PhD thesis

Memory-induced complex contagion in spreading phenomena on networks

Xavier Roderic Hoffmann

Memory-induced complex contagion
in spreading phenomena on networks

Memòria presentada per optar al grau de doctor
per la Universitat de Barcelona

UNIVERSITAT DE
BARCELONA

Programa de doctorat: Física

Línia de recerca: Física de la Matèria Condensada

Autor: Xavier Roderic Hoffmann

Director: Dr. Marián Boguñá Espinal

Tutor: Dr. Giancarlo Franzese

Barcelona, juny 2021

Fet a mà a Barcelona per trapitosalsol.es

Handcrafted in Barcelona by trapitosalsol.es

Versió digital disponible a github.com/xhoffmann

Digital version available at github.com/xhoffmann

Abstract

Epidemic modeling has proven to be an essential framework for the study of contagion phenomena in biological, social, and technical systems. Albeit epidemic models have evolved into powerful predictive tools, most assume memoryless agents and independent transmission channels. Nevertheless, many real-life examples are manifestly time-sensitive and show strong correlations. Moreover, recent trends in agent-based modeling support a generalized shift from edge-based descriptions toward node-centric approaches.

Here I develop an infection mechanism that is endowed with memory of past exposures and simultaneously incorporates the joint effect of multiple infectious sources. A notion of social reinforcement/inhibition arises organically, without being incorporated explicitly into the model. As a result, the concepts of non-Markovian dynamics and complex contagion become intrinsically coupled. I derive mean-field approximations for random degree-regular networks and perform extensive stochastic simulations for nonhomogeneous networks.

The analysis of the SIS model reveals a sophisticated interplay between two memory modes, displayed by a collective memory loss and the dislocation of the critical point into two phase transitions. An intermediate region emerges where the system is either excitable or bistable, exhibiting fundamentally distinct behaviors compared to the customary healthy and endemic phases. Additionally, the transition to the endemic phase becomes hybrid, showing both continuous and discontinuous properties.

These results provide renewed insights on the interaction between microscopic mechanisms and topological aspects of the underlying contact networks, and their joint effect on the properties of spreading processes. In particular, this type of modeling approach that combines memory effects and complex contagion could be suitable to describe ecological interactions between biological and social pathogens.

Chapter 1
Introduction

1.1 The history of epidemiology

Since its dawning, humankind has endured the burden of disease. The earliest evidence of smallpox, for example, traces back to 3rd century BCE Egyptian mummies [28]; however, it is believed to have appeared in the first agricultural settlements around 6000 BCE [86]. Despite major improvements in public health management, humanity of the 21st is far from overcoming the hurdle of infectious diseases. Global warming has driven the spread of malaria, dengue, and yellow fever into new regions [175, 137]. Misuse and overuse of antibiotics has accelerated the appearance of drug-resistant strains of tuberculosis, pneumonia, and ghonorrea [178]. And the ongoing AIDS and COVID-19 pandemics leave no doubt about the latent threat of novel infections [62, 49].

For centuries, shamans, menders, doctors, and scientists have attempted to fathom the outbreak and spread of these illnesses. Besides curing the sick, they devised and evaluated strategies such as inoculation and isolation plans in order to reduce mortality rates. Over the years, epidemiology became a cornerstone of public health and preventive medicine. Nowadays, epidemic modeling is one of the principal tools to study the spreading mechanisms of pathogens, predict the evolution of an outbreak, and assess containment protocols.

The first systematic epidemiological study appeared in 1662, when John Graunt published his analysis of births, deaths, and causes of death in London [146, 50]. A pioneer in demography and descriptive analytics, he presented one of the first life-tables and reported time-trends for many diseases.[1] Almost a century later, Europe was immersed in a devastating smallpox epidemic. Motivated by the controversy surrounding mass inoculation in France, Daniel Bernoulli developed a revolutionary analytical approach to quantify the benefits of eradicating the disease [58, 13]. His 1766 paper concluded that, in

[1] He also refuted the idea that plague outbreaks coincide with the reign of a new monarch.

absence of smallpox, the life expectancy at birth in Breslau would increase by around 3 years, and that the median age of the population would jump from about 11.5 to 25.5 years.

This brief review of early advances ends with John Snow, often credited as the founder of modern epidemiology. His investigations during the 1854 London cholera outbreak identified a well in Soho as the source of one of the infection hotspots [154, 71]. Although he lacked conclusive biological evidence about the harm posed by the water, the cluster of affected households around the water pump persuaded the local authorities to disable it (see Fig. 1.1). His work also refuted the then-prevailing miasma theory [105], which blamed high infection rates in impoverished areas on bad air quality instead of addressing the underlying issues of poor nutrition and sanitation.

