Epidemics on Small-World Networks

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Abstract

This paper reviews the use of graphs as mathematical descriptions of social networks, and discusses in particular some properties of the commonly used random (Erdős-Rényi), small-world (Watts-Strogatz) and scale-free (power-law) models, with particular focus on the small-world model. The applications of such networks to epidemiology are then briefly outlined with some comments pertaining to the analysis of the 2002-2003 outbreak of severe acute respiratory syndrome (SARS).

1 Introduction

> I read somewhere that everybody on this planet is separated by only six other people. Six degrees of separation. Between us and everybody else on this planet. The president of the United States. A gondolier in Venice. I find that A] tremendously comforting that we're so close and B] like Chinese water torture that we're so close. Because you have to find the right six people to make the connection. It's not just big names. It's *anyone*. A native in a rain forest. A Tierra del Fuegan. An Eskimo. I am bound to everyone on this planet by a trail of six people. It's a profound thought. [...] How every person is a new door opening up into other worlds. Six degrees of separation between me and everyone else on this planet. But to find the right six people.
>
> Ouisa Kittredge, in John Guare's Six Degrees of Separation.[13]

In 1967, sociology professor Stanley Milgram conducted a now-famous experiment proving that the world was indeed a small place.[27] He mailed letters to randomly selected people in Wichita, Kansas and Omaha, Nebraska, asking them to forward a particular letter to two target persons in Massachusetts. The catch was that each participant was not allowed to mail the letter directly to the target person, but instead were supposed to forward it to an acquaintance or relative whom they thought would be more likely to know the target person. Milgram discovered the counterintuitive result that that the median number of intermediaries was 5.5.[9, 27] The result was rounded off in the phrase "six degrees of separation", which immortalized the phenomenon in the eponymous play by John Guare[13] about a con artist.

Milgram's experiment brought to the forefront the realization that the nontrivial structure of the social fabric meant that societies cannot be modeled naïvely as ideal lattices. Generalizing this result to systems outside sociology, extrapolations from standard results from statistical

physics, which are almost exclusively from systems distributed on regular lattices, were bound to give qualitatively incorrect results for systems on networks with nontrivial topologies. For example, the one-dimensional Ising model was recently solved on a small-world network and shown to have a non-zero transition temperature, in contrast to the result obtained on the regular lattice.[43]

Milgram's experiment also popularized the modeling of societies as networks based on mathematical objects known as graphs, the study of which has formed the subject of various popular science works.[9, 45] A graph[13] is a mathematical object associated with two sets; the first set is a collection of nodes or vertexes isomorphic to points, while the second set is a collection of edges connecting two vertexes together, each of which is isomorphic to a line segment. The degree of a vertex is defined as the number of edges containing that vertex. Graphs form the mathematical framework for describing networks such as that of a society, whereby each vertex is an individual person or organism and each edge represents a social contact or interaction of some sort. In general, all sorts of networks following such a formalism can be used as fruitful models for complex systems.[5]

Oftentimes one is not just interested in the structure of the social fabrics of various communities, but also on processes that take place in them. The dynamics of such phenomena are then affected in a non-trivial manner on the complexity of the underlying network.[3]

One of the most important processes sociologically is the occurrence of disease. Epidemiology, which is the study of disease spreading, its causes, and implications for public health policy, is only just beginning to realize the fundamental importance of the underlying structures of society for the spread of diseases. In particular, the failure of traditional epidemiological models to describe the severe acute respiratory syndrome (SARS) outbreak of 2003 is arguably one of the driving factors for the current paradigm shift in the study of epidemics. The World Health Organization estimates that while SARS caused less than 800 deaths worldwide, the economic cost is a staggering 30 billion US dollars in health care costs and lost production.[48] Gaining a more accurate picture of diseases work so as to implement effective countermeasures is hence a matter that has far more than just academic interest.

This paper outlines the connection between epidemics and phase transitions in statistical mechanics, emphasising the effects of a non-trivial spatial topology, and briefly describes how traditional epidemiology fails to describe the outbreak of SARS. In this paper, we will focus on the structural aspects of complexity.

