UNIVERSITY OF TRENTO

INDUSTRIAL ENGINEERING DEPARTMENT

Master Degree Thesis

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

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Summary

La tesi si propone di presentare una delle tecnologie sviluppate per compiere ricerca sul funzionamento del cervello. Partendo da una presentazione e analisi dei principali sensori e MEMS prodotti per questo scopo, viene poi descritto in dettaglio un dispositivo realizzato basandosi su un array ad alta densità di microelettrodi, costruito con tecnologia CMOS.

L'elaborato è strutturato nelle seguenti parti: L'introduzione, dove è presentata la biologia del tessuto cerebrale, focalizzandosi sul neurone, l'unità cellulare principale che lo compone. Segue una parte in cui sono esposte e analizzate le tecnologie sviluppatesi negli anni, con lo scopo di illustrare quali siano le principali motivazioni che portino allo sviluppo di sensori molto differenti fra loro. Viene inoltre descritta la caratterizzazione elettrica della giunzione che si crea fra la cellula e il sensore. La parte centrale dell'elaborato presenta un sensore basato su tecnologia CMOS, indagando sulla logica alla base del suo funzionamento e su come esso sia costruito ed implementato nell'interezza del suo apparato strumentale. Viene inoltre confrontato con sensori simili e si evidenziano qui le sue peculiarità positive e negative al confronto con dispositivi che implementano architetture differenti. In conclusione si discute dei principali risultati raggiunti finora con l'uso di queste tecniche e di quali siano i filoni più attivi e di maggior interesse futuro per il mondo scientifico.

Contents

List of Tables

List of Figures

Part I

Introduction

Chapter 1

Introduction and main objectives

In human history the presence of disease, caused by parasite is always existed. Because of the lack of knowledge about medical science and the poor hygienical conditions the deaths caused by infections in history have had dramatic repercussion on population life. During the bubonic plague of the 14th century for example 25 million of deaths were reported in Europe out of a population of 100 million. During the colonization of the Americas the disease imported by the Europeans is one of the main causes of the genocide of the local population. Diseases like smallpox and cholera were unknown in these countries and native americans have not antibodies to them.

Not only diseases like these two have am incredible cost in term of human life. They have also effect on historical effect. Due to plague in fact in Europe starts the persecution against Jewish people, considered responsible of the illness spread. While in the Americas, the new infection were one of the element that permits at colonist to subdue the inhabitants. Others important epidemic, famous for their consequences were Spanish influenza, Smallpox, Typhus, HIV/AIDS and the more recent Covid-19. Because of these, is straightforward evidence of the effect that diseases have on our life.

Only in the last three centuries and just in the most developed countries, like Europe and North America, a significant increase in life expectancy have been observed. The mortality is decreased, but the modification in social patterns and the develop of large cities, have had some consequence. It was increased in the 18^{th} and 19^{th} centuries the frequency and magnitude of epidemics. For this reason diseases are an inevitable agents that is involved in our life. Their implications do not concern only our health status. When we are sick, there are modification in our relationship, work, social life. There is also an economic cost to be healed. Only in few nations worldwide treatment is covered free of charge by the state. In the majority, be ill can result in having to sustain excessive cost. Often due to this, people going into debit or to not take care. Nowadays, but also in the past when a new disease appears it is very important try to understand its origin, the biological mechanism under its spread, comprehend its resistance to existent drugs and so on. In practice collect all the information available to understand what is happening. These are part of the epidemiological investigation. Other key dimensions are for example

genetic resistance, selective pressure of a disease on different human communities, the mechanism underline the acquisition of immunity. Using this knowledge epidemiology try to make a step further of only reconstruct the cause behind the development of a disease: model how can evolute during time. There is a lot of interest about this topic both from

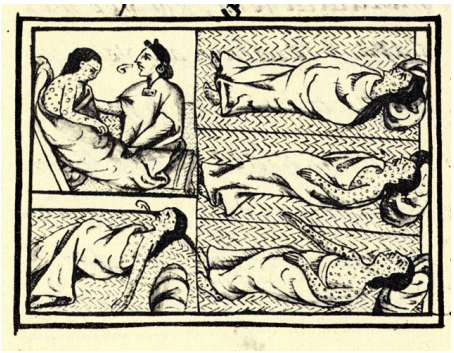


Figure 1.1: Representation of smallpox disease on the Mexican population in the XIV century. Figure from the Florentine Codex [**Sahagun1965**].

a scientific perspective and also for public society regulations. Develop a model that can give us the capability of estimate phenomena like the spread of a disease have a multitude of effects and outcomes on the society. Social and economic costs are related to every illness, but develop instruments to better understand its impact can avoid the losses of human life and permit to have less serious effect on the society. A first simple example, that can help to understand the beneficial effect of study epidemics is the possibility to develop instruments that can permits to consider the specificity of each different disease with "simple" parameters and obtain information like:

- is this disease so infective that can cause a pandemic?
- what are the threshold conditions that can cause and outbreak?

At a first look the problem can seem simple. Unfortunately, the creation of a model capable of perform an evaluation like the one explained above, suitable for every disease is a problem for which there is not still a solution. Every research about the creation of a model that try to represent a real phenomenon must confront with the necessity to simplify, while trying to not loss in accuracy. So, in a century of research, many different aspects regarding an epidemic have been considered. The scientist try to catch the most significant ones to develop their model and then figure out if it is capable to give them insights about the considered disease.

1.1 Epidemiological theory foundations

Definition of the theoretical basis and main concepts that will be used in the present work.