1.2 Mathematical modeling of infectious diseases

The previous examples—as well as many other efforts—preceded the development of modern germ theory during the late 19th century [21]. A major breakthrough came with the ability to differentiate infectious diseases, which can be passed between individuals (for instance, influenza), from noninfectious diseases, which develop over an individual's lifespan (think about arthritis, for example). In addition, advances in microbiology allowed to classify pathogens as either micro- or macroparasitic. The former are small (usually single-cell) organisms such as viruses, bacteria, protoza, or prions. The latter are any larger form of pathogens, including helminths, flukes, and other parasitic worms. Macroparasites exhibit a complex life-cycle within the host, which must be modeled explicitly. In contrast, microparasitic infections develop rapidly, so the internal dynamics of the pathogen within the host can often be safely ignored [98].

The comprehensive understanding of the biology behind the spread of infections allowed for evermore sophisticated models, which lead to the establishment of epidemiology as a core pillar of preventive medicine. Over time, the increasing demand in statistical rigor attracted a variety of scientists and prompted the foundation of a new interdisciplinary subfield, nowadays identified as mathematical epidemiology. For instance, in their attempt to describe the recurrence of measles outbreaks, Pyotr Dimitrievich En'ko (in 1899) and William Hamer (in 1906) independently introduced the hypothesis of homo-

Figure 1.1: **1854 Broad Street cholera outbreak.** Aided by Reverend Henry Widehead, John Snow tracked down the homes of the deceased (black stacks) and identified a pump on Broad Street as the source of the Soho outbreak (brown dot, other nearby pumps in green). It was later discovered that the public well had been dug less than a meter from an old cesspit that had begun to leak feacal bacteria. Map originally published in [154], adapted from [153].

geneous mixing [63, 50, 86, 152]. Inspired by the law of mass action, they assumed individuals interact uniformly with all others. Nobel Prize winner Ronald Ross used a similar approach in 1911, when he developed a host-vector model for the transmission of malaria between mosquitos and humans [86, 21].

The establishment of epidemic modeling is usually attributed to Kermack and McKendrick, who published a series of seminal papers between 1927 and 1933 [99, 100, 101]. In short, their modeling scheme consists in i) dividing the population into a discrete number of categories (i.e., homogeneous compartments), ii) translating all biological properties of the disease into mathematical parameters, and iii) specifying the rules that govern the transitions between compartments (see Fig. 1.2). The success of their approach lies in the balance

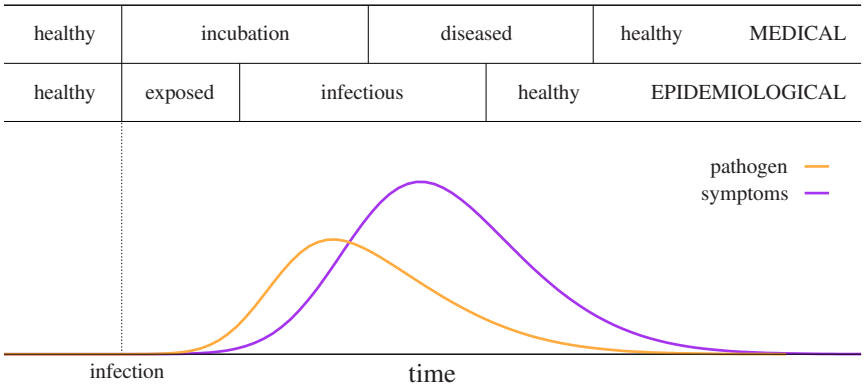

Figure 1.2: **Timeline and stages of an infection.** Schematic evolution of the pathogen dynamics (orange) and host immune response (purple). Medically, the host becomes diseased after a period of incubation. From an epidemiological point of view, the host first undergoes an exposed period, during which it is not yet infectious. Note that medical and epidemiological compartments are not necessarily correlated.

between generality and simplicity; this versatility allows to study a large variety of pathologies and gauge the effect of different preventive interventions. Nevertheless, the use of differential equations implicitly assumes continuous variables, which presents a major drawback when (some of) the population groups are small. For these cases, the model developed by Reed and Frost is more suitable [74, 72]. First mentioned in a Cutter lecture at Harvard in 1928, it was not deemed worthy of publishing by the authors.[2] Their microscopic, probabilistic approach provided an easy mapping between stochastic and deterministic formalisms, a feature that proved fundamental for later developments.

Mathematical epidemiology experienced a surge in the mid-to-late 20th century [6, 4]. The possibility of including a wide array of factors, such as age, birth, death, migration, or immunity, allowed for evermore sophisticated and accurate models [98]. A noteworthy development was the ability to thoroughly evaluate inoculation strategies, which could aid in achieving herd immunity. These theoretical advances led to the design of aggressive, global vaccina-

[2] In fact, it did not appear in print until 1976.

tion programs. Success—and worldwide joy—arrived on May 8, 1980, when the 33rd World Health Assembly officially declared the eradication of smallpox [28]. Nowadays, compartmental-like models remain the starting point of the majority of research efforts.

