the Recovered compartment, and it corresponds to the inverse of the average duration of immunity.

Threshold value

Although the SIR model is simple, it plays a fundamental role in predicting a key aspect of epidemics: the threshold value, a concept first introduced by Kermack and McKendrick in their pioneering work [23]. They showed that, in a fully susceptible population, an epidemic will only start if the basic reproduction number \mathcal{R}_0 is greater than 1, marking the birth of the term "threshold value" in epidemiology. In the SIR model the reproduction number value is equal to the ratio between the transmission and recovery rate:

$$\mathcal{R}_0 = \frac{\beta}{\gamma}. \quad (3.1)$$

Over the years, the dynamics of this system have been extensively studied and analyzed [14, 27–33]. The threshold effect highlights two distinct scenarios:

- **Free Disease Equilibrium ($\mathcal{R}_0 < 1$):** When \mathcal{R}_0 is less than one, the disease does not spread within the population. Although infected individuals make contact with

susceptibles, the rate of disease transmission is slower than the recovery rate. Mathematically, this means $\beta/\gamma < 1$, or $\beta < \gamma$, where β represents the transmission rate and γ the recovery rate. Consequently, the healing process is faster than the spread of infection, and the number of infected individuals quickly drops to zero. The majority of the population remains susceptible, and this state is globally asymptotically stable, as demonstrated by [10].

- **Epidemic spread ($\mathcal{R}_0 > 1$):** When the threshold is greater than one, the number of infected individuals grows until it reaches a peak and then declines toward zero. The peak number of infected people, as well as the final number of susceptibles, can be calculated using the system's initial conditions and the values of β and γ , as described by [34]. As reported in this work:

Let $s_s(t), i_s(t)$ be a solution of the system

$$\begin{aligned} ds/dt &= -\beta is \\ di/dt &= \beta is - \gamma i \end{aligned} \tag{3.2}$$

with $s(0) = s_0 \geq 0$, and $i(0) \geq 0$. The mass conservation assumption holds so $r(t) = 1 - s(t) - i(t)$. The triangle in the si phase plane given by

$$T(s, i) | s \geq 0, i \geq 0, s + i \leq 1$$

is positively invariant and unique solutions exist in T for all positive time, so that the model is mathematically and epidemiologically well posed. If parameter σ is defined as $\sigma = \beta/\gamma$ it holds that if $\sigma s_0 > 1$, the $i(t)$ first increase up to a maximum value $i_{max} = i_0 + s_0 - 1/\sigma - [\log(s_\infty/s_0)]/\sigma$ and then decrease to zero as $t \rightarrow \infty$. The susceptible fraction $s(t)$ is a decreasing function and the limiting value s_∞ is the unique root in $(0, 1/\sigma)$ of the equation

$$i_0 + s_0 - s_\infty - \log(s_\infty/s_0)/\sigma = 0.$$

This scenario illustrates how a highly aggressive infection can spread widely within a population. If no countermeasures are taken, it can lead to significant social and economic consequences. A typical epidemic outbreak has an infective curve that first increases from an initial I_0 near zero, reaches a peak, and then decreases toward zero as a function of time. The susceptible fraction $s(t)$ instead always decreases, but the final susceptible fraction s_∞ is positive. The epidemic dies out because, when the susceptible fraction $s(t)$ goes below $1/\sigma$, the replacement number $\sigma s(t)$ goes below 1.

Thus, even with its simplicity, the SIR model provides critical insights into the potential severity of an epidemic and highlights the importance of timely interventions to prevent widespread harm.

The SIR mean-field model equations

After introducing the basic framework of the model, the set of differential equations that describe the rates of change in the system's state is presented. The equations, along with

initial conditions, are necessary to fully define the model. The class sizes are expressed as fractions of the total, constant population.

The model is then

$$\begin{cases} ds(t)/dt = -\beta s(t)i(t), & s(0) = s_0 \gg 0; \\ di(t)/dt = \beta s(t)i(t) - \gamma i(t), & i(0) = i_0 > 0; \\ dr(t)/dt = \gamma i(t), & r(0) = r_0 = 0; \end{cases} \quad (3.3)$$

where

$$s_0 + i_0 + r_0 = 1. \quad (3.4)$$

Several works analyze this model and provide a detailed mathematical derivation of its solution [12, 28, 33]. However, this is not the focus of the present work. Instead, we provide an overview of the main results and a summary of the dynamics that emerge from the model.

Model behavior

To present the evolution of the model, a simple numerical simulation is carried out. The removal rate is set to $\gamma = 0.1$, meaning the infection lasts, on average, 10 days. The transmission rate is $\beta = 0.5$. At the start of the simulation, the majority of the population is in the susceptible class, with only a small portion of individuals already infected.

The initial reproduction number is $\mathcal{R}_0 = 5$, which is greater than 1. As discussed in Section 3.3.2, this indicates that the disease will evolve into an epidemic.

The key features that emerge from the model simulation are:

- **Slow initial phase:** The early stages of the epidemic are marked by a slow start, as seen in the first part of the curves in Figure 3.2a. The curves remain nearly flat because only a few individuals are initially infected, and it takes time for the infection to spread and reach a larger portion of the population.
- **Exponential growth:** In the second stage, the number of infections increases exponentially.
- **Peak infection:** Eventually, the number of infections reaches a peak.
- **Residual susceptible population:** By the end of the epidemic, a portion of the population remains susceptible, though this amount depends on the model's parameters.

An additional detail emerging from the model, as analyzed in [30] and visible in Figure 3.2, is the evolution of the reproductive ratio over time. The reproductive ratio is given by the formula $\mathcal{R}(t) = \frac{\beta}{\gamma} \cdot s(t)$. Initially, assuming $s(0) \approx 1$, we can simplify this to obtain the value of \mathcal{R}_0 . However, as the susceptible fraction decreases exponentially alongside the growth in infections, the reproductive ratio also decreases, and the infection peak occurs when $\mathcal{R}(t) = 1$.

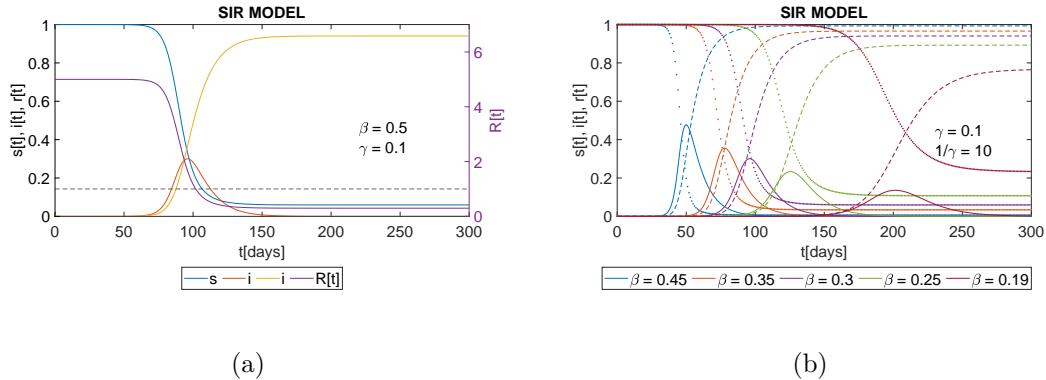


Figure 3.2: SIR system numerical solutions. Figure a) shows the evolution of compartments in the case of an epidemic. The violet dotted line represents the time-dependent $\mathcal{R}_0(t)$. It can be seen that when this parameter is equal to 1, the number of infected reaches its maximum value. In b) 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.

Two key metrics to consider during the emergence of a new disease are the rate of increase of infections and the final size of the remaining susceptible population by the end of the epidemic. The course an epidemic takes can vary dramatically depending on whether it spreads rapidly, overwhelming healthcare systems, or whether interventions succeed in "flattening the curve." The rate of infection growth depends on how quickly the disease spreads through the population. If an epidemic grows rapidly, a large number of people fall ill in a short period, potentially overwhelming healthcare resources. On the other hand, the final size of the susceptible population indicates how many individuals remain uninfected once the epidemic has run its course. This is influenced by factors such as herd immunity or interventions like vaccination. "Flattening the curve" refers to strategies aimed at slowing the spread of the disease, thereby reducing the peak number of infections and spreading cases over a longer period. This reduces the strain on healthcare systems without necessarily reducing the total number of infections.

Some strategies for flattening the curve include:

- Social distancing and quarantine to reduce contact between susceptible individuals and the infected. This decreases the transmission rate β .
- Vaccination, which immediately reduces the number of susceptible individuals, thus preventing the disease from spreading widely.

While flattening the curve may not lower the total number of infections (i.e., the cumulative number may remain the same), it reduces the peak number of active cases at any given time, which is crucial for ensuring that healthcare systems are not overwhelmed.

3.3.3 Other modeling techniques

There are several ways to model an epidemic, and the main categories of these models are now introduced. Many of the articles that will be reviewed in the next chapter 4 make use of one or more of these model types.

Stochastic models

This group of models, which originate from the mean-field approach, utilizes a different mathematical framework. In these models, the transition between states is governed by stochastic functions. Conceptually, they share the same foundation as ordinary differential equation (ODE) models but differ in application. These stochastic models are particularly useful when studying diseases with a lower number of infected individuals or when the epidemic's outcome is influenced by changes in individual dynamics, a phenomenon known as demographic variability. Demographic variability includes changes in transmission rates, birth rates, recovery rates, or mortality within the population. One approach to model such variability is through stochastic models paired with Monte Carlo simulations [35].

Networked models

In this class of models, disease dynamics are considered over complex and realistic networks, with a focus on understanding how the network structure impacts the epidemic's spread by analyzing parameters like the rate of infection. The model represents individuals as nodes in a graph, with edges illustrating interactions between them. Nodes can also represent subgroups, and the relationships between individuals or groups can be weighted, representing varying strengths of interactions [36, 37]. The larger the number of nodes and the more accurately the connections reflect real-world interactions, the better the model is at reliably simulating the spread of the disease.

Agent-based models

Agent-based models (ABMs) simulate the progression of a disease by focusing on the behavior and interaction of autonomous agents, which could be individuals or collective entities like organizations. This modeling approach is built on observing spontaneous interactions between individuals [38], creating a dynamic system where each person acts according to certain rules. The goal is to understand how these individual behaviors influence the overall evolution of the system.

One key advantage of ABMs is that they provide an intuitive way to interpret epidemic modeling by focusing on individual perspectives, making the model's results easier to understand. Additionally, because agents embed individual behavior, ABMs can deliver highly detailed simulations and offer insights into specific countermeasures that might help mitigate the spread of a disease.

However, ABMs require a large amount of detailed, reliable data to be effective, as their accuracy depends on the precision of the information integrated into the model.

Collecting and incorporating this data can be a challenging task, which is a potential limitation of this modeling approach [10].



Figure 3.3: Agent based network representation

Multilayer systems and networks

The complex dynamic of interactions existing in the real world, develops in multiple patterns, with complicated relationships. These interactions can change over time, and using the theory of multilayer systems 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. In 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 [17, 40] and can be helpful to describe complex systems like human relationships. An interesting result obtained is 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 [17]. 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 model that considers, for each agent, both its physical contacts with others, where the disease can be transmitted, and its network of relations, representing the social dynamics in which everyone is involved. An example of this network is shown in Figure 3.4. 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 [41]. Multiple systems can have either a single or coupled dynamic. In the case of a single dynamic, there is a top layer whose evolution occurs independently on top of a multilayer network. In contrast, a coupled structure describes phenomena in each layer evolving under the mutual influence of what is happening in the other layers.

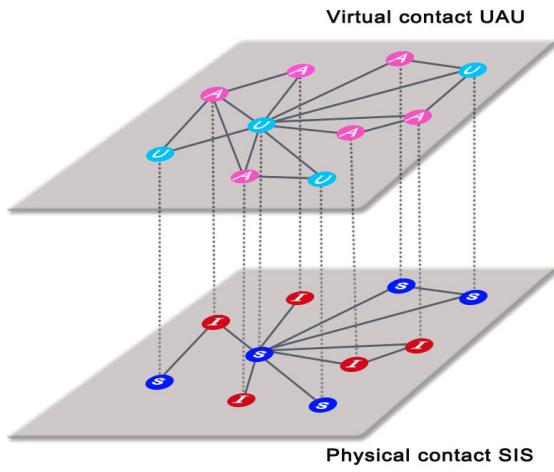


Figure 3.4: Representation of a multiplex structure. The figure is taken from the work of [39] and shows the network implemented in their model. There are two layers coupled together: one representing awareness and the other the epidemic state. In this case, nodes connected by interlayer connections represent the same individual. Thus, the model describes people with two attributes: awareness of a disease and health state. The mechanisms of infection and becoming aware are distinct, but both attributes influence how an agent's state evolves. For example, if an agent is aware of the disease, they may act more cautiously, thereby reducing their probability of becoming infected.

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 [42], 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 epidemiological initial works, the focus of scientists was primarily on presenting 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 infectious [10, 20]. It was only later, as epidemiological models proved effective and reliable in describing and predicting disease spread, that interest in their beneficial impact on population safety and well-being grew. Tools that integrate real data with epidemiological models emerged, helping inform decisions on matters such as school closures or travel restrictions, as described in [42].

Furthermore, new categories of models have emerged, such as agent-based models, net-worked models, and multi-layer/multi-system models. Despite their differing approaches, they aim to integrate various population characteristics, for example contact structure, age distribution, and movement patterns, to address the limitations of the original homogeneity assumption [13]. 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 is to incorporate changes in the structure of models that describe aspects of behavior or population composition. 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 uses this methodology for analyzing COVID-19 include [25, 26, 43].

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 capture both behavior effects 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 behaviors be integrated into pre-existing epidemiological theory? To better address this question, we follow the classification proposed in [44], 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.

The first two points focus on distinguishing between the various strategies implemented to model and integrate information dynamics, which play a crucial role in behavioral models. In this context, information acts as an infectious agent within the social layer. Models incorporate this effect in different ways, highlighting how messages are communicated—whether through media or conversation—and the type of information researchers choose to emphasize (e.g., fear of the disease or data on infection numbers). The last categorization is dedicated to the different strategies used to integrate human behavior into these models.

4.1 Information's sources

When analyzing the source of information, there is a clear distinction between works [45] that assume that governments and populations base their decisions on precise data, such as the number of infected individuals (prevalence) [46, 47], and those that consider more informal sources, such as conversations between people, public opinion, or media [48, 49]. 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 [50]. Several works analyze its effects to on the spread of infection [19, 51]. An example of how this phenomenon can occur is the case of an article originally published by a prestigious source. Although the thesis presented in this work, which claimed a correlation between the measles, mumps, and rubella (MMR) vaccine and autism or other gastrointestinal disorders, was later debunked by the scientific community and the article retracted [52], the negative impact in terms of spreading fear about vaccines has persisted. In many cases, this fear has become deeply ingrained, leading to a reduction in herd immunity and a resurgence of measles [42].

4.2 Classification of different types of information

Another interesting aspect relates to the different types of information used as a basis for developing a new model. Choosing a specific type of information leads to the creation of distinct models that focus on different aspects of behavior. Some studies focus on the influence of media on behavior [46, 53], while others examine peer-to-peer conversations, information exchange, and individual beliefs [47]. These are completely different approaches, even though they aim to achieve the same effect: simulating the evolution of people's opinions and behavior. Incorporating 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" [54]. To model how awareness spreads in the population, it is often treated like a disease [39, 41, 55–58]. 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 build their ideas [59, 60] and also analyze the possible formation of opinion distributions, such as perfect consensus, consensus, polarization, clustering, or dissension.

4.3 How to 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 [16, 41, 61].

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 discussed in [56].

There is also a broad class of mean-field models that explicitly consider the effects of behavior. In these cases, 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. Refer to paragraph 4.3.2 for further details on

this topic.

Another possibility involves modifying the structure or connections in the network used to simulate disease evolution [62]. In network-based models, data extracted from social network structures [63] or small-world models [64] 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 Individuals-based models

Multiple networks simulated with Markovian process

To begin the presentation of individual-based models, the first category discussed is network simulations using Markovian processes. A notable example is the article by Granell et al. [56]. In this work, a multiplex model is implemented with two distinct connectivity layers: the physical layer, where the disease spreads, and the virtual contact layer, through which "awareness" about the disease diffuses. Awareness is in the knowledge about how reduce the risk of infection, and diffuses through conversation or is due to becoming infected. The article then uses the Microscopic Markov Chain approach to simulate the interaction resulting from the coupling of the two layers. Interestingly, the authors observe the existence of a metacritical point for the onset of the epidemic, which depends on awareness dynamic and topology of the virtual network. There is, in fact, a parameter related to the ability to influence through communication and it is observed that such parameter impacts the onset of the epidemic only when it exceeds a certain threshold. A subsequent work of the same authors [39], considers also the effect of a global communication agent. In this case, the metacritical point disappears.

In the article by [65], there is a complete description of the stochastic process at the agent level, which is useful for understanding how agent interactions are modeled across different layers using a Markovian approach. Other works using this method include [55, 58, 62]. Here, a similar double-layer structure, composed of an SIR model coupled with an unaware-aware-unaware (UAU) process, is presentet. To simulate the evolution of the complex structure resulting from the coupling of the two models, these works develop transition trees for all possible state changes and their respective transition probabilities. An example can be seen in figure 4.2.