1.1.1 Epidemiologic research historical background

The research field regarding the development of technique to understand how epidemics can evolve during time has a history starting back in the 20th century. The first important discovery in this field must be attributed to the scientists that find the mechanism used by disease to spread. A first innovative work is the one done by John Snow, that during an epidemic of Cholera in London in 1854 successfully determined the source of the infection, even without knowing its etiological agent. Then advancing in the microbiological research is conducted by Pasteur and Koch. They found the etiological agent of disease, enabling the possibility to treat and prevent people from an infection. Then, Hamer work in 1906 added a first major theoretical contribution. He formulated a theory about the correlation between the course of an epidemic and the interaction, contact ratio, between susceptible and infectious individuals. It is the so called "mass -action" principle. The number of contacts between these two groups determines the spread rate of the disease. This law originally written in discrete time, is then updated in 1908 by Ross, that re-written it in continuous time. For the first time the problem can be studied using a clearly, well defined mathematical theory. Then the contributions of Kermack and McKendrick in 1927 add another fundamental principle to the modern epidemiology. They formulated a threshold theory explaining which condition can generate the development of an epidemic. The theorem affirms that a certain value must be exceed, depending on the proportion of susceptible and infectious individual. Controlling this value permits to understand if the number of infections will increase, until a peak is reached or if the epidemic is a descendent phase [**Mata2021**], [**Anderson_82**]. Their contribution with the mass action principle represents the base for the mean field model theory, that will be presented and analysed in section 1.2.1.

1.1.2 Epidemiological glossary

A list of principal concepts and terms is here presented.

Micro and Macro parasite

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

Disease transmission

How it is transmitted a disease.

Epidemic disease

It occurs when there is a disease with a rapid outbreak, in less than a year there is it development and a peak is reached.

Endemic disease

It is a disease that last for a longer period, in which must be considered a renewal in the population, and so in the number of susceptible.

Pandemic disease

Incubation, Symptoms, Infected and Infectious

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

Types of infectious diseases

For infectious disease it is indicated an ilness resulting from the presence of a pathogenic microbial agent. It is possible to distinguish a difference between *transmittable* and *communicable* disease. CONTINUA

Outbreak

Incidence and prevalence

Immunity and herd immunity

Virulence and Contagiousness

Overdispersion and Superspreading

CFR, IFR and mortality excess

Reproduction number R_0

Incubation period and serial interval

1.2 Epidemiological models categorization

There are several different typologies of mathematical model developed to describe the course of an epidemic. In this section the principal categories are introduced. There is then a focus, in 1.2.1, on the one used for the development of the present work, useful to introduce its main mechanisms and the first important conclusion that can be derived from it.

1.2.1 Mean field models

The mean field model, also known as compartmental models are the first and most studied typology of mathematical model used in epidemiology. The population considered in the model is divided in several subgroups, on the basis of the dynamic that want to be described. In this class of model the severity of infection usually is not considered. People

are infected or not. The transition from one compartment to another is determined by differential equations. There are transmission rates from one class to another.

SIR model

The first model we present is one of the simplest used to describe an epidemic. Here the population or density of individuals is divided in three groups: Susceptible, Infectious and Recovered. The sum of all three groups is N, the total number of people. Usually, the groups are normalized and in this way their sum is equal to 1.

$$\frac{S}{N} + \frac{I}{N} + \frac{R}{N} = 1 \tag{1.1}$$

The symbols used to indicate the density of each group are s, i, r, while the capital letters are used to specify both the name of the groups or the absolute number of participants in each one. Usually, the assumption that N remains stable is done. This is possible considering that the epidemic time span is much lower than the life duration of a person, and so the number of death and birth is neglected. Alternatively, we can consider that the number of births, which is an input in the S compartment is roughly equal to the number of deaths, which is an output. The net rate at which the number of infections increase is proportional to the number of encounters between S and I individuals, expressed by βsi, where β is a disease transmission coefficient. The simplest and easier way to initially explain the meaning of β is to consider that not every contact between a susceptible and an infected person can generate a contagion. The value of β is used to describe this parameter. Individuals pass from the infectious state to the recovered one at a rate γ. So the infection duration last an average time of $1/\gamma$ days. In this initial model the immunity acquired after recovering from the illness is lifelong. It is equivalent for the model if after being sick a person recover or die, because it considers that it will not transmit the infection any more. This assumption can be modified and there are often disease in which after a certain period individuals become again susceptible. Another initial simplification is the one of consider the coefficient β and γ constant. Also there is not a network structure defined in this model, but the population is considered homogeneously mixed. Most frequent alternatives groups used to expand these three initial categories are: Exposed, Asymptomatic, Vaccinated, Symptomatic.These are intermediate groups, while a possible final state that can be add is the one of the "Deceased" individuals. Although, SIR model is quite simple it can predict a very important aspect of an epidemic, the threshold value. Because of this, two phases can be distinguished in the disease: a free-disease scenario, while the contagion is almost disappeared and a second state in which there is a large number of infected, called endemic equilibrium.