1.3 Network epidemiology

The birth of network science in the late 90s revolutionized many fields, including epidemiology. Old-fashioned homogeneous mixing was gradually replaced by the idea that individuals interact with a limited number of peers. This is nicely represented through a network: a collection of nodes that are connected by a set of links [1, 123]. As it turns out, the structural properties of the underlying contact network play an essential role when it comes to disease propagation.

One of the first important findings was that many real-life networks are small-world [173]. This means that i) my contacts usually also interact between them,[3] and ii) I can reach any other individual with the help of only a small number of intermediary contacts.[4,5] In terms of spreading, the small-world effect significantly increases the propagation speed; consequently, weaker pathogens are able to evolve into endemic outbreaks. Another pioneering result was the discovery of fat-tailed distributions in real-life networks. While the large majority of individuals have very little contacts, only a handful are connected to many others. This scale-free property was found in a wide variety of social [142, 108], biological [114, 92], and technical systems [67, 2, 89]. A new, suitably modified theoretical framework showed that the epidemic threshold vanishes in scale-free networks, implying that even the weakest infection could cause a persistent outbreak [134].[6]

Faced with the threat of super-weak–but–mega-destructive pathogens, one could be tempted to give up on transmission-reducing interventions. Taking a look at the implications for vaccinations, however, reveals a much brighter picture. If we select individuals randomly, we would have to immunize 95% of

[3] In technical words, the networks have high clustering.

[4] In technical words, the networks have a small diameter.

[5] In the field of sociology, this feature was discovered in 1967 by Milgram in his famous "six degrees of separation" experiment [116, 160].

[6] This striking result sparked a heated debate between theorists and experimentalists alike [61, 16, 95, 149].

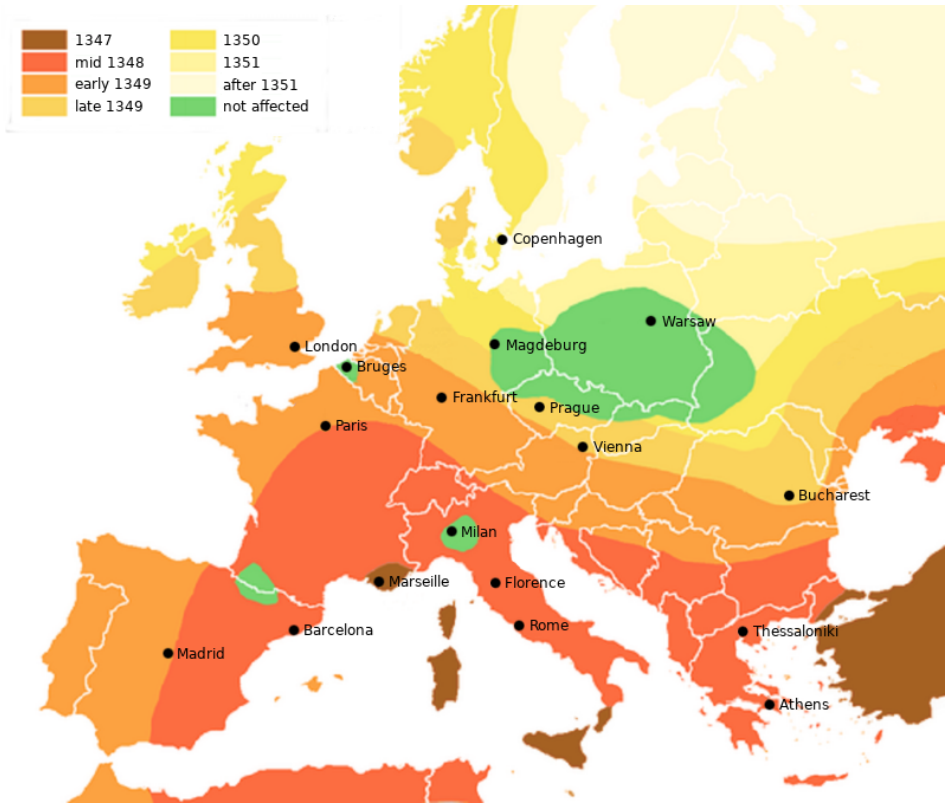

Figure 1.3: **Spread of the bubonic plague in Europe.** Merchant ships fleeing the Crimean port city of Kaffa transported the disease to Constantinople, Venice, Messina, Genoa, and Marseille. From there it slowly spread over land at an almost constant pace (see dates in legend). Green indicates areas with little or no incidence; white lines correspond to contemporary borders. Cities are included for reference. Image derived from [181].

the population in order to eradicate measles [4]. Similarly, random immunization strategies for computer viruses would require to install the appropriate antivirus software on more than 99% of devices [136]. On the other hand, only a small number of nodes would need to be inoculated if we choose those with the highest number of connections [135, 57]. Unfortunately, this targeted strategy requires a detailed map of the interactions, which we often lack. An alternative is acquaintance immunization, where we start at a random node and proceed iteratively by randomly selecting one of their neighbors [42]; given

the topological properties of the contact network we only need a few steps to reach a highly connected individual. By replicating this procedure in multiple parts of the network, eradication can be achieved with an immunization rate as low as 30%.