In [62], a slightly more complex situation is described, where two possible opinions—pro-physical distancing (P) and anti-physical distancing (A)—are considered. In contrast, [66] studies a simpler structure, where a SIS model is coupled with either adopting or not adopting self-protective measures.

Interesting results derived from these works include:

- The observation of the influence of opinions on transmission speed and the final epidemic size [62].
- The effect on epidemic spreading of authoritative information, publicizing epidemic prevention processes, and encouraging reasonable behavior, such as isolating when

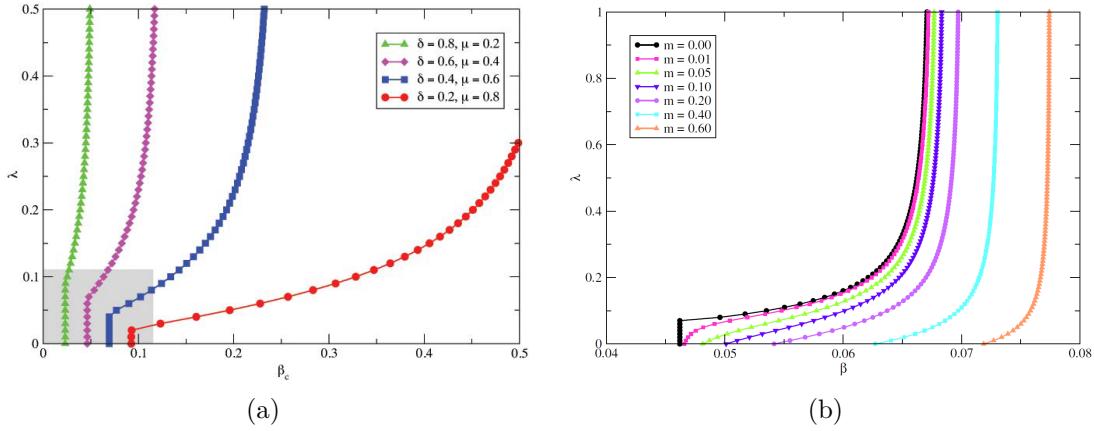


Figure 4.1: The effect of awareness communication on the onset of an epidemic is depicted in the studies by Granell et al. [39, 56]. In the first plot (a), the x-axis represents the minimum value of the transmission rate β that is necessary to trigger an outbreak, while the y-axis measures the level of awareness in the population, denoted by the parameter λ . The shaded region shows that below a critical threshold of λ , awareness has little to no impact on controlling the epidemic. However, once awareness exceeds this threshold, the value of β required for an outbreak increases significantly, indicating that the spread of awareness can effectively delay or prevent the epidemic. In contrast, in the second plot (b), a global communication agent, represented by the media parameter m , continuously influences the population. This parameter ensures that the awareness layer consistently impacts the epidemic dynamics, regardless of the value of λ , making it evident that a strong media presence can amplify the protective effects of awareness.

infected [58].

- The importance of self-awareness as a mechanism to reduce disease prevalence [55].

The investigation conducted in [66] provide a stability analysis of a two-layer model that links the decision to use or not use self-protection measures with the disease state. The equilibria of the system are explicitly calculated, including the epidemic threshold. Figure 4.3 illustrates their simulations, where a parameter representing risk perception is varied. The study shows how this parameter influences the emergence of periodic oscillations and identifies the conditions that lead to global convergence towards such periodic solutions. This is a significant result, indicating that during an epidemic, there is a collective behavioral response, and it specifies under what conditions the system stabilizes or becomes dynamic.

Game theoretical models

In the probabilistic framework, another area involves the use of game theory principles. These are used to explore strategic interactions between individuals, where participants act to maximize their utility, potentially influencing the actions of others. The concept

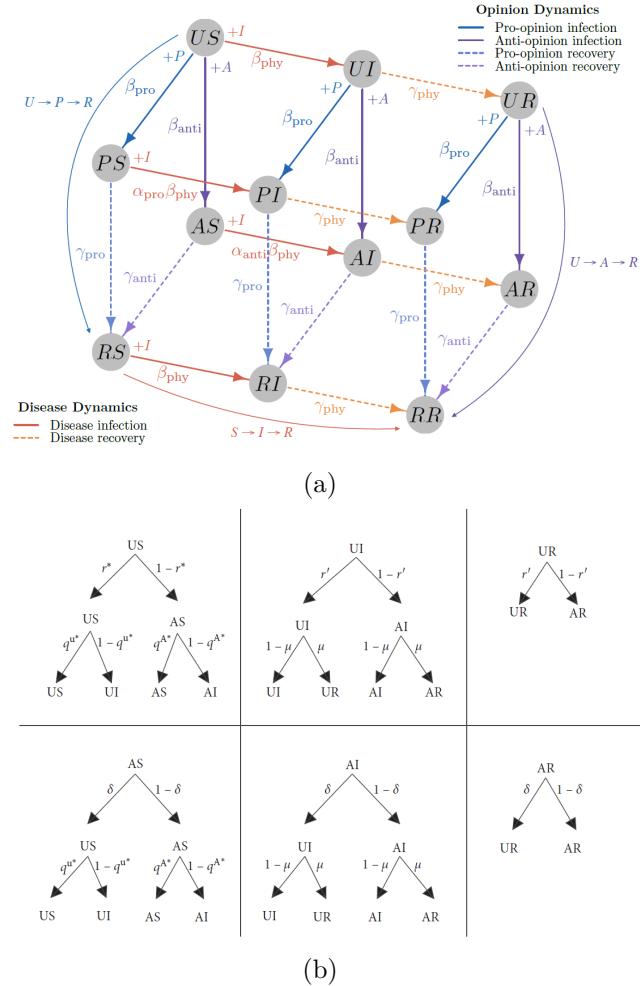


Figure 4.2: a) An example of a multiplex network structure resulting from the coupling of a SIR and a uninformed-pro/anti physical distance-recovered (U-P/A-R) model. b) The transition trees realized to describe the system of a SIR coupled with a UAU model using a Markovian process. Pictures taken from articles [55, 62].

of Nash Equilibrium is also important in this context. It is defined as "a set of strategies such that no player has an incentive to unilaterally deviate from the present strategy" [16]. That is, the Nash Equilibrium leads individuals to adopt strategies consistent with their goal of maximizing their benefit or utility in a perfectly rational way, forming the best responses to one another.

Many articles use this idea to model how populations adjust their behavior during an epidemic. One such example is [67], which focuses on the behavior of a population deciding between their sexual habits and the risk of HIV infection. The main result is derived by observing how population behavior changes as information about a possible

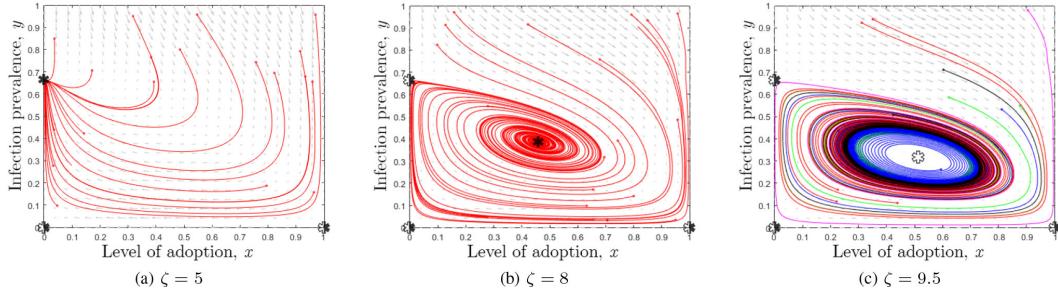


Figure 4.3: Simulations from the article [66] demonstrate the evolution of their model across various values of the risk perception parameter. In the visual representation, the x-axis shows the adoption level of precautionary measures, while the y-axis indicates the prevalence reached by the infection. The diagram highlights stable equilibria, saddle points, and unstable equilibria, which are marked with black, black-white, and white asterisks, respectively.

vaccine spreads. Optimistic news lead to a decrease in the number of contacts, while pessimistic forecasts cause an increase in risky behavior, even at the same level of risk, as shown in figure 4.4. A particularly interesting conclusion is that focusing public health messaging on dire forecasts may unintentionally lead to an increase in risky behavior.

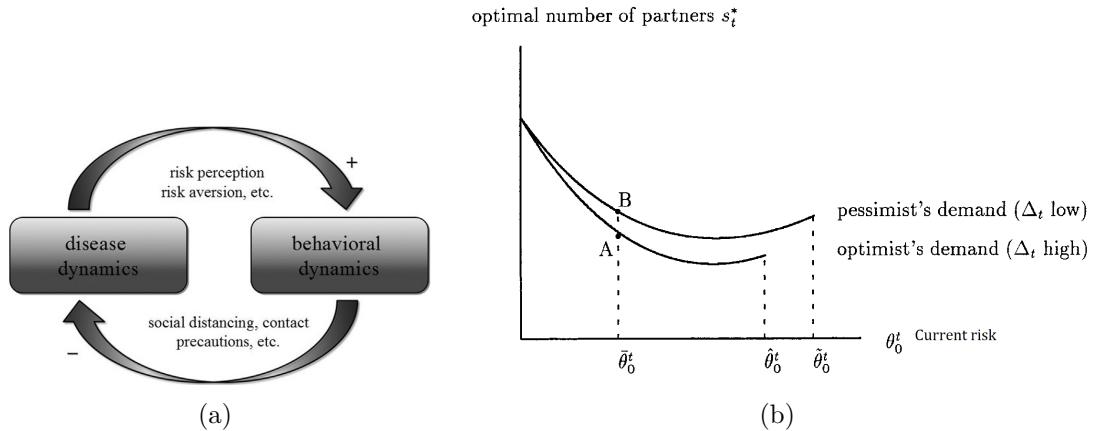


Figure 4.4: a) A representation of the feedback loop, taken from the article of [68], representing the trade-off between: advantages related to avoid the disease and social cost of behave using precautions. b) The effect on people’s behavior due to optimistic or pessimistic forecasts is described in the illustration presented in [67]. The population tends to act more cautiously if there is hope that the situation will improve in the future.

A different focus is the one from [69]. Here, the authors study behavioral change related to contact rates and especially social distancing, in the context of a pandemic situation like COVID-19 to understand the efficacy of policies for partial or full voluntarily contact reduction. They aim to realize more insight into the percentage of adoption from the

population of social distancing policies because increasing the quality of these estimations matters in the planning of strategies to handle a pandemic from a government point of view.

Another case study is [70], which defines different utility functions to model the trade-off between social well-being from maintaining connections, the fatigue of doing so, and the potential physical harm those connections may cause. The main results outlined in [70] confirm that a higher number of connections between individuals leads to greater disease transmission, resulting in more infections and a shorter epidemic duration. It also highlights that "the higher the (perceived) risks of a disease, the lower the net benefit of a tie, the stronger the social distancing, and consequently the smaller the epidemic size." Using this co-evolutionary approach, a highly correlated dynamics between the two layers emerges: a feedback loop between the spread of infection and behavioral adaptation, with structural modifications in the network occurring in the simulated scenarios. The introduction of network-based modeling further develops this work and leads to several key findings. First, including the benefit of social connection creates multiple transmission routes for the disease. Second, a reduction in the final epidemic size only occurs when the indirect benefits are relatively low and the costs of maintaining ties are high. Finally, small changes in social behavior can have large impacts on the epidemic. In the next paragraph, other similar studies that incorporate network models will be discussed. However, before that, a final case where the game-theoretical approach is often applied will be presented: vaccination. Many models examine the decision-making process behind vaccination, highlighting the trade-off between the benefits of getting vaccinated and the risks associated with it. In terms of modeling, the link between behavior and epidemic spread in this case is that individuals who choose vaccination are removed from the susceptible group, with a percentage reflecting the vaccine's efficacy, thereby reducing the potential for disease transmission. In the study developed in [51], a feedback loop is established between disease prevalence and individual strategic vaccination behavior. Their model successfully fits vaccine coverage data from both the pertussis and MMR vaccine scares and can also predict future trends in disease prevalence and vaccine coverage. Moreover, the article highlights the phenomenon for which the vaccine fear becomes more frequent as eradication goals for more vaccine-preventable diseases are approached.

Network based models

The inclusion of networks in the modeling process has gained popularity as a tool for scientists to enhance the accuracy of their models by simulating real-world connections between people. The main goal behind developing network-based models is to create a representation of society and then use it to simulate the spread of disease. A comprehensive example of this approach is presented in [37], where a method is introduced to simulate scenarios such as quarantine or regional barriers that limit population movement. By adjusting network connections—reducing contacts between nodes or cutting ties between specific regions—these models effectively demonstrate the impact of interventions like lockdowns or travel restrictions on disease transmission. They also enable analysis of how containment measures affect the trajectory of an epidemic. Works such

as [38] also fall into this category, utilizing urban mobility patterns as a proxy for modeling epidemics. Similarly, in [63], social networks are used as a proxy for connections, hypothesizing that people’s behavior in maintaining social contacts is analogous to how they might behave in the context of disease transmission. Another innovative approach involves the development of multilayer networks, such as in [64], where the social structure of a town is recreated. Each layer represents a different environment—ranging from homes to workplaces, distinguishing between various job types, and even considering a layer for friendships. Each individual exists across multiple layers and interacts with different groups depending on their social environment. This model found that the layer associated with friendship poses the highest risk for outbreak development, due to closer interactions and lower security measures. Consequently, even on the friendship layer, a lower transmission rate (β) compared to other layers can result in a significant epidemic involving many susceptible individuals.

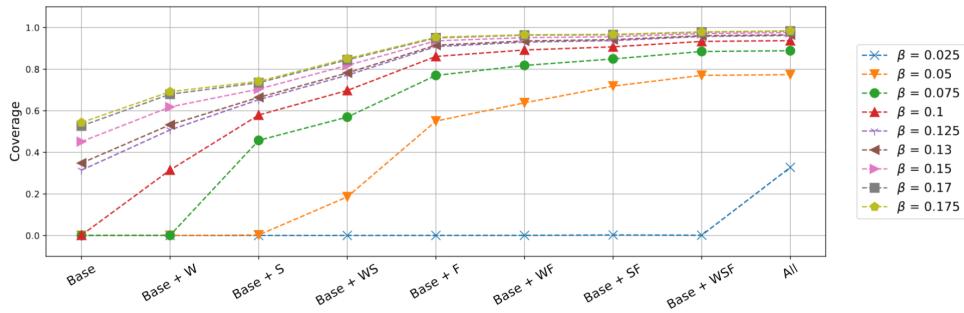


Figure 4.5: In the simulation presented in [64], a city is modeled with people divided into several social groups. The authors found that the layer associated with friendships is where the disease outbreak occurs with the lowest value of the infectivity parameter, β .

Threshold models

Another possible mechanism for modeling how individuals change their actions is by observing the behaviors and opinions of their neighbors [22, 71]. A well-known theoretical tool for this context is the Watts threshold model [72], which is foundational for studying such transitions. In [41], various threshold models are discussed, including the Watts threshold, which is linear. In their model, each node is assigned a random threshold value based on a given distribution. The threshold represents the point at which a node changes its opinion when a certain number of its neighbors adopt a different behavior. The structure of the network is crucial for determining how opinions spread. The authors found that opinion propagation is most favorable in networks with low randomness and a regular structure. Additionally, they analyzed the effects of network clusters, noting that well-connected clusters can act as opinion hubs, reinforcing the spread of opinions.

Ad-hoc rule-based models

The last category of individual-based models focuses on individuals acting according to specific rules designed to simulate particular situations. A clear example of this is found in [73], where disease propagation is modeled based on opinions for or against vaccination. The evolution of these opinions is determined by the interaction and exchange of ideas between agents and co-evolves alongside their health condition. A comprehensive set of rules is established to model all possible situations that lead to changes in both opinion and disease states. An example is visible in picture 4.6

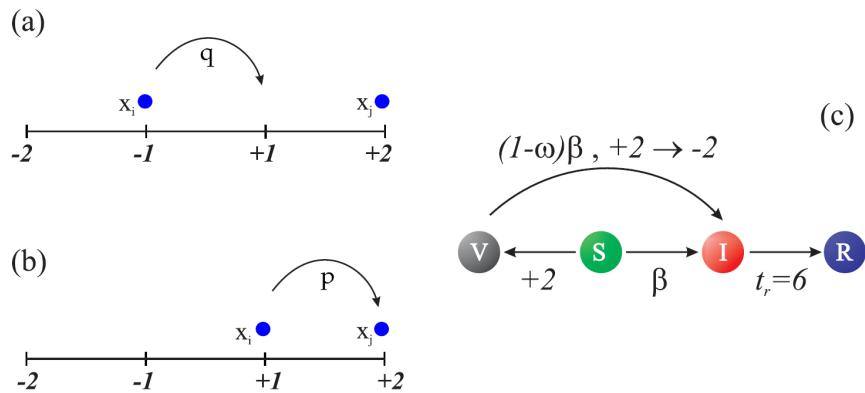


Figure 4.6: The picture, taken from the article [73], illustrates the mechanisms underlying the model's dynamics. The figures on the left depict opinion dynamics: when two nodes have opposing opinions, one adjusts its state to match the other's opinion with a probability $q(a)$. If both nodes share the same opinion, the opinion is reinforced with a probability $p(b)$. The figure on the right represents contagion dynamics: a susceptible individual S (green) becomes infected (red) with a probability β and recovers (blue) after a time t_r . A susceptible individual can also become vaccinated V (grey) upon acquiring an opinion state of $+2$. However, they can still become infected with a probability $(1 - \omega)\beta$ - with ω the efficiency of the vaccine- which causes their opinion to shift to -2 .

