The talk of the town: modelling the spread of information and changes in behaviour

Sebastian Funk and Vincent Jansen

Abstract Changes in host behaviour can influence the course of a disease outbreak. These changes can be triggered by public campaigns and mass media reporting, but also by person-to-person communication and influence from peers. Here, we describe a model in which awareness of the presence of a disease can spread in a population, and influence the spread of the disease itself through protective measures that people can take. We describe the dynamics of disease spread, focusing, in particular, on the relation between awareness and proximity of disease in the network.

1 Human Behaviour and Infectious Diseases

Human behaviour is intricately linked with the spread of infectious diseases[27, 9]. After all, transmission of an infectious disease depends on *contact* of some sort, either with another infected individual or with an environmental reservoir. The rate of transmission depends on the intensity and rate with which we make such contacts. For instance, the rate of transmission of a sexually transmitted disease is linked to the behaviour that governs the frequency with which sexual contacts, or the change in sexual partners. An element of human behaviour is therefore contained in any mathematical model for an infectious disease, in a way that may be as simple as a fixed *contact rate* in a traditional Susceptible-Infected-Recovered (SIR) model [3].

Sebastian Funk
Department of Infectious Disease Epidemiology and Centre for the Mathematical Modelling of Infectious Diseases, London School of Hygiene & Tropical Medicine, Keppel St, London WC1E 7HT, UK, e-mail: sebastian.funk@lshtm.ac.uk

Vincent A. A. Jansen
School of Biological Sciences, Royal Holloway, University of London, Egham TW20 0EX, UK, e-mail: vincent.jansen@rhul.ac.uk

There are situations, however, in which it may become desirable to model behaviour *explicitly*, that is to include it in the model dynamics and allow it to change over time. Such situations arise, for example, when behaviour depends on overall prevalence of a disease (so-called *prevalence-elastic* behaviour), on information which is communicated concurrently with the spread of an infection, or on extrinsic factors such as perceived adverse vaccine effects [16] or severe outcomes associated with a given disease. In these cases, behaviour can be an important source of heterogeneity in the population, it can change over time, and it can both affect and be affected by the dynamics of the disease itself.

While it might be impossible to model the behaviour of an *individual*, it has been suggested that *collective* human behaviour can be described using computational and mathematical models [13]. These have been applied, for example, in sociology [26], economics [20], anthropology [21], and to crowd behaviour [14, 15] and vehicle traffic [35]. In order to study the collective behavioural response to the spread of an infectious disease, one needs to consider the following questions:

- **What causes people to act?** We are all exposed to a variety of sources of information, and have different tendencies to act on them. Collectively, are we more likely to respond to public health messages, or to be influenced by the behaviour of our peers? Does it influence us to perceive high prevalence of the disease in our neighbourhood? It is known that humans tend to overestimate the risk of extreme outcomes [24]. How does this influence our response to an outbreak of a given disease? All of these factors will depend on the specific disease, the media and public health response, and a variety of other cultural and historical factors. It remains an open challenge to identify common patterns in the answer any of these questions.
- **How do the behavioural reactions influence the disease dynamics?** Depending on the disease being studied, behavioural changes can have an impact on the dynamics of the disease in a variety of ways. For airborne diseases, individual behaviour that has the potential to affect the dynamics of the disease can range from social distancing or voluntary quarantine to wearing face masks, hygienic practise, usage of prophylactic or other medication and vaccination. Beyond these, more extreme measures such as mass flight from an area in which a disease is present, or the erection of road blocks to stop a disease from expanding geographically have occurred in history. All of these have the potential to influence the epidemiology of an infectious disease in different ways.

