SOCIAL CONTACT PATTERNS AND DISEASE DYNAMICS

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Summary: Communicable disease has historically constituted a very serious threat to society. The AIDS epidemic, the SARS outbreak and the prospect of bio-terrorism have made clear that this threat is still present. The growing threat to contemporary society from infectious disease stems largely from our modern lifestyle, namely given the global time-compressed social interaction. More than ever, social contact patterns provide an essential key to understanding infectious disease dynamics. Here we discuss the main aspects of epidemiological modelling and contact patterns that should be taken into consideration, and we highlight the interplay between disease characteristics and social interaction patterns. We also discuss similarities and differences between patterns of social interaction in contemporary and historical society, and the implications for disease dynamics.

1. Introduction

Contemporary society is sometimes described as a 'risk society'. In recent years, the label has begun to ring true to many. Accelerating pollution and environmental breakdown, as well as the increasing threat of global terrorism have rapidly become two disturbingly real risks with potentially fatal consequences. A third risk is the re-emerging threat of communicable diseases. During most of the 20th century, pandemics (epidemics that spread throughout huge areas and large populations) were considered to be an historical threat to society; the advent of modern medicine had forever taken care of the plague, smallpox and other contagious horrors. At least that was the commonly held belief, until the AIDS epidemic struck at the beginning of the 1980s. More recently, SARS has convincingly demonstrated that communicable disease remains a real threat to society and humankind, and that we need to take protective measures.

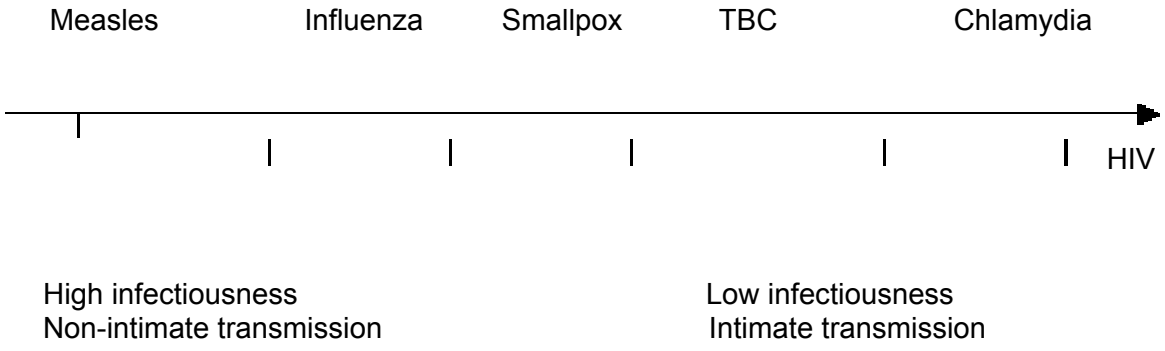

Figure 1. Some well-known infectious diseases arranged according to the degree of intimacy in transmission.

Our basic point of departure is that the importance of the actual contact pattern is a function of the disease's infectiousness. Infections spread from person to person, either directly or via air, or through food and water. The more intimate the contact required for

transmission to take place, the more important the contact pattern. Figure 1 shows some known infectious diseases, ordered on a one-dimensional scale ranging from high infectiousness and non-intimate transmission on the left hand side, to low infectiousness and intimate transmission on the right hand side. For our purposes here, it is sufficient to state that the lower the infectiousness the greater the attention that should be paid to social contact patterns. On the other hand, the higher the infectiousness, the larger the threat and the greater the cost of misrepresenting the importance of social interaction at all levels.

2. Models and Concepts of Epidemics

Theories about the nature of infectious disease have probably existed ever since man was able to formalize such theories in words. For example, a slow-moving cloud of bad air was put forward as one of many competing explanations behind the diffusion of the Black Death in the 14th century (Ziegler 1998). A famous early example of biological warfare, dating back to the siege of Kaffa in 1346, even suggests that the idea that diseases can be contagious might be older than the related field of science itself. The city of Kaffa was an important Genoese trading hub in the Crimean Peninsula on the Black Sea, which was laid siege to by a Mongol army (supported by Venice, Genoa's main rival). The Genoese locked themselves up behind the city walls and the city's decline was rapid. As Kaffa was located on a trade route, the surrounding area lay open to the plague that had ravaged China since the early 1330s and was now working its way westwards along the trade routes. The plague, eventually to become known as the Black Death, hit the besiegers with full force, obliging the Mongolian general Kipchak khan Janibeg to call off the siege and withdraw with what was left of his forces. Of course, not even the most powerful city walls could protect the people within from the plague. Even if men could not breach the walls, it is almost certain that black rats, highly effective carriers of disease, could. As a result, the citizens of Kaffa were also infected with the disease. However, legend holds that the plague came to Kaffa in a much more cunning fashion (Karlen 1996): the Black Death might have prevented Janibeg from bringing Kaffa to its knees by the use of military force but, before retreating, he ordered that the infested corpses of his soldiers be catapulted into the city.

D'Alembert was the first to attempt to describe the diffusion of infectious disease using a mathematical model in the 18th century (Dietz and Heesterbeek 2002). Nowadays, the analysis of epidemical processes has become an advanced field of research, in which sophisticated mathematical models are used that are often intractable for the layman (Anderson and May 1991; Diekman and Heesterbeek 2000). However, it is possible to get an intuitive feeling for the basic laws that govern the dynamic features of epidemiological processes without going into the mathematical details.

