Nine challenges in incorporating the dynamics of behaviour in infectious diseases models

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ABSTRACT

Traditionally, the spread of infectious diseases in human populations has been modelled with static parameters. These parameters, however, can change when individuals change their behaviour. If these changes are themselves influenced by the disease dynamics, there is scope for mechanistic models of behaviour to improve our understanding of this interaction. Here, we present challenges in modelling changes in behaviour relating to disease dynamics, specifically: how to incorporate behavioural changes in models of infectious disease dynamics, how to inform measurement of relevant behaviour to parameterise such models, and how to determine the impact of behavioural changes on observed disease dynamics.

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Introduction

Human behaviour may be influenced by a myriad of factors ranging from media to person-to-person communication. The behavioural response towards an infectious disease (e.g., whether to get vaccinated, or whether to stay at home during an epidemic) is shaped by a combination of these influences, and how people evaluate them with respect to the alternatives. Additionally, behavioural responses are influenced by various factors, such as religious or cultural beliefs and norms, that can be clustered both spatially and socially. Even within social groups, there is individual-level variability, and responses are constrained by our personal circumstances. For example, people may be asked or feel obliged to turn up for work irrespective of whether they feel at risk of infection.

The interrelationship between the spread of an infectious disease and the behaviour towards it is subject to a number of dynamic feedbacks. Specifically, an outbreak of an infectious disease can trigger behavioural responses, which in turn can affect the course of the epidemic. Mathematical models provide an invaluable tool to study such feedbacks. Yet, behavioural dynamics have, until recently, rarely been incorporated in models of infectious disease dynamics. Taking into account individual behavioural heterogeneities and shifts in such models can be important because (1) predictions may be unreliable if they fail to take into account behavioural dynamics and (2) most policies target individual-level behaviour and not macro-scale dynamics.

To formulate models in which infectious disease dynamics and behaviour are interdependent, we need to understand the mechanisms behind any mutual influence. To what extent do people themselves, their social “networks”, media opinion leaders, or health care providers affect individual behaviour? And how are the perceptions that determine behaviour influenced by properties of an infection, such as its prevalence or severity? There are often several ways of interpreting the same influence; in the case of disease prevalence, for example, people could respond to current prevalence, recent prevalence, or historical prevalence. Disease severity also affects behaviour (Sadique et al., 2013), but the relationship is not necessarily straight-forward: different responses will be prompted by a disease that infects 50% of a population and kills 1% of those infected versus an infection that infects only 0.5% but kills...
them all. Lastly, knowing that “No man is an island, . . . any man’s death diminishes me, because I am involved in mankind,” people might be aware of external risks, but are not necessarily good at estimating their chance of occurring.

The following challenges relate to the overarching questions of how to incorporate behavioural changes in models of infectious disease dynamics. We do not aim to provide a new perspective or comprehensive review on these topics, which can be found in numerous recent works (Ferguson, 2007; Funk et al., 2010; Bauch and Galvani, 2013; Manfredi and d’Onofrio, 2013). Instead, our goal is to summarise some open questions and challenges in the field that are an important focus of immediate research, and that we hope will serve as an entry point for those interested in getting involved.

1. Set the baseline and determine the effect of departing from it

A key challenge underlying many of the points addressed in this paper is to set an appropriate baseline of behaviour. Two important “baseline” behaviours stand out, one related to mixing, that is how people go about activities of daily life that involve some risk of infection (e.g., going to school, or having sex) and the other related to disease prevention and control. The contact baseline, or the “normal mixing” behaviour, can be disrupted by an epidemic through a number of mechanisms. For example, individuals can choose to change their behaviour in an attempt to reduce their risk (Auld, 2003), or their behaviour can be influenced by the nature of being ill (Lloyd-Smith et al., 2004; Van Kerckhove et al., 2013), both of which affect contact patterns. The other relevant “baseline” refers to people’s inherent willingness to partake in preventative behaviours; most people, for example, follow official recommendations and have their children vaccinated.

A “baseline” or equilibrium might be attained through game theoretic analysis (Gersovitz, 2013; Geoffrey and Philipson, 1997) under the assumption that people make rational decisions by weighing up the private benefits and costs of different options, yielding a certain fraction of the population seeking vaccination, or adopting safe sex. In the absence of data on such “baseline” behaviour, the theoretical equilibrium can provide a useful starting point. This can then be disrupted, by some event, such as the measles–mumps–rubella (MMR) scandal in the United Kingdom. How exactly and under which circumstances such disruptions manifest themselves is an open research question, and one that can only be answered by relating game-theoretical or other modelling approaches more closely with independent observations of behaviour.