Killing an estimated 200 million people world-wide, the Black Death is the deadliest pandemic recorded in human history [54]. People in the 14th century traveled by land and water, causing a slow, wave-like propagation of the bubonic plague (see Fig. 1.3). In our day and age, airplanes take us to the other side of the Earth in no time, and diseases fly with us [44, 43]. The potential dangers derived from an evermore globalized world materialized during the 2003 SARS epidemic. Being the first outbreak to be monitored live by the scientific community, thorough analysis of the disease dynamics and spreading patterns has demonstrated the crucial role played by human mobility (see Fig. 1.4). For example, 144 of Singapore's 206 cases (roughly 70%) were traced back to the wanderings of only four superspreaders [112, 125].

Luckily, the widespread use of digital technologies has enabled the acquisition of detailed, multi-scale mobility data. Combined with the theoretical advances of network epidemiology, this has lead to the development of powerful predictive tools such as the global epidemic and mobility (GLEAM) project [8, 9]. This team of scientists was successful in forecasting the peak of the 2009 swine flu pandemic—two months in advance [10, 159]. Additionally, these comprehensive models allow to accurately gauge and quantify preventive interventions such as travel bans. For example, the 40% drop in air traffic to and from Mexico delayed the global spread of the swine flu by only 2 days. Moreover, if restrictions had reached 90% the outbreak would have simply been shifted by 2 weeks [7]. Although still in its infancy, predictive epidemic modeling has consistently proven its potential[7] and is gradually becoming an indispensable tool to inform and shape public health policies.

1.4 Spreading beyond infectious diseases

As I mentioned in passing in the previous section, epidemic modeling can be straightforwardly applied to the propagation of digital viruses in computers and mobile devices [170, 180]. Nonetheless, the framework can be adapted

[7] Some examples are the 2012–2014 MERS [141] and 2015–2016 Zika [182] outbreaks, or the on-going COVID-19 pandemic [33].

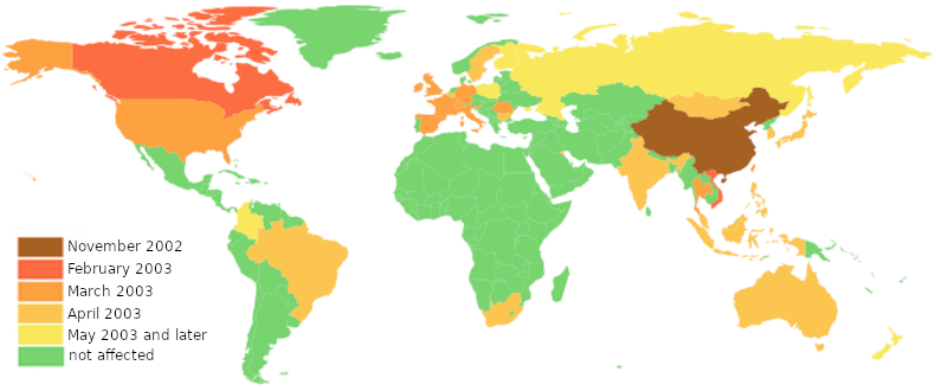

Figure 1.4: **Worldwide spread of the 2003 SARS epidemic.** The outbreak was first identified in mid November 2002 in Foshan, China; the disease had been exported to Vietnam and Canada by the end of February 2003. A few months later, cases had been confirmed in Brazil, Colombia, South Africa, United States, and various countries in Asia, Europe, and Oceania (see dates in legend). Green indicates countries that were not affected. Based on data from [56].

to a variety of "pathogens". Already in the 1960s various studies explored the mapping from diseases to information and rumors [79, 50]. In recent years, this approach has been redirected toward the spread of true and fake news [94, 167] and the circulation of memes and viral content on social media [5, 174]. Another relevant topic is the diffusion of social, cultural, and technological innovations. Examples include the uptake of menstrual cups [130], the adoption of VoIP services [96], and the dissemination of scientific advances [12, 55]. Last but not least, spreading phenomena also occurr in sociotechnical systems. Typical cases are routing and congestion in communication networks [126], and the spread of cascading failures in power networks [145].

