1

Introduction

In this chapter we provide a brief introduction to the remainder of the book.

The uninitiated may require a broader background to the topic of complex networks. Rather than overburden out current presentation, we refer interested readers to some good introductory books and papers [1–14] for more background information on complex networks and network science.

1.1 Motivation and background

Throughout history, infectious diseases have always been a serious threat to human health and life. It is therefore of great practical significance to study epidemic transmission and then to take effective measures to prevent and control them. Toward this end, much research has fallen within the field of epidemiology, which uses mathematical modeling as an analytical approach. Traditionally, epidemic models were based on uniformly mixing populations, which are unable to characterize epidemic propagation in large-scale social contact networks with disparate heterogeneity. However, the fact that most population-based epidemics spread through physical interactions raises contact networks as a basic tool for mathematical description of contagion dynamics. In the last decade, spurred by the availability of real data and the maturation of network theory, there has been a burst of research on network-based epidemic transmission [15–26].

Beyond ordinary infection diseases, recurring computer virus attacks (as well as computer worms and other malware vectors) on the Internet also illustrate the urgent need for knowledge about modeling, analysis and control of epidemic dynamics on complex networks.

The World Health Organization (WHO) announced in 2012 [27] that some time in the next couple of years Guinea worm will become only the second known

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disease, after smallpox, to be completely eradicated. The disease has been known to afflict humans for thousands of years. Unlike other diseases, the campaign against Guinea worm has focused not on developing a cure, but on educating people about how the disease spreads and how infestation can be prevented.

While Guinea worm may be almost eradicated, people worry that several other infectious diseases are re-emerging [28]: Tuberculosis: poor-quality diagnoses, treatment, and medicines contributed to the rise of 8.7 million new cases in 2011, particularly in Eastern Europe, India, China, and parts of Africa; Leprosy: 219 000 new cases were reported last year, mostly in Africa and Asia; and, Bubonic plague: the same Black Death that wiped out millions in Europe has cropped up in the United States, and between 1000 and 2000 cases of plague are still reported worldwide each year.

Information spread can also appear to propagate like a virus. In 2011, in the wake of the Fukushima nuclear disaster, rumors spread throughout China that iodized table salt could be used to help prevent radiation sickness. The subsequent rumors and panic-buying lead to a shortage of salt in both China and neighboring territories. Organized, or coherent, spread of rumors combined with lack of judgement on the part of public news agencies, led to official information sources appearing to lose credibility. Conversely, institutionalized and individual cyber-attacks have gained recent prominence. Naturally, network structure and propagation dynamics become key features in controlling and understanding such mechanisms.

Epidemics on networks is a rapidly expanding field of considerable contemporary interest to researchers in a broad spectrum of areas including applied mathematics, probability, physics, biology, and so on. There is a need for a book at an introductory research level that gives a balanced overview of the current state-of-the-art in this area. Concerning the advance of techniques, it has become clear that more fundamental knowledge is needed within the context of mathematical and numerical studies on how epidemic dynamical networks can be modeled, analyzed, and controlled. This book, based on existing research, aims to address this need. We discuss in detail different epidemic models on complex networks and a variety of applicable mathematical techniques. Using mean-field approximation we provide a detailed analysis of epidemic dynamics, the theory of complex networks, and qualitative theory and stability methods of ordinary differential equations. The current volume serves to present recent progress in the investigation of these important topics and some related topics.

1.2 A brief history of mathematical epidemiology

In this section we give a brief, largely descriptive, history of mathematical epidemiology. Many undergraduate texts provide extensive coverage of the details.

Epidemiological modeling is a large subject in mathematical biology, a single short section about its brief history is of course insufficient to give a complete picture of the field. So we here refer the readers to a book and a review article [29, 30] for more details.

1.2.1 Compartmental modeling

The recorded earliest mathematical epidemic model dates from the eighteenth century. In 1760, by using ordinary differential equations, Daniel Bernoulli studied smallpox vaccination, and gave the Bernoulli equations [31]. Bernoulli's results showed that universal inoculation against smallpox could increase life expectancy.

Later in 1889, En'ko built the chain-binomial model for measles and scarlet fever. To understand the recurring epidemics of measles, in 1906, Hamer gave a discrete mathematical model, and presented the mass-action principle [32, 33]. In 1911, Ronald Ross established and studied the malaria transmission model, and gave the standard incidence ratio and the basic reproduction number (sometimes called the basic reproductive number, basic reproductive rate, basic reproductive ratio, and denoted as R_0). In epidemiology, the basic reproduction number of an infection is the number of cases that one case generates on average over the course of its infectious period. The roots of the concept can be traced through the work of Alfred Lotka, Ronald Ross, and others, but its first modern application in epidemiology was by George MacDonald in 1952, who constructed population models of the spread of malaria. In 1926, by studying the spreading patterns of the Black Death in 1665–1666 and the plague in 1906, A. G. McKendrick and W. O. Kermack formulated a simple deterministic model that was the modern mathematical epidemic model–the SIR compartmental model, which was successful in predicting the behavior of outbreaks in many recorded epidemics. Based on this model, they presented the threshold theory to determine eventual endemic or disease-free status of a disease. In 1949, Bartlett's measles model [34] was built.

In a compartmental model of infectious disease, individuals are divided into several classes, for example, the compartments: susceptible (S), latent (E), infected (I), vaccinated (V), and/or removed (recovered) (R). The E status is also used to represent the stage when individuals have been *exposed* to a disease and are therefore infected, but not yet infectious. Depending on the propagation process, we can build various compartmental models by combining these different classes (or creating new ones). Examples of such include SI, SIS, SIR, SIRS, SEI, SEIS, SEIR, SEIRS, SIV, and so on. The sequence in which classes are listed typically corresponds to the infection pathway. In a compartmental SIS model, say, each individual can be in two discrete states, either susceptible to or infected by the virus particle, and susceptible individuals (S) may become infected (I) owing to contact wth other infected individuals, and infected individuals also may recover to susceptible state (S), with a certain recovery rate. Apart from percolation models [22], this book will discuss most such models.