We will restrict our discussion here to the dynamic features of diseases that only infect humans, such as measles or smallpox. One basic epidemiological principle for this type of disease is that, at a certain point in time, each infected individual must infect an average of at least one new individual in order for the epidemic to stay alive over a protracted time span. The average number of people infected by the preceding, already infected generation is called the *Reproduction Rate* (Giesecke 1994). One intuitive way of understanding the reproduction rate is to compare it with the reproduction of animal species. If each animal were to produce an average of less than one offspring, sooner or later the species would become extinct. If, on the other hand, each animal produces an average of more than one offspring, we will observe an explosion in the size of that species' population. For example, the Giant Panda's reproduction rate faces the constant risk of dropping below one, implying that the species is in danger of extinction. Conversely, cockroaches are a group whose reproduction rate is frequently much higher than one.

Basically, the same principle holds true for infectious diseases. In the case of smallpox, mankind has reduced the reproduction rate to below one through artificial and ambitious vaccination campaigns, so that the disease has now been eliminated, at least in the western world. One of the reproduction rate's most interesting features is that a vaccination campaign can be successful without cutting the rate to zero –ie, it is not necessary for all the members of a population to be immune in order to prevent an epidemic–. The key is that the reproduction rate must be less than one at any stage of the epidemic because, as a result, the number of infected individuals will decrease in successive generations, until a generation with no infected individuals is reached.

In fact, the potentially harmful side effects represent the greatest impediment to successful vaccination. When vaccination comes at the cost of painful or irritating side effects, which, to some extent, is almost always the case, people may start weighing up the costs and benefits. With vaccination at a cost, Adam might not be willing to accept being vaccinated unless Eve were to do so too. While if Eve were to accept the vaccination, then there would be no real need for Adam to be vaccinated. The reader will recognize that we have entered the realm of the classic free-rider problem (Olson 1971): if everyone else has been vaccinated, there is no need for me to follow suit because I can already benefit from their vaccination without paying the cost. Another recent hindrance to the success of vaccination programs is that some parents', mainly intellectuals, awareness of health matters has misguidedly led them to withdraw their children from certain vaccination programmes.

A central question in epidemiology is whether or not a disease will manage to gain a foothold in a population. In situations with a low variation in the number of contacts through which the disease can be transmitted from one individual to another, this question can be answered by considering the reproduction rate when a single infectious individual enters a totally susceptible population. Consider, for example, whether a returning traveller infected with SARS might cause an epidemic in his homeland or not. The reproduction rate at this initial stage is called the *basic reproduction rate*, denoted by R_0 in epidemiological literature (Giesecke 1994).

Another frequent and important assumption is that the infected person shares the same interaction pattern as the rest of the population. Mathematically, it has been demonstrated that it is impossible for a disease to gain foothold in a population, as long the R_0 is less than one, provided this assumption holds true. However, note that, if even a single member of the population is not immune, there is always the possibility that some individuals could become infected. In epidemiology, if it is impossible for a population to suffer epidemical outbreaks, it is said to exhibit *herd immunity* (Giesecke 1994).

It is not always appropriate to assume a low variation in the number of contacts through which the disease can be transmitted. The assumption is usually reasonable for very contagious diseases, such as measles and the flu, where just standing close to an infectious person is often enough (see Figure 1). Most of us have a huge number of such contacts every day –eg, on the bus, in the theatre or a restaurant–. It is therefore realistic to assume that the majority of the population has a very large number of contacts. Nevertheless, there is an obvious exception to this rule of thumb: sexual contacts, where the variation in the number of contacts is known to be extremely large (Anderson and May 1991). While the majority has a low number of these contacts each year, some individuals have a large number of contacts and a small group of people has numerous sexual contacts every year.

For these types of contact patterns, R_0 gives a conservative estimate on the likelihood of an epidemic outbreak from a single case of an infectious individual returning to a totally susceptible population. When a returning individual is infected with gonorrhea, for

example, he will probably visit a doctor on noting the symptoms and, if the number of yearly contacts is relatively low, the infection is unlikely to be transmitted. However, if the disease is endemic somewhere in the world, it is reasonable to assume that there will be several occasions (not just one) when an infected individual returns infectious. Therefore, there is a serious risk that an individual with numerous sexual contacts will return infectious sooner or later and that this individual will also transmit the disease to his/her partners. Research has shown that the partners of individuals with many partners are also likely to have numerous partners, etc. This situation is likely to lead to an epidemical outbreak among individuals with numerous partners that, on occasions, will also infect individuals who have few partners. This is a simplified example; a full account would also have to take into consideration that people display different risk behaviours.

The reproduction rate is determined by three different factors (Giesecke 1994): *c*, the average number of potentially infectious contacts per person; *b*, the probability of infection per contact between susceptible and infected; and *D,* the average duration of the infection. Unfortunately, none of these parameters can be estimated in a controlled experiment and, accordingly, the parameters for most diseases are associated with a degree of uncertainty. This is especially true in the case of smallpox, where both the probability of transmission and the duration of infectiousness are disputed.

The average number of potentially infectious contacts per person depends on the stage of the epidemic because the number of susceptible people decreases as the number of those already infected grows. This dynamic process is very often modelled with a system of differential equations.

In standard epidemiological models, individuals are assumed to be in one of three possible states. According to these states, the population can be classified into three categories: susceptible (S), infected (I), or resistant (R) persons. It is conventional to distinguish between SI, SIS, and SIR models and, in all, interaction between persons is assumed to be random (Anderson and May 1991). Children's diseases are best modelled using a SIR model because infection leads to life-long immunity. For most sexually transmitted diseases, the SIS model is more useful, since only a few sexually transmitted diseases confer any immunity after infection. HIV is a clear exception and is still appropriately described, at least in the Western world, with the SI model.

