

Epidemics addressed in several network situations

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Abstract—The study of epidemics has been widely used to look into the behavior of diseases among humans but also the behavior of viruses in computer networks. Many studies have shown that the behavior of such diseases differs when it occurs in different network situations such as interconnected networks and multiplex networks. Also heterogeneity in infection rates between nodes can play a major part in the behavior of a disease. This paper serves as an introduction to the field of epidemics, where basic epidemic models such as SIS and SIR are discussed. Besides this, we look into some results of studies in epidemics in several network models. In this way, this introduction creates a broad overview of the basics of epidemics, creating a good starting point for a researcher investigating this field.

I. INTRODUCTION

Epidemiology is the study towards diseases with the goal to predict, understand and develop strategies to control the spread of infectious diseases [1]. The oldest application of this study is in the field of health care, but nowadays the behavior of epidemics can also be applied to viruses in computer networks. One of the reasons why this is so, is because epidemiology is far older than computers are. [2] states that one of the first classical papers in epidemiology is that of Kermack and McKendrick [3], which originates from 1927 and introduced among other the SIR model. Computer science, in particular network science, began to play a prominent role in the study of epidemics from 2000. Before this, epidemic modeling hardly considered specific details of the underlying contact network. As will be clear from this paper, the underlying network can be of major influence on the behavior of an epidemic.

As an introduction to epidemics addressed in several network situations, we look into three cases where it is shown that the underlying network and its properties (behavior) has a great impact on the behavior of the epidemics.

First, where earlier studies assumed that all nodes of a complex network interact on a single network, the study of [4] looks into the behavior of epidemics on interconnected networks. Namely, societies are composed of many interconnected networks. For example, residents of two cities (two separate networks) can be connected to each other because they work in the other city. This results in the two separate networks to form one interconnected network. The study of [4] shows that the way how a disease spreads in such interconnected networks depends on how strongly these networks are coupled. For example in strongly coupled networks systems,

epidemics occur simultaneously across the entire system when having a critical infection strength.

Secondly, with the popularity of social media, researchers have looked into the influence of awareness and a mass media on the behavior of epidemics [5], [6]. In this study, one looks into multiplex networks, networks with different kind of nodes that are coupled together. Here, an information layer exist as a layer above the physical epidemics layer, representing the relations with people one regularly shares information with. The information exchange can make a individual aware of an disease and change the probability of infection. The addition of a mass media increases the probability of individuals getting aware.

Third, in ‘simple’ network models, infection rates, or possibility that one gets infected by a someone he had a link with, are constant and equal for each link. The notion of heterogeneous infection rates changes the behavior of an epidemic. [7] shows that in a network with heterogeneous infection rates there is a slow epidemic extinction due to the fact that people that highly interact with each other (thus have high infection rates among each other) keep the disease alive for a long time.

The studies introduced above where done using dynamics from different basic epidemic models, the ‘Susceptible-Infected-Susceptible’ and ‘Susceptible-Infected-Recovered’ model. We will start with an elaboration on these models at the beginning of this paper, because the understanding of these models is a requisite for starting research in the field of epidemics. Also the epidemic threshold will be introduced. After this, we will dig into some research results from [4], [6], [7]. Regarding these researches, the different network models that are used will be discussed to create a basis for the network science part.

For the interested readers, we provide more starting points to continue in this field or complete ones knowledge on the discussed topics.

II. EPIDEMIC MODELS

In epidemiology, two simple models form the basis on which many variants exist. These models are the ‘Susceptible-Infected-Recovered’ (SIR) and the ‘Susceptible-Infected-Susceptible’ (SIS) model. [1] Both models start with infected items that can infect their direct, healthy neighbors that are susceptible to the disease. In this section we elaborate on these

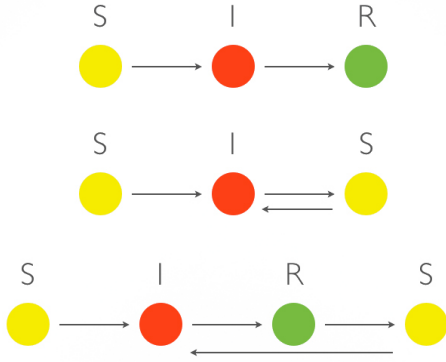


Figure 1: Graphical view of the SIR and SIS model.

models. An understanding of these models is a requisite for further understanding of the research results of the sections that follow.

