

# Modelling the influence of human behaviour on the spread of infectious diseases: a review

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Human behaviour plays an important role in the spread of infectious diseases, and understanding the influence of behaviour on the spread of diseases can be key to improving control efforts. While behavioural responses to the spread of a disease have often been reported anecdotally, there has been relatively little systematic investigation into how behavioural changes can affect disease dynamics. Mathematical models for the spread of infectious diseases are an important tool for investigating and quantifying such effects, not at least because the spread of a disease among humans is not amenable to direct experimental study. Here, we review recent efforts to incorporate human behaviour into disease models, and propose that such models can be broadly classified according to the type and source of information which individuals are assumed to base their behaviour on, and according to the assumed effects of such behaviour. We highlight recent advances as well as gaps in our understanding of the interplay between infectious disease dynamics and human behaviour, and suggest what kind of data taking efforts would be helpful in filling these gaps.

**Keywords:** epidemiology, infectious diseases, behaviour, vaccination

## 1. Introduction

Recent outbreaks of infectious diseases have brought pictures of empty streets and people wearing face masks to television screens and front pages, as fear of diseases of unknown fatality swept around the globe. Arguably one of the most striking aspects of these outbreaks were the reactions to the disease. During the outbreak of Influenza A (H1N1) in 2009, the effect on societies, partly through public measures but also through personal and uncoordinated responses, has been noticeable. The

public reaction to this disease was sustained and widespread, and interestingly, part of this reaction resulted from individual behavioural responses to the presence of the disease.

Historically, human behaviour has been intricately linked with the spread of infectious diseases (McNeill, 1976). In medieval times, the lethality of the bubonic plague caused people to “shun and flee from the sick and all that pertained to them, and thus doing, each thought to secure immunity for himself”, as Boccaccio vividly records in the *Decameron*. Equally compelling are the accounts of the citizens of the Yorkshire village of Eyam who voluntarily quarantined themselves to prevent spread of the plague from the village (Scott and Duncan, 2001). More recently, during the influenza pandemic of the early 20th century, people eventually stayed away from congregated places (Crosby, 1990). In 1995, a presumed outbreak of bubonic plague in Surat, India, caused widespread panic and flight of hundreds of thousands of people (Campbell and Hughes, 1995). When SARS broke out in the early 21st century, the usage of face masks became widespread in affected areas, and many changed their travelling behaviour (Lau et al., 2005). Prevalence-elastic behaviour, i.e. protective behaviour which is seen increasingly as a disease becomes more prevalent, has been observed both in the context of Measles (Philipson, 1996) and HIV (Ahituv et al., 1996).

While behavioural responses to the spread of a disease have frequently been reported anecdotally, there has been relatively little systematic investigation into their nature, or the effect they can have on the spread of the disease. Behavioural changes are sometimes cited in the interpretation of outbreak data to explain drops in the transmission rate (e.g. Riley et al., 2003; Nishiura, 2007), yet rarely is it detailed how these reactions can be quantified and captured in a systematic way. Mathematical models have been devised to study human behaviour in the context of, for instance, escape panic (Helbing et al., 2000), pedestrian trails (Helbing et al., 1997) and traffic jams (Wilson, 2008), but efforts to study human behaviour in the context of epidemics usually concentrated on judging the effectiveness of various institutionally enforced public health measures such as school closures (e.g., Hatchett et al., 2007; Bootsma and Ferguson, 2007). Recently, however, the impact of self-initiated actions on the progression of an infectious disease has received increased attention. After all, individual self-initiated behaviour can change the fate of an outbreak, and its interaction with disease dynamics requires proper understanding if we are to fully comprehend what happens when a disease spreads through human populations (Ferguson, 2007).

Here, we aim to systematically review the theoretical studies undertaken so far to study the impact of individual behaviour on the epidemiology of infectious

diseases. Our goal is two-fold. First, we propose a classification system that we believe is useful to help bring order to the complex diversity of models. Second, we aim to assess the contribution that the models can make towards understanding dynamics and control of human diseases, as well as the suggestions that follow as to how responses to outbreaks could be measured and quantified in a reasonable way.