2 Phase transitions and the onset of epidemics

In most network models of disease propagation, the feature of key interest is the occurrence of epidemics, which is the phenomenon where infected individuals transmit the disease in a rapid, widespread manner that grow uncontrollably over time. (The epidemiological literature draws a distinction between epidemics and outbreaks, which are essentially epidemics which eventually die out without any need for external intervention.) Outbreaks and epidemics understandably have great social and economic consequences, as illustrated by various throughout history and most recently with the SARS outbreak of 2003[38] and the current outbreaks of the H5N1 (Influenza A) avian flu virus.[47]

2.1 The epidemic threshold

In equilibrium, the transition describing the onset of an epidemic occurs at the so-called epidemic threshold. Below the epidemic threshold, only localized outbreaks occur. These are described in a graph formalism by the presence of components, which are finite-sized clusters of connected vertexes.

Above the epidemic threshold, the equilibrium state describes an epidemic: many clusters cross-link spontaneously to form what is termed a giant component. All nodes in the giant component are connected to each other with paths that are much shorter than those connecting nodes outside the giant component, if they are at all connected. The critical point also exhibits universality in that epidemiological specifics of various diseases become irrelevant as the dynamics in the critical region become dominated by interactions with connected nearest neighbors[41].

Newman, Watts and Strogatz have developed a generating function formalism which expedites calculations of topological properties from solely the degree distribution of a graph, which is defined as the probability p_k that a randomly sampled vertex in a graph has exactly degree k.[32] The epidemic threshold on a graph is then expressed elegantly in an equation exactly analogous to the critical isotherm:

$$z_1\{K\} = z_2\{K\} \tag{1}$$

where z_w is the expected number of neighbors of a vertex which are connected to it by a path of exactly w edges and $\{K\}$ represents formally the relevant set of coupling constants. Note that z_1 is equivalent by definition to the average degree of a vertex. The epidemic phase then follows intuitively as the region such that $z_2 > z_1$.

It is interesting to note that the onset of epidemics is formally identical to percolation problems, with the epidemic threshold analogous to the formation of a giant percolation cluster.[30]

3 Models for social networks

Random graph models are amenable to analysis using the usual methods of statistical mechanics by considering ensemble averages. Such a random graph exhibits a phase transition in their topological structure: below the transition, the graph consists of a disjoint collection of components, which are finite-sized clusters of connected vertexes; above the threshold, these components spontaneously cross-link to form what is termed a giant component. All nodes in the giant component are connected to each other with paths that are much shorter than those connecting nodes outside the giant component, if they are at all connected.[42]

This section of the paper outlines some properties of three commonly used models of networks used to describe communities.

3.1 Random networks

First discovered by Erdős and Rényi[11, 16], the random graph is the simplest model of a social network. There are only two assumptions built into this model:

1. There are a fixed number of vertexes N.

2. The vertexes are connected randomly with a uniform probability p of having any two vertexes connected by an edge.

Yet despite their mathematical simplicity, random graphs have surprisingly rich statistical properties. Random graphs have the property that if each node has on average one link, then there exists a unique giant cluster of connected vertexes. In other words, a random network has a phase transition occurring at $p = \frac{1}{2}$ and has an average connection length of $l \sim \ln n$[35]. It also exhibits a Poisson degree distribution in the limit $N \to \infty$, which can be derived as the limit of a binomial distribution from statistical considerations[34, 35].

While simple to describe, the underlying assumptions for random graphs are simply unrealistic to describe just about any real-life network. For example, in a social context, some people are more well-connected (in a very literal sense) than others. These would be people playing leadership roles, e.g. heads of household in families, teachers in schools, and healthcare workers in hospitals. Also, connections form and break over the course of a day. An worker commuting to his or her workplace interacts with a large number of groups of people, with physical contacts between people being constantly in a state of flux. From an epidemiological point of view, the presence of super-spreaders[34] in many diseases is also at odds with the Poisson degree distribution, which implies that the probability of observing an outlier is exponentially rare.