A. The SIR-model

The SIR epidemic model is a well established model that is used since 1927 [3] to describe several diseases. In this model, each node has three possible states, namely susceptible (S), infected (I) and recovered (R) - as the name of the model suggests.

The state transitions can be seen in Fig. 1. Given an network, each node starts in state S except for one node which starts in state I being the first infected node. Nodes in state I infect their neighbors in state S with probability β at each time step, which cause these neighbors to enter S . How infected nodes reach R is described in different ways. One way is to say that an infected nodes needs to spend an recovery time t_r in state I [4]. In other models, infected nodes can spontaneously recover with probability μ [6]. This doesn't change the function of the model.

In the SIR model, it is assumed that when a node is recovered from the disease, it has become immune for another infection (of the same disease). Another name for the R state can therefor be 'Removed' [2], because the infected item that recovers can be removed from the population and from the infection process.

Two diseases that can be describes using the SIR model are measles and chickenpox.

B. The SIS-model

The SIS model is a adaption on the SIR model, where it is assumed that nodes do not acquire immunity after infection. The state transitions can be seen in Fig. 1.

As in the SIR model, a susceptible node can be infected by another node with probability β and will recover spontaneously (probability μ) or after spending the recovery time t_r , depending on which precise model of this kind is used.

The SIS model is a generalization of the SIRS model (also Fig. 1), where infected nodes first reach the recovered state before it will return susceptible again with a probability α . In the SIS model $\alpha = 1$, leading to a superfluous state R .

Shortly, the only difference between SIS and SIR models is the absence of R and the flow of recovered individuals to the susceptible stage [1].

The SIS model can be used to describe epidemics such as the flu among humans and malware in computer networks [2].

C. The Epidemic Threshold

There are different phases an epidemic can be in. A network stays completely disease-free as long as no node is infected. As soon as one node is infected, the disease has the possibility to spread, as long as the infected nodes infect another node before recovery, which happens after t_r (or spontaneously with probability μ). The transmissibility $T_\beta = 1 - (1 - \beta)^{t_r}$ is the probability that a node infects a neighbor before recovery, as an example. In both the SIS and SIR model, the spreading of the disease depends on the infection rate β ; the bigger β , the bigger the chance that other nodes get infected.

Besides the disease-free phase, an epidemic can be in the absorbing phase, where the disease dies out due to a low infection rate. On the other hand, a disease reaches its epidemic (active) phase when the infection rate reaches a certain critical value β_c , such that the disease will not die out anymore. In this case, a giant cluster will emerge, where the infection between two nodes is almost certain. Because of this giant cluster, the disease will always be present in the network.

Given the different phases, one can look at what happens with the average density of infected individuals ρ , used in [7]. Namely, in the absorbing phase ρ will decay to zero (disease-free) and in the active phase ρ will reach a stationary value that is larger than zero.

β_c is called the epidemic threshold. Many studies are concerned in finding this threshold in specific situations. The behavior of the disease itself can be described with several models. For example, [4] relies on the parameter κ (described in Section III-A) and [6] uses a Markov Chain approach. However, these models don't change the fact that there exists an epidemic threshold.

III. EPIDEMICS ON INTERCONNECTED NETWORKS

As a first example of epidemic studies looking at different network topologies, we look at epidemics on interconnected networks. To gain full understanding of the concept, we first discuss interconnected network systems itself before we look into the consequences this has on the behavior of the epidemic and the epidemic threshold.

In the research of [4], the SIS model is used.

A. Interconnected Networks

Interconnected networks can alternately be seen as interconnected communities within a single larger network: Take as example a collection of cities on a island. An structured view of a interconnected network is given in Fig. 2, having two networks, A and B . Besides the intranetwork links within these networks, nodes can have internetwork links connecting them to the other network. A node can only belong to one of the interconnected networks.

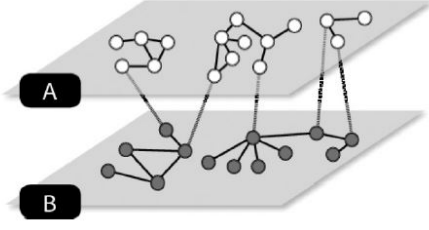


Figure 2: An interconnected network system, from [4].