We decided to apply two restrictions in selecting which models to include in this review. First, we will focus exclusively on self-initiated, voluntary behaviour, i.e., on scenarios where people first assess a situation based on both the information available to them and their beliefs, attitudes, norms etc., and then make a personal decision about how to respond to the given situation. This is different from scenarios where institutions make recommendations or impose new regulations and expect the public to comply with those recommendations and regulations. As an example, consider the decision to go to school during an outbreak of a disease. In the first case, a model could assume that individuals (or their parents) can make a personal decision about whether to go to school or not during the outbreak. In the second case, the model might make an assumption about school closure, and individual decisions about whether or not to go to school are implicitly excluded from the model. The second restriction is that we will focus exclusively on models that consider behavioural reactions to information from the outside world. Take again the school example: if a model assumes that the decision to go to school depends solely on whether an individual is infected or not (intrinsic factor), we would not include the model here. If, however, the model assumes that the decision depends at least to a certain extent on the outside world (e.g., how many people at the school are infected – an extrinsic factor), then we would include the model in the review. While the investigation of intrinsic factors and corresponding behavioural change has a strong tradition in modelling sexually transmitted diseases, particularly HIV and the psychological effects of different treatments, such as antiretroviral therapy (Baggaley et al., 2005; Blower et al., 2000), male circumcision (Williams et al., 2006; White et al., 2008) and a hypothetical vaccine (Blower and McLean, 1994), we chose not to include such studies for the sake of brevity and focus.

## 2. The SIR model and simple extensions

A simple model of infectious diseases is the SIR (Susceptible-Infected-Recovered) model (Kermack and McKendrick, 1927) which forms the basis of almost all disease models studied since. In the SIR model, the population is divided into three classes, where susceptibles (S) can be infected by those already infected (I) and subsequently recover (R), at which point they are immune to the disease or otherwise removed

from the population (Fig. 1). In the simplest SIR model, the transitions between these classes are all assumed to occur at a rate proportional to the number of individuals in the respective classes, as well as to constant rates of infection and recovery, respectively. That way, the incidence rate is proportional to  $\beta SI$ , where  $\beta$  is the infection rate, and  $S$  and  $I$  are the number of susceptible and infected, respectively. In this simple case, the dynamics of the system can be described by the following set of ordinary differential equations (Anderson and May, 1991)

$$\begin{aligned}\frac{dS}{dt} &= -\frac{1}{N}\beta SI, \\ \frac{dI}{dt} &= \frac{1}{N}\beta SI - \gamma I, \\ \frac{dR}{dt} &= \gamma I,\end{aligned}$$

where the last equation for the number of recovered ( $R$ ) is redundant as the number of individuals in the population  $N$  is constant and at any time  $N = S + I + R$ . More sophisticated models can explicitly include spatial or contact network structure, so that each individual in the population can be infected only by a constrained set of other individuals.

A number of studies have considered extensions of the simple SIR model in which the incidence rate is not bilinear in  $S$  and  $I$ , but a more general function  $f(S, I)$ , to include effects of saturation. One justification which was given for modifying the model this way is that "in the presence of a very large number of infectives the population may tend to reduce the number of contacts per time" (Capasso and Serio, 1978). While such models have been shown to yield rich complex dynamics (Liu et al., 1986, 1987), they rarely went into much detail about the precise impact of the behavioural reaction, or the dynamics of these reactions. In that sense, they form an intermediate step between the simple SIR model and the models reviewed in the following, which are more explicit in the incorporation of behavioural aspects to modulate the dynamics of the SIR model or the underlying contact network structure.

While beyond the scope of this paper, it is important to note that it is not a trivial exercise to associate the model parameters with values which accurately relate the model to data (Mollison, 1995). To do this, one usually needs sophisticated statistical methods allowing for proper estimation of model and parameter uncertainties. For example, statisticians and (control) engineers have developed sensitivity and robustness analyses, and recent developments in computational statistics (such as particle filters and sequential Monte Carlo in an approximate Bayesian

computation setting) combine the strengths of these approaches in a very flexible framework (e.g. Ionides et al., 2006; Toni et al., 2009).