Derivation of I evolution

The number of infected on the next day is in a discrete time Δt given by the equation:

$$I(t + \Delta t) = I(t) + [\beta S(t) I(t) - \gamma I(t)]\Delta t \tag{1.2}$$

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

$$\frac{dI(t)}{dt} = \lim_{\Delta t \to 0} \frac{I(t+\Delta t) - I(t)}{\Delta t} = \beta S(t) I(t) - \gamma I(t) \tag{1.3}$$

Observing the dynamic development, at $time = 0$ the population is almost composed by Susceptible, so $S(0) \approx N$, and in the first steps of contagion evolution this quantity remains stable. Considering this approximation, we have

$$\frac{dI(t)}{dt} = (\beta S(0) - \gamma) I(t), \tag{1.4}$$

which gives,

$$I(t) = I(0) \exp^{(\beta S(0) - \gamma)t} \tag{1.5}$$

and the final set of differential equations that describe the dynamic of infection is the following:

$$\begin{cases} dS/dt = -\beta SI \\ dI/dt = \beta SI - \gamma I \\ dR/dt = \gamma I \end{cases} \tag{1.6}$$

From the analytic solution of the infectious dynamic equation in 1.5, we can see what happens at the beginning of an epidemic. Furthermore, observing the exponential sign we can deduce the disease behaviour and how the situation can evolve. In fact if the exponential is greater than zero the number of sick grows exponentially. While, in the opposite case, infected people tend to zero.

The value $\beta S(0)/\gamma = 1$ is defined as epidemic threshold. This quantity, normalized, is called R_0 index:

$$R_0 = \beta/\gamma \tag{1.7}$$

It measure the intensity of the contagion, or alternatively the number of secondary infections a sick person can generate. Analysing the equation of susceptibles, with this model we see that it is always decreasing. In the SIR model, if the condition to start the epidemic is met after an increasing in the number of Infected, a peak is reached. Then, the disease begins its falling phase. It is the natural behaviour of an epidemic. This transition happens when the value of $R = \beta S(t)/\gamma$, the effective reproduction number become less of 1.

INSERIRE FIGURE DAL MATLAB FATTO DA TE!e dei commenti su quello.

Other two interesting quantities to consider when a new disease appears are, the rate of increase of the infectious and the final size of remaining susceptible at the end of the epidemic. In fact, there is a large difference when a population suffering for an epidemic, if this ends rapidly because a lot of people get sick or id this number can be controlled, and the infectious curve is flatter.

1.2.2 Stochastic models

In this typology of models, the transition from a state to another is determined using a function of probability. They can be conceptually derived starting from the framework represented by ODEs models. They are useful when the disease to study has a lower number of infected or if there is a connection between the epidemic outcome and changes

in individual dynamics. This is called demographic variability, and it concerns changes in transmission, births, recovery, or deaths within the population. Using stochastic models with Monte Carlo simulations can be useful to investigate epidemic models on networks. The two most important type of models using this approach consider the time variable as continuous, $t \in [0, \infty)$and then the state variable is either discrete (Continuous-Time Markov-Chain) or continuous (Stochastic Differential Equations). Referring to the SIR model to make a simple example here the S and I compartments are modelled as random variables. The probability to individuals to change group depends on infection and recovery, the possible events that can occur. It is called transition probability. In a Markov chain approach the transition probability is discretised, and there is no dependence on the history of the epidemic to know how it will evolve at time $t + \Delta t$. It is necessary to know only the current state of the process at time t. In the Stochastic differential equation, the random variables are continuous. The system of equation deriving from this method are usually simpler to solve that the ones find using the first approach[**Allen2017**].

1.2.3 Agent-based networks

Another representation of the disease evolution can be done using an agent-based model. Here, using the topology of a network, composed by nodes and edges is realised the simulation. Individuals are represented with the node and their interactions with the edges. So, the connections in the graph are responsible for the contagion. Using this framework, it is possible to simulate more realistic scenarios, because permits to represent large and heterogeneous systems. It is not easy however to understand how model a complex society in this way.

1.2.4 Multilayer networks

The complex dynamic of interactions existing in real word, develops in multiple patterns, with complicated relationships. This connection can change in time, and using the theory of multilayer network it can be improved the comprehension of such complexity. This is a more recent development of the research, the traditional network theory was revisited, to create a framework that can include multiple networks, that evolve and influence each other [**DeDomenico2016**]. One possible way to develop models with this structure is the one done imagining that each layer represent a different type of interaction. An epidemiological example is one layer in which the physical contact between people are simulated and another representing social structure, the network of relations that every person has. This case has been presented in multiple works in the last year, for example CITA. The dynamic realized in multiple networks can be single or coupled. In the first there is a top layer with its own dynamic evolution influencing another one. The coupled structure instead is the one in which the phenomena described in each layer evolve with the influence of what is happening in the other. There is a coupled connection with the presence of intra-layer connections.

Part II

Behavioural Disease Model

Chapter 2

Review of epidemiological behavioural and opinion models in literature

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

- deterministic/mean field models
- opinion models
- multilayer networks
- opinion-disease models

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

2.1 Opinion models

In the analysis performed by Wang [**Wang_2019**], are presented mechanism implemented to explain coevolution spreading in complex network. The principal methodologies created over time are threshold model, that can use a linear threshold or a "Watts threshold". Here each node has a random different threshold, based on a certain distribution. Using a threshold means that a node change opinion on the basis of its neighbours' belief. The shape of the network is then fundamental for an opinion to spread. The best scenario is the one in which there is a low degree of randomness, and the network is regular. Also, cluster can have a reinforcement effect, if they are sufficiently connected to the resto of the graph. Their work then report analysis based on competition or cooperation of opinions "contagions". And a SAR model is presented. Similar to a SIR, here the meaning of letter

A is "adopted". It means becoming convinced of a certain opinion, but with a probability or rate to then return to the previous behaviour. Also the work of [**Nunner2021**] define and test some different models based on trade-off between the benefit of having connections and the penalty for acquiring infections. It is showed that when the behaviour of people depends on maximizing their net benefit, the individual risk perception plays an important role in the formulation of a cost function. The models derived with this so called co-evolutionary approach, have an overall dynamic very correlated between the two strati: it is a feedback loop between infection spreading, people behaviour adaptation and consequently structural modification in the network.