The premise of random interaction is disqualified by Equation 1. This gives the SI model as a continuous model in its simplest form, consisting of a system of two differential equations:

$$\frac{dS}{dt} = \frac{-c\beta S(t)I(t)}{N}, \qquad \frac{dI}{dt} = \frac{c\beta S(t)I(t)}{N}. \tag{1}$$

There are two dependent variables in Equation 1: The number of susceptible persons, *S*, and the number of infected persons, *I*. At each point in time $S(t)+I(t)=N$, where *N* is the population size. Evidently, this model is homogenous for people, as each person is assumed to have the same number of contacts, *c*, so we can say that the model assumes random interaction.

For many diseases, like measles or flu, that spread through droplets of an infected person's breath, random interaction is a reasonable assumption and probably a good approximation. In other words, there is an abundance of everyday situations where a person is exposed to such infections, for instance on public transportation, in the workplace and in shops. The random interaction assumption offers an important

advantage in that it can easily be modelled with differential equations, and these models can be studied analytically (Diekman and Heesterbeek 2000).

The solution to Equation 1 gives an S-shaped trajectory, as shown in Figure 2, because the number of infected that can transmit the infection is low in the early stages of the process, while the number of susceptible is low in the late stages. As a result, the number of infected experiences the highest growth in the middle stage of the process.

The SIS model can be written as a system of two differential equations, as follows:

$$\frac{dS}{dt} = \frac{-c\beta S(t)I(t)}{N} + \frac{I(t)}{D},$$
$$\frac{dI}{dt} = \frac{c\beta S(t)I(t)}{N} - \frac{I(t)}{D}. \quad (2)$$

The equation for SIS model differs from the SI model in the way the term $\frac{I(t)}{D}$, which describes the rate at which individuals recover from the disease or become susceptible, is applied in both equations. The solution to the SIS equations also shows that we should expect an S-shaped trajectory in the number of infected. However, the SIS trajectory differs from the SI trajectory in that the number of concurrently infected people never reaches the total population (which does not rule out the possibility that each individual can become infected at some point in time). Instead, the process reaches equilibrium where exactly as many infectious individuals become susceptible again, as susceptible individuals become infected.

The last model we are going to discuss here is the SIR model, which in its simplest form can be formulated as a set of differential equations, as follows:

$$\frac{dS}{dt} = \frac{-c\beta S(t)I(t)}{N},$$
$$\frac{dI}{dt} = \frac{c\beta S(t)I(t)}{N} - \frac{I(t)}{D},$$
$$\frac{dR}{dt} = \frac{I(t)}{D}. \quad (3)$$

The SIR model describes the process in the three different states. The solution to the SIR model also shows an S-shaped form in the early stages of the epidemic. The SIR model differs from both the SI and SIS models in that it shows a tendency to end up with zero infected in the long run.

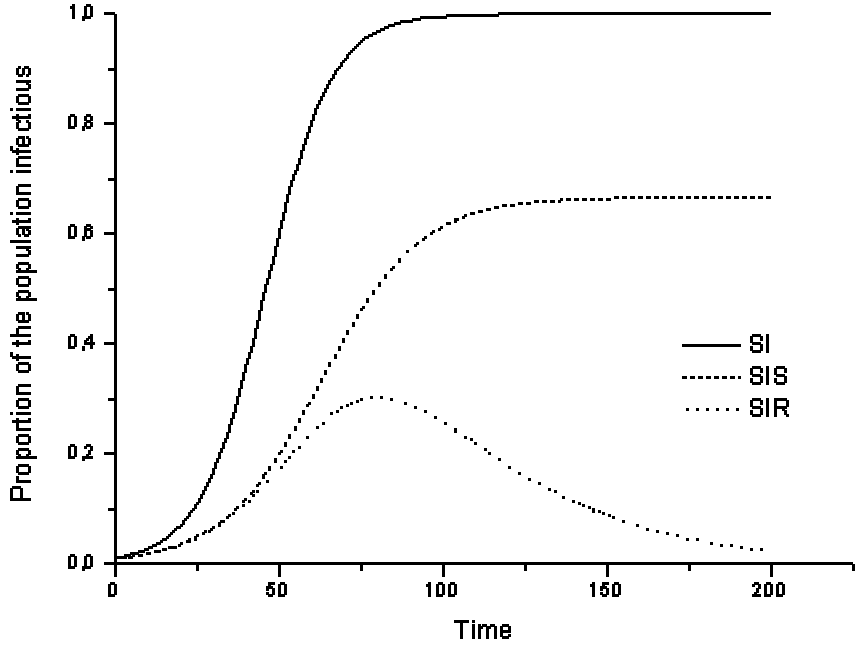

Figure 2. Growth trajectories (disease dynamics) for three epidemiological models.

These three basic models can be adapted to the characteristics of specific diseases. For example, it is possible to show individuals as being immune for a certain time interval, for new individuals to enter the population through birth and emigration, and for people to leave the population due to migration or death. Accordingly, we can generate more complex types of trajectories, such as cyclic behaviour and even chaotic dynamics, which characterize some types of infectious disease.

Looking back at Figure 1, physical space is an important factor, which could be significant in terms of reproduction rate, but is not covered in the models above. For our purposes here, we will treat the world as a simple matrix with a Moore neighbourhood (Figure 3). The reproduction rate will usually be lower if individuals are scattered in two-dimensional space, as the individuals will only transmit the infection to their neighbours.