### 3. Towards a classification of behaviour-disease models

To discuss and understand the implications of human behavioural responses to diseases, it is useful to recognise that there are two different processes at play. Human behaviour is based on attitudes, belief systems, opinions and awareness of a disease, and all these factors can change over time, both in an individual and in the population on the whole. The dynamics in these attributes is one element that is relevant to understanding the impact of behavioural responses to a disease. On the other hand, the transmission of the pathogen creates a second dynamic, as outlined above in the description of the SIR model. To fully understand the impact of human behaviour on infectious disease dynamics, we need to know how both these processes operate and interact.

There are various ways to model how certain types of behaviour change over time (Cavalli-Sforza and Feldman, 1981), but rather than going into the technical details of a particular way of modelling the causes and consequences of behavioural change, we will here focus on a few main aspects that are relevant for infectious disease dynamics in humans. We will not concentrate on the *mathematical* properties of the models or tools applied to analyse them, but instead focus on *conceptual* differences in the attempts to integrate elements of human behaviour in infectious disease models. With regard to the causes of behavioural change, all models that we are reviewing here make an assumption about the *source* of information, and an assumption about the *type* of information which people base their decisions on that will eventually result in a behavioural change (Table 1). With regard to the consequence of behavioural change, all models make an assumption about the *effect* of the change on the dynamics of disease spread (Table 2). We will now discuss these distinctions in more detail.

#### *Ia. Source of information*

Many models assume that the information on which people act – i.e., change their behaviour in a way that is relevant for the spread of disease – is available to everyone. Examples are any sort of news published by newspapers, TV stations, websites and other media channels, information published by public health authorities, etc. We will call this globally available information. On the other side of the spectrum, a few recent models assume that the information is taken from the social or spatial neighbourhood only. Examples are the spread of information by word

Table 1. *Classification according to source and type of information people base their behaviour on. Note that the studies of Epstein et al. (2008) and Funk et al. (2009, 2010) each appear in two categories because they consider both global and local spread of fear or awareness of a disease.*

	Belief-based	Prevalence-based
Local	Epstein et al. (2008) Salathé and Bonhoeffer (2008) Funk et al. (2009, 2010) Eames (2009)	Gross et al. (2006) Zanette and Risau-Gusmán (2008) Shaw and Schwartz (2008) Bagnoli et al. (2007) Perisic and Bauch (2009a,b)
Global	Tanaka et al. (2002) Bauch et al. (2005) Epstein et al. (2008) Coelho and Codeço (2009) Funk et al. (2009, 2010) Kiss et al. (2009) Tanaka et al. (2009)	Bauch et al. (2003) Bauch and Earn (2004) Del Valle et al. (2005) Chen (2006) Reluga et al. (2006) Codeço et al. (2007) d’Onofrio et al. (2007) Galvani et al. (2007) Vardavas et al. (2007) Basu et al. (2008) d’Onofrio et al. (2008)

of mouth, the assessment of the disease prevalence among acquaintances and in the local community, etc. We will call this locally available information. Thus, by source of information we mean whether individuals base their behavioural choice on publicly available information (*global*), or on information which comes from their social neighbourhood (*local*). The difference between globally and locally available information can be very important: in socially or spatially structured models, information can occur in clusters which in turn can have strong effects on disease dynamics. For example, the clustered occurrence of beliefs about vaccines against a certain disease can lead directly to the clustered occurrence of people susceptible to that disease (Salathé and Bonhoeffer, 2008). As another example, the local spread of awareness of a disease in the proximity of an outbreak can completely stop a disease from spreading (Funk et al., 2009, 2010). The importance of spatial effect on dynamics processes, in particular in an ecological context, has long been recognised (Durrett and Levin, 1994) and is directly applicable to the topic at hand.