2.2 Multilayer network

One work based on feedback between two networks concatenated is the one performed by Peng et all, [**Peng2021**]. Here there is explained a model based on two graphs, where one simulates the evolution of a disease, using a SIR or SIRS dynamic, and another explicit the behaviour of individual in a UPAU network. U means uninformed, P pro-physical distancing and A anti-physical distancing. In this network the people's conduct influence the β coefficient of the epidemic diffusion. They demonstrate the effectiveness of having an opinion in reducing the negative effect of a disease and that lengthening the duration time for which an individual maintains opinion can help suppressing the transmission. Study the effect of competition in a multilayer network is the objective of Teslya et all research [**teslya2022**]. At cause of interpersonal communication individual can change their opinion. They are divided in two main groups, positive or negative w.r.t health conduct. Here is also inserted an influence due to assortatively when contacting with others. Their principal results further than the fact that opinion influence disease, is realizing a model in which the two opinions can coexist at equilibrium. There can be oscillation of prevalence due to increased transmissibility of infection. In SIR model they demonstrate a reverse correlation between the rate of social contact and the peak magnitude of infectious. The causes of oscillations in the disease dynamic are a high infection rate and a pronounced difference in infection rate between individuals with different opinions. The others important factors are the high-rate opinion exchange and high sensitivity of population to prevalence. In the article [**Alvarez_Zuzek_2017**] the opinion about vaccination is taking in consideration, into a SIR+V mean field model. Conversating is the mean used by individual to modify their opinion. With a very positive opinion susceptible individuals can choose to take the vaccine. Interesting they use a r factor to describe the extremism in opinion. Varying this coefficient, they observe that the best scenario for delay the development of an epidemic is the one where the society is neutral. So, when there aren't compromise or persuasion, but the conversation is based on "rational" arguments. Another works analysing two competing opinion is [**Epstein_2021**]. Here population is sensitive to both fear of vaccine and disease. These two interact and the vaccination grow rate increases only if the fear of the disease is larger than the of vaccine. The infection curve is very influenced by the presented dynamic, and the best scenario is obviously the one in which the fear of vaccine does not exist. However, in the case where the two fears coexist there is an improvement in the number of infected, for multiple infection waves. The work by Auld [**Auld_2003**],

reflect an observed characteristic in society: pessimistic expectations over the future induce a more risky behaviours. This conclusion derives observing and simulation evolution correlated to the news about a vaccine. This knowledge causes a decrease in infection rate before the vaccine becomes available. Then there is a return to normal behaviour. If there are not information, pessimism cause more risky behaviour. In [**Sontag_2022**] there is another SIR and opinion model, with population that is divided in trusting and distrusting. They add in the model the effect of fading and a global force, that simulates central interventions. The main interesting conclusion of their work is that strong public intervention have a similar effect to the network to the ones obtained if the population is composed of trusting and compliant individuals. However, higher percentages of distrusting cause the model to pass a phase transition where outbreaks cannot be suppressed. A different approach in using a multilayer network is the one realised in [**Turker_2023**], where the social structure of a town is re-created. Every layer describes the places populated by individuals: from house, to work, distinguishing between different type of work, and considering a level for friendship. Each person is present to more than a layer and, in each layer, relates to different agents, based on the social group's provenience. Using this approach, they have found that the level in which is easier for an outbreak to develop is the one associated with friendship. Here the interaction is closer with others, the security level is lower. For this reason, a lower value of transmissibility rate β is sufficient to have an epidemic with many susceptible involved.

2.3 Opinion-disease model

The work done by Funk and its colleagues [**Funk_2010**], it is very interesting: they collect and explain systematically the behavioural reaction of population in response to a pandemic. They classify the human behaviour subject to different possible sources of information. An information can be global available or local. This reflects the way it radiates or if develops in social cluster. Another important difference is related to objectivity. Certain information is based on belief and can change with time. This typology can be influenced by the social connections of an individual or by the influence of external agents, like media. Cognitive bias also can have an impact on our opinions: amplification, confirmation, anchoring bias. They then focus on the influence of self-initiated action in the control of disease diffusion. When an individual change its behaviour, form a modelling point of view this can influence: its probability to change state (from S to I for example). The value of β or γ. Modification in the contact network, with a self-isolation or adherence to more cautious conduct. Fear has an important role in how people face epidemic. Due to this emotion, people can decide to get vaccinated for example (or not, if they are more frightened by vaccines). Another phenomenon observed and influenced by fear is saturation. When there is many infectious people tend to decrease their number of contact and this cause a decrement in the I curve. Another multilayer network with two opinion, 0 where individual not take precautions and 1 where the protective measures are used, is presented in [**Frieswijk_2022**]. This model is associated to a SIS disease one. The article studies the stability of asymptotically equilibria of the system. Assuming different value of a parameter used to describe risk perception they found a set of final possible states.

The most interesting is a stable asymptotical equilibrium in which there is a periodic epidemic outbreak and a consequently population behaviour response, changing behaviour to a safer. An analysis of people choices about vaccinations is done by [**Bauch_2012**], they study the feedback between the positive effect due to vaccination and the fear of being vaccinated. In fact, thanks to vaccines, the disease incidence can become very low, and the perception of risk related to them can seem larger. They implemented an approach based on game theory and using social learning. A possibility to integrate the effect of opinion in the dynamic of an epidemic, is creating different subgroups of susceptible. They are separated according to their level of opinion, and the less they belief in use of NPI, for example, the higher probability of being infectious they have. This is the approach used in [**Tyson_2020**]. They also implemented different functions describing the influence between opinion and the possibility to become infected. The influence of media has also been analysed. This is interesting, because it's a communication channel that can be used by government, and so it is an available control measure that can be implemented, to try control the behaviour of population. For example in [**Collinson2014**] a parameter depending on I value simulates the effect of media covering the news about the disease. Increasing the number of infectious cause, the creation of news and other media about it. These can have as effect to induce more people practice social distance for example. Study both the effect of media, see as a central node of communication joined with opinion evolution is done in [**Granell_2014**]. Nodes co-exist into two layer, one for disease spreading and one for awareness, (unaware-aware-unaware model). In their application the awareness process without media, must reach a certain level on the transmissibility of awareness to influence the onset of epidemic. Instead, with an influence of media, greater than zero, this "metacritical" point disappears. A central broadcast, even with a small communication influence power, as a direct effect on all the network dynamic.