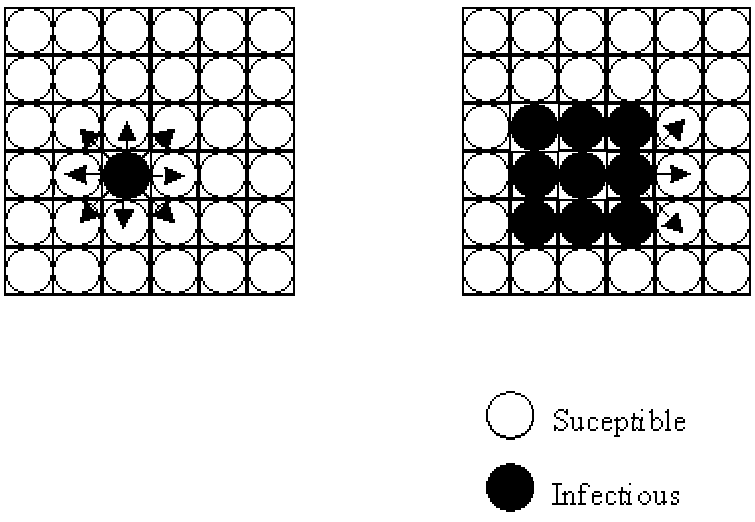

Figure 3. Disease contagion in two-dimensional space with Moore neighbourhood.

As Figure 3 clearly shows, the fact that individuals are structured in space increases the probability of an individual's neighbours being infected already. This would make the reproduction rate lower than is the case for an identically-sized population with randomly organized contacts between individuals (Eames and Keeling 2002).

However, the effect of geographical space is not necessarily as important as it might seem at first glance provided some contacts stretch over a greater distance, as is the case in Figure 4. This kind of interaction structure –where the vast majority of contacts are local and a few, but significant, number of contacts stretch over a longer distance– is usually referred to as the 'small world' phenomena (Watts and Strogatz 1998).

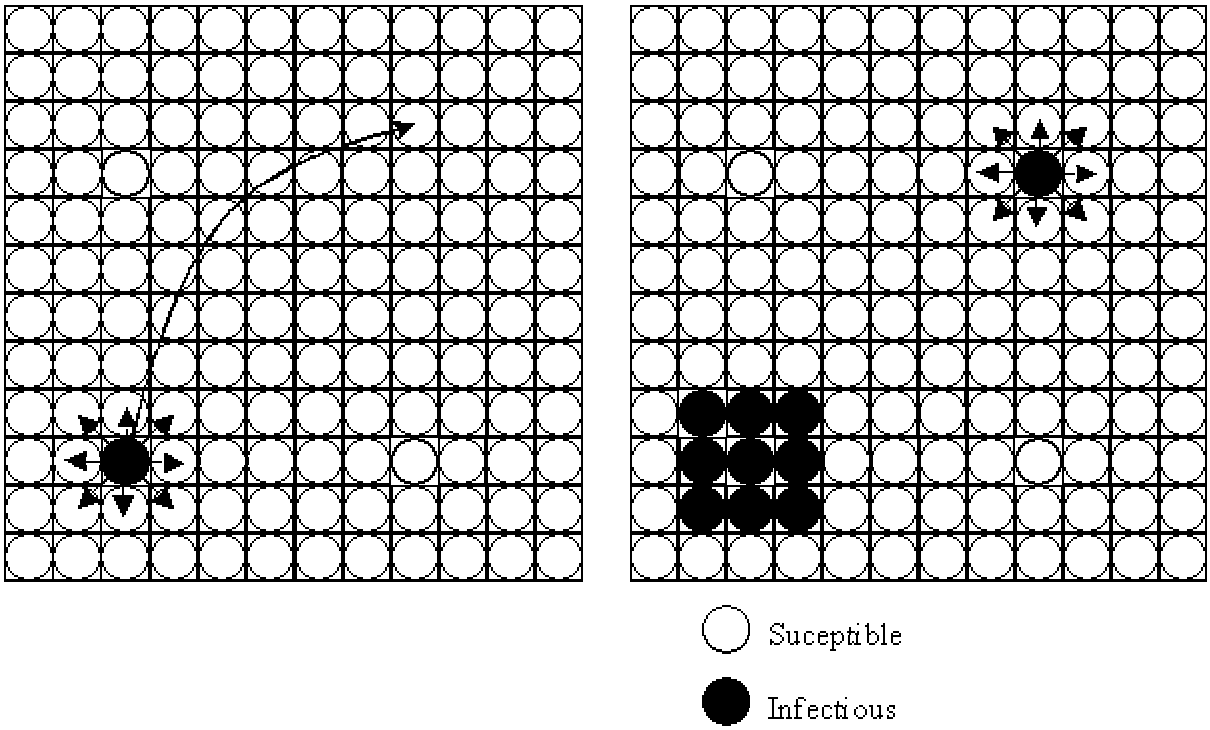

Figure 4. Disease contagion in a small world.

As stated above, the premise of random interaction assumption is not suitable for modelling the diffusion of a sexually transmitted infection (STI) because sex is simply not random (Laumann *et al.* 1994). As is the case in most social interaction, societal dimensions structure sexual contacts. An early approach to this problem of non-randomness in the spread of STIs is to divide the population into sub-populations, defined by gender and the level of sexual activity. The diffusion within and between subpopulations is then modelled as systems of differential equations. The rationale is that a small, very sexually active group can be modelled by conceptualising it as a subpopulation, usually referred to as a core group (Hethcote and Yorke 1984). These types of models have been used to study the difference between two specific scenarios.