#### *Ib. Type of information*

In the vast majority of models reviewed here, the information that individuals base a behavioural change on is the prevalence of a disease. However, there is a

plethora of other information that will possibly affect the decision to behave in a certain way, and such information can be completely independent of the actual disease prevalence. The mismatch between subjective and objective assessment of risk has been demonstrated experimentally (Young et al., 2008), and some of the key factors contributing to this mismatch – media misrepresentation (Frost et al., 1997) and social amplification of risk (Kasperson et al., 1988) – are well understood. We thus propose a simplifying classification of the type of information into "directly relating to disease prevalence" and "not directly relating to disease prevalence". The crucially important distinction between the two classes is that latter relates to information whose dynamics are at least partially independent of the actual disease dynamics (for example, a belief may initially have originated from prevalence-related information, but subsequently spread independently of current prevalence). From here on, we will use the terms "belief-based" and "prevalence-based" to separate the two types of information due to which behavioural change can occur.

There are many alternatives to such a classification, but we argue that the proposed classification captures the key differences between the two categories. One such difference is that belief-based behavioural change can occur at time points (and on time scales) that are very much detached from the temporal dynamics of infectious diseases. For example, decisions about vaccines can be influenced by opinions and beliefs that can spread much faster than the corresponding disease, and decisions about childhood vaccines are mostly made in the absence of actual disease outbreaks. Secondly, prevalence-based information is by definition objective, while belief-based information can be highly subjective. A case in point is the belief that the measles-mumps-rubella (MMR) vaccine can cause autism, a belief which has spread widely despite the overwhelming evidence that rejects such a causality (Stratton et al., 2004). Thirdly, prevalence-based behavioural change is based on a single piece of information (prevalence), while belief-based behavioural change can be caused by multiple (and even conflicting) pieces of information.

## *II. Effect of behavioural change*

If the behavioural change is to be relevant for infectious disease dynamics, it must affect either (a) the disease state (S, I, or R – see above) of the individual, (b) the infection rate or the recovery rate, or (c) the contact network structure relevant for the spread of disease. We classify the models accordingly. For example, in models where a behavioural change constitutes a decision to vaccinate, the individual will leave the susceptible state (S) and move directly into the immune state (R). As another example, consider a model where disease prevalence causes increased social

Table 2. Classification according to the effect of behavioural change. Note that the study of Epstein et al. (2008) appears in two categories because it considers both a change in parameters as fearful individuals avoid all infectious contacts, as well as a change in contact structure as individuals free from a disease.

Behaviour changes disease state of individuals	Behaviour modifies model parameters
Bauch et al. (2003)	Tanaka et al. (2002)
Bauch and Earn (2004)	Del Valle et al. (2005)
Bauch et al. (2005)	Bagnoli et al. (2007)
Chen (2006)	Epstein et al. (2008)
Reluga et al. (2006)	Funk et al. (2009)
Codeço et al. (2007)	Kiss et al. (2009)
d’Onofrio et al. (2007)	Tanaka et al. (2009)
Galvani et al. (2007)	
Vardavas et al. (2007)	Behaviour changes contact structure
Basu et al. (2008)	
d’Onofrio et al. (2008)	Gross et al. (2006)
Salathé and Bonhoeffer (2008)	Epstein et al. (2008)
Coelho and Codeço (2009)	Shaw and Schwartz (2008)
Eames (2009)	Zanette and Risau-Gusmán (2008)
Perisic and Bauch (2009a,b)	

distancing – such a behavioural change could be modelled either as a decrease in the transmission rate, or as reduction in the number of intensity of contacts, provided the model explicitly assumes a contact network structure.

The class of models that assumes a change in disease state as a consequence of a behavioural change are models dealing with vaccination decisions. In the simplest case, the decision to vaccinate results in moving directly from disease state S (susceptible) to disease state R (immune). The class of models that assumes a change in parameters or changes in population structure as a consequence of behavioural change are models dealing with the effect of people reducing their exposure to diseases as a reaction to the presence of either the disease or certain beliefs about the disease. In the following, we will discuss these two major classes of models.

