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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.

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

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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; Geoffard 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

consulting a doctor decreased radically over the course of the epidemic (Brooks-Pollock et al., 2011), and the increasing availability of online surveillance of influenza-like illness elsewhere opens promising avenues for similar studies. Likewise, doctors' diagnosis and reporting behaviour may change, depending on familiarity with a condition and perceptions about which infections are currently common. Changes may take place gradually as attitudes evolve, or suddenly in response to significant changes in the reporting system, for example the UK's introduction of a telephone and internet service midway through the 2009 pandemic (Harcourt et al., 2012).

Some models have attempted to draw together evidence from various sources to account for changing reporting behaviours (Birrell et al., 2011), but in general more information is needed. Laboratory testing of cases defined on the basis of symptoms alone provides a useful validation of doctors' diagnoses, but fails to capture those individuals who do not seek treatment. Work is required to better integrate medically attended case series with community surveillance, particularly surveillance that explicitly monitors trends in health-care seeking behaviour (Rubin et al., 2009; Brooks-Pollock et al., 2011), to "cut out the middle man" (the doctor) and better understand the true epidemic picture, and the behavioural drivers that may distort our measurements.

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 first 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 episodic 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 Khandelwal, 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 parametrising 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

disease spread, as well as simultaneous measurement of the possible drivers of behaviour change. However, collecting these data, even if key drivers are known, is very difficult and, consequently, rarely done (Rubin et al., 2009; Van Kerckhove et al., 2013).

An alternative approach may be the collection of data on hypothetical scenarios. The advantage of a hypothetical scenario is that researchers can gain information on many different sorts of events, and studies can be repeated among many different population samples. However, hypothetical studies can be expensive and the value of these studies remains controversial. In this situation, epidemiological models, in addition to being consumers of data on behavioural response and change, can also be used to inform real-time collection of data on behaviour. What sample sizes should be used to robustly detect changes in behaviour? Which observation window must be used to robustly parameterise models? Are there "sentinel" individuals that can be observed to minimise resources and maximise prediction accuracy? While making use of models when planning studies may not solve practical limitations of cohort recruitment, it will make the rare opportunities of real-time behavioural data collection a more efficient process so models can more accurately reflect behaviour and make better predictions.

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.

Conclusions

Behavioural heterogeneities and changes play an important role in many areas of infectious disease dynamics, from vaccine-preventable infections (Metcalf et al., 2015) and eradication efforts (Klepac et al., 2015), to network modelling and measurement (Eames et al., 2015; Pellis et al., 2015). Designing and validating models of behaviour towards infectious diseases and changes therein is challenging. Nonetheless, while it may be impossible to capture the behaviour of a given individual, it may be more feasible to predict behavioural averages and distributions. Consequently, population-scale behaviour may be amenable to modelling (Funk et al., 2010), and even where it is not, it remains important to identify the limits of predictability and propagate uncertainty onto model uncertainty.

With these challenges addressed, models of infectious diseases that include human behaviour can make the important transition from theoretical models of what–if scenarios to becoming relevant for policy decisions (Edmunds et al., 2013; Metcalf et al., 2015).