Flawed as it might be, the random graph model is still useful as a basis for comparison. One particularly insightful example is the calculation by Itzkovitz *et. al.* of the distribution of subgraphs in random graphs.[21] Deviations can then form the basis for uncovering unusual structural motifs for further analysis.

Dissatisfaction with the unrealistic description of random graph models formed the motivation for new models, such as the small-world model and the power-law model described below.

3.2 Small-world networks (Watts and Strogatz)

In 1998 Watts and Strogatz proposed a new model[44] which aimed to address the deficiencies that have shown up as a result of using the Erdős-Rényi model. The small world model is made from a regular network, e.g. lattices, that consists entirely of localized links, with edges connection only vertexes that are in close proximity with each other, up to some range κ, such that it has coordination number $z = 2\kappa$. Nodes are then rewired randomly with probability ϕ that any particular vertex was rewired. In contrast to the regular network, where only nodes in close proximity are connected to each other by edges, the random rewiring process introduces connections known as "shortcuts", which disregard the proximity (or otherwise) of nodes.

Newman and Watts proved that this model has a second-order phase transition in the limit of zero shortcut density, i.e. $\phi = 0$.[29] In the other limit, this model reduces to that of a random graph. The small-world model can therefore be seen as a way to interpolate between regular networks and random graphs.

A subtle point which is not always well appreciated is that in order to create a small-world network, one requires a notion of distance on a graph in order to distinguish between "close by" and "far apart". In mathematical terms, this is accomplished formally by introducing a measure or even a metric[7] on the graph. Random models have a uniform measure whereas small-world models take on the measures of the original regular lattice from which they were constructed.

3.2.1 Properties

Small-world models exhibit two signature characteristics:[44, 42, 34, 35]

1. Small-world networks have a high degree of clustering, which is the conditional probability that if a vertex A is connected to another vertex B by an edge, which in turn is connected to another vertex C by another edge, that A is connected to C by yet another edge. In lay terms, clustering is the phenomenon whereby your friend's friend is also your friend[35].

2. Small-world networks exhibit the so-called "small-world effect", that the average shortest path length between vertexes l scales in a logarithmic manner with system size, i.e. $l \sim \mathcal{O}(\ln N)$, or sometimes in a fashion that is similar in that it grows very slowly with increasing system size. The small-world effect is sometimes referred to colloquially as the "six degrees of separation" phenomenon.

By explicitly considering systems of finite size N, Newman and Watts discovered[29] that there exists some intermediate system size $\xi = (\phi\kappa)^{-\frac{1}{d}}$[30] for fixed κ and ϕ such that the expected number of shortcut links is equal to one. This length scale then diverges as ξ at the second-order phase transition. From this result, they obtain a scaling relation[29] for l and hence conclude that l has two different scaling limits in various régimes:

$$l \sim \begin{cases} \frac{Nd}{4} & , N \ll \xi \\ \xi \ln \frac{N}{\xi} & , N \gg \xi \end{cases} \tag{2}$$

Newman *et. al.* also obtained an mean-field solution[31] for l on a one-dimensional ring (a periodic lattice with periodic boundary conditions) which goes as