In the light of this wide range of possibilities for behavioural influences and outcomes, it is important to identify their common elements, in order to understand the overall influence of human behaviour on infectious diseases. Previously [9], we suggested to distinguish between *prevalence-based* behaviours, based on information directly related to the disease prevalence, or *belief-based* behaviours based in on information not directly related to disease prevalence. Belief-based behaviour can have its own dynamics independently of the disease dynamics, as the behaviour can be copied from one person to the next. This is the case, for example, for behavioural changes that are based on the spread of some sort of information, be it a

rumour, awareness or fear. Moreover, we suggested to distinguish whether individuals source their social neighbourhood for (*local*) information to act on or behaviour to imitate, or whether they act on publicly available (*global*) information. Lastly, for the influence on disease dynamics, we suggested to distinguish whether a given behaviour would change the state of an individual with respect to a disease (e.g., by turning someone from being susceptible to being immune via vaccination), whether it would change the parameters of spread itself (e.g., by leading to speedier recovery from infection), or modify the contact structure between individuals (e.g., if people avoid contact with those infected). Of course, all these distinctions are somewhat arbitrary, and in reality our reactions will rarely fit perfectly in either of these categories.

2 The Spread of Awareness

Ideas, innovations, rumours or a cultural practice can spread in a way not entirely dissimilar from the spread of a disease: those who have not yet been "infected" (i.e., convinced or informed) can become so by coming in some form of (not necessarily physical) contact with someone who has [10, 4]. The spread of rumours or ideas has been described as "infection of the mind" [30] or "thought contagion" [25]. The analogy between the spread of information and communicable diseases seems to have been first proposed by Landau [23] and later, independently, by Kendall [18] and Goffman and Newill [11]. Generally, studies on models of rumours have concentrated on similar questions to epidemic models, i.e. the probability of it affecting a large part of the population and the fraction which hears of it over a given period of time. The work of Landau [23] is based on the epidemic model of Kermack and McKendrick [19] and considers cases where probability of transmission depends on the age of the rumour, or the time since a given spreader heard it first. A similar model was proposed by Landahl [22], who had individuals transmit a message an average of f times, f being a function of time. The stochastic model of Daley and Kendall [7, 6] added a "stifler" class for those who carry the rumour have lost interest and no longer spread it, just as Goffman and Newill [10, 11, 12] did in their deterministic models.

After the flurry of activity on models of rumour spreading in the 1950s and 60s, interest resurged in the past 10 years, in line with increasing interest in network theory. A number of studies applied variants of the model of Daley and Kendall [6] to different network settings [36, 37, 29, 28] to study the interplay between topology and model dynamics. Nekovee et al. [30] extended this to include the possibility that individuals lose interest or forget about the rumour. Agliari et al. [1, 2] proposed a model in which the information contained in the rumour decays as it spreads through the population, an idea we will get back to in the following.

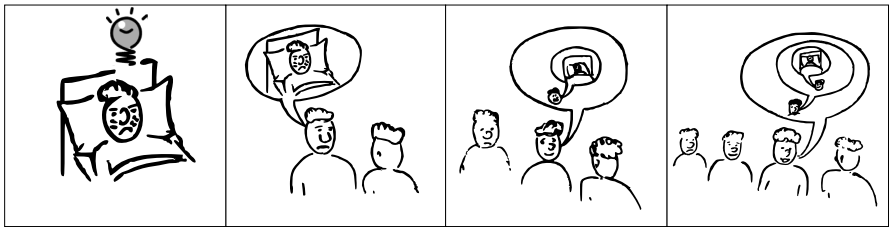

Fig. 1 A model of awareness spread with decay of information. Left to right: Awareness originates in an infected case. As it spreads from person to person, the level of awareness gets updated with the distance from the source, and in this way loses some quality, in the sense that it will cause less of an incentive to change the behaviour of the recipient.

3 Spreading Awareness and Behavioural Changes

We are interested in a situation were people change their behaviour upon becoming *aware* of the presence of a disease. In particular, we want to investigate what happens when awareness can *spread*, in the sense outlined in Sect. 2. We understand this to be awareness of the (perceived) presence of the disease, and assume people to change their behaviour once they become aware, by protecting themselves from getting infected. We consider a scenario where first-hand information originates via acutely infected cases but subsequently spreads independently of the disease.

There is anecdotal evidence that this kind of word-of-mouth and person-to-person spread of awareness can occur when an infectious disease is around. From the lepers' bell to notes on a nursery door, from the millions of text messages exchanged during the outbreak of severe acute respiratory syndrome (SARS) in Ghuangzhou in 2003 [34], to online health fora [5] and the exchange of twitter messages concerning vaccination against pandemic influenza H1N1 [32], examples for the exchange of information relating to the presence of an infectious disease are numerous.