B. Strongly and Weakly Coupled Networks

In [4], strongly coupled and weakly coupled interconnected networks are defined. Given k_A and k_B , the average degree of network A and B , the expected number of nearest neighbors that a node chosen by following an arbitrary link will have is defined as $\kappa = k^2/k$. Whether a network system is strongly coupled or weakly coupled is decided using this κ . In a strongly coupled network system κ_T (κ calculated over the entire coupled network system) will be greater than κ_B and κ_A (both calculated disregarding internetwork connections).

[4] defines a interaction strength $(k_{AB})_c$ that separates strongly coupled from weakly coupled networks as

$$(k_{AB})_c = \frac{\sqrt{2k_A k_B - (k_A)^2} - k_A}{2} \quad (1)$$

Given Eq. (1), one can see that having to identical intranetwork degrees leads to $(k_{AB})_c = 0$. This means that identical networks always form strongly coupled network systems. This formed network system will behave as a single network, where two halves are labeled A and B - which has no effect on the physical properties of the network.

For this reason, in the notion of interconnected networks, it is assumed that the average degree k_A and k_B of the networks A and B are not the same. In the remainder of this paper, following [4], we assume that $k_B > k_A$, thus that network B is the more intraconnected network.

C. Epidemics on interconnected networks

The effect on the spread of an epidemic depends on the parameters of the individual networks and their interconnections. Connecting one network to another can have a profound or small effect, which depends on whether this leads to a strongly or weakly coupled networks. [4] shows that in a strongly coupled network, all networks are simultaneously either disease free or part of an epidemic. In weakly coupled networks a ‘mixed’ phase can exist, where the disease is epidemic on one of the networks.

First we look into the strongly coupled case. Using the definition of the T_β in Section II-C and κ , the epidemic threshold of a single networks is

$$\beta_c(\kappa) = 1 - [1 - (\kappa - 1)^{-1}]^{1/t_r} \quad (2)$$

Given Eq. (2), it can be seen that for the strongly coupled case ($\kappa_T > \kappa_B$) the critical value $\beta_c(\kappa_T)$ is smaller than

both $\beta_c(\kappa_B)$ and $\beta_c(\kappa_A)$. This means that the disease spreads across the interconnected network system as it is a single network. In this case, internetwork connections will bring an epidemic into existence before any intranetwork connections can do so independently.

In weakly coupled systems, it follows using Eq. (1) and Eq. (2) that $\beta_c(\kappa_B) < \beta_c(\kappa_T) < \beta_c(\kappa_A)$. [4] shows that the network system will enter a ‘mixed’ epidemic phase when the β is above the individual epidemic threshold of network B , but not above the epidemic threshold of the entire system. In this case, an epidemic occurs in B , but the disease will not spread to more than isolated small clusters of A . In this mixed phase, the addition of interconnections between two networks is only affecting epidemic spreading on the network with the weaker intranetwork connections, with the epidemic on the other networks unchanged by the internetwork links.

As soon as β is increased to above $\beta_c(\kappa_T)$, network B becomes capable of spreading the disease to network A and both network will enter the epidemic phase.

IV. THE INFLUENCE OF AWARENESS

Now we look at the results on the behavior on epidemics when there is awareness involved. In the study of [6] a multiplex network with the presence of a mass media is introduced which is used to model the network when nodes can be aware of certain situations, which can lead to different infection rates. This research is a continuation on [5].

A. Multiplex Networks

A multiplex network can be seen as a layered simple network where nodes will be in different states at the same time, where particular states are ‘part’ of a different layer. All layers thus contain the same nodes, but different links.

The multiplex network as used in [6] is shown in Fig. 3. This system contains a information layer and epidemics layer.