Chapter 3

Models description and analysis

The model developed for the thesis work is a behavioural disease multilayer system. Before describe the full multilayer network, a discussion about the SIRS and behavioural model implemented is presented.

3.1 Behavioural model

The behavioural network alone is composed of three compartments. These are Compliant, Co, Careless Ca, Against Ag. The differential equations describing the model evolution are 3.1:

$$\begin{cases} \dot{Ca} = -k_1 CaCo - k_2 CaAg + \lambda_1 Co + \lambda_2 Ag \\ \dot{Co} = k_1 CaCo - \lambda_1 Co \\ \dot{Ag} = k_2 CaAg - \lambda_2 Ag \end{cases} \tag{3.1}$$

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

- vaccine denialism;
- misinformation diffusion;
- refusal about existence of the disease;
- lack of trust on doctors and government policies.

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

- k_1 influence rate between Ca and Co;
- k_2 influence rate between Ca and Ag;
- λ_1 rate of leave compliant behaviour due to fatigue;
- λ_2 rate of leave against behaviour due to fatigue.

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

$$R_i = \frac{k_i}{\lambda_i} \tag{3.2}$$

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

Equilibrium and stability analysis

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

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

The equations become

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

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

$$y = \frac{x(k_2 - k_2 x + \lambda_2)\lambda_2}{x(k_2 - k_1) + \lambda_1 - \lambda_2} \tag{3.3}$$

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

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

The choice of the right R_i to use for the second nullcline depends on the comparison between the two reproductive ration values. The larger is the one that must be used. Now are presented four main possibilities of the system evolution, and the stability of the found equilibria are studied.

I case: $R_1 > 1$ and $R_1 > R_2$
The plots of the system evolution in this case is:

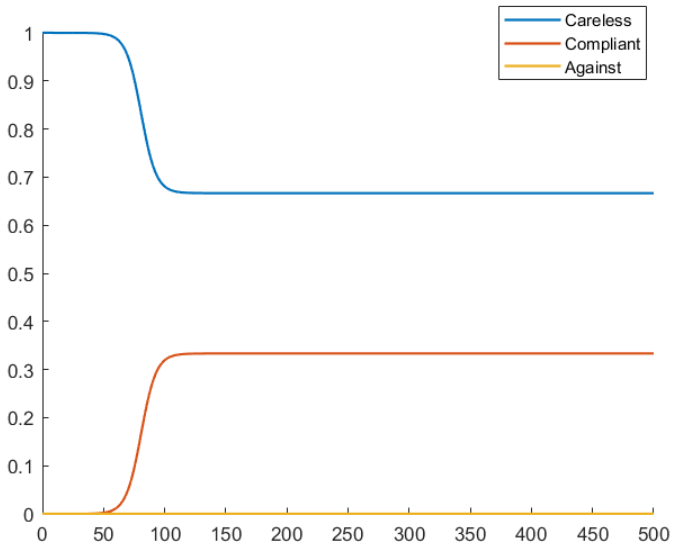


Figure 3.1: The behavioural system dynamic with $R1 > R2$ and $R1 > 1$.

In this first scenario, as visible in figure 3.1, the Against compartment tend to zero, so the equilibrium point can be calculated as $Ca = \lambda_1/k_1$ and $Co = 1 - \lambda_1/k_1$. The nullcline resulting plot is visible in 3.2.

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

- trace(J) < 0

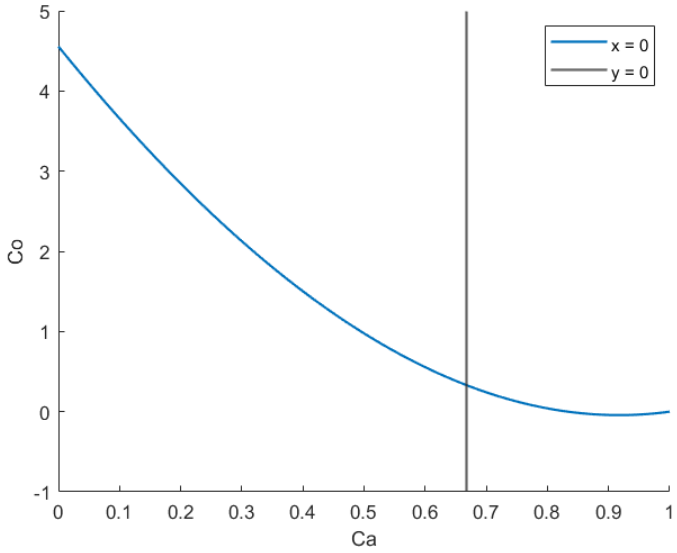


Figure 3.2: The behavioural system nullcline lines with $R1 > R2$ and $R1 > 1$.

- $\det(J) > 0$

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

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

The plots of the system evolution has an opposite behaviour w.r.t the first case. So here, is the Compliant compartment that tend to zero at equilibrium.