In the first scenario –known as assortative interaction– most contacts take place within the different groups, ie, sexually active individuals have sexual encounters with other sexually active people, while those with low sexual activity tend to have sex with other people with low sexual activity. In the second scenario –known as disassortative interaction– most contacts take place between the groups, ie, sexually active individuals have sexual encounters with people with low sexual activity. Theoretical analyses have demonstrated that assortative interaction initially spreads the STI more quickly, but results in a smaller-sized total epidemic. On the other hand, a dissasortative interaction pattern generates a

slow initial spread, but a large epidemic. The mixed-scenario –known as symmetric association– where interaction takes place both within and between the subgroups, gives rise to an intermediate pattern of diffusion.

Most empirical studies indicate that assortative interaction is the closest match to empirical reality (Granath *et al.* 1991). The distinction between assortative and disassortative interaction has also been used for other variables besides gender and sexual activity, such as socio-economic status, age, etc. These studies demonstrate that assortative interaction is structured through sociological variables, ie, people are more likely to sexually interact with others from the same social class, age group, or ethnic group (see Liljeros, Edling and Amaral 2003, for an extensive review).

A further step away from the standard model, and the assumption of a homogenous *c*-parameter, is to study the distribution of the number of contacts people have. Several empirical studies have demonstrated a very large variation in the number of sexual partners (Laumann et al. 1994; Liljeros et al. 2001). While most people report between one and four sexual partners during their lifetimes, some point to several hundred or more. Again, this means that the effective R_0 need not be greater than one for an epidemic to occur in a population.

Even though modelling groups of people with different levels of sexual activity is more realistic than assuming random interaction for the entire population, these models do not consider key factors such as the formation and/or duration of partnerships, and concurrency (Potterat, Muth and Brody 2000). Theoretically, these factors can be incorporated into a deterministic model by representing each unique combination as a differential equation in a system of differential equations. However, adding factors quickly overloads the system with too many dimensions, making analytical solutions extremely difficult.

3. The Complexity of Social Contact Patterns

Clearly, modelling structured interaction by means of flow equations quickly becomes fairly cumbersome. Still, to bring leverage to intuitive ideas, analytical tools are needed to conceptualise interaction structure at the level of persons. A simple depiction of social life starts with conceptualising each human being as a dot or node (or vertex). Social and physical relations between people can be conceptualised as links between dots, or edges between nodes (or arcs between vertices). Mathematically, the set of the nodes and edges in a social system defines a graph, and the analytical treatment thereof is known as Graph Theory. This has proved to be a catalyst in the development of social network analysis that now provides the basic tools for analysing sociograms (Wasserman and Faust 1994).

The key to social network analysis is that the focus of study shifts from people to the relations between them, as well as to the overall patterns of relationships in a population. These relations can connect individuals to form dyads, triads or, in the present context, larger networks. When shifting the unit of observation from people to interrelated persons we should consider several issues, although we have only briefly covered the most important.

An ego network starts with a specific person, for whom relations to other persons are mapped. Thus, to collect data for an ego network of sexual contacts, we would start with an identified person (possibly infected) and would re-construct all his/her different sexual contacts, as well as the contacts between the other people in that ego network. However, to collect data for a complete sexual network, we would start with all the (possibly

infected) individuals identified in a community and then attempt to re-construct all the different sexual connections between them.

To facilitate an understanding of the matters covered below, we will first look at some key concepts and ideas. Firstly, a network consists of the set of edges between a set of identified nodes, also referred to as a graph. This implies that a node can be part of the network (group or population) without being connected to any other node. This type of node is called an isolate. Secondly, a node's degree is determined by the number of its edges, so a degree of four means that a node has four edges. In the present context, we are most interested in people with a high degree, as they have many sexual contacts. Thirdly, degree centrality measures the degree of a node relative to the degree of other nodes in the network. Nodes with the highest centrality have the largest number of edges. Fourthly, the network density is given by the observed number of edges in the network relative to the possible number of edges in the network. Lastly, a network component refers to a set of nodes in a network that is connected by a set of edges (which may be a cluster or, if forming a completely connected sub-graph, a clique). Think of components as isolated subsets within the network; epidemics can only take place within components. Note that a component differs from the idea of a cluster as used in epidemiology. In epidemiology, a cluster is defined by the set of infected persons believed to be related by a common etiologic process, regardless of their personal interrelationship.

Social scientists have studied social diffusion along lines that are very similar to the aforementioned SIS models for quite some time, but the details of interaction structure have only recently been considered seriously. They did not fully realize that a network perspective was crucial to understanding disease dynamics until the outbreak of the HIV/AIDS epidemic, when serious empirical studies began to appear (Klovdahl 2001).

In the late 1990s, physicists became interested in the study of complex networks. In contrast to much of the traditional network analysis, which focused on the relationship between single nodes and their network, this new line of research has concentrated on identifying classes of networks and their properties. The classification is based on studying the degree distribution, ie, the number of links, *k*, connecting to a node, which relates back to our comments on the number of contacts in Section 3. So far, three types of networks have been identified based on the their degree distributions, *P(k)*: single-scale, broad-scale and scale-free networks (Amaral et al. 2000). In a scale-free network, nodes do not have a typical number of edges. Most nodes only have a small number of edges, although a significant number of these nodes have a large number of edges and all the intermediate frequencies of edges are represented. Consequently, the standard deviation from the mean is extremely large and grows with added observations. Consider the following comparison with a well known-single scale parameter (with a Gaussian distribution): if the height distribution of humans were scale-free, we would have 100m-tall men and women walking around among us. The mathematical form of a scale-free degree distribution is shown as a power law, $P(k) \sim k^{-\alpha}$. Typically, a power-law distribution is identified by plotting the frequency distribution of connectivity in a graph with logarithmic axes. In such a graph, the power law will take the form of a straight line, as shown in Figure 5 below.