A similar approach is developed in the article [18], where an explicit mechanism is implemented to govern the competition between different health opinions. Individuals with a positive $+$ opinion may switch to the opposing $-$ opinion after interacting with others, following a switch rate function. By varying the parameters of this function, its behavior can become linear, saturating, or sigmoidal similar to established functions used to describe predator responses to prey population density.

Finally, the article by [46] extends the SEIR model by incorporating the effect of mass media on disease spread, using a specific set of functions. These functions account for disease prevalence, recovery rate, and media impact. The goal is to conduct a sensitivity analysis on the parameters influencing the epidemic's peak magnitude, timing, and ending.

4.3.2 Homogeneous population models

This section presents the works that have most contributed to shaping the development of the thesis, the mean-field models. The assumption that the population is homogeneously mixed results in models capable of describing phenomena nationwide, which is difficult to achieve when modeling individual behavior.

However, the effectiveness of this class of models relies heavily on the modeling principles applied. Models are powerful tools, but they represent the aspects the modeler chooses to emphasize. Therefore, selecting and integrating the most promising features is crucial for creating a useful instrument. By analyzing prior works, we gain insights into what has been previously explored and the outcomes achieved. The most interesting characteristics of various models are now presented, followed by an explanation of how they have contributed to the development of the model in this thesis.

The article [47] was one of the first studied for its interesting modeling approach. It integrates two dynamics: the epidemic evolution influences the parameters governing people's behavior, and, conversely, the population's behavior affects the spread of the disease. This bidirectional interaction allows for a more realistic simulation of how behavioral changes and disease dynamics influence each other. A SIR model is associated with an opinion dynamic that occurs only within the S compartment. This compartment is divided into four subgroups, representing different attitudes toward prophylactic behavior. In this way, more cautious individuals have a lower probability of becoming infected. The opinion dynamic focuses on the phenomenon of influence, modeled by a specific parameter, and on opinion amplification, a cognitive bias where confronting someone with the same belief strengthens that belief. The most interesting aspect of this work is the concept that opinion spreads through conversation, not through a utilitarian or contagion process like fear diffusion.

A similar hypothesis of social learning is explored in the article [74]. In this model, both risky and cautious behaviors coexist in the population and can be transmitted. The model also incorporates the effects of clustering and the phenomenon of "cultural bias." This bias suggests that the risky trait is more likely to be adopted by cautious individuals than the reverse. Additionally, the authors introduce the concept of uncertainty regarding the infection causes, meaning that people are unsure of the best way to behave to avoid contracting the disease.

In the article [75], compliance with the use of NPIs (Non-Pharmaceutical Interventions) is the central focus of the behavioral component of the model. In this case, non-compliance is modeled as a social contagion: the population is divided into two groups, compliant (c) and non-compliant (nc). Using the mass-action mixing property, compliant individuals become non-compliant, but there is no recovery once their status changes. The primary goal is to understand the interplay between the stringency of lockdown measures, non-compliant behavior, and the spread of the disease. Vaccine adoption and awareness diffusion are the main arguments developed in [76]. Awareness is present only in the Susceptible compartment, and there is a term, $M(t)$, that represents the accumulated density of awareness programs driven by various information sources. This term is influenced by several factors: awareness generated by neighboring individuals, the intensity of awareness

programs in response to the prevalence of the disease, and a waning effect due to the decreasing quality or effectiveness of the information over time. Their complete model and the interplay between disease and behavior is shown in figure 4.7. An interesting aspect of this article is that the authors evaluate their model using data from the COVID-19 vaccination campaign in China. They observe how their model effectively reproduces the population behavior and government policies during different phases of the epidemic.

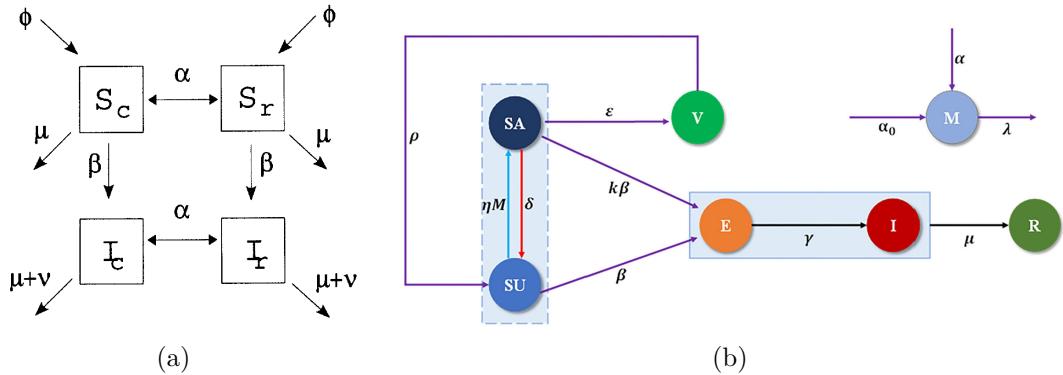


Figure 4.7: a) The model presented in [74] shows horizontal layers representing behavioral diffusion, while vertical layers represent disease spread. Behavior diffuses between both infected and susceptible individuals, but this is not depicted for visual clarity. b) The model developed in [76] incorporates behavior dynamics only in the susceptible (S) layer, influencing both contagion rates and the probability of vaccination. The M compartment, which is the accumulated density of awareness programs, follows its own dynamics, observing the state of the disease and the distribution of public opinion, and it influences the diffusion of awareness.

An interesting article on the subject of behavior and vaccines is [19]. In this model, an initially susceptible population can split into two opposing compartments, depending on what they fear more: the disease or vaccination. The model includes six compartments, with the fear dynamics occurring only in the susceptible (S) compartment.

In this scenario, fear of vaccination can undermine outbreak control. Initially, people may get vaccinated, but they stop too early as their fears reverse. The study also conducts a sensitivity analysis on the contact rates for the two fears, showing that transmission speed significantly affects the model's outcomes. The results range from multiple infection waves to complete disease extinction without an outbreak, depending on the conditions.

4.4 Perspective review of the literature

To conclude this chapter on the literature review, an evaluation of the models discussed is presented, with a focus on how they relate to the scope and aim of this thesis. This evaluation wants to explain the key insights gained from the literature and highlight also what are the differences and novelty introduced in this work.

4.4.1 Individual state models

Referring to the individual-based models presented in the previous section 4.3.1, most face the challenge of developing complex simulations to model the evolution of disease and individual behavior, but struggle to scale these simulations to the nationwide level. In many cases, small groups of agents are used, such as the 50 nodes mentioned in [70], and even in works where a larger number of nodes is implemented, the count is typically in the thousands [39], not in the millions, as would be necessary for national-scale modeling. To overcome this difficulty, some articles use mean-field approximations, considering the limit of an infinite population size and employing statistical approaches [66].

Another critical issue these models face is the need for large amounts of data to accurately represent how populations behave during unusual situations like epidemic outbreaks. Without this data, it becomes difficult to draw precise conclusions from the models. While these models can still provide useful insights, their application for making precise, real-world predictions remains limited. They are better suited as scientific tools for exploring theoretical scenarios rather than for offering actionable advice on a larger scale. An article that demonstrates the volume of data required to test a model with real-world scenarios is [77]. In this study, data from three different countries—Luxembourg, Austria, and Sweden—was collected, including information on total detected cases, hospitalized individuals, people in intensive care units (ICUs), and deaths. This comprehensive dataset was used to fit the model.

In the case of behavioral-epidemic models, even fewer data were available until the recent COVID-19 pandemic. As explained in [78], this lack of data was a significant challenge. However, they show how it was partially addressed by implementing models based on population behavior with respect to influence. Despite these improvements, the availability of data today is certainly better, offering more robust insights. As they say: *"The issue is that research has not yet provided empirical benchmarks for endogenous contact rates in disease scenarios, so it is unclear how such policies can be evaluated scientifically: ideally a policy is benchmarked against a set of counterfactuals given the disease, not compared with what was before the disease"*.

4.4.2 Well-mixed population models

As stated earlier in paragraph 4.3.2, a major critique of developing complex mean-field models is that if they are not supported by consistent observation of the phenomena they aim to reproduce, their capacity to generate meaningful insights can be significantly limited. For this reason, confront and develop a model using empirical data as reference, is one of the main objectives pursued in this thesis work. The comparison of the model's emerging dynamics with real-world data is a reliable method to test it. Reading through works in this field, a common approach emerges for modeling systems that aim to incorporate two distinct dynamics, such as behavior and disease spread. Most of these models introduce additional compartments, which represent subgroups of homogeneously mixed individuals sharing common characteristics—typically, their disease state and course of action. The primary distinction lies in how modelers handle the flow between these compartments: while some works focus on the influence of behavior solely within

the Susceptible layer [19, 47, 76], others implement a full double-layer model [48, 74, 75]. Another key difference is whether the change in behavior dynamics is unidirectional, as in [75], or bidirectional, as seen in [19, 47, 74, 76], where individuals can adjust their actions in both directions.

Notably, some of the most influential aspects drawn from the literature for this thesis include the "double fear" structure developed by [19] and presented in 4.3.2, and the awareness element that influences behavior change, modeled through an external node $M(t)$ in [76], and seen in paragraph 4.3.2. Additionally, this thesis seeks to implement a fully coupled double-layer model for behavior and epidemic spread, incorporating elements such as memory waning and fatigue, which combine with conversational mechanisms to create bidirectional flows in the behavioral model. Unlike [74], which uses a simpler epidemic model, this work employs a more complex one to accurately reflect COVID-19 dynamics. Furthermore, the models developed in [48] and [79] share structural similarities with this thesis. However, [48] assumes faster information diffusion than disease transmission, decoupling the two layers, while [79] focuses on homophily and polarization of compartments—factors not considered here due to data limitations.

4.4.3 Final remarks

All the concepts presented in this chapter are useful for understanding the current state of epidemic behavior modeling and for explaining why the development of a new model was necessary for this thesis.

To summarize, the main features sought in a model are:

- Comparability to empirical data collected during a real epidemic, both for the disease and the behavioral layer.
- In a mean-field system, the ability to model disease incidence, peer pressure between individuals with different behaviors, fatigue in maintaining certain habits, and the possibility to modify these dynamics by introducing a mean-field intervention, such as government actions, to encourage the transition to more compliant behavior.
- Avoidance of using awareness as a factor in the model, as it is too abstract and difficult to observe or measure within a population; instead, focusing on behavior dynamics and peer-to-peer imitation based on which behavior appears more convincing.
- Exclusion of vaccination dynamics, as it is a well-known aspect and, in the case of COVID-19, vaccines were only available on a large scale after more than a year.

All these characteristics have been incorporated, and the following chapter will explain how the model was developed and how these features were integrated.

Part II

Behavioral-Disease Model

Chapter 5

Model development and justification

Because the main contribution of this thesis work is the presentation of a new multi-system model, understanding the reasons that led to its development is crucial. Otherwise, the reader might question: "Why not use an already developed and analyzed model?"

The primary answer lies in the observation made while studying the literature: the connection between empirical data and epidemiological models is often missing. Most works relating social and epidemiological aspects are purely theoretical models based on ad-hoc assumptions; probably, because constructing a framework grounded in empirical data related to individual behavior is challenging [70], and, until the Coronavirus pandemic, data on this topic were scarce.

The availability of research such as the one conducted by Meta during COVID-19 [80] serves as both a major source of inspiration and data. This research allows exploration of different behaviors related to how people react to and manage the necessity of living with an infectious disease. It also provides this valuable information as a dynamic time series.

Often, what emerges from this data is the non-linear dynamic evolution of behavior. While many models not incorporate this characteristic [81], this feature is here addressed. The behavioral-epidemic mean-field model developed aims to interconnect these two fea-

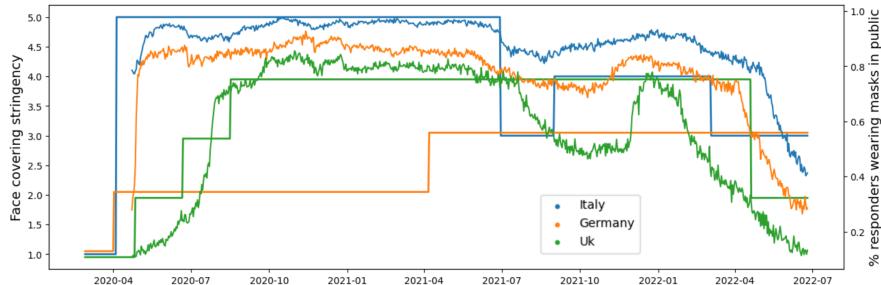


Figure 5.1: The evolution of mask wearing behaviors. Figure from [84].

tures, linking the theoretical framework with empirical evidence. Often, in other works, this connection is realized using proxy models that attempt to reconstruct agent behavior, spatial motion, or opinion datasets extrapolated, for instance, from social networks, as done in [82, 83]. The problem with these approaches is that people's opinions do not always align with their actual behaviors, and the lack of a necessary and direct correlation between the two is another concept the model attempts to overcome.

For example, consider the evolution of mask-wearing behavior in different European countries, as shown in figure 5.1. It is immediately evident that, at a certain point, there is a sharp increase in the use of this self-protective device, resembling a step function. This effect results from regulatory prescriptions introduced by authorities, with behavior closely following the evolution of these stringency measures.

Phenomena like this have been incorporated into the model's development using a coefficient parameter, ψ , to represent the effect of centralized interventions and modify the basic persuasion rate of different behaviors. Additional empirical evidence supporting the development of the model can be found in [84].

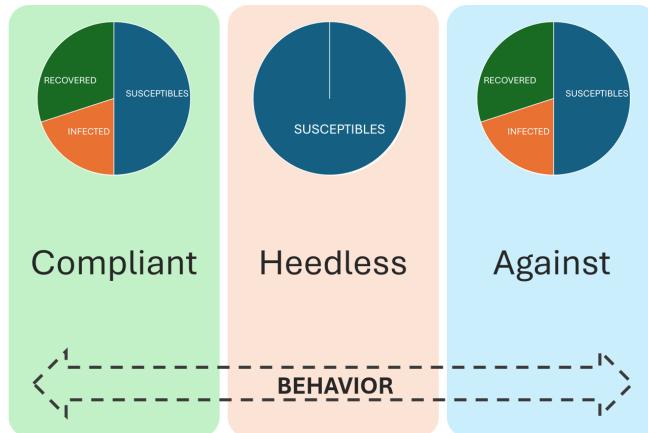


Figure 5.2: Representation of the model with individuals divided into different behavioral categories, each of which can correspond to a specific disease state, except for the Heedless group, which is characterized solely by Susceptible individuals.

To introduce the model and begin its description, the next chapter first presents and analyzes the two layers that together form the complete model: a SIRS epidemic model and a behavioral model consisting of three compartments: Compliant, Against, and Heedless. Then, the full model is presented, described and analysed in Chapter 7. Then the full model is presented, described and analysed. The full model comprises seven compartments, as the heedless behavior is not considered while individuals are infected or recovered. This assumption stems from the reasoning that it is highly improbable for an individual to act heedlessly when infected (unless, potentially, when completely asymptomatic). Figure 5.2 provides a compact representation of the population subdivision.

Infection can be transmitted across all three behavioral groups, but the Compliant group is more cautious. A parameter, ρ , models their reduced probability of being infected, while another parameter, ϵ , accounts for their compliance with self-isolation while

infectious. This reduces the number of infected Compliant individuals (I_C in the model equations presented later) contributing to new infections in the epidemic layer.

Behavior is "transmitted" through peer-to-peer influence ($k_i, i = 1, \dots, 6$), and fatigue parameters (λ_i) are included to model the dropout rate caused by the difficulty of maintaining a certain behavior over time.

For the epidemic model, classical coefficients are used: β (transmission rate), γ (recovery rate), and δ (immunity waning rate).

Chapter 6

Epidemic and Behavioral model alone: a presentation

To develop a multi-layer system that combines an epidemiological layer with a behavioral one, we first present the dynamics of each layer independently. This section briefly introduces the SIRS model, focusing primarily on the reasons for its selection. Then, the Heedless, Compliant, Against behavioral model is introduced, simulated, and analyzed. Understanding the underlying dynamics of this model is crucial for gaining insights into the complex interactions that emerge within the multi-layer structure.

6.1 SIRS model

To describe the epidemic evolution, a SIRS model is implemented. It is an extension of the most famous SIR (see discussion above). Its main addition is the possibility for individuals to become again susceptible after a certain period of time beyond the end of the infection. There are four main characteristic parameters in this 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.
- \mathcal{R}_0 is the reproduction number, derived as in the *SIR* model as the ratio between the β , and γ coefficients.