$$l = \frac{N}{\kappa} f(N\kappa\phi) \tag{3}$$

$$f(x) = \frac{1}{2\sqrt{x(x+2)}} \tanh^{-1} \frac{x}{\sqrt{x(x+2)}} \tag{4}$$

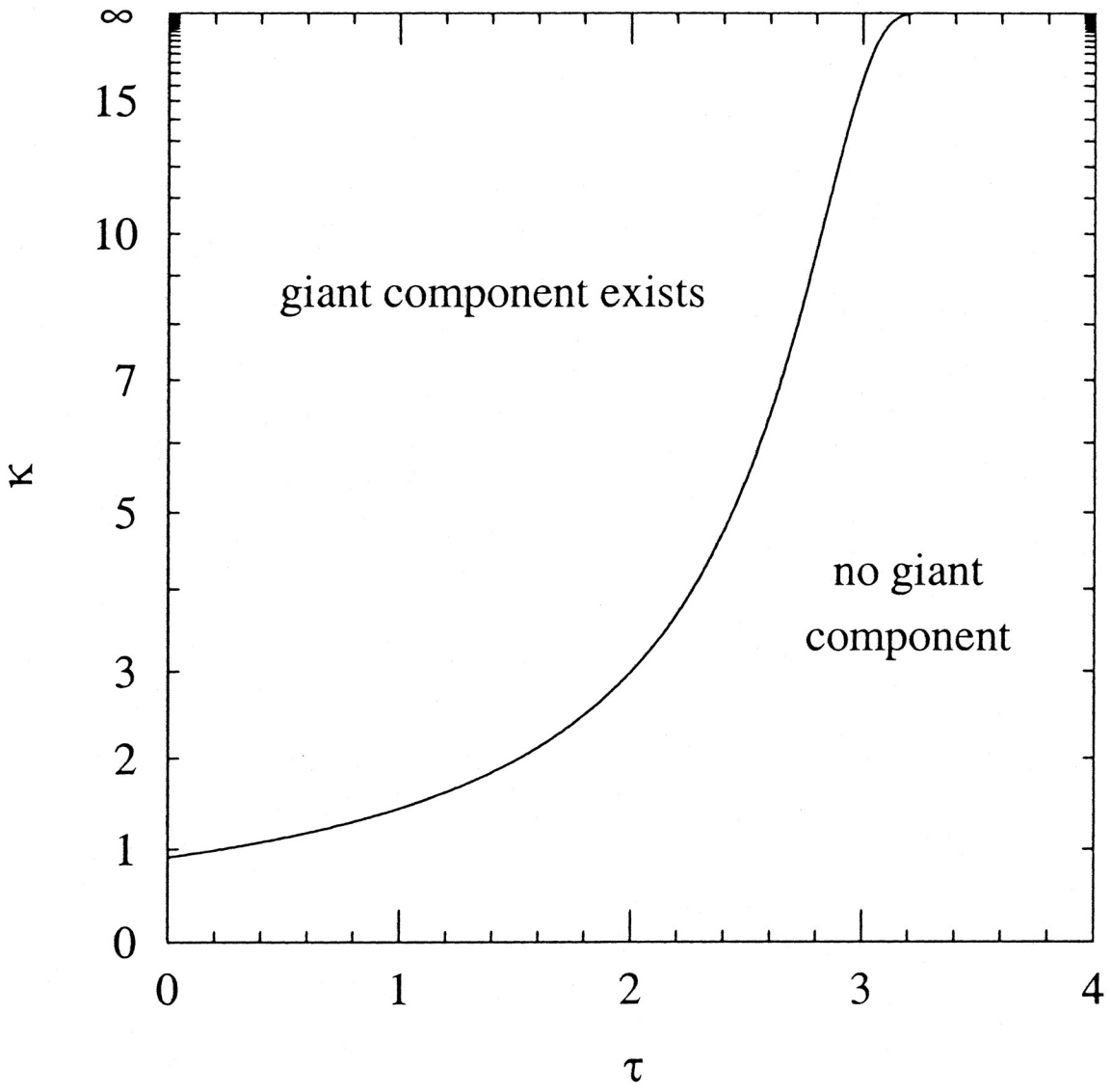
3.3 Scale-free (power-law) networks

Networks with power-law degree distributions[8] have attracted much attention as accurate models for other real-world systems such as the World Wide Web, in which growth by addition of nodes gives rise to important structural features[2, 14]. The degree distribution is (up to a normalization constant C)[35]

$$p_k = \begin{cases} Ck^{-\tau} e^{-\frac{k}{\kappa}} & , k \geq 1 \\ 0 & , k = 0 \end{cases} \tag{5}$$

The power-law network thus distinguishes itself from a random network by the so-called "long tail" of nodes with very high degree. This feature of the World Wide Web has captured the popular imagination since the idea was popularized in an influential online article[6]. Power-law networks have a two-parameter phase diagram which is shown in Figure 1. Interestingly, there exist two non-trivial intercepts in the phase diagram at $(\tau, \kappa) = \left(0, (\ln 3)^{-1} \approx 0.910\right)$

Figure 1: Phase diagram for the scale-free (power-law) network. Reproduced from Ref. [34]. The axes κ and τ are defined in Eq. 5. Copyright 2002 National Academy of Sciences, U. S. A.



and $(3.479\cdots, \infty)$. This implies that for $\tau > 3.479$, the fluctuations are so large that it is impossible for a giant component to form, and for $\kappa < 0.910$, the system has too small a length scale to support a giant component.