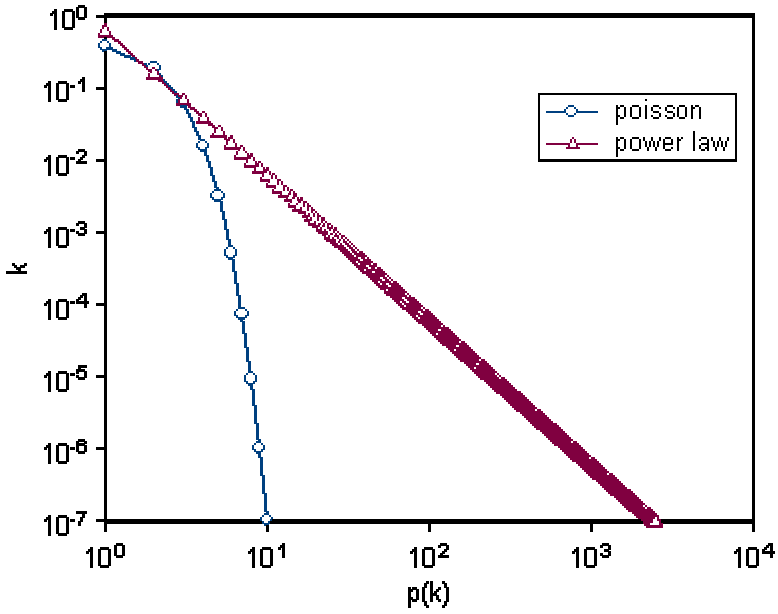

Figure 5. Comparison of the functional form of Poisson and power-law distributions for connectivity, *k*, with logged axes.

As can be seen from Figure 5, the tail of the power-law distribution declines at a much slower rate than other probability distributions, such as the Poisson distribution shown here. If dealing with power-law phenomena, we should bear in mind the possibility that an extreme number could occur from a random sample. The concept that the network of sexual contacts is scale free, with a degree distribution following a power law, helps explain why we observe such a wide spread in the number of sexual contacts (Liljeros et al. 2001).

It is interesting to note that a slope $a<2$ means that the standard deviation becomes infinite in populations of infinite size. While the human population is clearly not infinite, this matter also has implications for finite populations. Recent studies have demonstrated that an epidemic can develop under conditions of very low contagiousness in a scale-free network (Pastor-Satorras and Vespignani 2001). Another property of scale-free networks is that, despite their high susceptibility, they are very sensitive to the strategic removal of nodes. This is a significant factor in preventing the spread of STIs because a network will quickly separate into distinct components if just a few active individuals are isolated (or modify their behaviour) (Albert, Jeong and Barabasi 2001), thus preventing the emergence of epidemics. Recent refined analysis, however, concludes that this does not hold true for assortative mixing (Newman 2002). It is also worth noting that epidemics can reproduce via a considerably lower number of infected persons at any given point in time in a scale-free network than in the case of random mixing (Pastor-Satorras and Vespignani 2001).

4. War, Trade and Global Tourism

Clearly, social interaction is key to our understanding of disease dynamics. The random interaction assumption was a parsimonious mathematical construct, but it is at odds with people's intuitive ideas about social life and, as we have seen, with scientific knowledge on epidemics and disease contagion. For STI-clinicians in many countries, contact tracing has become standard procedure. Hopefully, it will also become normal practice in more countries and on an international scale. Moreover, a growing understanding of the

importance of contact patterns is also slowly filtering through to the work of epidemiologists around the world. As this is happening, it is highly important that social and behavioural scientists catalogue and deliver their cumulative knowledge on social behaviour and social interaction, and the way in which it is changing.

The interplay between human behaviour and disease dynamics and evolution is interesting. The history of mankind is full of examples where changes in human practice bring about new diseases, and where movement and new interaction patterns between people serve to introduce new microbes into virgin populations. Conversely, history gives as many examples where diseases have a dramatic impact on social life, and ultimately, on human life itself. Trade and wars used to be the engines of this interplay.

For as long as man has fought wars, invading armies have been effective carriers of disease and have provoked large migrations, with people fleeing the fighting and the invaders. It is believed that when the Huns came sweeping in from China in the first century they sparked a series of epidemics that eventually undermined the Roman Empire. Disease has also changed the odds of war: consider, for example, when a large part of Napoleon's army perished from typhus in the Russian campaign in 1812, or when the plague broke Athens' domination over Sparta in around 430BC (Karlen 1996). It has also been demonstrated that smallpox, rather than military or technological superiority, was the main reason behind the Europeans' rapid and full domination of every corner of the New World. Indeed, the concept of coupling of war and epidemics rose to such dominance in the last century that some claim it has become a constraint to historians of social history and medicine (Cooter 2003). For example, it is now suggested that cargo ships were responsible for bringing leprosy to the Mediterranean region in around 400BC, rather than Alexander the Great's army returning from the Indian campaign (Mark 2002).

Trade ships were excellent containers for nurturing diseases 'because both passengers and crew typically had to endure poor hygiene, poor nutrition, and cramped quarters, especially on long voyages. All of these factors provided an excellent environment for the incubation and spread of disease (Mark 2002)'. Apart from the crew and passengers, cargo ships would occasionally carry livestock and domestic fowl, which are also carriers of diseases that are potentially hazardous to humans. Another, and certainly more terrifying, passenger was the rat, specifically the black rat. This rat, which could climb mooring ropes, was known to carry many diseases, such as the plague given the infected-flees to which it was host. Through trade and exploration, disease could cross oceans and thus travel from continent to continent.