The choice of an SIR-like model is made because these models are well-known for their ability to describe diseases like COVID-19, and the literature provides numerous examples that use this model [25, 85]. The SEIR model could also be a viable choice due to the relevance of the "Exposed" compartment, which effectively captures the disease progression for infections such as COVID-19. In these cases, an incubation period occurs after exposure before symptoms appear and the individual becomes contagious. However, this compartment was excluded because it has been shown [25] that a simpler SIR model

can still accurately represent the disease's dynamics. When comparing model simulations with real data, a delay can be incorporated to account for the lag between symptom onset, testing, and reporting. This delay reflects the time needed for symptoms to manifest, conduct testing, and report cases to relevant authorities.

The model includes the possibility of reinfection, which is important when studying long-term scenarios. Considering the effect of individual behavior on disease progression, two critical phases are hypothesized to influence this evolution: the initial stages of the epidemic and the period following the first peak.

In the initial stages, the SIRS model behaves similarly to a typical SIR model because reinfection is unlikely to occur in a short time frame. However, over time, as reinfections become possible, individual attitudes and behaviors will increasingly impact the disease's spread.

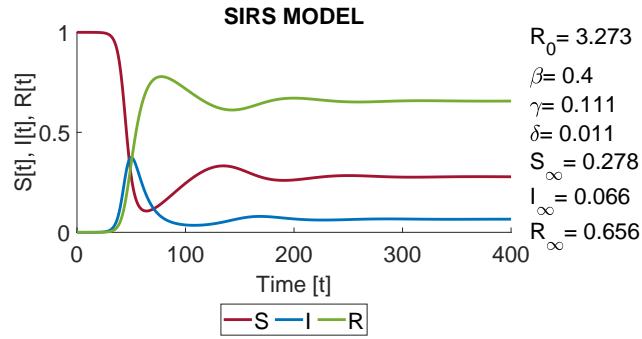


Figure 6.1: Simulation of the SIRS model. The parameters of the model, which meaning is presented in section 3.3.2, are chosen to resemble those of the initial stages of the COVID-19 pandemic [86] and are the same as those used for simulations with the full epi-behavioral model.

6.2 Behavioural model

The development of the behavioral model builds on several works already presented in the literature. In particular, the following mechanisms are considered the most relevant:

- The competition between two opposing behaviors/opinions, driven by peer pressure [19].
- Non-compliance viewed as a form of social contagion [75].
- The unaware-aware-unaware opinion model class [62, 76], for how the compartments are linked together and for the idea of social pressure between individuals.
- The fatigue mechanism, where maintaining a certain behavior leads to a spontaneous loss of compliance [19].

To integrate all these aspects into a mean-field model, the first step is to define the compartments used to segment the population. The population is divided into three compartments: Heedless, Compliant, and Against, denoted respectively as H, C, and A. The meaning of each compartment is as follows:

- H*: Individuals who behave without much regard for guidelines and are careless about the risks associated with the infection.
- C*: Individuals who actively seek to avoid infection or spreading the virus by following guidelines and taking precautions.
- A*: Individuals who do not consider the infection a risk to their safety and do not use protection or modify their behavior during the epidemic. They disregard risk-mitigating guidelines and do not align with safer behaviors as the epidemic unfolds.

Initial conditions

As an initial condition, the hypothesis is that, at the start of the dynamics, most of the population is in the Heedless compartment. This assumption is based on the idea that when a new disease emerges, it is poorly understood, and the population has limited information about it. The hypothesis is that people in the Heedless compartment may be clueless about the risks of becoming infected. This lack of knowledge causes them to maintain their usual behavior, making them susceptible to infection. This assumption is also supported by data and literature [87]. As an example of this initial configuration, the case of COVID-19 in Italy is considered. In the early stages of its spread, when the disease was primarily affecting China, it was not viewed as a significant threat by much of the population in Western countries. It was perceived as a distant issue affecting a faraway nation. Therefore, when the epidemic reached Europe and Italy, both the population and government were caught off guard. There was an initial delay in the implementation of countermeasures, as well as in the dissemination of reliable information about the disease's progression to the general public.

Then, there are two opposing behavioral groups, which in the initial phase of the model comprise a small fraction of the population: Compliant and Against.

The Compliant group actively seeks to reduce their chances of becoming infected. They practice self-protection measures like wearing face masks, sanitizing their hands, and voluntarily limiting their presence in public spaces to reduce contact with others.

In contrast, the Against group consists of individuals who, for personal reasons—such as anti-scientific beliefs, low trust in policymakers, or other concerns—do not take action to minimize their chances of infection or the possibility of infecting others. This category encompasses phenomena such as:

- vaccine denialism;
- misinformation spread;
- denial of the existence of the disease;
- distrust of doctors and government policies.

The inclusion of the Against compartment stems from the fact that, especially in the early stages of a new disease outbreak, there is often a lack of reliable knowledge. As documented by [88], this can lead to the spread of false beliefs in the population. It has also been demonstrated [89] that misinformation, especially when associated with fear, can have lasting effects. A notable example is the belief that the measles, mumps, and rubella (MMR) vaccine can cause developmental disorders in children. Despite the fact that the original publication making this claim has been scientifically discredited [52], this idea remains popular and has contributed to a rise in vaccine skepticism [89].

Social contagion dynamic

The evolution of the model is governed by two principal mechanisms:

- Heedless individuals transitioning to either Compliant or Against compartments.
- Compliant and Against individuals returning to the Heedless compartment.

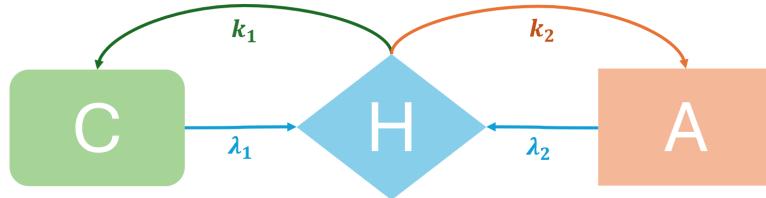


Figure 6.2: The figure represents the behavioral model developed, featuring three compartments: Heedless, Compliant, and Against, abbreviated as H, C, and A. The arrows indicate the inflows and outflows between these compartments.

The first mechanism is driven by peer pressure: the size of each group and the level of "persuasion" are the parameters that govern this process. It is mathematically modeled similarly to person-to-person disease transmission, as seen in the SIR-like mean-field model described in section 3.3.2. Instead, the return to the Heedless compartment is modeled as a "spontaneous decay" process: individuals naturally leave the Compliant and Against compartments and return to Heedless, transitioning spontaneously depending on the level of "fatigue" associated with maintaining the behavior. The flow between an intermediate compartment, represented by H, rather than a direct transition between A and C (and vice versa), is a modeling choice stemming from the idea that heedlessness can initially signify a lack of awareness about a new disease. Over time, it can represent the state of people after a period of coexistence with the disease, where the fatigue of maintaining compliance or resistance increases, or when indifference toward the disease grows. To describe these transitions, different coefficients are introduced. The k_1 and k_2 are persuasion rates, while λ_1 and λ_2 represent fatigue rates. Their meanings are as follows:

- k_1 : persuasion rate from Heedless to Compliant;
- k_2 : persuasion rate from Heedless to Against;

- λ_1 : rate of leaving the Compliant behavior due to fatigue;
- λ_2 : rate of leaving the Against behavior due to fatigue.

Finally, the resulting differential equations describing the model's dynamic are:

$$\begin{cases} \dot{H} = -k_1 HC - k_2 HA + \lambda_1 C + \lambda_2 A \\ \dot{C} = k_1 HC - \lambda_1 C \\ \dot{A} = k_2 HA - \lambda_2 A \end{cases} \quad (6.1)$$

Another assumption made to describe the model is the principle of mass conservation, meaning that the relationship $H + C + A = 1$ holds. Additionally, the initial conditions described in the previous section are translated as follows:

$$\begin{cases} H(0) = 1 - C_0 - A_0 \\ C(0) = C_0 > 0 \\ A(0) = A_0 > 0 \end{cases} \quad (6.2)$$

Behavior conversion number

To simplify the understanding of the system's underlying dynamics, an analogy can be drawn with the reproduction number in epidemic models. By examining the system equations 6.1, a relationship can be identified. From both the second and third equations, we can isolate the two coefficients (specifically k_1, λ_1 , and k_2, λ_2) and derive a new parameter, called "Behavior Conversion Rate", \mathcal{B} . This rate is the result of the ratio between the persuasion rate and the fatigue decay rate, and can be viewed as a measure of the transmission potential of social contagion. The general formula to calculate it is::

$$\mathcal{B}_i = \frac{k_i}{\lambda_i} \quad \text{with } i = 1, \text{ or } 2. \quad (6.3)$$

In the model presented here, \mathcal{B}_1 represents the Behavior Conversion Rate associated with the Compliant compartment, while \mathcal{B}_2 , corresponds to the Against compartment. The results of different numerical simulations will now be displayed to demonstrate how the relationship between these two values influences the evolution of social contagion.

6.2.1 Model simulation

To represent different dynamics, four main cases are now presented. The coefficient values have been set appropriately to highlight different interesting situations in which the system can evolve. These cases represent the majority of possible scenarios:

I case: $\mathcal{B}_1, \mathcal{B}_2 < 1$, $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 > \lambda_2$.

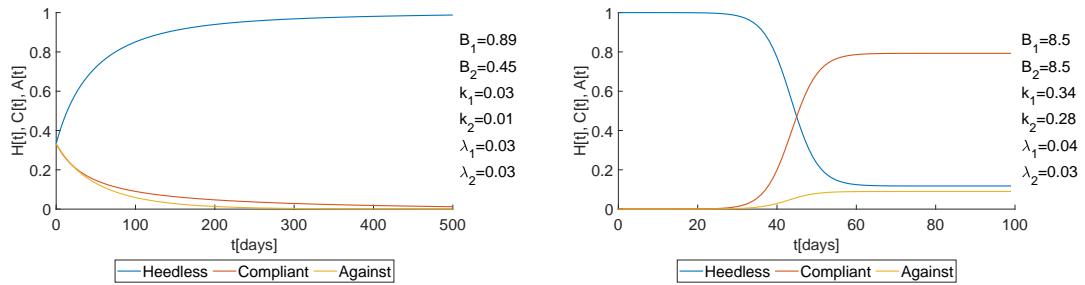
II case: $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 = \mathcal{B}_2$, and $\lambda_1 < \lambda_2$.

III case: $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 = \lambda_2$.

IV case: $\mathcal{B}_1, \mathcal{B}_2 > 1$ and $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 < \lambda_2$.

The values k_1 , and k_2 are calculated from the formula of \mathcal{B}_1 , and \mathcal{B}_2 .

In Figure 6.3, it is evident that when both Behavior conversion numbers are less than one, social contagion does not spread: even though, in this case, the Compliant and Against compartments together represent 60% of the total population at the beginning of the simulation, they clearly tend to zero over time. In contrast, the right panel shows the case where the two \mathcal{B} values are equal and greater than one. To emphasize the importance of the fatigue rate, it is shown that with a lower λ_2 value than λ_1 , the Against compartment becomes dominant by the end of the simulation.



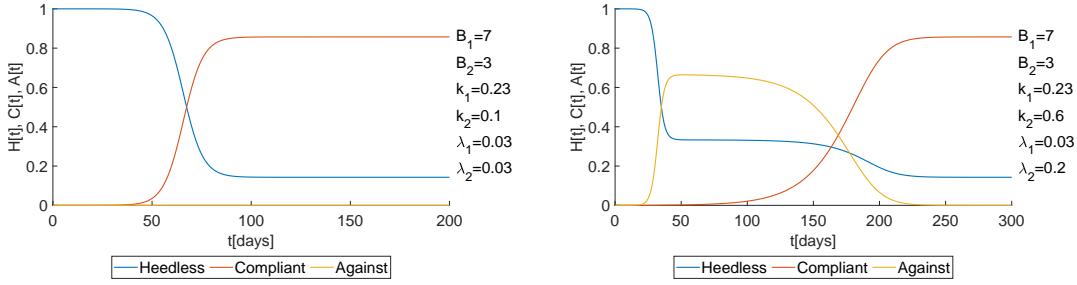
(a) $\mathcal{B}_1, \mathcal{B}_2 < 1$, $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 > \lambda_2$. (b) $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 = \mathcal{B}_2$, and $\lambda_1 < \lambda_2$.

Figure 6.3: Behavioral system dynamics first two cases. In the left panel there is the case in which both B_1 , B_2 are less than one. The system tends to an equilibrium in which all individuals tend to H compartment. In the right panel, instead the case in which the conversion numbers are equal, but because $k_1 > k_2$ the Compliant compartment becomes greater than the Against one.

Figure 6.4 illustrates two other interesting scenarios. On the left, we observe the dynamics when one of the \mathcal{B} values is greater than the other, and both λ values are the same. It is straightforward to understand that this dynamic would also occur if, with the same values, the λ of the dominant behavior were greater than the other, as a larger λ would result in a higher k (persuasion rate). The right panel, however, presents a particularly intriguing situation. Here, a lower λ_1 compared to λ_2 , combined with $k_2 > k_1$, leads to an initial rapid spread of the Against group, even though $\mathcal{B}_2 < \mathcal{B}_1$! It is only after some time that the system evolves to the final equilibrium, which matches the left scenario's result, as the \mathcal{B} values are the same in both simulations.

Equilibrium and stability analysis

To enhance understanding of the system, equilibria are searched and their stability is studied. As observed, the system's final equilibrium values vary according to parameter values. Specifically, the coefficients were combined to produce two Behavior conversion numbers, \mathcal{B}_1 and \mathcal{B}_2 . One way to identify and visualize the system's equilibrium for a specific parameter set is through nullclines. The nullclines are used in an autonomous


 (a) $B_1, B_2 > 1$, $B_1 > B_2$, and $\lambda_1 = \lambda_2$.

 (b) $B_1, B_2 > 1$ and $B_1 > B_2$, and $\lambda_1 < \lambda_2$.

Figure 6.4: Behavioral system dynamics: the second two cases. In the left panel, the scenario depicts Compliant becoming the dominant group, while Against gradually tends toward zero. In the right panel, with the same value for the conversion number but a higher persuasion rate for Against compared to Compliant, the system converges to the same equilibrium as in the left figure. However, it first goes through a phase where the Against group becomes dominant.

system of differential equations (DE) to sketch the phase plane of such a system. In a system of two DEs:

$$\frac{dx}{dt} = f(x, y) \quad (6.4)$$

$$\frac{dy}{dt} = g(x, y) \quad (6.5)$$

There are two types of nullclines: x -nullcline, and y -nullcline. The x -nullcline is a set of points in the phase plane so that $\frac{dx}{dt} = 0$, and graphically can be represented as a set of vectors that go either straight up or down. Instead, the y -nullcline is a set of points in which $\frac{dy}{dt} = 0$. In these points the vectors are horizontal, going either to the left or to the right.

The original system of three equations, 6.1, has been reduced to a system of two equations. This transformation allows the visualization of nullclines in a two-dimensional graph, known as a phase-plane plot. The dimensional reduction is achieved using the mass conservation assumption, which is based on the relationship $1 = H + C + A$. By substituting the A term into the first two equations, the system is rewritten as a two-equation system with two unknowns. The resulting reduced system is as follows:

$$\begin{cases} \dot{H} = -k_1 HC - k_2(1 - H - C)H + \lambda_1 C + \lambda_2(1 - H - C) \\ \dot{C} = k_1 HC - \lambda_1 C \end{cases}$$

To simplify the readability and use a notation more familiar for plotting, the H, C symbols

have been substituted with respectively x, y . So equations become:

$$\begin{cases} \dot{x} = -k_1yx - k_2(1-y-x)x + \lambda_1y + \lambda_2(1-y-x) \\ \dot{y} = k_1yx - \lambda_1y \end{cases} \quad (6.6)$$

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 $\dot{x} = 0$:

$$y = \frac{x(k_2 - k_2x + \lambda_2) - \lambda_2}{x(k_2 - k_1) + \lambda_1 - \lambda_2} \quad (6.7)$$

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

$$x = \frac{\lambda_1}{k_1} = 1/\mathcal{B}_1 \quad \text{or } y = 0.$$

The first nullcline existence condition can be calculated, imposing that the denominator must be not equal to zero. The result is

$$x \neq \frac{\lambda_2 - \lambda_1}{k_2 - k_1}$$

This value of x is in the interval $[0,1]$ only if $\lambda_2 > \lambda_1$ and $k_2 > k_1$, or $\lambda_2 < \lambda_1$ and $k_2 < k_1$. The second nullcline instead, exists always if $k_1 \neq 0$.