In our model (see box below for details), we consider the population to be connected in a *contact network*; that is, any two members of the population are connected if they could potentially transmitted the disease between each other. In addition, we people are connected on a second network over which awareness spreads. Connections can be present over both networks or only on one of them, that is people could be connected on an online forum but not be able to transmit disease between each other because they never get into contact, or vice versa, or they could be connected on both networks.

Lastly, we use assume the *quality* of information, or the probability of individuals to act on it, to decay as it spreads in the population (Fig. 1), an idea first formulated by Agliari et al. [1, 2]. This reflects that we are interested in local and timely information, which will lose its value both with time and (network) distance.

Mathematical details of the model

As described in [8], we divide the population of size N into susceptibles (S), infected (I) and recovered (R) [3]. Further, we divide the population according to the level of awareness they possess, here understood as awareness that the disease is present nearby. The level of awareness is denoted i, with $i = 0$ denoting the highest level of awareness, decreasing as i increases. Awareness spreads at rate α and is lost (forgotten) at rate λ. Each time awareness is passed on to someone else, its level increases by 1 (in other words, a bit of quality is lost every time awareness is passed on). The infection spreads at base rate β, and recovery from disease occurs at rate γ. Susceptibles of awareness level i are assumed to reduce their susceptibility (i.e., their infection rate) by a factor ρ^i, so that $0 < \rho < 1$ is the decay constant of awareness. New generation arises in infected individuals at rate ω.

The resulting set of equations is (see also Fig. 2)

$$\frac{dI_i}{dt} = -\lambda I_0 + \omega(I - I_0)$$

$$\frac{dR_i}{dt} = +\gamma I_0 - \lambda R_0$$

$$\frac{dS_{i>0}}{dt} = -(1-\rho^i)\beta\frac{S_i}{N}I - \alpha\frac{S_i}{N}\left(\sum_{j=0}^{i-2} N_j\right) + \alpha\frac{N_{i-1}}{N}\left(\sum_{j=i+1}^{\infty} S_j\right) - \lambda S_i + \lambda S_{i-1}$$

$$\frac{dI_{i>0}}{dt} = +(1-\rho^i)\beta\frac{S_i}{N}I - \gamma I_i - \alpha\frac{I_i}{N}\left(\sum_{j=0}^{i-2} N_j\right) + \alpha\frac{N_{i-1}}{N}\left(\sum_{j=i+1}^{\infty} I_j\right) - \lambda I_i + \lambda I_{i-1}$$

$$\frac{dR_{i>0}}{dt} = +\gamma I_i - \alpha\frac{R_i}{N}\left(\sum_{j=0}^{i-2} N_j\right) + \alpha\frac{N_{i-1}}{N}\left(\sum_{j=i+1}^{\infty} R_j\right) - \lambda R_i + \lambda R_{i-1}$$

where $I = \sum_i I_i$ and $N_i = S_i + I_i + R_i$.

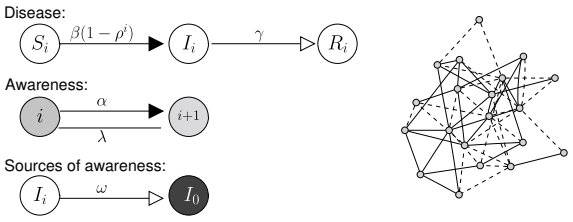

Fig. 2 Left: Schematic diagram of the model. Transitions are marked occur within nodes (empty caps), or across the disease (solid) or awareness (dashed) network (solid caps). Right: A network with two types of edges (disease, awareness)

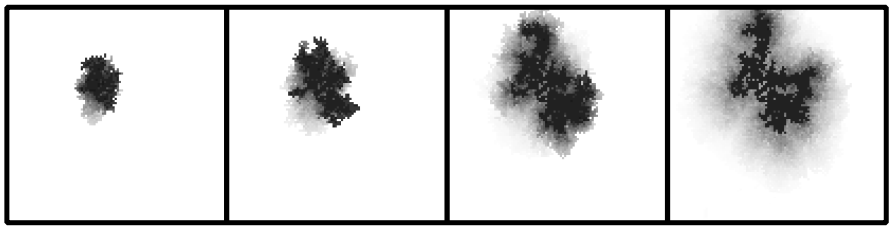

Fig. 3 Snapshots from a simulation of the disease-awareness model on a triangular lattice, progressing in time from left to right. The black patch in the centre is where the disease has reached, surrounded by susceptibles in white to dark grey, with increasing awareness levels the darker they are plotted. In the rightmost panel, the outbreak has stopped. Coloured figure and movies available in the supporting online material of [8].