By the 18th century, Europe had spread its disease pool across the world and domestic European diseases have been endemic since then. On the other hand, new and hostile microbes from the rest of the world are still occasionally introduced into European populations, without causing much sustained harm so far. However, the relatively harmless disease environment that has emerged in Europe over the last 300 years also poses a formidable threat. For example, there had been no known population in the world without immune defences to smallpox since the day Columbus set foot on the American continent, at least until the dawn of the 21st century. Now, continental Europe constitutes such a virgin population.

Although global trade is more widespread than ever and despite the fact that the world still sees more than its share of war, terror, and streams of refugees, global tourism and business travel probably constitute the most lethal and hard-to-control threats to Europe's open societies. In the Western world, millions and millions of people travel on a regular basis. Moreover, they now travel more frequently, cover longer distances and at faster speeds than ever before. Of course, the same applies to goods, which are transported in larger volumes, longer distances and in less time. From a structural perspective, the

growing interaction between societies, organizations, groups, and individuals is contemporary society's distinguishing characteristic. In the fields of politics, business, and academia, there are virtually no boundaries to interaction and exchange. Nevertheless, increasing interconnection may not be the most noteworthy, or even the most significant, of the current changes.

Referring back to Figure 2 and the small world network, it seems highly likely that the world has been 'small' for quite some time and at least since the age of reason. Thus, the remarkable change that has and is taking place is not that clusters are being connected, even across great distances, but rather the time-scale of these connections. Journeys once took weeks or months by ship, but are now covered in less than 24 hours –eg, Jakarta is a mere 15 hours away from Amsterdam by KLM–. Alas, we are living in a time-compressed, small world. It is highly unlikely that SARS would have spread outside rural China had it not been for the speed and efficiency of modern air travel, in conjunction with international aid concerns in this case. If the disease, which has a very short incubation period and an acute course of illness, had been borne in a caravan of mules, it would probably have reaped its last victim among the caravan drivers. However, in 2003 SARS could spread from Hong Kong to North America and Europe, claiming several lives, within a fortnight (Leung and Ooi 2003).

The number of people travelling to, from and within Europe by air each year is formidable. Looking at passenger traffic alone, four of the world's ten most-frequented international airports are European (London Heathrow came in third, while Frankfurt, Paris, and Amsterdam ranked seventh, eighth and ninth, respectively). Considering arrivals, departures and direct transits in 2002, a total of 63mn passengers passed through Heathrow, while Frankfurt and Charles de Gaulle had 48mn each, and Schiphol saw 40mn passengers. Madrid-Barajas was ranked 13th, with close to 40mn passengers in 2002 (ACI 2003).

Stockholm's remote Arlanda airport had 16.5 million passengers in 2002, with a total of 123,000 landings, including direct connections from Europe mainly, but also from the US, Central America, Northern Africa, Russia and Asia (LFV 2003). Given the efficiency of the air traffic network, most of the world is within reach via just a few connections. Returning to Madrid's Barajas airport, Figure 6 depicts the number of airports in the world reached as a function of connections. Barajas has direct connections to 105 airports, while just one transfer puts another 1,100 airports within reach and a mere three connecting flights enables 2,500 different domestic and foreign airports to connect to Madrid.

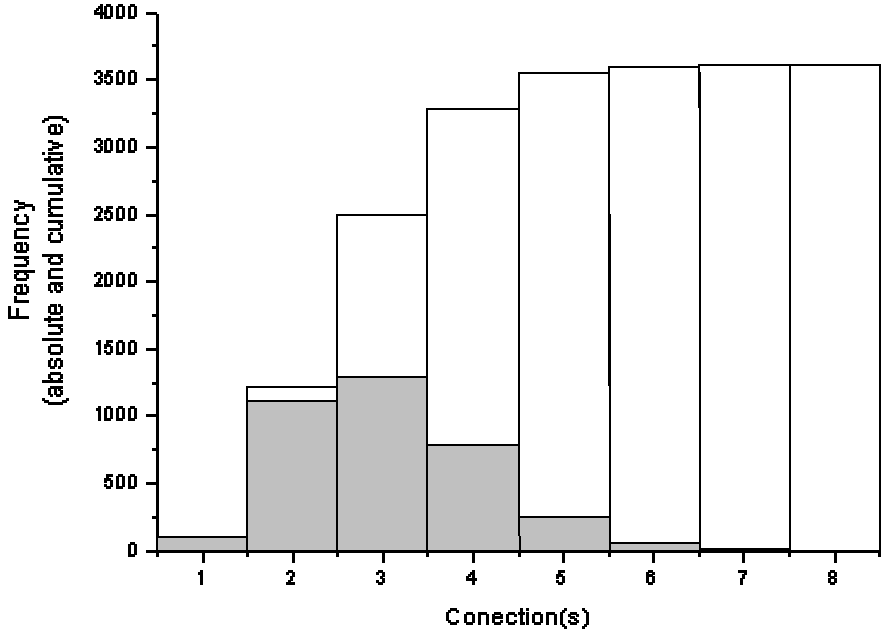

Figure 6. The number of airports worldwide reached from Madrid-Barajas airport as a function of the number of connecting flights (numbers reproduced by courtesy of Luis Amaral).

