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Thirteen challenges in modelling plant diseases

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ABSTRACT

The underlying structure of epidemiological models, and the questions that models can be used to address, do not necessarily depend on the host organism in question. This means that certain preoccupations of plant disease modellers are similar to those of modellers of diseases in human, livestock and wild animal populations. However, a number of aspects of plant epidemiology are very distinctive, and this leads to specific challenges in modelling plant diseases, which in turn sets a certain agenda for modellers. Here we outline a selection of 13 challenges, specific to plant disease epidemiology, that we feel are important targets for future work.

Introduction

Certain of the issues that are important in modelling diseases of humans, livestock and wild animals are equally important to plant disease epidemiology. Generic questions surround the effects of population structure and stochasticity upon epidemic dynamics, and how models can be parameterised from data that are all too often limited. The extent to which different aspects of the complex biology underlying spread need to be captured in models can be unclear, and this ambiguity in what must be represented naturally leads to a focus on model parsimony. Methods to propagate uncertainties in model structure and/or parameter values to uncertainty in model prediction are also required, irrespective of whether the pathogen has a plant, human or agricultural or wild animal host.

Nevertheless, many aspects of plant disease epidemiology set a distinctive agenda for plant disease modellers. Most obvious is that, in the absence of human-mediated movement, individual plants are sessile, although there are complex heterogeneities in the availability of hosts for infection in both space and time. Equally characteristic, however, are infection rates that are strongly controlled by environmental conditions, and disease that is frequently cryptic (i.e. undetectable) and/or poorly reported (particularly in natural environments). Extensive prophylactic control, interactions among multiple hosts and/or pathogens, and complex pathogen life cycles must also feature prominently in any meaningful discussion of plant epidemiology.

Here we outline a selection of 13 challenges that are specific to plant disease, and that we feel are particularly important. We particularly focus on challenges relating to disease prediction and disease control using epidemiological models. These challenges can be partitioned into those relating to modelling the plant host(s) (Challenges 1–4), modelling the pathogen(s) (Challenges 5–9) and modelling control (Challenges 10–13). We have necessarily been selective in the challenges we identify, constrained by a tight word limit and a fixed quota of references. *Giving a broad overview of the challenges faced by modellers of plant disease within the constraints of a single article has itself been a significant challenge.*

1. Linking epidemiological models to crop yield and ecosystem services

Crop pathogens are important primarily because they cause loss of yield. However, models concentrating on yield (e.g. Madden et al., 2000a) typically only include very simple epidemiology, e.g. logistic growth of epidemics. *Models should incorporate sufficient epidemiological realism in order to analyse and predict the effects of disease and host dynamics on yield.* An attractive metric to capture transients in

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the outputs of compartmental models was proposed by Hall et al. (2007),

$$\text{Yield} = \int_{t=0}^{T_{\max}} \omega(t)S(t), \quad (1)$$

where $S(t)$ is healthy tissue, T_{max} season length and $\omega(t)$ a weighting function. However to be useful in practice this would require realistic dynamics for host growth, and appropriate definition of $\omega(t)$ to account for the effects of the timing of loss of healthy tissue relative to grain filling or fruit production. The central role of within-host severity in yield indicates individual plants may need to be distinguished in the epidemic model to allow for variation in severity between hosts, with a more careful treatment of autoinfection than is typical. Ideally models would also account for compensation via reduced competition from diseased plants on remaining healthy individuals, and for the effects of pathogens on the full range of ecosystem services (Boyd et al., 2013). *Recent high-profile introductions of invasive forest pathogens underline the need for models that incorporate and quantify impacts of ecosystem services*.

2. Temporal changes in host availability, from plant organs to populations

Amounts of susceptible tissue can vary over orders of magnitude within a single season. It is therefore surprising that changes in the number or density of hosts are most often ignored. When host population sizes do vary in models, this is typically via a simplistic caricature. On longer timescales, perturbations due to harvesting for crops or seasonal defoliation in perennial hosts are rarely considered. *The default assumption of constant population size should be replaced: models should more routinely include realistic within-season host dynamics and synchronous removal at the end of each season*. Within-season dynamics are required for a proper treatment of yield, and to explain paradoxical reductions in severity when host growth out-paces that of the pathogen. Host growth can also make populations more invasible by reducing distances between individuals, particularly for soil-borne pathogens, a phenomenon accentuated by disease-induced growth. Madden et al. (2002) showed how to define invasion thresholds over multiple seasons using semi-discrete models. *Models capturing long-term behaviour by tracking pathogens over multiple growing seasons in both annual and perennial hosts should be extended, particularly to include a more detailed treatment of host growth and recruitment, overwintering and alternate hosts*.

3. Capturing host spatial structure, even when data are limited

Plants are sessile, and this means the spatial structure of host populations is of paramount importance. However host location data is expensive to collect and often incomplete. Proxies including maps tracking groups of species or results of environmentally-driven species distribution models (e.g. maximum entropy) are often all that are available. *The key challenges are to assess the impact of incomplete or inaccurate host data on the predictive accuracy of models, and to develop methods to account for the additional uncertainty to which this leads*. Loss of small-scale spatial detail often creates artificially-extended regions in which the host may erroneously be assumed to be contiguous; the potential bias of this in overstating spread remains unclear.