4 Dynamics of the Model

In the following, we describe the phenomena observed in simulations of the model. Readers interested in analytical backing of these results are referred to [8].

4.1 Relative Timescales of Spread

The dynamic interaction between awareness and disease that results from our model depends highly on the relative timescales of the two processes. If awareness spreads much faster than disease, it will reach its final distribution among the population before the disease spreads widely. In this case, awareness provides merely a uniform backdrop that is static on short timescales. This is the scenario we would expect for information disseminated by the mass media in response to an outbreak of a novel disease. If, on the other hand, the disease spreads much faster than awareness, it will encounter an unaware population which only retrospectively might receive information on the outbreak. In other words, this situation is similar to one in which awareness does not exist at all. In both of these cases, there is no need to model the dynamical interaction of awareness and disease explicitly, and any impact of awareness on spread can be subsumed in the parameters of the disease model.

If, on the other hand, both spread on similar timescales, the effect of the dynamic interaction between awareness and disease becomes more sensitive to the details of network and spatial structure, as we will describe in the following sections.

4.2 Local Quenching of Disease Outbreaks

If disease and awareness operate on similar timescales, the dynamical interplay between the two can result in them having a strong impact on each other, with network

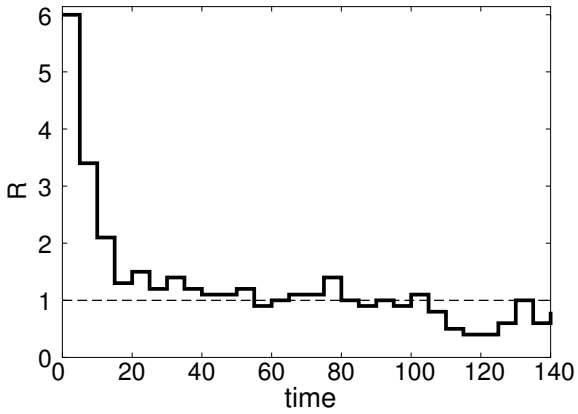

Fig. 4 Change of the effective reproductive number R in time in a simulated outbreak on a triangular lattice, starting with a single infected individual.

structure and overlap becoming more important. Let us first assume that the networks of both infection and awareness are the same. In that case, as soon as awareness originates in those infected and spreads in the population, it starts to quench the outbreak locally because high-quality information (which has a high tendency of changing the behaviour or people) is near the outbreak itself. This results in a lot of awareness appearing around infected cases, which can slow down an outbreak until the disease reaches another unaware part of the population, or it can even stop an outbreak altogether (Fig. 3).

If the behavioural reaction is not strong enough to stop an initial outbreak completely in its tracks while, on the other hand, it is strong enough to slow down the spread of the disease locally, the course of the outbreak is changed: if we follow the the reproductive number R over time it moves around 1 for a long time during the outbreak instead of declining monotonically, as would be expected without the effect of the behavioural response (Fig. 4). This is not dissimilar from patterns observed for the influenza pandemic of 1918, where similar variation of R in time has been attributed to the possible impact of individual reactions [31], or the irregular pattern in the epidemic tail of the 2001 UK Foot and Mouth Disease epidemic [17]. The changing dynamics are reflected in the spatiotemporal pattern which changes from a simple diffusive spread with radial outward progression from the source of the outbreak to a much more irregular, patchy shape, characteristic of critical phenomena (Fig. 3) [33].