#### 4. Rational decisions and voluntary vaccination

The epidemiology of many well-known vaccine-preventable diseases is subject to human behaviour. Common childhood diseases, such as chickenpox and measles (Phillipson, 1996), provide a timely example: The decision whether to vaccinate a child or not is ultimately a personal decision and thus has a strong behavioural com-

ponent. An arguably even more striking example of behaviour affecting infectious diseases is provided by the so-called measles and chickenpox parties, where parents expose their susceptible children directly to other infected children. The vaccination policies of a large number of countries are based on voluntary compliance, and drops in vaccination coverage have led to increased interest in so-called rational vaccination decisions and their effects on the epidemiology of vaccine-preventable infectious diseases. While psychological studies have led to considerable insight into causes for behaviour with regard to disease (Norman and Conner, 2005), and to the concept of *health belief models* to explain the concepts and notions underlying behavioural choices, epidemiological modellers have turned to game theory and focused on a dilemma introduced by voluntary vaccination. If vaccination is perceived to come with risks or side-effects, it can be presumed better to opt out and not take any risk while relying on the rest of the population to keep the coverage high and provide herd immunity. Besides refusal of vaccination due to religious or other beliefs and the risk of simply forgetting to vaccinate due to a lack of awareness, the choice to “free-ride” and exploit the vaccination behaviour of others can reduce the general level of vaccination. Concerns about proclaimed risks of vaccines can drive widespread refusal of vaccination and consequent drops in vaccine uptake, as has been the case with pertussis in the 1970s (Gangarosa et al., 1998) and, more recently, with the MMR vaccine (Jansen et al., 2003).

By making a rational choice of whether to vaccinate themselves or their children, individuals weigh up the costs and risks associated with vaccination with its benefit of removing or reducing the risk of infection. Game theory (von Neumann and Morgenstern, 1944; Weibull, 1995) provides a tool to study simple conflicts of individuals choosing between actions of different costs and benefits while acting perfectly rationally to maximise their own gain. If game theory is applied to vaccination decisions, one finds that the vaccination level attained from individuals acting only in their best self-interest is always below the optimal for the community (Fine and Clarkson, 1986) because of the protection gained by others that do vaccinate and the avoidance of potential risks associated with a given vaccine (see Box). This would make it impossible to eradicate a disease under voluntary vaccination (Bauch and Earn, 2004; Chen, 2006).

Early studies of the impact of this conflict attempted to parametrise risks and benefits associated with a given vaccine and potential infection using realistic estimates. Using a model for the disease under study, the fraction of the population infected in an outbreak and the associated individual risk of infection can be estimated and weighed up with the risks of a given vaccine to yield a best rational strategy. Feeding the outcome of this strategy back into the disease model one

**Box: The vaccination game**

A simple version of the vaccination game (Bauch and Earn, 2004) has each member of the population of size  $N$  play the strategy “vaccinate with probability  $P$ ”. The ratio of the perceived vaccination risk to the risk associated with vaccination is denoted by  $r$  and assumed constant in the population. If  $\pi_\theta$  denotes the risk of infection if a fraction  $\theta$  of the population is vaccinated, the expected payoff of an individual playing strategy  $P$  is

$$E(P, \theta) = -rP - \pi_\theta(1 - P). \quad (1)$$

In game theory, a strategy or set of strategies is a *Nash equilibrium* if no player, knowing the strategies of all other players, can improve his or her payoff by changing strategy if all other players stay with theirs. In the vaccination example, because all players are equal, this implies that if there is a Nash equilibrium, all players play the same strategy. In other words, a strategy  $P^*$  is a Nash equilibrium if no player can improve their payoff (1) by switching to a different strategy  $P$ . In this case, the fraction of the population vaccinated is  $\theta^* = P^*$ . To find such a strategy, note that  $\pi_\theta$ , the risk of infection, decreases with increasing  $\theta$ , i.e., it is lower the higher the fraction vaccinated, and that  $\pi_{\theta \geq \theta'} = 0$  if  $\theta'$  is the threshold vaccination level for herd immunity,  $\theta' = 1/R_0$  (Anderson and May, 1991). The gain in payoff from changing strategy from  $P^*$  played by the other players to  $P$  is

$$\Delta E = (\pi_{(N-1)P^*+P} - r)(P - P^*) \quad (2)$$

Now, if  $r \geq \pi_0$ , i.e. if the vaccine is perceived to be more risky than infection, then  $r \geq \pi_\theta$  for all  $\theta$ , and the only way to achieve  $\Delta E \leq 0$  for all  $P \in [0, 1]$  is  $P^* = 0$ . That is, the Nash equilibrium is never to vaccinate.