Albeit their similarities, biological and social contagions are fundamentally distinct [30, 29]. While repeated contact with an infected individual can increase the likelihood of catching the disease, this is not the case when we think about the adoption of a novel technology. For instance, I would never buy a fax machine if only one of my friends owned one —even if they insisted on repeated occasions. However, if my whole group of friends switched to faxing I would need to buy one in order to stay in touch. Therefore we distin-

guish simple contagion, for which a single contact may suffice, from complex contagion, which requires reinforcement from distinct contacts.[8]

These two contagion mechanisms interact very differently with the mesoscale structure of the underlying network. Real networks are organized in communities; roughly speaking, nodes are more densly linked within their community than outside of it [73]. On one hand, links between communities (weak ties) allow simple contagions to permeate the network more rapidly [82, 127]. On the other, the existence of links within a community (strong ties) facilitates social reinforcement, as required by complex contagion [30, 20, 174]. Combining spreading mechanisms with network topologies offers a virtually endless array of possibilities, as reflected by the vast amount of modeling schemes that have appeared in the last decade (see [26, 84] for examples).

Earlier I described the diffusion of news, information, and ideas, but I purposely omitted the topic of opinion formation. For example, imagine that you and I have opposing opinions about a particular issue. Through dialog and discussion, each of us can cause the other to switch their opinion; i.e., both you and I can transmit our opinion. However, this is not the case for infections. While an infected individual is capable of transmitting the pathogen to its contacts, a healthy individual is unable to spread antibodies that can heal the ill. From a modeling point of view, opinions are symmetric and contagious diseases are not. This fundamental difference warranted the development of a parallel field of spreading phenomena, generically identified as opinion dynamics [26]. One of the most noteworthy contributions is the voter model, which describes the formation of opinions by random imitation [40, 88]. Recently, this simple mechanism was used to explain the statistical fluctuations observed in 1980–2012 U.S. presidential elections [70].

Unfortunately, the simplicity of mathematical models is often overthrown by the complexities of human behavior. In 2016, 74% of world-wide deaths were due to noncommunicable diseases (NCDs); i.e., pathologies that are not transmitted through an infectious agent [177]. That same year, the lead cause of death was cardiavascular disease, accounting for a staggering 27% of global deaths. This and other NCDs are associated to unhealthy practices such as smoking, excessive alcohol consumption, or lack of exercise. These risk factors possess a high social component: people smoke and drink more abundantly at social gatherings, and physical activity is often performed in

[8] Interestingly, some memes spread as simple pathogens while others are governed by the rules of complex contagion [174].

groups. Research has shown that bad health attitudes such as obesity [37], smoking [38], and substance abuse [39] are transmitted as a complex contagion and facilitate contracting NCDs. Additionally, the coupling between opinion dynamics and disease propagation is very relevant in the context of social awareness. For instance, the dissemination of prophylactic measures for preventing HIV [41], initiatives to quite smoking [122], or mass vaccination campaigns [81] are heavily reliant on their acceptance by the general public; resistance by rebel factions (e.g., anti-vaccination movements) can cause tremendous harm in terms of public health [148, 25]. Finally, studies have shown competitive dynamics between positive (jogging) and negative (smoking) health practices [104]. Altogether, these advances pave the way toward a framework of ecological interactions among lifestyle choices, health attitudes, and biological pathogens.

1.5 The assumption of Markovianity

The evolution of epidemic outbreaks is highly nondeterministic; therefore, their randomness can be adequately modeled via stochastic processes. The Markov property requires that the conditional probability of future states, conditioned on both past and present states, depends only on the present state; the sequence of preceding events is irrelevant. Stochastic processes that satisfy the Markov property are called Markovian or, in less technical jargon, mermoryless; mathematically, they are characterized by exponential interevent time distributions [48, 83].

A widespread approach when modeling spreading phenomena is to assume Markovian dynamics; nevertheless, empirical observations contrast starkly with this assumption. The clearest evidence is found for infectious periods, i.e., the lag between infection and recovery: instead of being exponentially distributed, recovery times typically follow a bell curve and have a well-defined average and bounded spread[9] [123]. Aditionally, nonexponential incubation periods[10] have been measured for HIV [14], Ebola [36], and foot-and-mouth disease and smallpox [157]. On the other hand, social contagion is strongly influenced by the fact that human activity patterns are predominantly bursty, showing stretches of high activity separated by long periods of inactivity [168,

[9]For example, 2–7 days in the case of dengue fever [176].

[10]The lag between being exposed and being infectious.

169]. Mathematically, this translates into heavy-tailed interevent time distributions,[11] which have been found in email responses, online gaming sessions, and financial transactions [97].

Although acknowledging the blatant discordance between model and data, most research efforts continue to apply the Markovian assumption. The reason for this apparent contradiction is simple: non-Markovian dynamics are much harder to tackle; their analytic tractability is low, and their computational complexity is high. Recent advances in terms of processing capacity have encouraged some researchers to surmount this hindrance. These early explorations have yielded promising results and lead the way toward a scarcely explored but potentially transformative area of epidemic modeling.