As George E. P. Box said, "Essentially, all models are wrong, but some are useful." Certainly, all the models we include here are wrong as they are mean-field approximations for the spreading of real epidemic diseases. Nonetheless, this is a useful approximation and many of these models have helped people to plan effective actions against various serious epidemic diseases.

After building a model, we need then to study it by qualitative, analytical, experimental (including numerical), and theoretical methods. Research methods for modern epidemic dynamics models can be summarized briefly as follows.

For a deterministic compartmental model, that is built based on uniform mixture hypothesis, we may study it by either theory or methods [35, 36] developed in ordinary differential equations, partial differential equations, delay differential equations, impulsive equations, and difference equations. The trends in these research areas are for higher model dimension and deeper and more refined analysis.

For a stochastic model, we may apply stochastic processes and stochastic dynamical methods. In contrast to the above, the trends with these models are toward specific diseases and toward deterministic and stochastic mixed models.

1.2.2 Epidemic modeling on complex networks

Many epidemic systems can be represented as a graph or network, where nodes stand for individuals and a link connects a pair of nodes – indicating interaction between individuals.

Patterns of this type can quickly become very complex and it is usually not sufficient to describe the connectivity between two nodes as uniform or homogenous. Heterogenous contact rates reflect that the node degree k, the number of contacts with other individuals for a given individual, are not uniform. Instead, such heterogeneity can be represented by the degree distribution p_k. Real networks underlying disease transmission have been represented not only by conventional graphs such as lattices, regular trees, or classical random graphs, but also by complex networks, such as the WS (Watts–Strongatz) small-world networks [14] or the BA (Barabási–Albert) scale-free networks [3].

Traditional epidemic models are useful for uniformly mixing populations with homogenous contacts. However, these are unable to characterize epidemic propagation in large-scale social contact networks with distinct heterogeneities. As is well known, all models are inaccurate simplifications of nature. By taking the heterogeneity into account, complex-network modeling of epidemics provides a somewhat more accurate viewpoint. A compartmental model based on uniform mixing can be viewed as a networked model with Delta degree distribution, an approximation to the Poissonian or power-law degree distribution. Conversely, if we take the degree distribution as the Delta distribution, a networked model will then become a uniformly mixing compartmental model.

Networked epidemic models are typically considered with networked mean-field theory, which was pioneered by two physicists, Pastor-Satorras and Vespignan [23, 37, 38], although some earlier results were already reported in a mathematical textbook [29]. The basic idea is, according to traditional compartment models, to

classify all nodes on the network by disease states and, based on this, subdivide nodes according to their degrees, such that nodes with the same degree belong to one class, which has the same dynamics patterns. The core here is the dynamical behavior for the nodes with the same degree can be represented by the average behavior. Based on such contact networks, many epidemic models, such as SI [39], SIS [23] and SIR [40], have been investigated.

To understand the mechanism of disease spreading and other similar processes, such as rumors spreading [20], networks of movie actor collaboration [3, 13] and science collaboration [41], WWW [42, 43], and the Internet [44], it is of great significance to inspect the effect of complex networks' features.

In China alone, some early research on networked epidemic transmission models was carried out by many researchers [4, 45–57]. Some results on propagation and immunization of infection on general networks with both homogenous and heterogenous components, and influence of dynamical condensation on epidemic spreading in scale-free networks [49, 52, 53, 58, 59, 60], global stability analysis of networked epidemic models [61–67] are obtained, to mention only a few.

After building a mathematical model, we may then apply the following cycle: run algorithms to compute with the model; analyze errors where results differ from data; create modifications of the mathematical model; (develop pure mathematics theory that is perhaps increasingly irrelevant); analyze improved model, and so on… In this book, however, we will concentrate on theoretical analysis of the models we build. In Chapter 4 we consider the problem of comparing these models with the real data.

Apart from the difficulty caused by very high dimension in networked epidemic models, some other problems for these models are that they did not properly take the population dynamics into account. These factors include the impact of birth, death, and migration on the network topology and the spreading patterns of diseases [23, 29, 30, 37, 38, 68–70]. Finally, networked models based on pair approximation [71] seem a further step to make networked models more accurate.

1.3 Organization of the book

This book consists of 11 chapters. Chapter 1 gives an introduction, motivation, and background for this work. In this chapter we present a brief history of mathematical epidemiology, and epidemic modeling on complex networks. Chapter 2 discusses different epidemic models on complex networks, such as staged progression models, models with population mobility, or effective contacts, models on weighted networks, or directed networks, discrete epidemic models, and stochastic SIRS epidemic models. Chapter 3 details some threshold analyses by the direct method and by using spectral properties. Chapter 4 analyzes networked models for SARS and H1N1, and sets up plausible models for propagation of the SARS virus and avian influenza outbreaks. This provides a reality-check for the otherwise abstract mathematical models of this text. We show that such models do match well the reality of current emerging diseases. Chapter 5 deals

with various infectivity functions, including constant, piecewise-linear, saturated, and nonlinear cases. Chapter 6 concentrates on the case for SIS models with an infective medium; Chapter 7 discusses the roles of human awareness in epidemic control; Chapter 8 reveals adaptive mechanism between dynamics and epidemics; and Chapter 9 summarizes methods for epidemic control and different immunization strategies. Finally, Chapter 10 demonstrates global stability analysis; and Chapter 11 investigates information diffusion and pathogen propagation on complex networks, and discusses some differences between information and epidemic spreading.

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