2. Assess how and to what extent behaviour should be modelled explicitly

During model development, an investigator must decide whether to treat a given quantity as a dynamic one which evolves in response to other quantities (a model “variable”), or as a fixed value that is exogenously imposed by the modeller (a model “parameter”). Traditional epidemic models account for behaviour implicitly through parameters such as the basic reproduction number. In contrast, modelling the dynamics of behaviour towards infectious diseases requires endogenising behaviour by making it a model variable. However, this leaves questions about which aspects of behaviour should be endogenised, and which should remain as model parameters. This is more than just a technical decision, because it has implications for how we understand and interpret behavioural dynamics. A relevant question is: To what extent is vaccination behaviour determined by response to disease dynamics, and to what extent is it determined by vaccine availability and social norms? In other words, to what extent are vaccine scares historical accidents (exogenous treatment), and to what extent are they enabled by the inherent instability of high vaccine coverage caused by vaccine-generated herd immunity (endogenous treatment)?

Intuitively, if behaviour depends on quantities that change rapidly, such as disease dynamics in a fast-expanding outbreak, then behaviour should probably be represented endogenously. If behaviour depends on quantities that change more slowly, such as social norms or vaccine supply, then it might be possible to represent behaviour through a model parameter. Which of the two scenarios applies, however, also depends on the timescales considered, as social norms and vaccine supply do evolve, yet over long periods. The question of whichever approach is most appropriate in a given scenario can be addressed more rigorously by formulating a collection of variant models where different aspects of behaviour are treated as variables or parameters, and then using model selection methods to determine which variant model best explains the data.

3. Determine the minimal level of detail required to model differences in behaviour

How much psychological detail is required for models to be able to capture the dynamics of population-level behaviour? There are many different models of health-related behaviour in psychology, but for epidemiological purposes a crude understanding of the major drivers and their relative strength is probably sufficient. In the same way that thermodynamic laws are not formulated to depend on the details of molecular-level dynamics, can we model population-level behaviour in a simple, aggregate way without explicit reference to individual-level dynamics?

The key challenge then becomes heterogeneity. How well does the simple model work for everybody? Are there identifiable groups whose response is predictably different, and how important are they epidemiologically? Is there a “landscape” of predispositions to certain behaviours (i.e., will some people be more inclined to follow official guidelines than others)? If yes, do people fall into discrete groups or is that landscape continuous? For example, are risk–averse versus risk-seeking tendencies bimodal, or distributed across a more continuous distribution? How do individuals perceive risks of both infection and adverse effects from control measures and how does the perception of risk change with disease prevalence in the population?

Many of these questions have been studied in econometrics (Gersovitz, 2013), but it remains an open challenge to translate these insights into mechanistic models of infectious disease dynamics. Exploring these questions in mechanistic models and testing different scenarios could yield the limits as well as strengths of “simpler” models, as well as suggest appropriate studies (e.g., through population surveys) that would directly inform model parameters.

4. Quantify changes in reporting behaviour

Data used to track an epidemic typically rely on reporting by individual doctors or hospitals, and therefore depend on how many people seek medical care, how likely doctors are to identify a case correctly, and how likely they are to report it. How does people’s health-seeking behaviour change during the course of an outbreak? The propensity to visit a doctor is likely to depend on levels of concern and on public health messages, both of which are subject to change as an outbreak progresses. Evidence from the 2009 flu pandemic in the UK suggested that individuals’ likelihood of
5. Predict the response to interventions and health campaigns

With better availability of drugs and vaccines, successful control of infectious diseases is increasingly dependent on compliance of individuals with implemented measures. Improving the design and evaluation of control strategies therefore requires deeper understanding of human behaviour, its variability and the drivers of its change. Can we predict the response to a health campaign?