4.3 The Importance of Clustering and Network Overlap

The local quenching of outbreaks described in Sect. 4.2 can only occur when there exists a notion of locality in the population in which disease and awareness spread. For this to be the case, the network needs to possess a *clustered* structure. In network science, clustering traditionally denotes the probability for there to be a connection

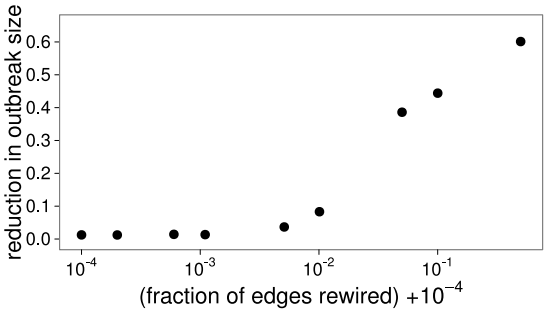

Fig. 5 Relative reduction in outbreak size in simulations of the disease-awareness model on a triangular lattice, with a part of the disease-edges randomly rewired, given as (mean outbreak size with awareness) / (mean outbreak size without awareness). The averages are over 100 simulations on a lattice of 10,000 nodes.

between two individuals who are both connected to a third individual (or, the probability of two friends of someone to be friends amongst themselves). Here, we mean, more generally, the fact the distribution of shortest paths from a given individual to other individuals in the networks has a steep slope or, in other words, that very few individuals are close (only a few hops on the network away), while most are distant (many hops away).

This alone, however, is not enough to guarantee a strong impact of the spread of awareness on outbreaks. For this, we need the networks over which awareness and disease spread to display a strong degree of overlap, in the sense that contacts on one network need to be contacts on the other, too. This guarantees that the individuals closest to those infected (which also act as sources of high-quality information) are the ones with the best information. Clustered structure then allows this information to be spread to individuals who themselves are not distant from the source of infection, protecting these before the disease can get to them.

This effect can be observed clearly when considering the model on an (overlapping) triangular lattice (i.e., a very clustered structure with a strong sense of locality) in which some of the disease edges are randomly rewired. As a consequence of this rewiring, the potentially infectious connections of an infected node have a certain probability of pointing to a region in the disease network which is not local to that node on the awareness network. If that is the case, the disease can escape regions of the network where it is locally suppressed as people around an infected cluster protect themselves. The greater the probability of such escape, the weaker the effect that awareness spread can have in containing outbreaks (Fig. 5).

5 Conclusions

We have described the dynamics of a model for the concurrent spread of an infectious disease and awareness to its presence, and assumed this awareness to be the trigger for behavioural reactions. Local interactions between disease and awareness only become relevant when the two spread on similar timescales. In that case, we

can observe local quenching of disease outbreaks as those that are most at risk become aware and protect themselves. When this happens, the spatial progression of an outbreak changes from a simple diffusive process to a situation where small outbreaks flare up before they get contained locally. This effect is the strongest when the networks over which infection and awareness spread are overlapping and clustered. If this is not the case, for example when the infection can escape to unaware populations with a certain probability, the behavioural reactions become less effective in quenching outbreaks.

Whether any of this happens in reality remains an open question. While all parts of our model have been informed by anecdotal evidence, it can be quite difficult to quantify the different components and their relative impact. Still, there are some things to be learnt from the kind of study we present here. Recent studies of health behaviour show that the structure of networks of influence can play a role in how such behaviours become adopted in a population [5]. Here, we show that, if people are indeed influenced by their peers, it is the interplay between the network of influence with the network of infection that determines the effect on outbreaks. Moreover, if behavioural reactions can change the epidemiology of a given disease, one must be careful in extrapolating from observations in a disease-free situation to one where a disease is present. How exactly peer and media influence, the particularities of any given infectious disease, and the type and strength of behavioural reactions interact is notoriously difficult to establish. Still, it seems that innovative theoretical approaches used hand-in-hand with careful observational studies, for example using the digital traces we leave in our on-line interactions, have a role to play in shedding some light on what shapes our reactions to disease outbreaks, and how this, in turn, can affect the fate of an outbreak itself.

Acknowledgements The authors would like to thank Erez Gilad, Marcel Salathé and Chris Watkins for many useful discussions. S.F. would like to thank the EU FP7 funded integrated project EPIWORK (grant agreement number 231807) for funding. This work was supported by the UK Engineering and Physical Sciences Research Council grant number EP/D002249/1.

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