The equilibrium point can be calculated as $Ca = \lambda_2/k_2$ and $Co = 1 - \lambda_2/k_2$. The nullcline resulting plot is visible in 3.4. The equilibrium is asymptotically stable.

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

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

From the nullcline plot 3.6, it can be seen that there is not an intersection. The plot of the second nullcline result in a vertical line with a value grater than one. In this condition the only equilibrium is the one for which $Ca = 1$ and both Ag and Co are equal to zero. The equilibrium is asymptotically stable.

IV case: $R_1 = R_2$

This final situation is the most difficult to analyse. In fact, due to the equal value of the two influence processes, the final equilibrium of the compartments cannot be calculated using only the previous relations, but depends also on the initial condition. The Careless compartment can be calculated using the same equation of previous cases, and the same value is found using both $Ca = \lambda_1/k_1$ and $Ca = \lambda_2/k_2$. As it can be seen from the system evolution and nullcline plots 3.7, 3.8,at the equilibrium the Against and Compliant groups are formed by a subdivision of the $1 - Ca$ part. The initial condition have an influence on how this subdivision is composed. Using the Routh-Hurwitz criterium nothing can be said on this equilibrium because the determinant of the Jacobian have a null value.

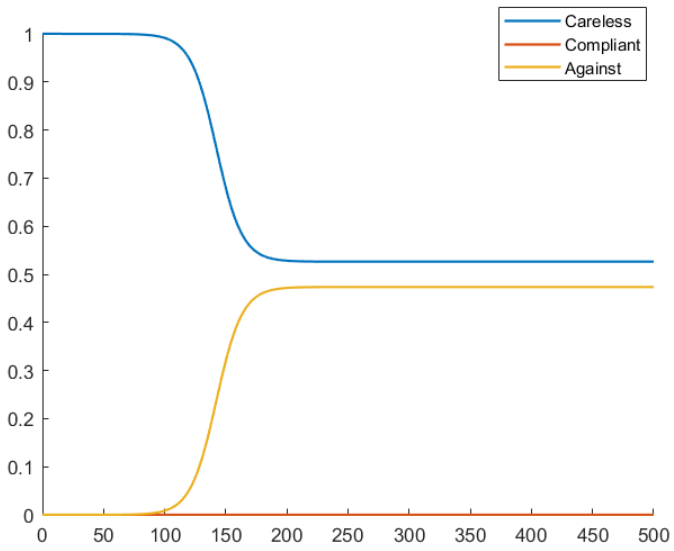


Figure 3.3: The behavioural system dynamic with $R2 > R1$ and $R2 > 1$.

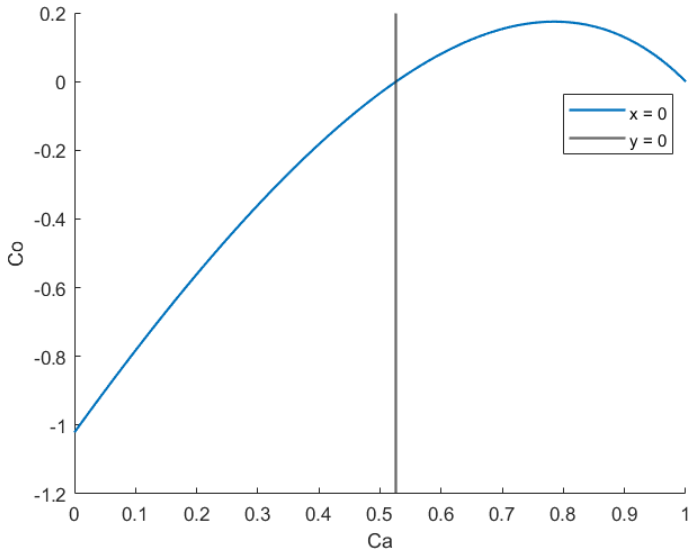


Figure 3.4: The behavioural system nullcline lines with $R2 > R1$ and $R2 > 1$.

Behavioural model experiment

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

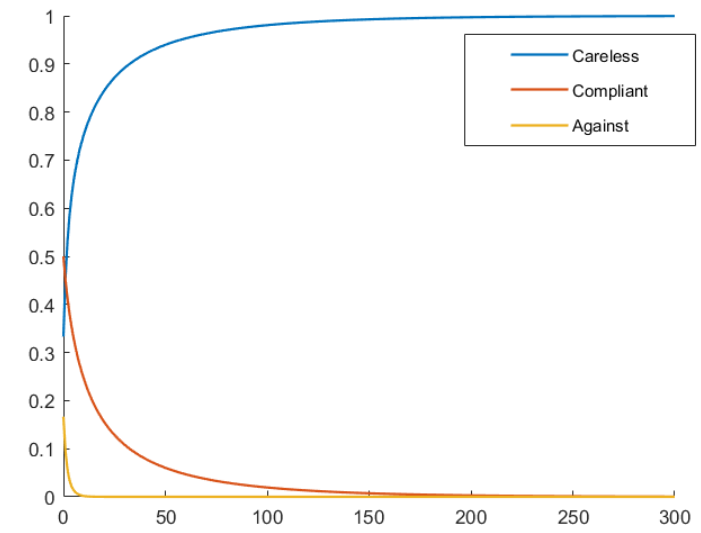


Figure 3.5: The behavioural system dynamic with $R1 < 1$ and $R2 < 1$.