Equilibria of the system

From the results find for the y -nullcline, it is possible to calculate explicitly the equilibrium value, as the intersection point of the two nullclines, from the equation 6.7. In fact if $x = \frac{\lambda_1}{k_1}$, the value is $y = 1 - \frac{\lambda_1}{k_1}$. Instead if $y = 0$, two values are found: $x = 1$, and $x = \frac{\lambda_2}{k_2}$. The three equilibria points, indicated with the letters A, B , and C are then:

- $A = (\frac{\lambda_1}{k_1}, 1 - \frac{\lambda_1}{k_1})$
- $B = (1, 0)$
- $C = (\frac{\lambda_2}{k_2}, 0)$

Equilibria stability analysis

To verify the local stability of the equilibrium points, the Routh-Hurwitz criterion is applied, requiring the Jacobian matrix of the system evaluated at each equilibrium. It is deemed locally stable if it meets the following conditions based on the Routh-Hurwitz criterion:

- A negative trace of the Jacobian, $\text{tr}(J) < 0$
- A positive determinant of the Jacobian, $\det(J) > 0$

When these conditions are fulfilled, the equilibrium point satisfies the Routh-Hurwitz criterion, indicating local stability. The Jacobian of the system 6.6 is

$$J = \begin{bmatrix} -k_1y - k_2 + k_2y + 2k_2x - \lambda_2 & -k_1x + k_2x + \lambda_1 - \lambda_2 \\ k_1y & k_1x - \lambda_1 \end{bmatrix} \quad (6.8)$$

The trace of J is

$$\text{tr}J = -k_1y - k_2 + k_2y + 2k_2x - \lambda_2 - \lambda_1 + k_1x \quad (6.9)$$

Its determinant is instead

$$\det J = k_2\lambda_1 + \lambda_1\lambda_2 + 2k_1k_2x^2 - k_1k_2x - k_1\lambda_2x - 2 \cdot k_2\lambda_1x + k_1\lambda_2y - k_2\lambda_1y \quad (6.10)$$

For each defined equilibrium point, an analysis is performed using the Routh-Hurwitz (R-H) criterion to deduce stability conditions expressed as relations between coefficients. This provides a simplified relation under which the system tends toward a specific equilibrium configuration. These relations are expressed in general as relationship between the coefficients k_1 , k_2 , and λ_1 , λ_2 .

Stability of point A: Considering the point $(\frac{\lambda_1}{k_1}, 1 - \frac{\lambda_1}{k_1})$, the trace evaluated with this value is $\text{tr}J(A) = -k_1 - k_2 + k_2\frac{\lambda_1}{k_1} - \lambda_2 + \lambda_1$. The criterium requires this value to be less than zero, and manipulating the terms it becomes: $-k_1 + \frac{\lambda_1}{k_1}(k_1 + k_2) - \lambda_2 < 0$. From this it is obtained the relation:

$$\frac{\lambda_1}{k_1} < \frac{k_1 + \lambda_2}{k_1 + k_2}$$

Instead, the determinant is $\det J(A) = k_1\lambda_1 - \lambda_1\lambda_2$. Simplifying the λ_2 the relation $\lambda_1 < k_1$ is found, that can be inverted, meaning $\frac{k_1}{\lambda_1} = B_1 > 1$. Furthermore, both the conditions must hold to verify the local stability and this relation can be used also within the trace. Due to $\frac{\lambda_1}{k_1} < 1$, it can be assumed the relation on the trace holds certainly if $\frac{k_1 + \lambda_2}{k_1 + k_2} > 1$, and so $k_1 + \lambda_2 > k_1 + k_2$. From this last inequality the relation $\lambda_2 > k_2$ can be derived, from which $B_2 < 1$. In conclusion, point A is certainly locally stable if $B_1 > 1$, and $B_2 < 1$.

Stability of point B: The second equilibrium has coordinates (1,0). Calculating the Jacobian determinant at this point yields the expression $\det J(B) = -k_2\lambda_1 + \lambda_1\lambda_2 + k_1k_2 - k_1\lambda_2$. By grouping terms and analyzing the inequality necessary to satisfy the Routh-Hurwitz criterion, this simplifies to $k_1(k_2 - \lambda_2) - \lambda_1(k_2 - \lambda_2) > 0$, which further reduces to $(k_1 - \lambda_1)(k_2 - \lambda_2) > 0$. This inequality is met if both terms in the product are either positive or negative:

- Case I : $k_1 > \lambda_1$, and $k_2 > \lambda_2$
- Case II : $k_1 < \lambda_1$, and $k_2 < \lambda_2$

Evaluating the trace at this point, the result is $\text{tr}J(B) = k_2 - \lambda_2 - \lambda_1 + k_1 < 0$. Rearranging terms gives the condition $k_1 + k_2 < \lambda_1 + \lambda_2$, which is true only under the Case II identified above. Thus, it can be concluded that equilibrium point B is locally stable only if $\frac{k_1}{\lambda_1} < 1$, and $\frac{k_2}{\lambda_2} < 1$, which correspond to $B_1 < 1$, and $B_2 < 1$.

Stability of point C: The coordinates of this point are $(0, \frac{\lambda_2}{k_2})$. The determinant at this value is given by $\det J(C) = k_2\lambda_1 - \lambda_1\lambda_2 + k_1\frac{\lambda_2^2}{k_2} - k_1\lambda_2 > 0$. Also here, rearranging terms simplifies the inequality to: $k_2[\lambda_1 - \lambda_1\frac{\lambda_2}{k_2} + k_1\frac{\lambda_2^2}{k_2} - k_1\frac{\lambda_2}{k_2}] > 0$, $k_2[\lambda_1(1 - \frac{\lambda_2}{k_2}) - k_1\frac{\lambda_2}{k_2}(1 - \frac{\lambda_2}{k_2})] > 0$, which further reduces to:

$$k_2[(\lambda_1 - k_1\frac{\lambda_2}{k_2})(1 - \frac{\lambda_2}{k_2})] > 0$$

To satisfy the inequality both terms must have the same sign, as in the previous point *B*:

- Case *I* : $\frac{k_2}{\lambda_2} > \frac{k_1}{\lambda_1}$, and $k_2 > \lambda_2$
- Case *II* : $\frac{k_2}{\lambda_2} < \frac{k_1}{\lambda_1}$, and $k_2 < \lambda_2$

The trace value instead corresponds to $\text{tr}J(C) = -k_2 + k_2\frac{\lambda_2}{k_2} + k_1\frac{\lambda_2}{k_2} - \lambda_1 < 0$, leading to inequality $\frac{\lambda_2}{k_2}(k_1 + k_2) < k_2 + \lambda_1$. It is found the relation:

$$\frac{\lambda_2}{k_2} < \frac{k_2 + \lambda_1}{k_1 + k_2}.$$

By choosing Case I, we ensure the trace inequality is satisfied. If $k_2 > \lambda_2$, the ratio $\frac{\lambda_2}{k_2}$ is less than one. So, if the right part of inequality is larger than one, the trace condition holds. The expression rewritten in this way is $\frac{k_2 + \lambda_1}{k_1 + k_2} > 1$, $k_2 + \lambda_1 > k_1 + k_2$, and finally $\lambda_1 > k_1$. But, if the initial assumption was that $\frac{k_2}{\lambda_2} > 1$, this results can be extended and concluding, point C is certainly stable if $B_2 > B_1$, $B_2 > 1$.

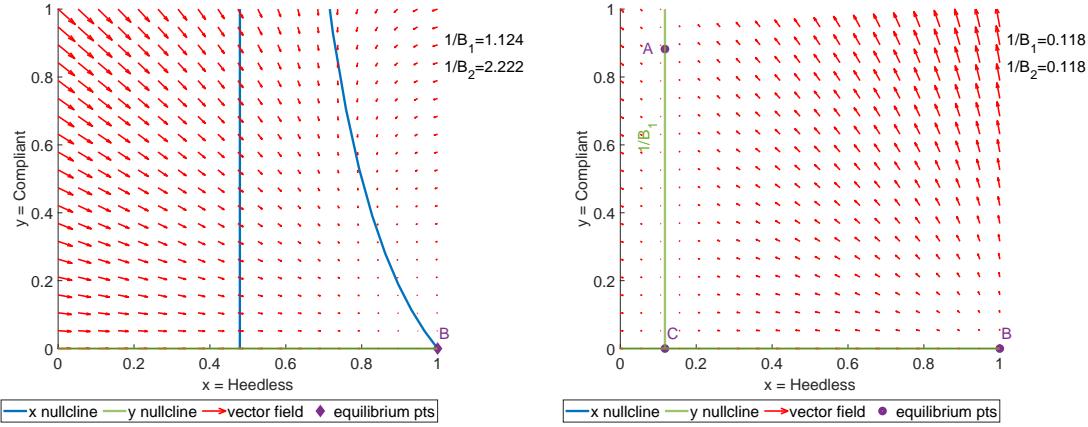
Equilibrium simulations

Based on the equilibrium analysis, we now understand how the model behaves with various parameter values. To confirm these results, nullcline plots for the four previously simulated cases are generated, and the different scenarios are examined. First, it is important to describe what can be visualized in the nullcline plot. The blue curve is the expression found solving the x-nullcline, and the vertical line in the plot correspond to the point of discontinuity of this expression, as discussed before. The green line is instead the y-nullcline, composed of a vertical and horizontal line, representing the two possible solutions. In purple are visualized the three equilibria points. If stable, they are marked with a diamond, will if unstable with a circle.

I case: $B_1, B_2 < 1$, $B_1 > B_2$, and $\lambda_1 > \lambda_2$.

If both the Conversion numbers are less than one, there is only one equilibrium in the phase plane, where both Compliant and Against tend toward zero.

In the left figure 6.5, the nullcline plot shows an intersection between the two nullclines only at the point $(1,0)$. Under this condition, the only equilibrium is at $H = 1$, with both A and C equal to zero. Using the notation from the system equation, this corresponds to $x = 1$ and $y = 0$. Calculating the trace and determinant of the Jacobian at this point yields


 (a) $\mathcal{B}_1, \mathcal{B}_2 < 1$, $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 > \lambda_2$.

 (b) $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 = \mathcal{B}_2$, and $\lambda_1 < \lambda_2$

Figure 6.5: Nullclines plots of the first two situations analyzed.

$\text{tr}J(1,0) = -\frac{209}{12000}$ and $\det J = \frac{121}{2400000}$. Thus, the equilibrium is locally asymptotically stable, as it satisfies the Routh-Hurwitz condition.

II case: $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 = \mathcal{B}_2$, and $\lambda_1 < \lambda_2$.

This second situation is the most complex to analyze. Due to the equal value of the two influence processes, the final equilibrium of the compartments cannot be determined solely by the previously established relations but also depends on the initial conditions.

The Headless compartment can still be determined using the equations from previous cases, and the same value is obtained for both $x = \lambda_1/k_1$ and $x = \lambda_2/k_2$. Thus, the final equilibrium values of x is $\bar{x} = 0.12$. As it can be seen from the system evolution 6.3, and nullcline plots 6.5, at the equilibrium the Against and Compliant groups are formed by a subdivision of the $1 - \bar{x}$ part. This division depends on the initial conditions of the model. Applying the Routh-Hurwitz criterion does not yield information on this equilibrium because the Jacobian determinant equals zero. An explanation for this is that, given the equilibrium alignment of points A and C and the equality of their conversion numbers,

the influxes and outfluxes between the Compliant and Against compartments balance. Consequently, the system's evolution is influenced by the initial numbers of Compliant and Against individuals, as once the system reaches the equilibrium value of Headless, it remains in this configuration. Observing the 3D plot 6.6 of the complete system, with surfaces for H and C , one can visualize a slope where an imaginary inertia-free ball would roll until it settles at the $1/\mathcal{B}_1 = 1/\mathcal{B}_2$ value.

III-a case: $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 = \lambda_2$.

In this scenario, as shown in Figure 6.7, there is an intersection between the two nullclines, at the point A . The equilibrium has coordinates equal to $x_A = \lambda_1/k_1$ and $y_A = 1 - \lambda_1/k_1$, derived by solving the nullcline expressions as previously described. Both R-H

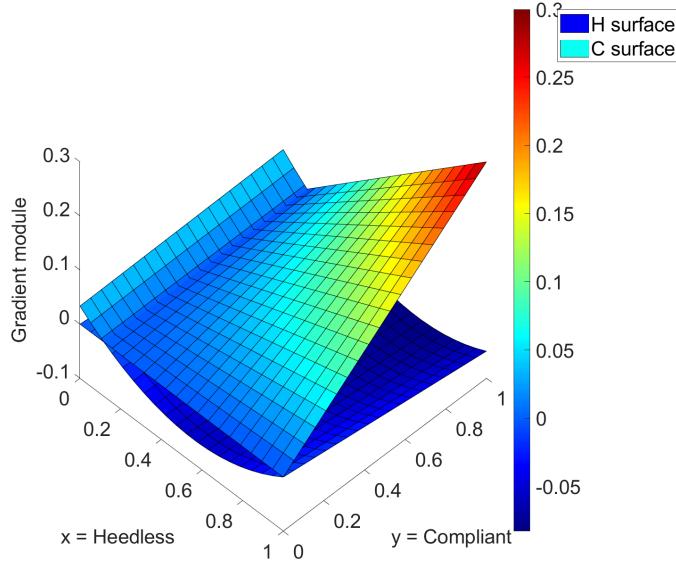


Figure 6.6: The nullcline functions represented as surfaces in a tridimensional space.

conditions hold: $\text{tr}J(\bar{x}, \bar{y}) = -23/105$, and $\det J(\bar{x}, \bar{y}) = 2/525$, so the solution is locally asymptotically stable and does not depend on the initial conditions.

III-b case: $\mathcal{B}_1 < 1, \mathcal{B}_2 > 1, \mathcal{B}_1 < \mathcal{B}_2$. The system's evolution shows opposite behavior compared to the previous case, with the Compliant compartment tending to zero at equilibrium. The right panel of Figure 6.7, shows two intersections in the phase plane at points B , and C . At both points, $y = 0$, but only point C is locally stable, as it satisfies the R-H conditions. The equilibrium point is calculated as $x_C = \lambda_2/k_2$ and $y_C = 0$.

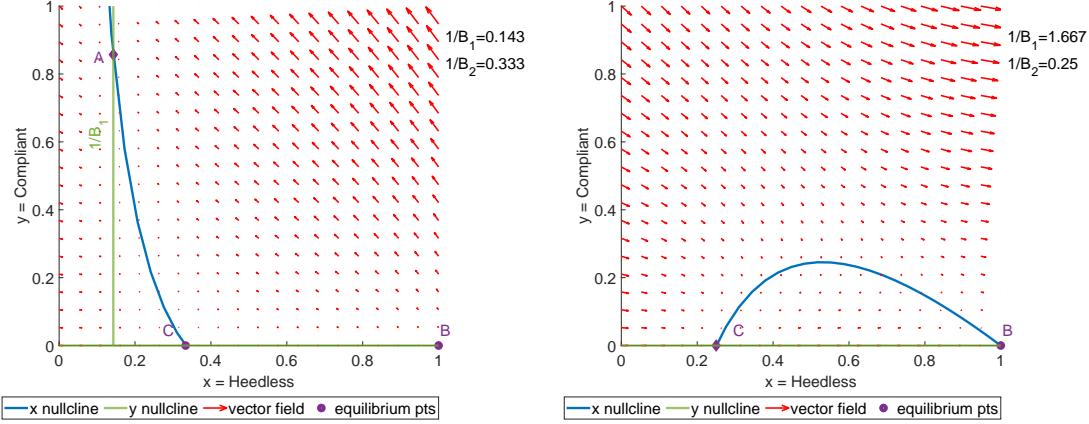
IV case: $\mathcal{B}_1, \mathcal{B}_2 > 1$ and $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 < \lambda_2$.

In this situation, the equilibrium has the same value, of the III-a case, and also the stability condition are verified. However, the nullcline plot is very different, referring to Figure 6.8. The phase plane is much more complex: there are two lines and the discontinuity point for the x-nullcline (the blue line), and the trajectory of convergence to the point A , the only locally stable is more complex.

In fact, looking at the model simulation in Figure 6.4, the system initially evolves to what seems as a first equilibrium, corresponding to $x_A = \lambda_2/k_2$, $y_A = 0$, but then, and it is confirmed by R-H, this equilibrium is unstable, and so the model continues its evolution after reaching the real locally stable configuration.

Behavioural model experiment

To better understand all possible scenarios emerging from the behavioral model, a set of simulations is conducted. Four vectors are defined, one for each model parameter, and a separate simulation is performed for each parameter combination. During each


 (a) $B_1, B_2 > 1$, $B_1 > B_2$, and $\lambda_1 = \lambda_2$.

 (b) $B_1 < 1$, $B_2 > 1$ and $B_2 > B_1$.

Figure 6.7: Nullclines plots of the second two situations analyzed.

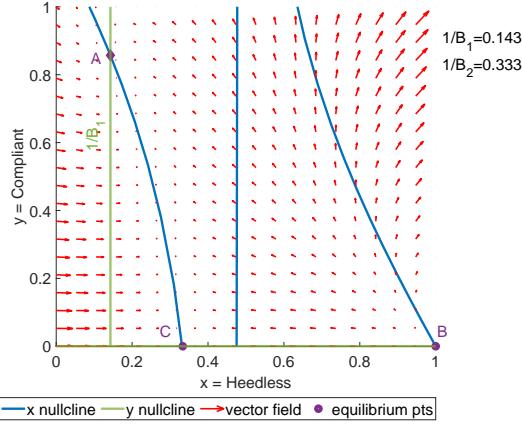


Figure 6.8: IV case of nullcline simulation. The A point is the locally stable one.

simulation, the parameter values remain constant. The variation range for each parameter is as follows:

- k_1 between 0.1 and 0.99
- k_2 between 0.1 and 0.99
- λ_1 between $1/2$ and $1/40 d^{-1}$

- k_1 between $1/2$ and $1/40\ d^{-1}$

The resulting $\mathcal{B}_1, \mathcal{B}_2$ have a range spanning between 0.5 , and 29.7 . These ranges are chosen based on the assumption that the "fatigue" rate realistically spans between two and forty days, a range supported by prior research, such as the study in [90]. For the behavior persuasion rate (k_1, k_2), both low and high values for transition rates are included. We observe the dynamics across all states, recording key metrics for each simulation, such as the final compartment values, peak values, and the time of peak occurrence. Additionally, for generating the sensitivity plots, the Conversion number derived from the coefficient combinations in equation (6.3) are applied.