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References

Auld, M.C., 2003. Choices beliefs and infectious disease dynamics. J. Health Econ. 22 (3), 361–377, http://dx.doi.org/10.1016/S0167-6296(02)00103-0.
Bauch, C.T., Galvani, A.P., 2013. Social factors in epidemiology. Science 342 (6154), 47–49, http://dx.doi.org/10.1126/science.1244492.
Birrell, P.J., Ketsetzis, G., Gay, N.J., Cooper, B.S., Presanis, A.M., Harris, R.J., Charlett, A., Zhang, X.-S., White, P.J., Pebody, R.G., De Angelis, D., 2011. Bayesian modeling to unmask and predict influenza A/H1N1pdm dynamics in London. Proc. Natl. Acad. Sci. U. S. A. 108 (45), 18238–18243, http://dx.doi.org/10.1073/pnas.1103002108.
Brooks-Pollock, E., Tilston, N., Edmunds, W.J., Eames, K.T.D., 2011. Using an online survey of healthcare-seeking behaviour to estimate the magnitude and severity of the 2009 h1n1v influenza epidemic in England. BMC Infect. Dis. 11, 68, http://dx.doi.org/10.1186/1471-2334-11-68.
Butler, D., 2013. When Google got flu wrong. Nature 494 (7436), 155–156, http://dx.doi.org/10.1038/494155a.
Eames, K.T.D., Bansal, S., Frost, S., Riley, S., 2015. Six challenges in measuring contact networks for use in modelling. Epidemics 10, 72–77.
Edmunds, W.J., Eames, K., Keogh-Brown, M., 2013. Capturing human behaviour: is it possible to bridge the gap between data and models? In: Manfredi, P., d'Onofrio, A. (Eds.), Modeling the Interplay between Human Behavior and the Spread of Infectious Disease. Springer, Heidelberg, pp. 311–321, http://dx.doi.org/10.1007/978-1-4614-5474-8_19.
Fenichel, E.P., Kuminoff, N.V., Chowell, G., 2013. Skip the trip: air travelers' behavioral responses to pandemic influenza. PLOS ONE 8 (3), e58249, http://dx.doi.org/10.1371/journal.pone.0058249.
Ferguson, N., 2007. Capturing human behaviour. Nature 446 (7137), 733, http://dx.doi.org/10.1038/446733a.
Ferrari, M.J., Grais, R.F., Bharti, N., Conlan, A.J.K., Bjørnstad, O.N., Wolfson, L.J., Guerin, P.J., Djibo, A., Grenfell, B.T., 2008. The dynamics of measles in sub-Saharan Africa. Nature 451 (7179), 679–684, http://dx.doi.org/10.1038/nature06509.
Funk, S., Salathé, M., Jansen, V.A.A., 2010. Modelling the influence of human behaviour on the spread of infectious diseases: a review. J. R. Soc. Interface 7 (50), 1247–1256, http://dx.doi.org/10.1098/rsif.2010.0142.
Geoffard, P.-Y., Philipson, T., 1997. Disease eradication: private versus public vaccination. Am. Econ. Rev. 87 (1), 222–230.
Gersovitz, M., 2013. Mathematical epidemiology and welfare economics. In: Manfredi, P., d'Onofrio, A. (Eds.), Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases. Springer, Berlin, Heidelberg, pp. 185–202, http://dx.doi.org/10.1007/978-1-4614-5474-8_12.
Ginsberg, J., Mohebbi, M.H., Patel, R.S., Brammer, L., Smolinski, M.S., Brilliant, L., 2009. Detecting influenza epidemics using search engine query data. Nature 457 (7232), 1012–1014, http://dx.doi.org/10.1038/nature07634.
González, M.C., Hidalgo, C.A., Barabási, A.-L., 2008. Understanding individual human mobility patterns. Nature 453 (7196), 779–782, http://dx.doi.org/10.1038/nature06958.
Harcourt, S.E., Smith, G.E., Elliot, A.J., Pebody, R., Charlett, A., Ibbotson, S., Regan, M., Hippisley-Cox, J., 2012. Use of a large general practice syndromic surveillance system to monitor the progress of the influenza A (H1N1) pandemic 2009 in the UK. Epidemiol. Infect. 140 (1), 100–105, http://dx.doi.org/10.1017/S095026881100046X.