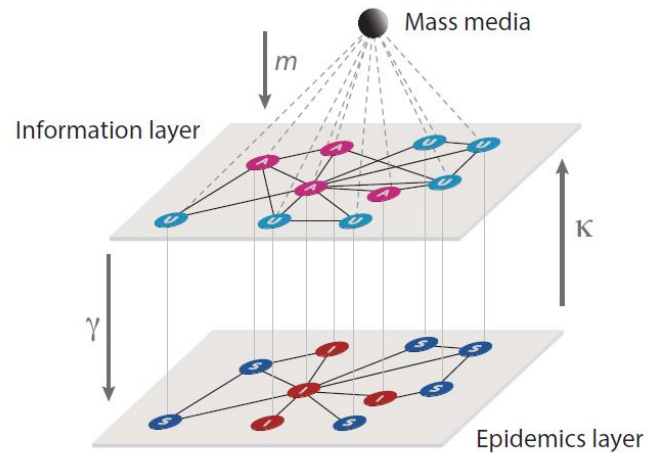


Figure 3: Example of a multiplex network, including the presence of a mass media, from [6].

Looking at simple epidemics, a disease such as the flu may spread in the network by physical contacts. Following the SIS-model this is represented in the epidemics layer. On the other hand, using all kind of devices such as mobile phones or computers, people can exchange information. The contacts people have are represented in the information layer. This layer follows a Unaware-Aware-Unaware model, which functions exactly as the SIS-model.

Fig. 3 also shows the relationships between the different layers. A node that is infected in the SIS layer will become Aware in the UAU layer with probability κ . This probability accounts for the possibility that the nodes may not know they are infected or may choose not to spread information about it. It is clear that this probability κ does not change the infection rate β in the information layer, because a node can only become Aware when it is already infected.

On the other hand, a node that is aware of the disease can take measures to prevent infection. The parameter γ regulates the probability of a node to get infected. With relation to the infection rate, take β^U as the probability of a unaware node to get infected, then $\beta^A = \gamma\beta^U$ as the probability of a aware node to get infected. From this follows that the fact that a node can be aware of the situation before it is infected can have a influence on the infection rate.

Both γ and κ are parameters between 0 and 1. It can be seen that $\gamma = 0$ represents total immunization. When $\gamma = 1$ and $\kappa = 0$ both interactions are disabled and the multiplex network will run both processes in single layer independent networks [6].

In [6] also the presence of a mass media is introduced. The mass media can bring every node with probability m in the aware state, independent of the communication links in the information layer. The mass media represents the fact that information can also have a global impact on the system.

B. Epidemics on multiplex networks

Using the multiplex network model, [6] shows that the immediacy of awareness κ has almost no effect on the dynamics, while the degree of immunization of aware nodes γ and the presence of the mass media (m) do change the critical aspects of the epidemics spreading. In [6] the authors specifically focused on the onset of an epidemic.

First looking at κ , in Section IV-A it was stated that κ doesn't influence the infection rate β . From [6] it also follows that κ is absent in the equations that determine the epidemic threshold. Thus, self-awareness is not a key factor for the dynamical behavior of this multiplex network system.

From Section IV-A it followed that γ has a influence on the infection rate β . The smaller γ , the better an aware node can protect itself from infection, which suggests that the epidemic threshold β (in fact $\gamma\beta$) should be higher to get in the active phase. The results of [6] confirms this. There is observed that for low values of γ the final incidence of the epidemics is lowered and the critical point for the onset of the epidemics is shifted to higher values of β .

The influence of the mass media is coupled with γ . It is shown that for a low γ the mass media effect is very pronounced, while when $\gamma = 1$, the mass media has no effect whatsoever. It is easy to understand this behavior. Namely, the mass media has the capacity to make people aware of the epidemic, but the effectiveness of this awareness depends on how good people can prevent themselves from the disease once they are aware. When $\gamma = 1$, the epidemic layer has effectively been disconnected from the information layer, which explains why awareness has no effect any more.

V. HETEROGENEOUS INFECTION RATES

As a third variation on the single network system model, we look into the effect of heterogeneous (and over time changeable) infection rates. The notion of heterogeneous infection rates is important because human contacts are very heterogeneous, making networks functioning with such rates very realistic.

In the research of [7], they use the SIS model. Having infection rates that can change, over time it will be possible that the epidemic reaches different phases. The simple case that $\beta > \beta_c$ applies forever doesn't apply here. One of the first results of [7] is that as the rate of infection increases, the SIS model exhibits a transition from an absorbing (disease-free) phase where the infection dies out to an active phase where the infection spreads over a large fraction of the population and becomes persistent.