If, on the other hand  $r < \pi_0$ , we have that  $r > \pi_{\theta'} = 0$  (excluding the trivial case where the vaccine is perceived to be risk-free). This implies that there exists a unique  $\theta^* \in (0, \theta')$  such that  $\pi_{\theta^*} = r$ . Remember that  $\theta^*$  corresponds to a universally played strategy  $P^* = \theta^*$ . Now, since  $\pi_\theta$  decreases with  $\theta$ , a switch from  $P^*$  to  $P > P^*$  will reduce the resulting risk of infection to  $\pi_{(N-1)P^*+P} < \pi_{P^*}$ , such that  $\Delta E < 0$ . A switch from  $P^*$  to  $P < P^*$ , on the other hand, will increase the risk of infection to  $\pi_{(N-1)P^*+P} > \pi_{P^*}$ , again resulting in  $\Delta E < 0$ . Therefore, for any positive risk of infection  $r > 0$ , there exists a Nash equilibrium  $P^*$  yielding a suboptimal vaccinated fraction  $\theta^* < \theta'$ .

can then study the consequences, a method which has been applied to the study of vaccination against smallpox to prepare for bioterrorism (Bauch et al., 2003), to childhood diseases (Bauch and Earn, 2004) and to yellow fever (Codeço et al., 2007).

In society, however, there can be a significant imbalance between perceived and real risks, especially during vaccine scares such as with the alleged link between the MMR vaccine and autism in the 1990s (Nicoll et al., 1998; Stratton et al., 2004).

Studies on influenza (Galvani et al., 2007) or human papillomavirus (HPV) (Basu et al., 2008) parametrised their models using the results of population surveys and confirmed the problem that with individuals acting rationally according to their perceived risk one would not be able to achieve vaccination levels that minimise disease prevalence in the population.

Even though the dynamics of vaccination behaviour are usually slow with respect to the disease dynamics, negative feedback between these two dynamical systems can cause interesting overall dynamics. For example, if vaccine scares significantly reduce coverage, subsequent outbreaks can increase the perceived risk of infection and prompt higher uptake of vaccines. Prevalence-elastic behaviour, i.e. protective behaviour which is seen increasingly as a disease becomes more prevalent, has been observed, for instance, in the context of Measles (Philipson, 1996). Modelling studies based on game theory have shown that the dynamics of rational behaviour and disease prevalence can lead to oscillations with outbreaks following upsurges in vaccination coverage and subsequent epidemic troughs. This is the case when vaccination decisions are assumed to be made by imitating others at a rate dependent on the individual benefit (Bauch, 2005; Reluga et al., 2006), as well as when decisions are based on past prevalence of a disease (Reluga et al., 2006; Vardavas et al., 2007; Breban et al., 2007; d’Onofrio et al., 2007, 2008). The dynamic belief model of Coelho and Codeço (2009) included random occurrences of adverse vaccine effects and media amplification of such events and found their results to be in line with a yellow fever scare in Brazil in 2008.

A different picture emerges if vaccination decisions are assumed to be made very quickly, that is at the timescale of an outbreak, and to be reactive to infected cases in the social neighbourhood rather than in the whole population. In that case, it has been shown that voluntary ring-vaccination of individuals can curtail local outbreaks if contacts are sufficiently local and the response is fast enough (Perisic and Bauch, 2009a,b).

Most of these models hinge on the assumption that vaccination decisions are fully rational, and the extent to which this is true remains unclear. In fact, at least some of the recent outbreaks of vaccine-preventable disease are known to have occurred in groups opposing vaccination on ideological grounds (Hanratty et al., 2000) or in communities beyond the reach of health authorities (Cohuet et al., 2009). Models have recently been proposed to base vaccination behaviour on the spread of opinions in a social neighbourhood (Salathé and Bonhoeffer, 2008; Eames, 2009) rather than individual rational behaviour. In that case, clusters of unvaccinated individuals can make outbreaks more likely even if population-wide vaccination coverage levels would be expected to provide herd immunity.