1.6 Thesis outline

Roughly speaking, the forefront of research in epidemic spreading can be divided in three areas. In terms of modeling transmission mechanisms, the current focus is on cooperation and competition between pathogens [31, 140, 90]. These ecological interactions are also of prominent interest in social contagion [121, 93] and coupled opinion-disease dynamics [75, 161]. Regarding the topological properties of the underlying contact structures, attention is distributed among embeddings and metric spaces [22, 129, 76], multilayer [53, 51, 138] and temporal networks [158, 162, 163, 140, 179], and descriptions that go beyond pairwise interactions [91, 11]. Finally, concerning the technical details of the temporal dynamics, many efforts are directed toward overcoming the assumption of Markovianity. This can be achieved by introducing nonexponential recoveries [103, 52, 109] or infections [165, 155, 110, 164], or both [102, 151, 68]. Alternatively, memory effects can be included indirectly. For instance, through two-step infection processes [35, 34], time-varying transmission probabilities [59, 113, 171, 183], or non-Markovian activation times in temporal networks [158, 179].

This thesis contributes to the examination of memory effects in spreading phenomena. However, the premise is to deviate from the typical framework of edge-based transmissions and recast the infection process as a node-centric mechanism. In short, the memory-induced complex contagion model describes individuals that aggregate past exposures to multiple infectious sources.

[11] By definition, heavy-tailed distributions are not well described by an exponential.

A notion of social reinforcement/inhibition arises organically, and the concepts of non-Markovian dynamics and complex contagion become intrinsically coupled. This combined approach could be particularly suitable to describe ecological interactions between biological and social pathogens. Notwithstanding, the motivation behind this research is of a more theoretic nature. Besides exploring potentially novel phenomenology, this work also challenges and validates the robustness of established modeling assumptions.

The focus of the thesis is the systematic analysis of the memory-induced complex contagion infection mechanism and its interaction with other components and properties of various spreading phenomena. I begin in Chapter 2 with the SI model, the simplest description of epidemic spreading, where the only possible transition is from healthy to infected. Afterward, in Chapter 3 agents are allowed to recover (SIR model), and I include reinfections (SIS model) in Chapter 4. Throughout the thesis, I combine analytic approximations with extensive simulations. Chapter 5 is dedicated to studying the interplay with nonhomogeneous network topologies, and in Chapter 6 I explore the effects of the memory-induced complex contagion mechanism in the voter model, a paradigmatic description of opinion dynamics. Chapter 7 investigates the merits and shortcomings of epidemic modeling by analyzing an empirical dataset of airport delays. Finally, Chapter 8 summarizes the results and comments on potential ways forward. Regarding additional material, mathematical derivations are detailed in Appendix A, and Appendix B provides notes on the generation of synthetic networks. Furthermore, Appendix C outlines various computational implementations, and supplementary figures are included in Appendix D.

As I mentioned earlier, epidemic-like models are employed for a variety of dynamics, such as opinion formation, rumor spreading, and innovation adoption. Although I use the original disease-specific terminology throughout this work, the scope and applicability of the analysis extends to all these fields.

Chapter 8
Conclusions

8.1 Background

Epidemic modeling has proven to be a powerful tool for the study of contagion processes in biological, social, and technological systems. Variations of the benchmark susceptible-infected-recovered (SIR) and susceptible-infected-susceptible (SIS) models have been applied to study the spreadof diseases, opinions, information, rumors, and innovation. Various adaptations and extensions—some very simple and clean but others rather elaborate and complicated—have provided valuable insights into the nature of spreading mechanisms, the dynamics of outbreaks, and the viability of containment protocols. More recently, the inclusion of real-life contact networks and mobility patterns has led to astonishingly accurate results, prompting the use of epidemic models as real-time predictive tools.

For years, the go-to modeling scheme for contact-based contagion assumed Markov processes and isolated transmissions. The Markov property translates into exponentially distributed interevent times and renders the system memoryless—its evolution is independent of its history. Although this approximation is most often justified because of the reduced mathematical (and computational) tractability of nonexponential distribution, its inappropriateness is widely supported by empirical evidence. Prototypical examples include the peaked distributions of infection periods of numerous diseases and the bursty human activity patterns in social networks, well described by heavy-tailed distributions.

On the other hand, assuming isolated transmissions leads to infection channels that are not influenced by their local environment; consequently, the infection likelihood can be written as the sum of statistically independent exposures. Nevertheless, experimental observations support the existence of more complex, nondyadic mechanisms in a variety of scenarios. The most cited example is social contagion, but evidence has also been found in fungal and bacterial pathogen colonization and—paradoxically—the proliferation of

noncommunicable diseases. Clearly, the time has come to overcome these outdated modeling limitations.

In recent years, an important amount of research has ventured into the scarcely explored area of memoryful and complex epidemics. Regarding memory, a wide array of modifications has been analyzed, such as two-step infection models, nonexponential distributions, and time-varying transmission probabilities. Similarly, a plethora of complex contagion schemes has been proposed to mediate the assumption of independent transmissions; examples include correlated, nonlinear transmission channels, extended neighborhood effects, and deterministic threshold models. So far, not many endeavors have focused on tackling both modeling assumptions simultaneously, and little is known about how these two features interact. Such a combined approach is of particular interest for contagion phenomena that include a social component, such as awareness and vaccination campaigns or the spread of noncommunicable diseases (e.g., obesity, anxiety, and substance abuse).