Heat map Final Equilibrium states

The first plots 6.9 are heat maps about the final value reached by various compartments, varying \mathcal{B}_1 and \mathcal{B}_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 Heedless. Considering the left panel in figure 6.9, if the heat map of Compliant is pass through vertically, clearly until $\mathcal{B}_1 > \mathcal{B}_2$, at the equilibrium there are still compliant. Then, when $\mathcal{B}_1 > \mathcal{B}_2$, the compliant abruptly tend to zero.

Another figure in which this threshold effect can be observed is 6.10.

The plots show how, for a fixed values of λ_1 and k_2 , the size at equilibrium of the system changes, varying the k_1 coefficient. To highlight the threshold effect due to the comparison of reproduction rates, on the x-axis is plotted the \mathcal{B}_1 coefficient, that can be calculated knowing the value of λ_1 and k_1 . For the same reason, different \mathcal{B}_2 situations are represented.

The threshold effect is clearly visible here as well. When examining the final values for the Compliant and Against compartments, it is evident that once the \mathcal{B}_1 reproductive coefficient becomes dominant, the increase in the final size observed in the Compliant compartment results from a decrease in the Careless compartment.

Heat map of max compartments value

Additionally, the peak values reached by the Compliant and Against compartments are observed. Figures 6.11, and 6.12 illustrate the maximum value reached by the Against compartment. Two situations are compared: in the first $k_1 \sim k_2$, while in the other the difference between the two parameter is higher. In the first situation, there are visible three possible situations:

- B_2 greater than B_1 , and the max value correspond to the value at Equilibrium. It is the bottom right part of the picture.
- $B_2 < B_1$, but $\lambda_2 > \lambda_1$. These cases are located on the diagonal threshold of the heat map, and are the situations in which there is first a peak of A, but then the C is the dominant group at equilibrium

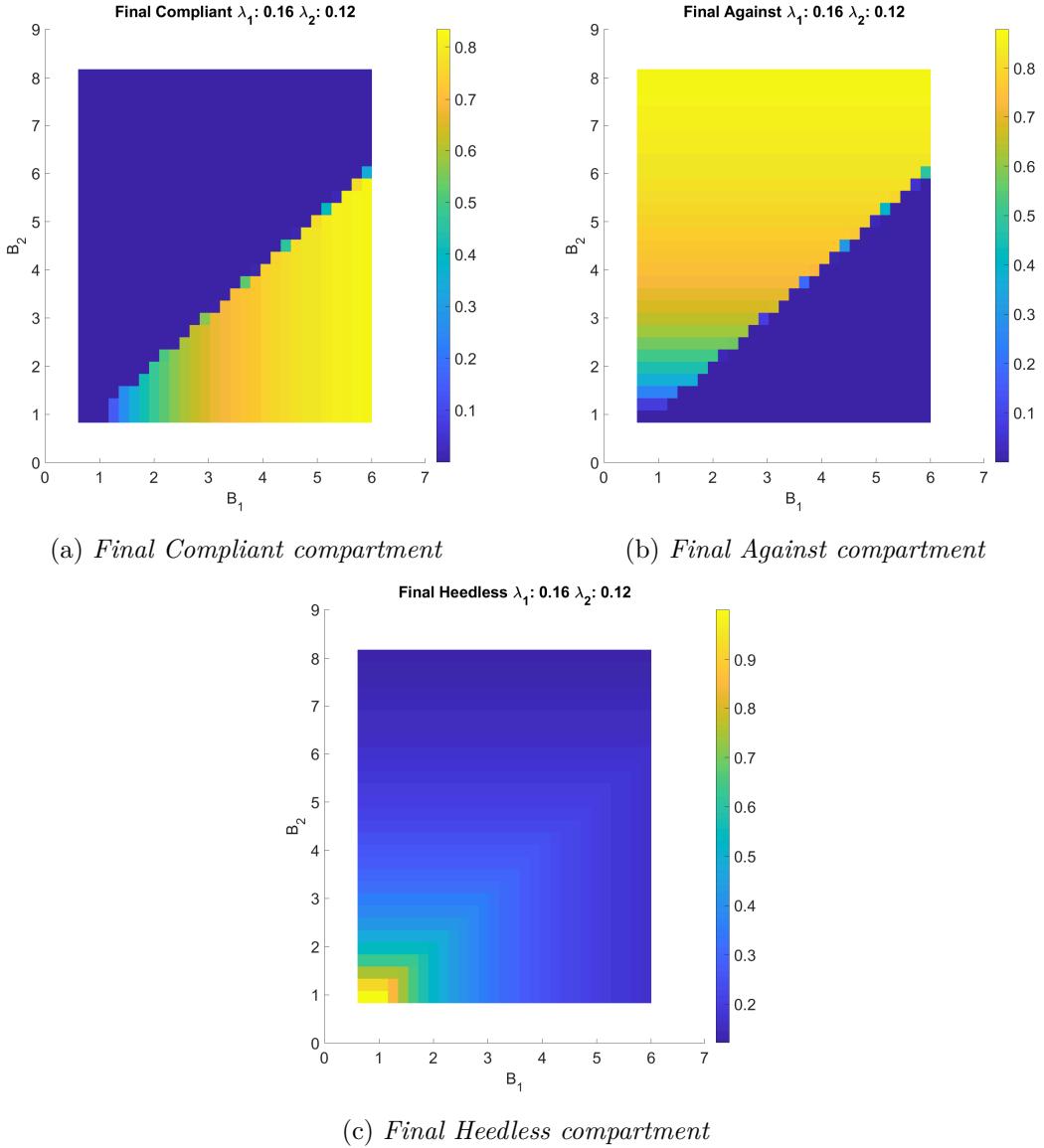


Figure 6.9: The final value reached at equilibrium by every compartment in the behavioural model.

- $B_2 < B_1$ and also $\lambda_2 < \lambda_1$. Here there is no peak, and A tends always to zero, or to remain approximately zero, depending on the initial conditions.

These three phenomena are visible also in Figure 6.12, in the left panel. Both the right panels in the two figures need as a comparison, to highlight the situation in which, having a larger persuasion rate, the A is always dominant on the other compartment. As a consequence the peak correspond always to the value at equilibrium, and in no case, the C compartment at equilibrium is different to zero. The first case confirms the dynamics

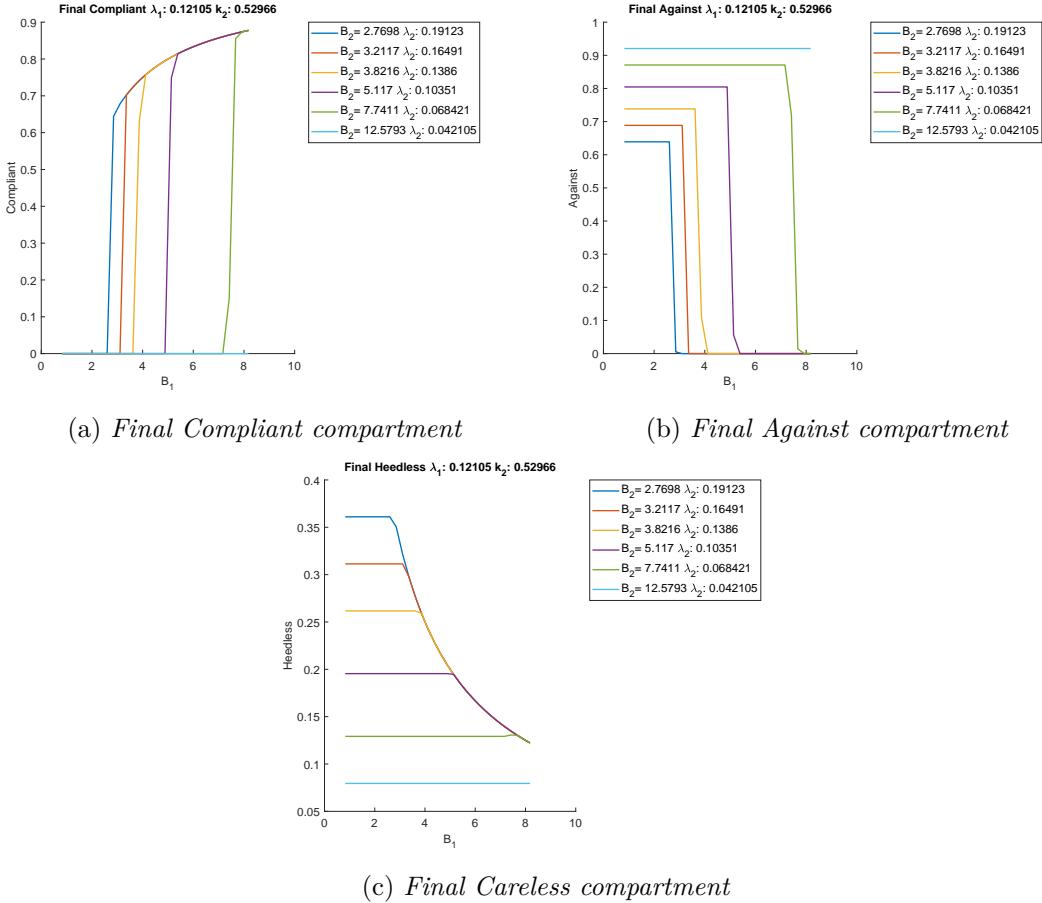


Figure 6.10: The final value reached at equilibrium by every compartment in the behavioural model varying the R_1 coefficient w.r.t different B_2 values.

presented earlier in the model simulation in section 6.2.1. This relationship between the departure rate associated with a certain behavior and the persuasion rate can lead to an initial phase where a behavior not dominant at equilibrium gains traction, reaches a peak, and then gradually declines. In some simulations, this diminishing phase can extend over a significant portion of the simulation time, as illustrated in Figure 6.4. The red line, called "section line" in Figure 6.11, represents the part of the heat map, with the same parameters values used in the simulation of the below figure 6.12.

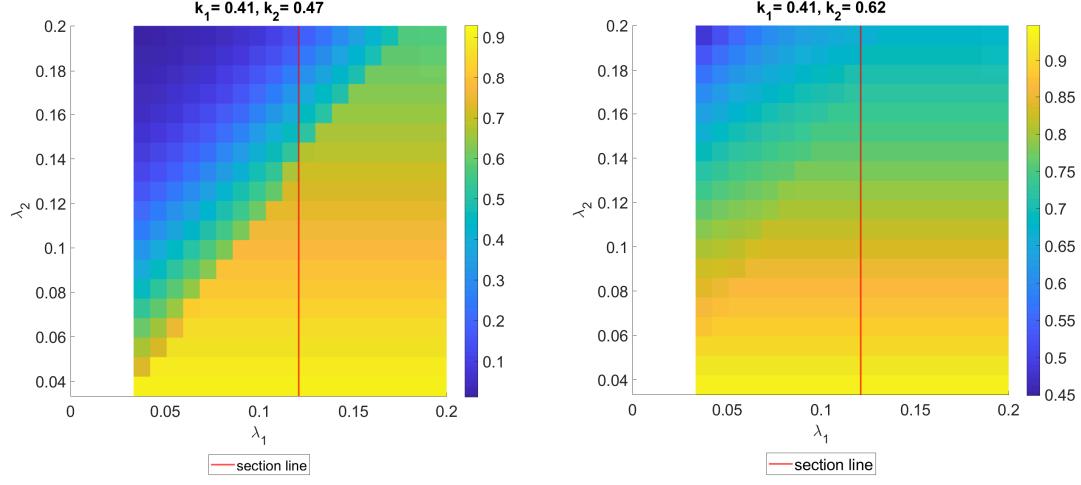


Figure 6.11: a) The max value of Against varying the λ_1, λ_2 . b) The evolution of several Against compartment dynamic, fixing k_1, λ_1 , and k_2 and varying only the λ_2 .

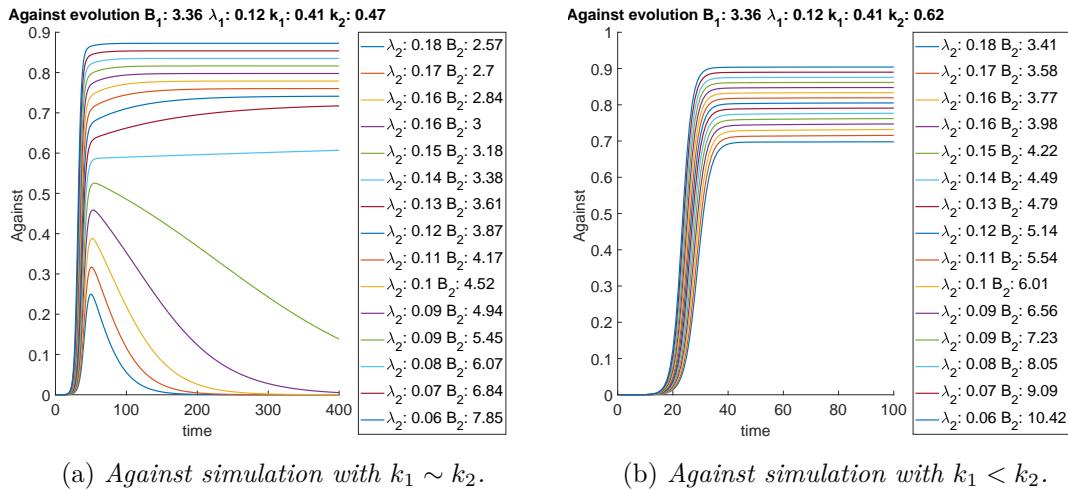


Figure 6.12: a) The max value of Against varying the λ_1, λ_2 . b) The evolution of several Against compartment dynamic, fixing k_1, λ_1 , and k_2 and varying only the λ_2 .

Chapter 7

Behavioral-epidemic model

7.1 Model description

The newly developed model in this thesis, the Susceptible-Against-Heedless-Compliant-Infected-Recovered (SAHCIR) model, combines both epidemic and behavioral components, establishing an innovative approach to disease modeling. This model aims to bridge the epidemiological and social dynamics of an outbreak, integrating empirical observations to compare model outcomes with real-world data, as discussed in the article [84]. While some existing models combine these aspects, such as the one presented in [48], they often rely on predefined assumptions about specific behaviors rather than direct empirical validation.

The SAHCIR model incorporates a variety of behavioral stances towards safety measures, reflecting both proactive (pro-precaution) and non-compliant (anti-precaution) attitudes. The model also recognizes that, particularly during an epidemic's initial phase, a significant portion of the population may not follow safety measures—not out of skepticism, but due to a lack of awareness about the severity of the outbreak. Additionally, the model allows for government intervention through parameters that influence the spontaneous transition rate from the "Against" to the "Compliant" group, mirroring real-world public health policies aimed at promoting preventive behaviors.

This model thus provides a more empirically grounded framework for understanding the interplay between public health dynamics and social behaviors, filling a gap in the literature by providing a model designed for direct confrontation with real-world data rather than solely hypothetical scenarios.

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, as seen in Chapter 6.2: Heedless, Compliant, and Against.

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

C: Compliant, people that want to avoid becoming infected or infecting others

- A: Against, people who do not see the epidemic as a risk and do not use protections or change their behavior during the epidemic.

In the model, behavioral dynamics are combined with a SIRS epidemic model to create seven distinct, mutually exclusive compartments that capture both disease states and behavioral responses. The Heedless behavior can only be adopted by individuals who are susceptible to infection. This reflects the assumption that when a new disease emerges, individuals lack sufficient information and, consequently, they behave "normally," without adopting safety measures.

As people transition through stages of infection and recovery, the model assumes they can gain awareness of the disease's risks and recognize the importance of infection prevention. Thus, after infection or recovery, it is considered unrealistic for them to remain Heedless. Non-heedless individuals are divided into two categories: Compliant, those who adopt behaviors to minimize further spread, and Against, who are aware but do not actively prevent transmission, possibly due to personal beliefs or low risk perception.

Overall, we can recognize the following compartments:

S_H : 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 therefore the people in this compartment have no fear of becoming infected and do not modify their behaviors.

S_C : Susceptible Compliant, the group composed of those who actively avoid becoming infected and use non-pharmaceutical interventions to limit the possibility of getting sick and of spreading the contagion.

S_A : Susceptible Against, the people that do not comply with the recommendations provided 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.

I_C : Infected Compliant, people infected by the virus. This group receives infections coming from both S_C and S_H compartments, because it is considered that even those who have a "neutral" opinion about the risk associated with the infection change their minds when they become infected. The main behavior associated with this group is that safety measures such as quarantine are respected, which limits disease spread.

I_A : Infected Against, compartment composed of the Against Susceptibles who became sick. They do not respect self-isolation, and spread the disease.

R_C : Recovered Compliant, compliant people that are healed from the infection and contribute to raising awareness about the risk associated with the disease.

R_A : 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 do not believe of needing protection.