Klepac, P., Funk, S., Hollingsworth, T.D., Metcalf, C.J.E., Hampson, K., 2015. Six challenges in the eradication of infectious diseases. Epidemics 10, 97–101.
Lloyd-Smith, J.O., Getz, W.M., Westerhoff, H.V., 2004. Frequency-dependent incidence in models of sexually transmitted diseases: portrayal of pair-based transmission and effects of illness on contact behaviour. Proc. Biol. Sci. 271 (1539), 625–634, http://dx.doi.org/10.1098/rspb.2003.2632.
Manfredi, P., d'Onofrio, A. (Eds.), 2013. Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases. Springer, Berlin, Heidelberg.
McIver, D.J., Brownstein, J.S., 2014. Wikipedia usage estimates prevalence of influenza-like illness in the united states in near real-time. PLoS Comput. Biol. 10 (4), e1003581, http://dx.doi.org/10.1371/journal.pcbi.1003581.
Mesnard, A., Seabright, P., 2009. Escaping epidemics through migration? Quarantine measures under incomplete information about infection risk. J. Public Econ. 93 (7–8), 931–938, http://dx.doi.org/10.1016/j.jpubeco.2009.05.001.
Metcalf, C.J.E., Andreason, V., Bjørnstad, O.N., Eames, K.T.D., Edmunds, W.J., Funk, S., Hollingsworth, T.D., Lessler, J., Viboud, C., Grenfell, B.T., 2015. Seven challenges in modelling vaccine preventable diseases. Epidemics 10, 11–15.
Mossong, J., Hens, N., Jit, M., Beutels, P., Auranen, K., Mikolajczyk, R., Massari, M., Salmaso, S., Tomba, G.S., Wallinga, J., Heijne, J., Sadkowska-Todys, M., Rosinska, M., Edmunds, W.J., 2008. Social contacts and mixing patterns relevant to the spread of infectious diseases. PLoS Med. 5 (3), e74, http://dx.doi.org/10.1371/journal.pmed.0050074.
Pellis, L., Ball, F., Bansal, S., Eames, K.T.D., House, T., Isham, V., Trapman, P., 2015. Eight challenges for network epidemic models. Epidemics 10, 58–62.
Rubin, G.J., Amlôt, R., Page, L., Wessely, S., 2009. Public perceptions anxiety and behaviour change in relation to the swine flu outbreak: cross sectional telephone survey. Br. Med. J. 339, b2651, http://dx.doi.org/10.1136/bmj.b2651.
Sadique, M.Z., Devlin, N., Edmunds, W.J., Parkin, D., 2013. The effect of perceived risks on the demand for vaccination: results from a discrete choice experiment. PLOS ONE 8 (2), e54149, http://dx.doi.org/10.1371/journal.pone.0054149.
Salathé, M., Bengtsson, L., Bodnar, T.J., Brewer, D.D., Brownstein, J.S., Buckee, C., Campbell, E.M., Cattuto, C., Khandelwal, S., Mabry, P.L., Vespignani, A., 2012. Digital epidemiology. PLoS Comput. Biol. 8 (7), e1002616, http://dx.doi.org/10.1371/journal.pcbi.1002616.

Salathé, M., Khandelwal, S., 2011. Assessing vaccination sentiments with online social media: implications for infectious disease dynamics and control. PLoS Comput. Biol. 7 (10), e1002199, http://dx.doi.org/10.1371/journal.pcbi.1002199.

Signorini, A., Segre, A.M., Polgreen, P.M., 2011. The use of twitter to track levels of disease activity and public concern in the U.S. during the influenza a H1N1 pandemic. PLoS ONE 6 (5), e19467, http://dx.doi.org/10.1371/journal.pone.0019467.

Simini, F., González, M.C., Maritan, A., Barabási, A.-L., 2012. A universal model for mobility and migration patterns. Nature 484 (7392), 96–100, http://dx.doi.org/10.1038/nature10856.

Van Kerckhove, K., Hens, N., Edmunds, W.J., Eames, K.T.D., 2013. The impact of illness on social networks: implications for transmission and control of influenza. Am. J. Epidemiol. 178 (11), 1655–1662, http://dx.doi.org/10.1093/aje/kwt196.