It is worth mentioning that vaccination decisions are often irreversible, i.e. while they render a previously susceptible individual immune, a subsequent change in attitude towards vaccination cannot bring that person back into the susceptible class. However, many vaccines require renewal at regular intervals because of waning immunity, or because of pathogen evolution (e.g., seasonal influenza). On top of that, a change of attitude can still affect the overall dynamics in the population when an opinion spreads to influence the decisions of others.

## 5. Other types of behavioural changes

The study of rational decisions is useful in identifying and explaining vaccination behaviour and its interaction with the epidemiology of a disease. However, there is a multitude of other behavioural changes that can influence the spread of infectious diseases, such as reductions in the number of potentially infectious contacts, wearing of face masks or practice of better hygiene. A number of studies have recently considered behavioural changes which do not completely remove those that change their behaviour from the susceptible population, but instead assume the actions to either change disease parameters or change networks of infectious contacts.

Behavioural traits which affect disease transmission can be transferred between individuals. Tanaka et al. (2002) studied a model where two different types of behaviour exist and their frequencies in the population change over time according to social interaction. The authors were particularly interested in the evolution of behaviour and found that new behaviour can establish itself in a population when it is less risky than the current norm, and that behaviour which protects from disease has an inherent evolutionary advantage if riskier behaviour leads to faster progression to infection and death. At the same time, belief in the use of certain practices can spread in a population even if these are inefficient in curing a given illness, simply because people then stay ill for longer and can be observed applying their inefficient treatment during that period (Tanaka et al., 2009).

Such behavioural dynamics can be linked to the more immediate reactions in an emerging epidemic. If fear or awareness of a disease spreads and causes protective behavioural change, the impact on disease dynamics can be quite remarkable. If people remove themselves from the circulation of a disease completely when they are affected by fear from the epidemic, subsequent return into circulation as the fear subsides can lead to multiple waves of infection (Epstein et al., 2008). The diffusion of health information, on the other hand, can reduce the prevalence of infection if individuals avoid infection or seek treatment earlier (Kiss et al., 2009). If people enter a class of low activity representing reduced travel behaviour or similar measures, with a given rate depending on the prevalence of a disease, this

can reduce both the basic reproductive number of the disease and the number of infected cases (Del Valle et al., 2005).

Individuals in direct contact with infected individuals would usually be expected to react most strongly to an outbreak. It is therefore particularly interesting to study the behavioural reactions to a disease in models with population structure. If, for instance, the susceptibility of individuals is reduced as a direct consequence of having of infectious contacts in a social network, it has been shown that a disease can be brought to extinction if the protection is strong enough (Bagnoli et al., 2007). If awareness of a disease is assumed to originate in infected cases but spreads independently to cause protective behaviour in the immediate surroundings of an emerging epidemic, the consequent reduction in susceptibility of those at risk can curtail an epidemic under certain conditions, and do so particularly effectively if the network of information spread overlaps with the contact network of disease transmission (Funk et al., 2009, 2010).

Another way of looking at the impact of reactions to the spread of a disease is to consider changes in behaviour to affect the structure of the network of disease transmission itself. The individual-based spatial model of Epstein et al. (2008) considers people fleeing from a disease location, and the problem that infected fleeing individuals could spread the disease into parts of the population which would not have been reached otherwise. Another set of studies has considered changes in network structure in response to a disease outbreak more explicitly. People who stay at home or avoid infected peers can be seen as cutting links of possible contagion, which in turn affects the progression of the disease. In a series of theoretical studies (Gross et al., 2006; Shaw and Schwartz, 2008; Zanette and Risau-Gusmán, 2008), it was assumed that healthy individuals cut contact with infected peers while forming a new link with a random person from the remaining population at the same time. Under these assumptions, the potential of a disease to invade the whole population is reduced, although this comes with an increased risk of outbreaks into spontaneously formed and strongly connected groups of susceptible individuals, or oscillations between these two scenarios (Gross et al., 2006).