Such responses can vary greatly both within and between populations, and depend on cultural circumstances, details of the infection, and the health campaign in question, as the examples of polio in Pakistan or measles in the UK (with differences in behaviour before and after the perfidious Wakefield study alleging a link between the MMR vaccine and autism) demonstrate. Moreover, the successes of a health campaign may vary due to the passive (requiring members of population to seek health measures) or active (bringing health measures to the individuals of a population) nature of the campaign. How much do the details of implementation matter? Can a single spokesperson make a difference? Can a campaign end up doing more harm than good? When we model interventions, how do we account for change in behaviour in response to interventions?

Analysis of uptake statistics linked to knowledge of campaigns would be a starting point to answer these questions, but how these are best translated into models for infectious disease dynamics remains an open question.

6. Identify the role of movement and travel

Infectious diseases and their dynamics are tightly linked with movement and travel. On one hand, population movement can drive local disease transmission and its seasonality, as in sub-Saharan Africa where increases in urban density during the dry season cause epidemic measles outbreaks (Ferrari et al., 2008). On the other hand, disease can be a strong driver of movement by causing people to flee disease-hit areas, especially dense urban centres (e.g., plague and cholera in historical London). This can have negative consequences for destination locations, especially if migrants are unknowingly infected (Mesnard and Seabright, 2009). Alternatively, people may choose to minimise their trips in response to outbreaks and engage in self-protecting behaviour by cancelling their flights, indicating that they value the reduction in perceived risk of infection more than the money spent on airfare (Fenichel et al., 2013). The ability of people to flee will depend on various factors including socioeconomic conditions, family structure, and non-local contacts, highlighting the importance of understanding the heterogeneity in causes and effects of disease-driven movement.

As a result of lower density of the remaining population, individuals’ contact networks may shrink, reducing local transmission. In contrast, medical and emergency response personnel are likely to experience an increase in their numbers of contacts. But how exactly do contact networks change in response to infection and to what extent are these dynamics dependent on where an infection is? Given that most contact patterns are measured in the absence of disease (Mossong et al., 2008), how useful are these studies for predicting disease spread and assessing control measures? Combining our understanding of basic human mobility and migration patterns (González et al., 2008; Simini et al., 2012) with the behaviour “baseline” (see Challenge 1) offers a good starting point to model the effects of changes in movement and travel on diseases.

7. Develop models that can be verified against data from digital sources

Data on individual and population behaviour concerning infectious diseases have historically been scarce. However, the recent advent of new digital sources may change that (Salathé et al., 2012). These sources include online social media, mobile phone data, Bluetooth data, electronic texts, search engine data, sales data and other sources of data now routinely collected by companies and institutions. For example, researchers have used data from Twitter to study awareness and sentiments regarding influenza outbreaks and vaccines (Salathé and Kandelinwal, 2011; Signorini et al., 2011). Other promising digital sources include usage data for websites like Wikipedia (McIver and Brownstein, 2014), and search engine data, such as available through Google Flu Trends (Ginsberg et al., 2009), although challenges clearly remain (Butler, 2013). Any scientific model must ultimately be testable against data, hence we must develop models that can be tested against the kind of data that are available. Relevant challenges include: How can we be more creative about using “new” data sources to develop models? Can we use digital media to set up our own experiments to answer some of the challenges posed here and thus inform model development? Which statistical models can be used, and which new ones need to be developed, to synthesise information derived from digital media with information derived from more traditional sources, such as cross-sectional population surveys, inside or outside of a modelling framework?

In this way, the weaknesses of one type of data may be compensated by the strengths of the other. Existing data on behaviour were often not collected with the purpose of model parameterisation in mind, so it can be difficult to find appropriate data for parameterising behavioural models; therefore modellers will often have to collect the data themselves. Because digital data sources are often resolved at the individual level, individual-based models and network simulations lend themselves naturally to such applications.

8. Inform real-time data collection

Data on behavioural change in response to an epidemic (or similar) shock are key for parametrisers models of infectious disease dynamics. In an ideal situation, collecting these data during a real epidemic would provide the rare opportunity to measure behaviours in response to local and global information about
Conclusions

9. Engage in dialogue across disciplines

Many of the issues discussed in this work touch on research that is being done in a number of different disciplines, from psychology to sociology, economics, epidemiology and mathematics. Different approaches are traditionally used in different fields, and rarely do results attained in one area get used in another. A recent book has gone to laudable lengths to include chapters from economists as well as mathematical biologists (Manfredi and d’Onofrio, 2013), but, clearly, much work remains to be done in an area where clearly there is great scope for cross-fertilisation of ideas and methods.

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