For the single network system with heterogeneous infection rates, the authors of [7] take $\beta_{ij} = \lambda w_{ij}$ as the effective rate of infection between two individuals i and j . The weight w_{ij} can be seen as the amount of contact between the individuals, and λ is a free parameter that acts as a transformation scale of these contact intensities into infection rates.

In the research of [7] there has been experimented with different distribution of weights, but it is easy to directly come up with a realistic situation such as a classroom of students. In a clique of friends the contact intensity is much higher than between two students that only meet in class. As is shown in [7], this will result in different behavior of an epidemic inside this single network.

The most important result looking at the heterogeneous infection rates is that besides the transition between different phases, there is a large new region inside the absorbing phase. In this region, the change in the average density of infected individuals ρ exhibits an anomalous slow decay. This behavior is very robust and is mostly facilitated by the high-link weights. Namely, the high-weight links facilitate the spreading of infections, while low-weight links hinder the spreading. In the network there will be isolated regions where the disease is locally active, with infection rate $\beta_{ij} \geq \beta_c$, which are able to sustain the activity for very long times. In other words, once a group of highly interacting individuals gets infected, they are able to continuously reinfect each other at a high rate, keeping the infection inside the group for very long times.

VI. DISCUSSION & CONCLUSION

The study of epidemics has been used to predict, understand and develop strategies to control the spread of infectious diseases and has applications in field such as health care and computer networks. To illustrate the influence the underlying network can have on the behavior of epidemics, in this paper three network situations and their consequences were discussed. Before this, the reader was introduced with important basics towards epidemics.

The most common epidemics models are the SIR-model and SIS-model. In both models, a node starts in the ‘Susceptible’ state, where it can be infected by a node it shares a link with, with a probability β . The models differ in what happens when the node is recovered from the infection: It will reach the ‘Recovered’ state or the ‘Susceptible’ state again. A recovered node in the SIR-model is assumed to be immune for the disease, while susceptible nodes are able to get infected again. The SIS model can also be seen as a generalized SIRS-model where the probability to get from the R to S state is 1.

An epidemic can be in different phases, according to the infection rate β . When β is bigger than the epidemic threshold β_c the epidemics reaches an active phase where the disease is always present in the network. In this active phase the density of infected nodes ρ will converge to a constant value greater than zero.

This paper described the epidemics behavior on interconnected network systems, multiplex network systems and single network systems with heterogeneous infection rates, all on the SIS model. For the first category one can make the distinction between strongly and weakly coupled network systems. In strongly coupled systems, epidemics occur always across the entire interacting network systems. Here, the presence of interconnections will enhance the epidemic spreading. In weakly coupled systems a ‘mixed’ phase exist. In this case epidemics can occur on several networks of the interacting system. When the infection rate is high enough, the more intraconnected networks become capable of spreading the disease to the less intraconnected networks.

Secondly, a multiplex network system with two layers, an information layer and epidemics layer was discussed, where the influence of awareness on the epidemics behavior was examined. Three principles were examined: self-awareness, the degree of immunization once aware and the influence of a mass media. It turns out that self-awareness has almost no effect on the dynamics of epidemics, while the degree of immunization of aware individuals and the mass media do change the critical aspects of the epidemics spreading.

Last the influence of heterogeneous infection rates in a single network system is discussed. A network system with such infection rates an epidemic will exhibit a very slow extinction in the absorbing phase. This is due to the fact that even in the absorbing phase, where ρ will decay toward zero, there will be small clusters composed by links with high infection rate which will remain infected for very long times, allowing the disease to survive in the network.

VII. FURTHER RESEARCH

This paper serves as a broad introduction to the field of epidemics. Besides having explained the core models like SIS, SIR and the epidemics threshold, we elaborated on some behavior of epidemics in different networks situations. The information and results in this paper can be used as a primer for further investigations in the field of epidemics.

For the complete experiments and derivations of the results explained in this paper, one is advised to directly turn to the research papers. More information regarding epidemics on interconnected network, one should read [4], [8]. Epidemics on multiplex network systems is discussed in [6] and their previous research [5]. [7] discusses the heterogeneous infection rates. All papers will provide more literature on these topics for the interested reader.

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