My thesis contributes to the analysis of the synergies between memory and nondyadic interactions in spreading processes. I develop a model that bypasses the usual framework of edge-based transmissions and recasts the infection process as a node-centric mechanism. Specifically, the memory-induced complex contagion (micc) model describes diseased nodes as infectious sources that spread doses of pathogen to their entire neighborhood. Healthy nodes, on the other hand, gather the toxins from all their neighbors—present and past—and become infected given the total viral load they have amassed. As a consequence of this infection mechanism, a notion of social reinforcement/inhibition arises organically, and the concepts of non-Markovian dynamics and complex contagion become intrinsically coupled.

The micc model provides a generic description of memoryful and complex spreading phenomena. However, in order to actually investigate its properties, one must particularize two ingredients: the infection probability density and the accumulation and decay of the viral load. I have chosen to describe the former by the versatile Weibull function, characterized by its shape parameter α. For starters, $\alpha = 1$ recovers the exponential distribution of the standard Markovian formulation, which enables the comparison to a well-known benchmark. Other values of the shape parameter interpolate between power law–like fat-tailed distributions ($\alpha < 1$) and bell curve–like peaked distributions ($\alpha > 1$). Regarding the viral load, I have chosen a simple linear accumulation and an exponential decay, with characteristic time ζ. This choice allows me to inter-

polate between a short-term memory mode ($\zeta = 0$) and a long-term memory mode ($\zeta \to \infty$). Furthermore, I have limited the analysis to the basic epidemic models (SI, SIR, and SIS) and a short exploration of the voter model. Finally, when the prescription of recoveries is required (SIR and SIS models), I maintain the exponential distribution of the standard Markovian formulation; this choice allows me to focus unequivocally on the effects of the memory-induced complex contagion infection mechanism.

8.2 Results

The SI model only allows the transition between the susceptible and infected states, thus any healthy population will eventually become completely diseased. The temporal dynamics of this evolution varies greatly in the miccSI model, where agents are equipped with the memory-induced complex contagion infection mechanism. Fat-tailed distributions describe nodes that are "impulsive"—reactionary, even—and require very little viral load to become infected. The outbreaks initially grow very rapidly but then decelerate and converge slowly toward the fully infected state. With peaked infection distributions, on the other hand, nodes are more "meditative" and become infected with a higher value of the viral load. In this case, outbreaks present a gradual initial growth and afterward saturate very quickly; additionally, they are more uniform and show a notably regular pattern. Although somewhat expected, these results lay the foundations for upcoming—more spectacular—features.

In the SIR model, infected nodes may overcome their ailment, transitioning to a final recovered compartment. On a whole, the system evolves towards a terminal state that is characterized by the absence of infected agents. Depending on the parameters of the pathogen, encapsulated via the effective spreading ratio, the outbreaks can be either very short-lived or develop into havoc-wreaking disruptions. These two phases are separated by a continuous phase transition at the epidemic threshold.

The short-term memory mode of the miccSIR model causes a displacement of the epidemic threshold toward higher values as the shape parameter of the infection probability grows. Thus more reactive agents (smaller values of the shape parameter) enable the existence of sustained outbreaks that are caused by weaker pathogens (lower values of the spreading ratio). In accordance with our findings for the miccSI model, meditative individuals re-

quire a longer build-up but cause a more concentrated explosive effect. On the contrary, the outbreaks for fat-tailed infection distributions are slightly less virulent and affect a smaller fraction of the population if agents possess an additional long-term memory. This phenomenon reveals the crucial role played by dormant nodes, which hinder the infection chains when the outbreak revisits previously afflicted areas of the network. In contrast, with peaked infection distributions their previously accumulated viral load facilitates the expansion of the outbreak, which becomes more violent.

Things get more interesting with the introduction of reinfections. In the SIS model, infected nodes that recover become once again susceptible and may reinfect at a later stage. This feature results in a nontrivial active steady state, where nodes repeatedly infect and recover, but overall the fraction of infected nodes remains more or less constant; nonetheless, less virulent outbreaks are still eradicated very quickly. With the conventional assumptions of Markovianity and simple contagion, the phase transition between the healthy and endemic phases is typically continuous.

The miccSIS model with peaked infection distributions can be studied with the use of an adequate analytical framework—and by applying (many) suitable approximations. When agents are endowed solely with a short-term memory, the system displays a tricritical point where the customary second-order phase transition gives way to a discontinuous transition. Then the customary healthy and endemic phases are separated by a bistable region where two stable attractors coexist. Additionally, as agents increase their memory span, this intermediate region already emerges for lower values of the shape parameter α. Notice that bistability in SIS-like models is usually obtained by explicitly prescribing some sort of coordination—either cooperative or competitive—between neighboring nodes. However, here it arises organically from the memory-induced complex contagion infection mechanism.