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

$$\begin{cases} \dot{S}_H = -\psi k_1 S_H \cdot C - k_2 S_H \cdot A + \lambda_1 S_C + \lambda_2 S_A + \delta(1 - \phi) R_C - \beta S_H \cdot I \\ \dot{S}_C = \psi k_1 S_H \cdot C + \delta \phi R_C - \lambda_1 S_C - \beta \rho S_C \cdot I \\ \dot{S}_A = k_2 S_H \cdot A - \lambda_2 S_A - \beta S_A \cdot I + \delta R_A \\ \dot{I}_C = \beta \rho S_C \cdot I + \beta S_H \cdot I + \psi k_3 I_A \cdot C - \lambda_3 I_C - k_4 I_C \cdot A + \lambda_4 I_A - \gamma I_C \\ \dot{I}_A = \beta S_A \cdot I - \psi k_3 I_A \cdot C + \lambda_3 I_C + k_4 I_C \cdot A - \lambda_4 I_A - \gamma I_A \\ \dot{R}_C = \gamma I_C - k_6 R_C \cdot A + \lambda_6 R_A + \psi k_5 R_A \cdot C - \lambda_5 R_C - \delta R_C \\ \dot{R}_A = \gamma I_A + k_6 R_C \cdot A - \lambda_6 R_A - \psi k_5 R_A \cdot C + \lambda_5 R_C - \delta R_A \end{cases} \quad (7.1)$$

where

- $A = S_A + I_A + R_A$ is the total fraction of Against individuals.
- $C = S_C + I_C + R_C$ is the total fraction of Compliant individuals.
- $I = \epsilon \cdot I_C + I_A$ is the fraction of infected people contributing to spreading the infection.
- ψ is a parameter that represents an increased (if its value is larger than 1) incentive to transition to the Compliant group. It can be regarded as an intervention from an external mean-field global agent.
- ϕ is a normalized parameter, used to split the population while re-entering in the susceptible class in the Headless 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 wanes (so that recovered people become susceptible again).
- ϵ specifies the fraction of compliant infected that participate in the infection process.

7.2 Basic reproduction number calculation

The first analysis that can be performed on the Behavioral Disease model is to compute its basic reproduction number. It is defined as the spectral radius of the next-generation matrix. Using the method outlined in [91] and now briefly described, this quantity is calculated. To distinguish between this value, that is related to the whole model, and is thus influenced by both the social and epidemic layer, and the classic epidemic only Reproduction number, described in Section 3.3.2, we denote this quantity with the symbol E_0 . It is the "Epidemic reproduction number". Consider $x = (x_1, x_2, \dots, x_n)^T$ a vector describing

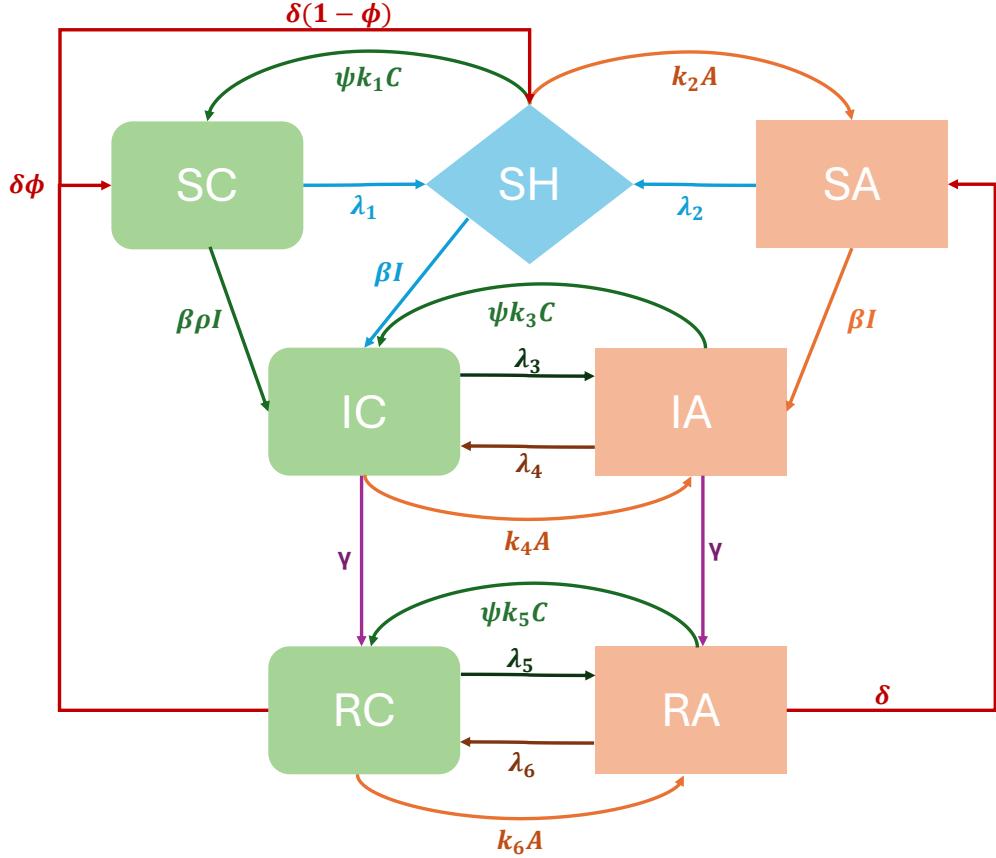


Figure 7.1: Epidemic behavioral model with compartments and fluxes.

the number of individuals in each compartment. The compartments of infected are m , and the relation $m < n$ holds. Assume that the system has a disease free equilibrium (DFE), denoted as x_0 : x_0 exist and is stable in the absence of the disease, so when the infected compartments have value equal to zero. Furthermore in the DFE the linearized equations for x_1, x_2, \dots, x_m decouple from the other equations. Thanks to these assumptions it is possible to write the m infected equations in the form:

$$\frac{dx_i}{dt} = \mathcal{F}_i(x) - \mathcal{V}_i(x), \text{ for } i = 1, 2, \dots, m \quad (7.2)$$

The two terms appearing in equation (7.2), are $\mathcal{F}_i(x)$, the rate of appearance of new infected in the compartments i , and $\mathcal{V}_i(x)$ the rate of other transitions between compartment i and other infected compartments. It is assumed that each function is continuously differentiable at least twice in each variable.

To describe the evolution of the system, the following matrix notations is adopted:

- $F = [\frac{d\mathcal{F}_i}{dx_j}(x_0)]$ with $1 \leq i, j \leq m$.

- $V = [\frac{dy_i}{dx_j}(x_0)]$ with $1 \leq i, j \leq m$.

The matrices are both evaluated at the disease free equilibrium, where $x = x_0$, and have dimension $m \times m$, with m being the number of infected compartments in the model. Using the defined matrices, according to [91, 92], the Epidemic reproduction number E_0 for the model (7.1) at a disease free equilibrium, where all infected densities are zero, is given by:

$$E_0 = \text{spec}(FV^{-1}) \quad (7.3)$$

The disease-free equilibrium is defined as a locally stable equilibrium of the system in which there is no disease. The matrix FV^{-1} is called next generation matrix, while $\text{spec}(M)$ is the spectral radius of a matrix M. To understand the entries of the matrix, consider perturbing a population at the DFE by introducing one infected individual in compartment k .

The (j, k) entry of V^{-1} is the average time that the individual spends in the compartment j . Instead, the (i, j) entry of F is the rate at which infected in compartment j produce new infections in i . In F only individuals that become infected for the first time must be inserted, while other influxes in the i th compartment, must be considered in the V matrix. Finally the entry (i, k) in FV^{-1} is the expected number of new infections in i produced by the infected individual originally introduced in k .

Considering the system dynamics presented in (7.1), matrices F and V are

$$F = \begin{bmatrix} \beta\epsilon(\rho S_C + S_H) & \beta(\rho S_C + S_H) \\ \beta\epsilon S_A & \beta S_A \end{bmatrix} \quad (7.4)$$

$$V = \begin{bmatrix} \lambda_3 + k_4(S_A + I_A + R_A) + \gamma - \psi k_3 I_A & k_4 I_C - \lambda_4 - \psi k_3 (S_C + I_C + R_C) \\ \psi k_3 I_A - \lambda_3 - k_4(S_A + I_A + R_A) & \psi k_3 (S_C + I_C + R_C) - k_4 I_C + \lambda_4 + \gamma \end{bmatrix} \quad (7.5)$$

Once the matrices composing the next-generation matrix are defined, the value of the epidemic reproduction number is calculated, resulting in:

$$\begin{aligned} E_0 &= \frac{\beta}{\gamma} \cdot \frac{\text{num}}{\text{den}} \\ \text{num} &= S_A(\gamma + \lambda_3 + \epsilon\lambda_4) + (S_H + \rho S_C)(\lambda_3 + \epsilon\gamma + \epsilon\lambda_4) + \dots \\ &\quad (S_A + S_H + \rho S_C)[(I_A - \epsilon I_C)(k_4 - \psi k_3) + k_4(R_A + S_A) + \psi\epsilon k_3(R_C + S_C)] \\ \text{den} &= \lambda_3 + \lambda_4 + \gamma + k_4(A - I_C) + \psi k_3(C - I_A) \end{aligned} \quad (7.6)$$

To compute the value of E_0 , the above expression must be evaluated at the DFE equilibria. Now it is determined how to compute them.

7.2.1 DFE calculation

To calculate the value of the DFE points of the system, consider the equations in system (7.1), referring to them as $\dot{x}_i = f_i(x)$, with $i = 1, \dots, 7$, corresponding to the seven

different compartments that form the model. With j instead defined the compartments of infected in the model, and the condition that $j < 7$ holds. Disease-free equilibria are solutions of the system in which no infection is present, so $x_j = 0$, and the differential expressions are equal to zero. Imposing these conditions, along with mass conservation, leads to the following system to solve:

$$\begin{cases} \dot{x}_i = 0 & i = 1, \dots, 7 \\ x_j = 0 & j \geq 1, j < 7 \\ \sum_{i=1}^7 x_i = 1 & i = 1, \dots, 7 \end{cases}$$

Solving the system of equations symbolically using Matlab, three disease free equilibria are found:

	S_C	S_H	S_A	E_0
type-A	0	1	0	$\frac{\beta \lambda_3 + \epsilon \gamma + \epsilon \lambda_4}{\gamma \lambda_3 + \lambda_4 + \gamma}$
type-B	0	$\frac{\lambda_2}{k_2}$	$1 - \frac{\lambda_2}{k_2}$	$\frac{\beta \gamma + \lambda_3 + \epsilon \lambda_4 + S_H \gamma (\epsilon - 1) + k_4 S_A}{\gamma \gamma + \lambda_3 + \lambda_4 + k_4 S_A}$
type-C	$1 - \frac{\lambda_1}{k_1}$	$\frac{\lambda_1}{k_1}$	0	$\frac{\beta (S_H + \rho S_C) (\lambda_3 + \epsilon \gamma + \epsilon \lambda_4 + \psi \epsilon k_3 S_C)}{\gamma \lambda_3 + \lambda_4 + \gamma + \psi k_3 S_C}$

For all equilibria, the corresponding values of R_C and R_A are equal to zero.

7.3 Model simulations

We now present an analysis of the model through a set of numerical simulations to observe how the system reacts and evolve with different sets of parameters is performed. Performing an analysis similar to the one conducted for the behavior model alone is in fact too complex, as it involves numerous equations and parameters. Even though models similar to the one presented here have been analyzed in other works, such as [48], those analyses often rely on strong assumptions. E.g. in [48] the hypothesis is the existence of different time scales between the epidemic and behavioral layers, enabling the simplification of the analysis. However, this assumption was deemed unreliable during the development of this model. Models intended to use empirical data, such as those related to epidemic evolution, typically rely on data collected on a daily basis. For this reason, it is assumed that the time scales of the epidemic and behavioral layers comparable. Although adopting the time-separation hypothesis might simplify the analysis, it would essentially yield the same results found in the behavior model alone. Thus, extensive simulations become crucial to enhance understanding of the model.

A first evaluation of E_0 is performed to assess its capability to provide indications about the stability of the equilibrium. As stated in [91, 92], the DFE is locally asymptotically stable if $E_0 < 1$. Conversely, if $E_0 > 1$, a fully susceptible population, when perturbed by the introduction of infectious individuals, will progress toward an epidemic.

The same four cases of parameter configurations used in the previous chapter for the behavioral layer are employed here. For the epidemic layer, the value of R_0 is fixed at 3.2727, based on the assumption of an average recovery period lasting 9 days and a transmission rate similar to that estimated for diseases like COVID-19 at the early stages of its spread [86]. An additional V case is introduced, in which the magnitudes of \mathcal{B}_1 and \mathcal{B}_2 are inverted relative to those in case IV.

To represent a wide range of scenarios, the epidemiological framework is kept constant, maintaining the same values for β , γ , and δ across all five cases. What varies is the intensity and relationship between the conversion numbers \mathcal{B}_1 and \mathcal{B}_2 .

All computations are performed using the following fixed set of parameters:

$$\begin{array}{llll} \beta = 0.40 & \gamma = 0.35 & \delta = 1/9 & \epsilon = 0.15 \\ \epsilon = 0.15 & \rho = 0.65 & \psi = 1 & \phi = 0.5 \end{array}$$

With these coefficient values, considering separately the epidemic and behavioral layers the basic epidemiological-only reproduction number of the SIRS model is:

$$R_0 = \frac{\beta}{\gamma + \delta} = 3.2727$$

For the other behavioral coefficients it is assumed $k_3 = k_5 = k_1$, and $k_4 = k_6 = k_2$. Also $\lambda_3 = \lambda_5 = \lambda_1$, and $\lambda_4 = \lambda_6 = \lambda_2$. The values of these coefficients change across the five cases.

	B_1	$\lambda_1 [d^{-1}]$	B_2	$\lambda_2 [d^{-1}]$
case I	0.89	1/30	0.45	1/40
case II	8.5	1/40	8.5	1/20
case III	7	1/30	3	1/30
case IV	7	1/30	3	1/5
case V	3	1/30	7	1/2

The values of the variables k_1 and k_2 are derived using the expression $\mathcal{B}_i = k_i / \lambda_i$, where $i = 1, 2$. The results of all simulations and the corresponding E_0 values are summarized in Table 7.2.

It is immediately noticeable how the value of E_0 changes and is influenced by both the behavioral parameters and the considered DFE. The initial distribution of the population across compartments is chose by perturbing the computed DFE, through the insertion of some infected. The DFE type-*C*, where the population is divided between the Compliant and Heedless Susceptible compartments, is always locally asymptotically stable. In contrast, the type-*B* equilibrium is always unstable, with $E_0 > 1$. A possible explanation for this observation lies in the effects of the parameters ρ and ϵ , which respectively reduce the likelihood of S_C becoming infected and the ability of I_C to infect others. In the next paragraphs, the plots of the most relevant simulations for each of the five cases defined by these parameters are shown. In the simulations conducted using the Runge-Kutta second-order method, the number of infected individuals at time zero is slightly greater than zero: $I_{C0} = I_{A0} = 10/60e6$.

Case #	DFE type	SC	SH	SA	E_0
1	A	0	1	0	1.142
2	A	0	1	0	0.951
2	C	0.8824	0.1176	0	0.5148
3	A	0	1	0	1.1138
3	B	0	0.3333	0.6667	2.7191
3	C	0.8571	0.1429	0	0.567
4	A	0	1	0	0.8361
4	B	0	0.3333	0.6667	2.6257
4	C	0.8571	0.1429	0	0.5091
5	A	0	1	0	0.6983
5	B	0.6667	0.3333	0	0.524
5	C	0	0.1429	0.8571	3.1669

Figure 7.2: The results of the simulations illustrate how E_0 , the Epidemic Reproduction Number, varies across different possible scenarios.

7.3.1 I case: $\mathcal{B}_1, \mathcal{B}_2 < 1$, $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 > \lambda_2$

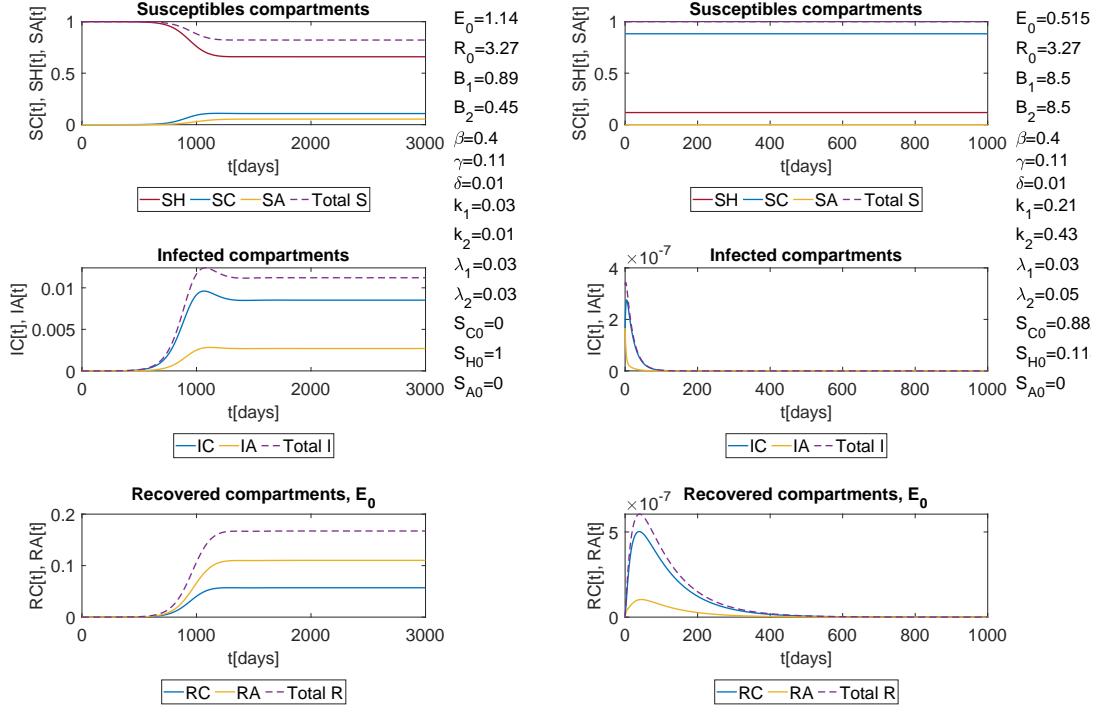
When calculating the Disease-Free Equilibrium (DFE), only one solution is found. It corresponds to the state $S_{C_0} = 0$, $S_{A_0} = 0$, and $S_{H_0} = 1$. In this equilibrium, the value of $E_0 = 1.142$, is greater than one. Therefore, as stated in the theorem presented in [91], the DFE is unstable, and a disease can evolve into an epidemic from this initial condition.