On closer inspection, the models that assume a changing network structure generally make two assumptions about how the transmission network changes. First, an existing link is removed. In practice, this is a possibility only in cases where the wish to distance oneself from the infection overrides the reasons for the existence of the transmission link in the first place. In certain extreme cases this is defensible as realistic, for instance, where the contraction of a sexually transmitted disease such as HIV by a partner in a sexual relationship can motivate the uninfected partner to stop such a relationship. If such a removal is the only assumption in the

model, then the effect is very similar to a reduction in the transmission parameter as discussed above. Some of the models go one step further and assume that after a link has been removed, a new one is formed (link rewiring) with a random new contact. For such rewiring to have an effect on infectious disease dynamics, the rate of change to the network would need to be of a similar magnitude as the spread of disease which is often not the case. More importantly, however, based on the available evidence on social network formation in humans, random link formation is of limited importance in network structure evolution. Thus, in our view, current models that take into account a change in the network structure can only be applied with great caution to infectious diseases in humans. Future work on this topic should take into account social processes such as homophily, i.e. the formation of new ties due to matching individual traits (McPherson et al., 2001), and triadic closure, i.e. formation of new ties between individuals A and C because of existing ties A – B and B – C (Granovetter, 1973).

## 6. Discussion

Reactions to infectious diseases have varied a great deal in history, and the difference in the assumptions and outcomes of the models reviewed here highlights the difficulty in quantifying human behaviour. Which subset of potential actions is taken by people depends greatly on temporal and societal context and may be particularly hard to predict in our times of rapid dissemination of information and opinion. At the same time, modern technology opens up completely new possibilities to measure behaviour directly (Lazer et al., 2009), for example by means of mobile phones (González et al., 2008; Eagle et al., 2009), web-based surveys (Jones and Salathé, 2009), monitoring of web content and traffic (Ginsberg et al., 2009; Bentley and Ormerod, 2009) or even disease outbreaks in virtual gaming worlds (Lofgren and Fefferman, 2007).

The modelling efforts undertaken so far to study the impact of human behaviour on the spread of infectious diseases were based on varying amounts of anecdotal evidence and common sense, but almost never validated with quantifiable observations. Still, these efforts have at least shed some light on the potential impact of behavioural changes on disease dynamics. That in itself can prove a valuable contribution to help inform centralised efforts of controlling future epidemics, and it can guide our thinking about how to measure human reactions to disease. The increasing number of studies that emphasise the importance of the social neighbourhood for individual decisions with respect to a given disease is a promising development in that regard. This choice is affected both by opinions held and behaviour adopted in the circle of acquaintances of one person. At the same time, changes in behaviour

are often contingent on the disease being present or at least perceived to be present nearby. The focus on the impact of the social neighbourhood can produce fundamentally different results from homogeneously mixing populations.

Although there is a wealth of studies in the sociological, psychological and public health literature considering human reactions to the presence of the disease, it has rarely been studied how these affect the disease dynamics. At the same time, few efforts have been made to parametrise the models discussed here from observations. Despite all difficulty in quantifying human behaviour, some potential sources of data have yet to be exploited in the context of infectious diseases. Based on the variety of theoretical studies and the classification scheme we propose, the data which would be needed to inform the choice and parametrisation of model for a given scenario would include: where people obtain their information from, which of the information available to them they trust, if and how they act upon that information, and how effective this reaction is.

Efforts to measure the interaction between individual behaviour and the epidemiology of an infectious disease could start at the most immediate level in collaboration with individual health care providers and hospitals (Olguin et al., 2009). Bluetooth-enabled devices allow to measure the number of close-proximity contacts during a given day (Natarajan et al., 2007) and changes therein if an infectious disease is present in the neighbourhood. Wireless sensor networks promise similar uses (Herman et al., 2009). Web-based social networks provide an underlying structure of friendships and acquaintances and can be used as basis for surveys, opinion polls and epidemic simulations (Salathé and Jones, 2010). A variety of indirect outcomes can be measured which translate to responsive behaviour, such as attendance of public gatherings or web traffic.

Ultimately, mass human behaviour remains to some extent unpredictable, and particular so in the face of disaster. Still, a concerted approach of observational studies and further mathematical modelling can be expected to lead to valuable insights into the way epidemics spread in human populations.

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Figure 1. Schematic representation of the SIR model with the transitions of infection (rate  $\beta$ ) and recovery (rate  $\gamma$ ).

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