International tourism accounts for am increasingly large part of the volume of passenger air traffic. Since 1990, international tourism has grown on every continent: in 1990 Africa had 15mn international tourist arrivals compared with 29mn in 2002. Over the same period, the number arrivals increased from 58mn to 131mn in Asia and the Pacific, from 93mn to 115mn in the Americas, from 10mn to 27mn in the Middle East and from 280mn to 400mn in Europe (WTO 2003). Clearly, intra-continental tourism accounts for a sizeable proportion of this growth, but inter-continental tourism is also bound to have grown over the period. In 1995 the fraction of long-haul tourist arrivals varied on a regional basis: in Africa, the Middle East and South Asia most arrivals came from within the region, but 20% percent of arrivals in the Americas was from outside the region, while almost 60% of the international tourist arrivals in Europe came from outside Europe (WTO 2003).

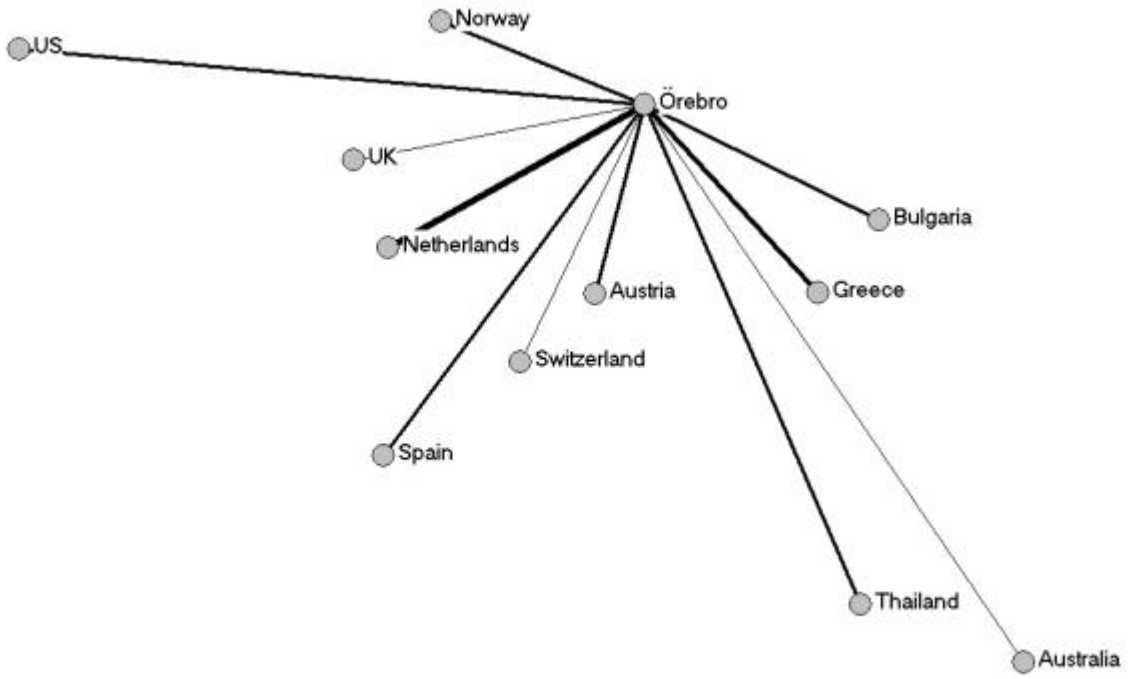

Figure 7. Country where Chlamydia was contracted by 22 patients at Örebro STD clinic in 1999-2000 (Falk *et al.* 2003).

With respect to disease contagion, the SARS epidemic recently reminded the world of the potential downside of international air travel. The STD-clinic of a small county hospital (Örebro) in Sweden provides another, less dramatic illustration of the risks posed by global interaction. A study of 231 patients who tested positive for genital *Chlamydia trachomatis* at the Örebro STD clinic between 1999 and 2000 found that 22 patients had contracted Chlamydia outside Sweden (Falk *et al.* 2003).

As shown in Figure 7, these 22 Chlamydia patients contracted the disease in 11 different countries, some of which are within very short distances –like Norway– while others are further afield –like Australia–.

5. Conclusion

Much of the threat to contemporary society from infectious disease stems from our modern lifestyle. As interaction and contact between people become increasingly compressed in time and space, communicable diseases will find it easier to spread both quickly and across huge geographical distances. In the case of certain diseases, it will no longer make sense to talk of different populations. Indeed, with respect to SARS, the world population seems to define the disease's target population. In this context, what preventive measures can be taken to protect society against this threat?

In closed societies, closing borders could, of course, be an option. Closing borders is an effective way to hold off foreign diseases, but it is not failsafe, as the citizens of Kaffa found to their cost when corpses came flying over the city walls. Border control is already a reality and, for example, has so far managed to protect Australian crops and animals from alien maladies. However, it is one thing to scan goods and baggage for food and other organics at airports, but quite another to scan potentially infected passengers. During the SARS epidemic, many countries set up screening stations for passengers arriving from the Far East, but no official evaluation of these measures is available yet.

In the light of previous discussions, we note that monitoring is essential. Almost all the available knowledge on epidemics derives back to observations of actual epidemics. Communicating risks to the public effectively is ever more crucial when individual freedom is high. Contact tracing is becoming harder in our contemporary society, but will still have an important role to play.

Achieving a good level of surveillance requires efficient cross-national cooperation in epidemiological research and practice. As with many other contemporary problems, issues related to diseases are best dealt with on-site. Thus, the best protective measures that Europe and the rest of the western world can take is to support the prevention against and treatment of diseases in countries were potential epidemics linger.

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