As with the small-world model, the power-law network also has an average shortest path length that also exhibits logarithmic growth with system size.[34]

4 Epidemiology and the SARS outbreak

The mathematical formalism of graphs as outlined in the previous section forms the basis of epidemiological models, which aim to capture the physics of the spread of infectious diseases. Conventional epidemiological theories make use of both kinetic models and a factor R_0 known as the basic reproductive ratio; both these elements are flawed in the presence of a nontrivial network topology.[24] This section reviews each of these elements and in turn summarizes the reasons for the breakdown. In the final section some analyses on the SARS outbreak are discussed.

4.1 The SIR kinetic model

Conventional epidemiological models employ kinetic models in terms of various classes of individuals in a population. One popular model is the SIR model, where the population is divided into susceptible (S), infected (I) and removed (R) classes.[24] In terms of rates of two elementary processes, namely g for the rate of infection and c for the rate of making contacts, the model is then governed by the differential equations

$$\dot{S} = -cSI \tag{6}$$
$$\dot{I} = (cS - g)\, I \tag{7}$$
$$\dot{R} = gI \tag{8}$$

The fact that time is the only dynamical variable of importance is equivalent to making an assumption of spatial homogeneity, or making a mean-field approximation. The limitations of this so-called full mixing approximation have already been acknowledged in the theoretical biology literature[15, 24, 26]. Durrett and Levin showed that fundamental discrepancies between homogeneous models and particulate models, and homogeneous models and reaction-diffusion models of predator-prey interactions mean that such mean-field approximations are unreliable to give even qualitatively correct answers.[15] This approximation also completely ignores spatial heterogeneity and implicitly assumes a Poissonian degree distribution.[35] Hence, any meaningful discussion of such models on graphs must be properly generalized.[24, 28, 33, 35]

It is interesting to note that one such generalization of the SIR model with four classes of individuals has been shown to give rise a model with an upper critical dimension of 5 and exhibits tricritical percolation behavior with both compact and fractal clusters.[22]

4.2 The basic reproductive ratio R_0

Traditional epidemiology focuses on a key parameter R_0 called the basic reproductive ratio, defined as the average number of secondary cases emanating from one primary case in a sus-

ceptible population.[23] When $R_0 > 1$, the population is vulnerable to a large scale epidemic, although not guaranteed to experience one. Conversely, when $R_0 < 1$, the population only experiences small, local outbreaks. Hence R_0 serves as an order parameter for the phase transition.

While R_0 is an intuitively appealing order parameter, its utility is derived from the Erdős-Rényi model[46]. Hence, analysis based on R_0 breaks down completely in the presence of phenomena not captured in the random graph model, such as the effect of super-spreaders[26] (described below) and the possible mixing of local and global factors in the transmission of disease[4], such as spatial heterogeneity in population and traffic densities[26], and the possibility of multiple modes of transmission[40].

The reason for the breakdown was shown to be because R_0 conflates the influence of both the transmission probability T intrinsic to the disease (the conditional probability that a vertex is infected given that a connected nearest neighbor is also infected) as well as the topology of the underlying network as a function of the moments of the degree distribution[26]:

$$R_0 = T\frac{\langle k^2 \rangle}{\langle k \rangle - 1} \tag{9}$$

Analyses that treat both factors separately were then shown to give rise to correct descriptions of the epidemic threshold[26].