Observing the behavior layer, since both Conversion numbers \mathcal{B}_1 and \mathcal{B}_2 are less than one, the majority of the population remains in the Heedless compartment in this scenario. This facilitates disease propagation because only a small portion of individuals are in the Susceptible-Compliant (S_C) category, reducing their likelihood of infection, governed by $\rho\beta$ with $\rho < 1$. Meanwhile, S_A and S_H share the same probability of contracting the disease, β .

In the left panel of Figure 7.3, it is evident that the dynamics are dominated by the Heedless compartment. Furthermore, an infection peak is observed, and the final equilibrium reached by the system is not disease-free: a relatively small portion of the population remains infected.

7.3.2 II case: $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 = \mathcal{B}_2$, and $\lambda_1 < \lambda_2$

If both behavioral layers have the same behavior conversion number, $\mathcal{B}_1 = \mathcal{B}_2$, as shown in the right panel of Figure 7.3, the epidemic evolution is heavily influenced by the initial compartment values. This property holds even when the balance of the Behavior reproduction number is achieved with different coefficient values. Recall that k_1 and k_2 determine which behavior becomes dominant, while λ_1 and λ_2 impact the persistence of a certain behavior. In this case, the Compliant course of action is more dominant and slightly less persistent. However, the initial conditions play a critical role in shaping the model evolution. When perturbing the type-C equilibrium, where the majority of the population is in the Compliant compartments, the population remains largely Compliant. Even when some infected individuals are introduced to perturb the disease-free state, the infection is rapidly suppressed. The equilibrium DFE, with a value of $E_0 = 0.515$, is



(a) Case I: Both \mathcal{B}_1 and \mathcal{B}_2 are less than one. None of the possible behaviors become dominant.

(b) Case II: \mathcal{B}_1 is equal to \mathcal{B}_2 . Starting from a type-C DFE, we have an E_0 that confirms the local asymptotic stability of the equilibrium. No epidemic develops in the system, as shown in the simulation.

Figure 7.3: Cases I and II of the simulations performed. In the left panel, there is an epidemic outbreak leading to an endemic equilibrium, while in the right panel, the disease dies out, and the population remains in the Susceptible compartment at equilibrium.

locally asymptotically stable.

7.3.3 III case: $\mathcal{B}_1, \mathcal{B}_2 > 1$, $\mathcal{B}_1 < \mathcal{B}_2$, and $\lambda_1 = \lambda_2$

Figure 7.4, explores the impact of compliance with rules and the adoption of self-precautions on the diffusion of an epidemic. Starting from the disease-free equilibrium type-A, where $S_{C0} = 1$ and all other compartments are equal to zero, the introduction of infected individuals triggers the spread of an infection. The value of $E_0 = 1.11$ indicates that the DFE is in fact unstable.

Although the disease can spread in a fully susceptible population, the protection provided by compliant behavior significantly reduces its spread. With \mathcal{B}_1 larger than \mathcal{B}_2 , compliant behavior quickly spreads throughout the population and persists longer than the infection duration. As a result, the majority of the population transitions to the S_C

compartment, while S_A tends toward zero. The epidemic fades after reaching a small peak and infecting only a minor fraction of the population.

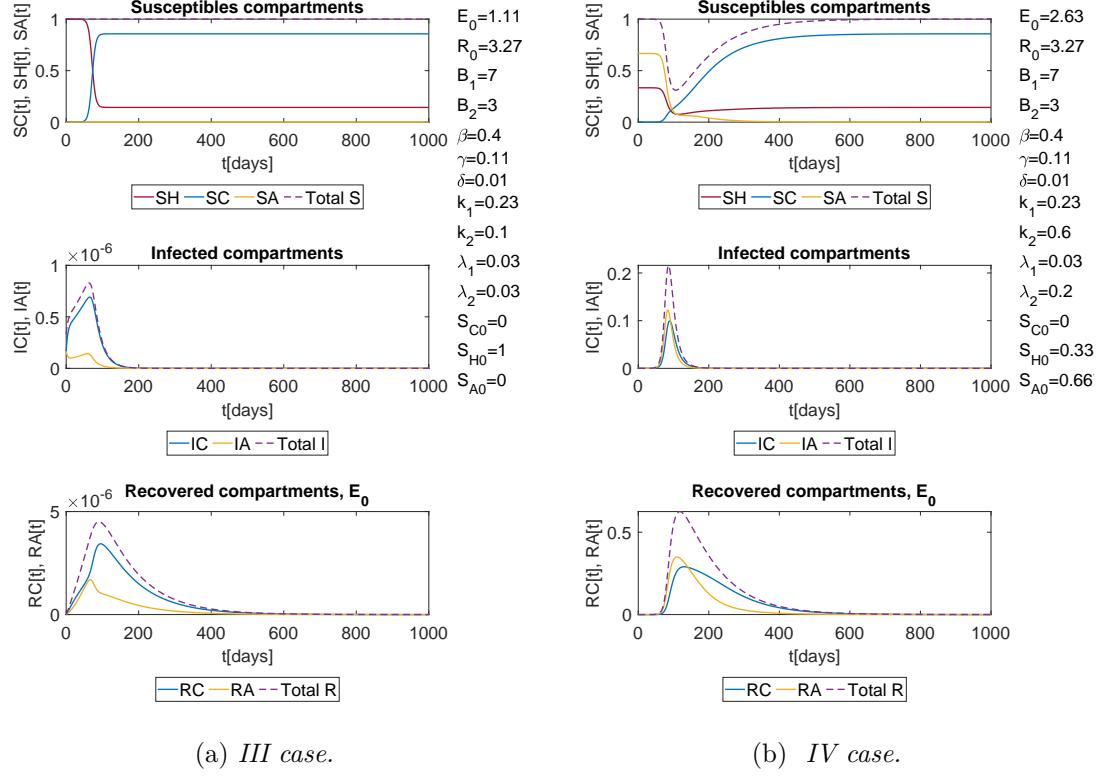


Figure 7.4: III and IV cases.

7.3.4 IV case: $\mathcal{B}_1, \mathcal{B}_2 > 1$ and $\mathcal{B}_1 > \mathcal{B}_2$, and $\lambda_1 < \lambda_2$

The fourth case highlights and describes the negative effects of non-compliant behavior. Even though $\mathcal{B}_2 < \mathcal{B}_1$, the condition $k_2 \gg k_1$ allows the Against behavior to initially spread more rapidly. As observed previously with the behavior model alone in Section 6.2.1, this dynamic results in significant early non-compliance. The simulation begins close to the DFE corresponding to type-B, where the majority of the population is in S_A . These initial conditions lead to a higher epidemic reproduction number, with $E_0 = 2.63$. Consequently, the number of infected individuals reaches a much higher peak compared to the third case. However, as the simulation progresses, the Compliant group gradually gains prevalence due to $\mathcal{B}_1 > \mathcal{B}_2$. The population tends to shift toward compliance, and after the peak, the disease diminishes toward zero.

7.3.5 V case: $\mathcal{B}_1, \mathcal{B}_2 > 1$ and $\mathcal{B}_1 < \mathcal{B}_2$

The final case represents the worst scenario for epidemic spread. The majority of the population adopts the Against behavior, which prevents the epidemic from being mitigated by NPIs or self-isolation. These measures are only adopted by an increasingly smaller portion of individuals. As a result, the epidemic trajectory closely resembles that of an SIRS-only model. With $E_0 = 3.17$, the highest value among all the cases presented, the infection peak is large, and the disease does not disappear. Instead, the system reaches an endemic equilibrium.

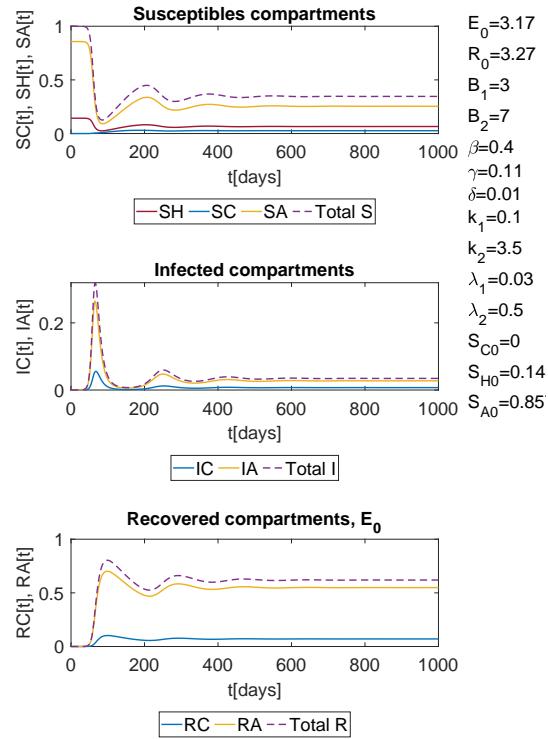


Figure 7.5: V case.

7.3.6 Influence of the ψ parameter

To model how government policies can influence the behavior of the population and create a distinction between spontaneous behavior and reactions to regulations, the parameter ψ was introduced. It acts as a central mean-field intervention, multiplying the coefficients k_1 , k_3 , and k_5 .

The value of ψ is greater than one ($\psi > 1$) when the simulations consider enforced policies that individuals must follow, and equal to one ($\psi = 1$) when no such regulations are in place.

In Figure 7.6, the evolution of S_C and the total number of infected individuals as ψ varies is represented. It is evident that increasing the value of ψ changes the timescale of compliance, causing S_C to reach its maximum more quickly, while also increasing the final number of S_C . Furthermore, it significantly reduces the peak value of infected individuals.

This effect is very pronounced when comparing cases: with $\psi = 1$, the peak value of I is around 2%, whereas in other cases, it drops to approximately 0.0007%.

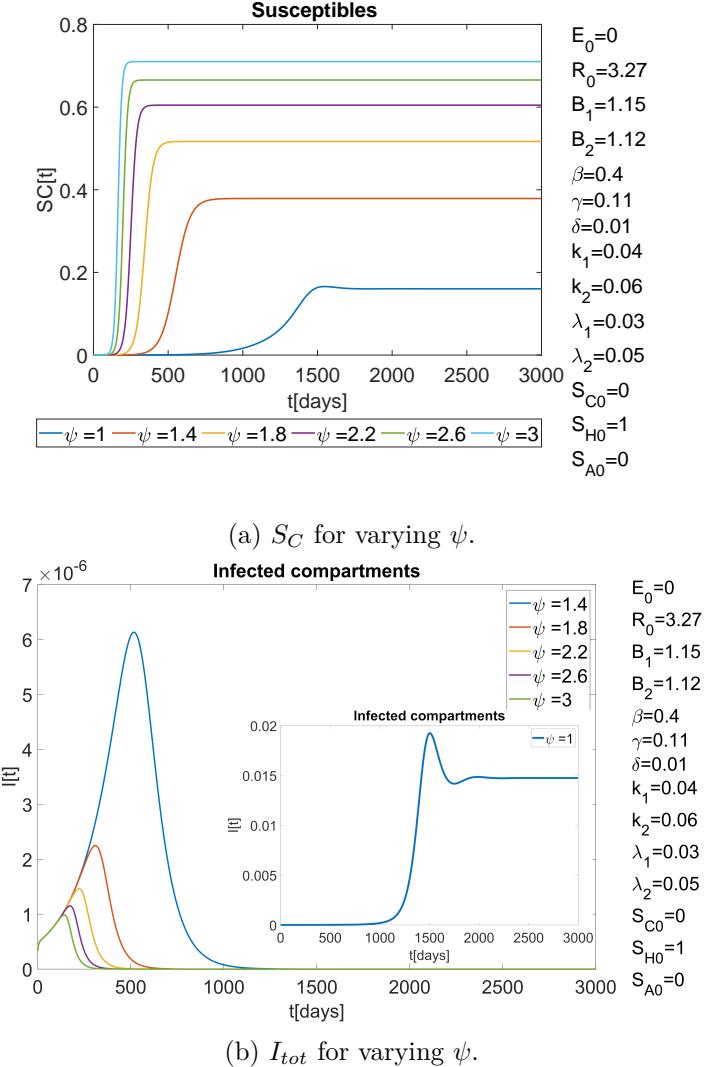


Figure 7.6: Simulations of the epidemic-behavioral model with varying parameter ψ . In the first figure, the changes in the S_C curve are shown. The maximum is reached more quickly when $\psi > 1$. In the second figure, the corresponding $I = I_C + I_A$ curves are presented. The small panel represents the case with $\psi = 1$. For $\psi > 1$, the infection peak becomes significantly smaller.

Overall, the implemented multi-system model demonstrates its ability to adapt effectively to a variety of scenarios. Specifically, it captures both situations: one where Compliant behavior significantly mitigates the disease's impact, and the opposite scenario, where the dominant presence of the Against group triggers a disease outbreak. These outcomes arise under the same disease parameter values, β, γ, δ . The estimated value of E_0 proves to be a valuable indicator of the stability of the system DFE.

Chapter 8

Conclusions

The work conducted during this thesis has led to the development of a novel epidemiological-behavioral model that couples an SIRS model with a behavioral mean-field model. Both opinion and behavioral modeling have been studied, and the most significant contributions in this field from recent years have been presented to provide a comprehensive perspective on how social dynamics can be integrated into disease progression models.

The main contributions of this study are summarized as follows:

- **Behavioral-Epidemic Interactions:** The work highlights the complex dynamics that emerge when a disease interacts with human behavior. A behavioral model with three possible behavioral states during the onset and progression of an epidemic is implemented. From its coupling with the disease model, the role of social dynamics in shaping epidemic outcomes becomes apparent.
- **Insights from Simulations:** Through analyses of individual model components and subsequent simulations, key parameters such as the Epidemic Reproduction Number (E_0) were identified. The evolution of E_0 and its effectiveness as an indicator to predict whether an epidemic will evolve were explored. Furthermore, the model demonstrates various dynamics: while compliance diffusion can mitigate the epidemic impact, the spread of misinformation, which alters individual behavior, can lead to more severe outcomes. The interplay of behavioral parameters, in particular, creates intriguing influences on epidemic evolution, which are explained in this work.
- **Framework Flexibility:** Developed with empirical data in mind, the resulting model is a versatile tool that can be adapted to different scenarios and reflects a wide range of behavioral and epidemiological dynamics.

8.1 Limitations

The proposed multi-system epidemiological-behavioral model is based on certain assumptions that may pose limitations. For instance, the homogeneous mixing assumption assumes that individuals interact with equal probability with anyone in the population.

Incorporating features like clustering or homophily—the tendency of individuals to associate with those who are similar to themselves—could add depth and realism to the model.

Another limitation is the presence of the Heedless compartment only in the Susceptible layer. This restriction can limit the model’s accuracy for the infected and recovered compartments since all individuals in S_H transition to I_C . Exploring alternative subdivisions of S_H could address this issue, but it would require a thorough and detailed analysis to ensure consistency and reliability.

8.2 Future perspective

Possible improvements and refinements include:

- Conducting an analysis based on empirical data to evaluate the model’s ability to replicate real behavioral patterns observed during the COVID-19 pandemic.
- Developing a more complex ψ parameter that incorporates information about the disease state (such as incidence or prevalence) or accounts for aspects like the economic costs associated with the disease, to better model the stringency of policies implemented by governments.
- Extending the simulations by exploring different values for the behavior transmission rates, k_i , and fatigue to maintain a behavior, λ_i terms.

To conclude, the present work is significant because it introduces a novel multi-system model that effectively integrates epidemiological and behavioral dynamics, providing insights into how individual and collective behaviors influence the spread of diseases. The model’s flexibility allows it to adapt to various scenarios, offering a valuable framework for understanding complex interactions between disease transmission and human behavior. Moreover, the potential to incorporate empirical data and compute the parameters value in this way add interesting future possible developments for the model.

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