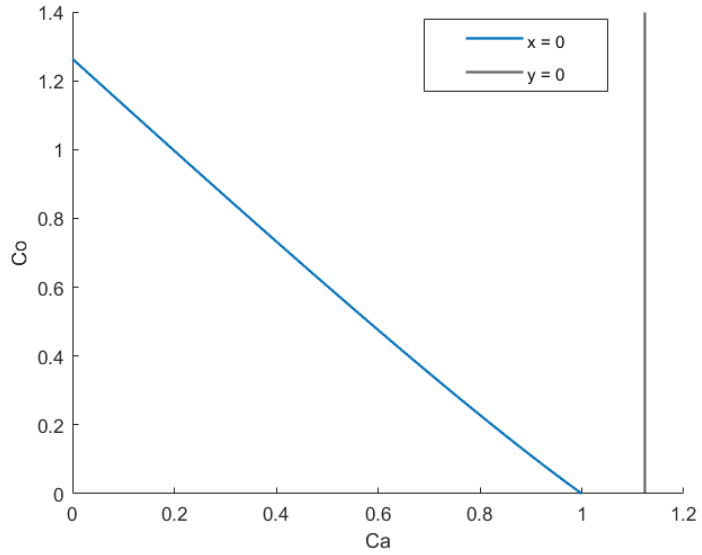


Figure 3.6: The behavioural system nullcline lines with $R1 < 1$ and $R2 < 1$.

of variation of each parameter is the following:

- k_1 between 0.1 and 0.99
- k_2 between 0.1 and 0.99
- λ_1 between 1/5 and 1/30 d^{-1}

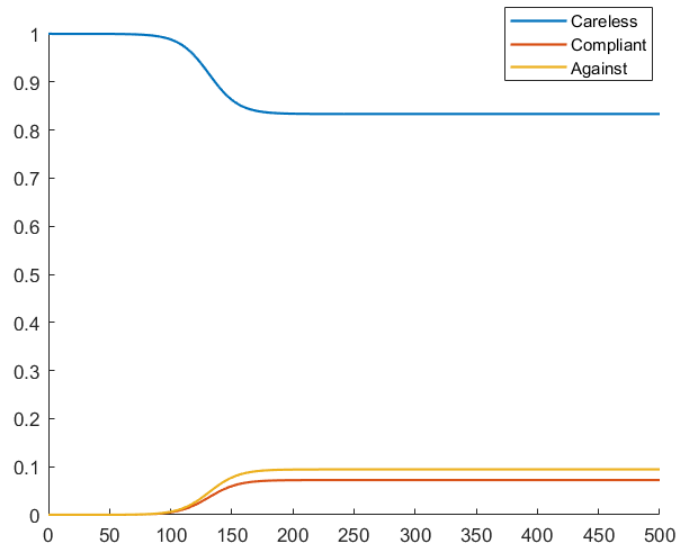


Figure 3.7: The behavioural system with $R1 = R2$.

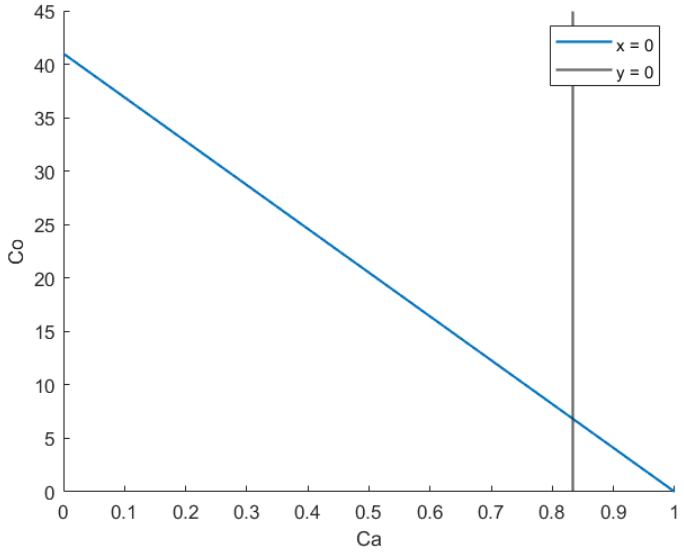
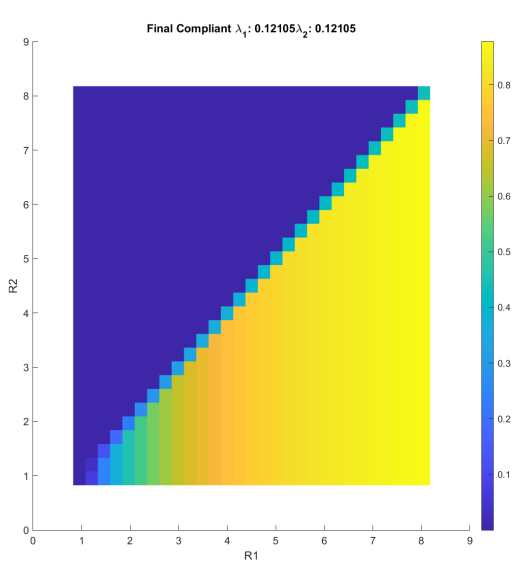


Figure 3.8: The behavioural system nullcline with $R1 = R2$.