Even though this analytical framework paints an exquisite qualitative picture, stochastic simulations are required to fill in the details. In particular, they reveal that the width of the previously discussed bistable region shrinks as the memory span of individuals increases. They are also necessary to analyze the miccSIS model with fat-tailed infection distributions. In this case, if agents only have short-term memory, the system behaves very similar to the standard Markovian model; conversely, the healthy and endemic phases are separated by an excitable region when agents possess a long-term memory mode. In this intermediate phase, outbreaks initially expand rapidly through the system but

afterward slow down and are eventually eradicated, surprisingly resembling a SIR-like curve.

Concluding, if nodes are equipped with a long-term memory mode, the system experiences a global memory loss. Specifically, agents that have an infinite memory span behave on aggregate as if they had no memory at all. Nevertheless, important differences with respect to the Markovian model are revealed when looking at the dynamical properties of the outbreaks. The three order parameters—average coverage, endemic probability, and late-time prevalence—are identical if agents are memoryless; when the long-term memory mode is activated, this delicate balance is broken and a second critical point emerges. In particular, the system first transitions from the healthy phase to an either bistable or excitable intermediate region, followed by a hybrid transition to the endemic phase.

In terms of phenomenology, the standard SIS model in unstructured networks yields rather unexciting results, but more interesting properties arise when a nontrivial contact structure is added. The miccSIS model, on the other hand, shows a plethora of features in degree-regular networks, revealing that network structure is not an essential ingredient in order to obtain a richer phenomenology. Unexpectedly, this variety is reduced with the introduction of nonhomogeneous networks. Particularly, clustering mitigates the impact of the short-term memory mode while node heterogeneity partially suppresses the effects of the long-term memory mode—the excitable region for fat-tailed infection distributions survives, but with peaked distributions the intermediate bistable phase vanishes.

Finally, the voter model—a paradigmatic description of opinion dynamics—can be recast as a spreading phenomenon with symmetric transitions between the two opinion states. In its Markovian formulation, the population becomes trapped in a transient quasi-stationary state but eventually evolves toward one of the two consensus states. When introducing the memory-induced complex contagion “infection” mechanism, it is important to distinguish between the processes of persuasion and reaffirmation. In the former, a node is “convinced” to change its opinion, while the latter corresponds to a node increasing their confidence in their belief. It turns out that the specific numerical values of the reaffirmation process are irrelevant—the dynamics is aptly described in terms of the persuasion process. Depending on the response of an individual to new stimuli (either meditative or impulsive), the system reaches consensus in a very short time or becomes trapped in a dynamic equilibrium.

8.3 Outlook

All in all, my analysis of the stylized yet feature-rich micc mechanism evidences a crucial role of non-Markovianity in the spread of epidemic outbreaks. In particular, agents' memory span dramatically alters the effect and impact of newly introduced pathogens. Currently, this topic is a very active field of epidemic modeling, with applications that range from the appearance of exotic diseases to the dissemination of fake news on social media.

However, I believe it is important to stress that the inclusion of memory is in no way imperative. Most on the interesting phenomenology has already been found in Markovian models, albeit via elaborate schemes or intricate network topologies. This conformity evidences the robustness of previous results and supports the appropriateness of their employment in many scenarios. Notwithstanding, memoryful epidemics add a nuanced layer of complexity that can lead to novel behaviors. Even so, to the extend of my knowledge, the only feature that has not been widely reported in previous studies is the appearance of excitable outbreaks in SIS-like models.

Inasmuch as time and space are limited, I had to exclude some topics from this work. Personally, I am intrigued by the perfect overlap of the endemic probability and the late-time prevalence in the Markovian SIS model, and I think that elucidating this apparent coincidence is definitely worth the while. Moreover, the development of a adequate analytical framework to study the temporal properties of outbreaks in the miccSIS model could shed (a lot of) light on the peculiar excitable intermediate region that appears with fat-tailed infection distributions. In addition, the fact that the results for the voter model are (practically) independent of the reaffirmation parameters enables the possibility of studying the problem with an analytical approach. Finally, an exhaustive exploration is required to find suitable data sets and provide supplementary empirical analyses, without excluding the possibility of designing and executing a controlled experiment.

I finish with the usual list of potential extensions and ways forward. The analysis of additional network characteristics—such as degree correlations or meso-scale structures—would supply renewed insights on the relevance of microscopic mechanisms and topological aspects in contagion processes. Another interesting variation consists in using mixed population groups, which would allow us to gauge the effect of demographic features on the dynamical and late-time properties of the system. Last but not least, we could explore

other infection probability distributions or include realistic nonexponential recoveries. All in the hope of increasing our understanding of epidemic spreading and improving our ability to shield the world from catastrophe.