The phase transitions describe in the preceding section can be seen to be analogous to the epidemic threshold described (albeit incompletely) by R_0. Below the epidemic threshold, the finite nature of the clusters mean that an outbreak of the disease on any one cluster is bound to die out due to the finite extent of each cluster. Above the epidemic threshold, the presence of a giant connected structure with short paths between arbitrary nodes means that diseases can spread in a rapid, widespread fashion - in other words, an epidemic is formed. Thus for an epidemic model on a graph, graphs, the epidemic threshold should not be taken to be simply $R_0 = 1$, but instead taken to be the criteria reported in the preceding section, or in general from Eq. 1.

4.3 Implications for disease control

The interruption of the spread of disease in an epidemic is analogous to the study of network resilience.[14, 35] Power-law networks such as the World Wide Web[2] have nodes of high connectivity which form network hubs; search algorithms biased to traverse hubs in descending order of degree have been shown to be superior to random-walk searches.[1] It has also been shown that removing hubs from such networks hubs dramatically slows down the search efficiency as compared to removing a regular vertex.[1, 12, 14]

In this vein, it is useful to conclude by analogy that the most effective disease control measures for curbing the extent of an epidemic would be to identify potential super-spreaders and concentrate prevention efforts upon such individuals. Such considerations have led to the implementation of control measures such as contact tracing for the express purpose of identifying bottlenecks which can be targeted specifically. This is an example of how epidemiological studies are relevant to public health policymaking.[35] Interestingly, the notion of looking at network structure to look for choke points to curb the spread of disease was advanced as early as 1985 in a seminal example studying the contact network described by the transmission of acquired immune deficiency syndrome (AIDS)[25].

4.4 Traditional epidemiology and the 2002-2003 SARS epidemic

Having discussed the mathematical formalism and theory underlying modern epidemiology, we now turn our attention to real data from the 2003 SARS epidemic. A brief chronology of events in the SARS epidemic of 2002-2003 can be found in Ref. [38].

The R_0 calculated for SARS is 2.2-3.6,[26] which is well above unity. Conventional epidemiology predicts a global pandemic, which did not occur. In contrast, the spread of SARS was restricted to localized areas.[38] Public health control measures decreased R_0 to 1.2 in Hong Kong and Singapore[26] but this still does not correctly explain the lack of an epidemic.

4.5 The super-spreader effect

A super-spreader is defined (somewhat tautologically) as an infected person who shows an abnormally high rate of transmission of a disease to other people[49]. Incidents of super-spreading have been blamed on everything from lack of hygiene to use of aerosol generators in hospital equipment to as-yet undiscovered modes of transmission.[38] The super-spreader phenomenon is known not to be due to unusual virulence of certain strains of SARS, since genetic evidence shows that viruses from all documented cases outside Guangdong province are related to the samples found in the first (index) patient.[20]

Super-spreaders are thought to be responsible for 70% of all cases of infection during the SARS outbreak[50]; these outliers are of immense epidemiological significance. While it is possible that super-spreaders represent a qualitatively different kind of interaction, it is also possible that super-spreaders are merely the result of the "long tail" of a skewed degree distribution such as that of a power-law network.

5 Summary and Outlook

The preceding sections have presented ample evidence that processes occurring on spaces with nontrivial topologies can give rise to complex phenomena which are at odds with conventional experience on regular lattices. In particular, traditional tools used to study the spread of diseases fail completely to describe real epidemics (or the lack thereof) because of the inherent assumptions used to establish their utility.[15, 24]

Despite intensive research, SARS phenomena are in general still poorly understood.[50] The origin of the super-spreader effect is still unknown, although the properties of scale-free networks allow an appealing interpretation as the effect arising from highly connected nodes on the network.

While the SARS outbreak is over, the suspected animal origins of the SARS coronavirus[19] means that wild animals may still harbor the virus and may serve as seeds for a future outbreak. [36] This has profound implications for possible resurgence[46] of SARS in the future from animal populations. Future models for disease propagation would probably want to treat the effects of having a reservoir of dormant etiological (causative) agents for long-term studies of SARS, in a manner analogous to the study of dissipative dynamical systems. Doing so may yield new insights into preventive measures that would reduce the probability of another epidemic occurring.

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