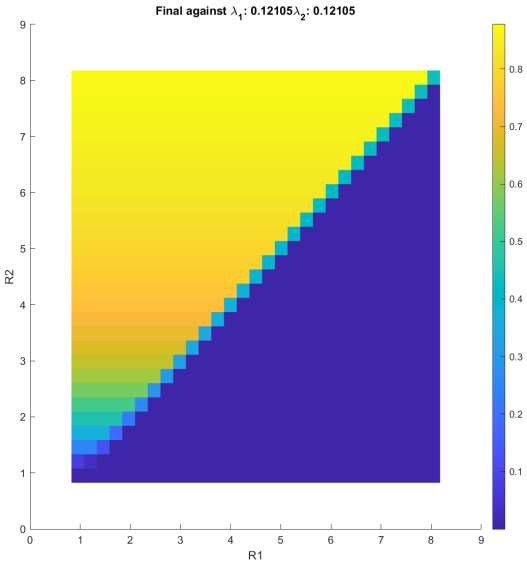
- k_1 between $1/5$ and $1/30$ d^{-1}

We observe the evolution of the dynamics of all the states, and to present a summary of the effects we collect for each simulation data such as the final value of the compartment, the max peak value and the corresponding time in which the peak occur. Also here, for the sensitivity plots realization, the reproduction rates deriving from the combination of coefficients, equation 3.2, are used.

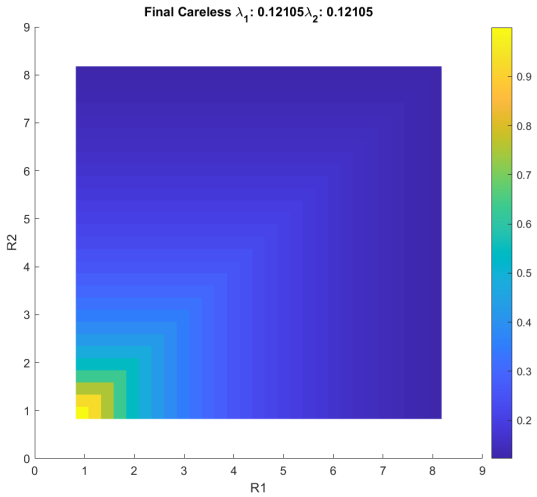
The first plots 3.9 are heat maps about the final value reached by various compartments, varying R_1 and R_2.



(a) *Final Compliant compartment*



(b) *Final Against compartment*



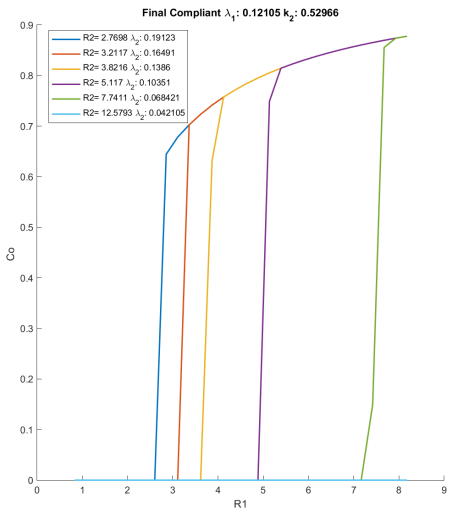
(c) *Final Careless compartment*

Figure 3.9: The final value reached at equilibrium by every compartment in the behavioural model.

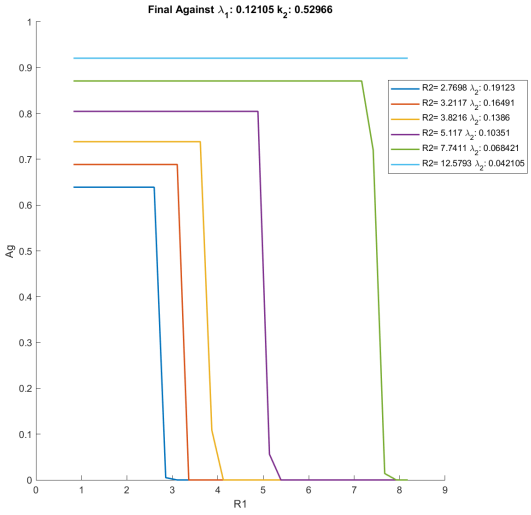
In these pictures is clearly visible the threshold effect observed in the stability analysis performed earlier. While one of the reproduction ratios becomes larger than the other, the system equilibrium is composed by the dominant group and a portion of Careless individuals.The greater is the ratio,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

Another figure in which this threshold effect can be observed is 3.10.

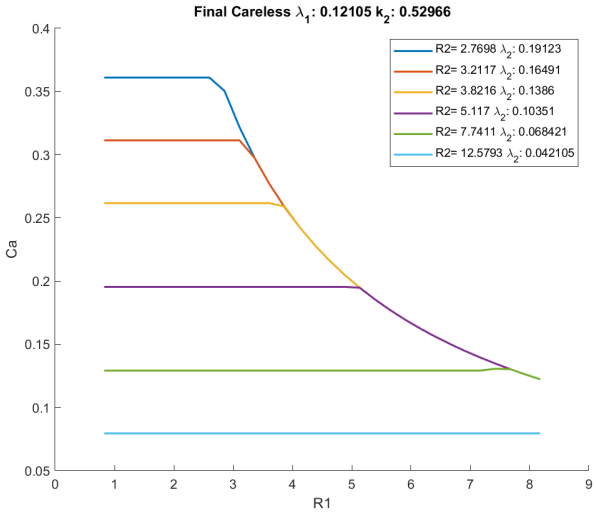
The plots show how, for a fixed values of λ_1 and k_2, change the size at equilibrium



(a) *Final Compliant compartment*



(b) *Final Against compartment*



(c) *Final Careless compartment*

Figure 3.10: The final value reached at equilibrium by every compartment in the behavioural model varying the R_1 coefficient w.r.t different R_2 values.

of the system, varying the k_1 coefficient. To highlight the threshold effect due to the comparison of reproduction rates, on the x-axis is plotted the R_1 coefficient, that can be calculated knowing the value of λ_1 and k_1. For the same reason, different R_2 situations are represented.

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