Including spatial dynamics reveals the role of asynchrony and dispersal among populations in influencing both host and pathogen polymorphism and rates of evolutionary change (Thrall and Burdon, 2002). Representing the continuum of connectivity from large continuous populations to small separate populations will allow more explicit predictions regarding the influence of fragmentation on host and pathogen evolution (Carlsson Granér and Thrall, 2002). Focusing on patch size also mimics historical interest in how fields can be arranged to give landscapes resilience to pathogens (van der Plank, 1948). An attractive analytic approach would involve adapting the work of DeWoody et al. (2005) by including crop rotation and overwintering in a spatially-structured metapopulation-type model of the agricultural mosaic. *We require metrics to quantify how the invasibility of landscapes is conditioned on the interplay between spatial structure and pathogen dispersal, particularly to allow limited control resources to be allocated, and to assess potential evolutionary implications*.

4. Beyond a single species: multiple and alternate hosts, spillover and community ecology

Many plant pathogens can infect multiple host species, and some require more than one host species to complete their life cycle. Although crop mixtures are well studied (Mundt, 2002), the area of multiple hosts has been neglected. Recent theoretical advances (e.g. type reproduction numbers to identify host species with most significant effects on spread (Heesterbeck and Roberts, 2007)) have not yet been applied. *The challenge is to validate simplifications including host indices or functional traits to capture host heterogeneity while avoiding parameter explosion*. This will help us to understand control, and allow impacts on ecosystem function and species coexistence to be quantified.

For multi-host pathogens, asymmetries in transmission, seasonal refuges, and relative densities of hosts are all critical, particularly when there is transmission between natural and domesticated hosts (Borer et al., 2009). Host heterogeneity is likely to influence the evolution of multi-host pathogen virulence, exacerbating apparent competition (Betancourt et al., 2013), and affecting whether generalist or specialist pathogens are favoured (Gudelj et al., 2004). Pathogen introductions, particularly to threatened tree species, mean it is urgent to understand spillover, in which epidemics in a host population of interest are driven not by transmission within that species, but by transmission from a different host (Power and Mitchell, 2004). Spillover can also drive the spread of invasive species (Flory and Clay, 2013), particularly when the invader is less affected by the pathogens it carries. *Models exploring how dynamics on pathogens' hosts in their natural range translate into an exotic range are clearly required*.

5. Realistic dispersal models, including meteorological and anthropomorphic drivers

Landscape-scale models often link locations via a dispersal kernel (e.g. Meentemeyer et al., 2011). The major attraction is parsimony: a simple function controls how transmission probabilities decay with distance. However, this is clearly a significant simplification. We need to understand how non-isotropic, time and space varying kernels impact on epidemic dynamics.

Including more realism in kernels would require sub-models of processes underlying heterogeneities in dispersal. A number of transmission routes are important, including wind-borne long-distance spore transport, rivers, trade networks, shared machinery and other anthropomorphic pathways. Of these, trade networks are increasingly well-studied (see Challenge 6, below), and long distance wind-borne dispersal has received significant attention, both via phenomenological models (e.g. Aylor, 2003) and more detailed models taking account of meteorological data (e.g. Isard et al., 2005). *The challenge is matching the complexity of dispersal to the purpose of the model and, crucially, to the quality of data available for parameterisation*. While a more detailed treatment of dispersal is attractive, this can only be meaningful if given statistical support via fitting, for which available data are typically rather sparse.

6. Network models for human-mediated spread

Driven by an increasing acknowledgment of the role of the plant trade in spreading disease (Brasier, 2008), there has been an interest in the use of network models to characterise the movement of inoculum by trade and transportation networks (Jeger et al., 2007). Understanding the spread of disease in these networks could help to identify network characteristics that exacerbate spread and also

be used to test sampling and control strategies (e.g. (Moslonka-Lefebvre et al., 2012) and Challenges 10 and 11). *Although some insight has already been gained from adaptations of network-theoretic models, in which the network structure is chosen for reasons of mathematical convenience, the real challenge is to understand the epidemiologically-important characteristics of real trade networks*. This is of course confounded by commercial sensitivities surrounding the data needed to parameterise network structure. Models must also consider the interaction between natural and artificial spread pathways. For example, many plant nurseries operate in open-field environments that are subject to import and export of inoculum via natural means as well as by human-mediated movement (Harwood et al., 2009).

7. Accounting for time-varying infectivity

Both seasonal drivers of the pathogen life-cycle (e.g. sporulation may be temperature dependent), and time-scales inherent to the life-cycle of the host or pathogen (e.g. growth and/or patterns of autoinfection) can result in time-varying patterns of infectivity. These have pronounced effects on dynamics and control (Cunniffe et al., 2012). *Capturing empirical patterns in models is a major challenge because infectivity is so difficult to measure in natural systems, and approaches for scaling from growth chamber measurements to natural conditions are not obvious*. Climate change also raises the possibility of inherent non-stationarity in all these processes, which poses additional challenges in modelling dynamical outcomes and implications of control.

8. Effects of vector preference on transmission

Recent experimental work has shown how transmission by vectors depends on the infection status of host plants (Mauck et al., 2012) and of the vectors themselves (Ingwell et al., 2012). Differences revolve around probabilities of vectors probing host plants and/or choosing to settle for an extended feed. Although simulation models have included vector "preference", conflating probing and settling into a single parameter (e.g. Sisterson, 2008), this is insufficient to represent fine distinctions, particularly the expected interaction with transmission type (i.e. non-persistent vs. semi-persistent vs. persistent).

The compartmental model of Madden et al. (2000b) is an attractive framework, particularly since it can represent different types of transmission via parametric changes. Vector preference could be included quite simply in that model. The probability with which vectors settle on individual host plants could be easily adapted to reflect differences in host preference, and the effects of changes in the numbers of extended feeds would be to affect the parameter controlling the number of visits made per vector per day. *Adding vector preference to compartmental models would allow us to understand interactions between host and vector infection status, transmission type and vector preference, together with any evolutionary implications*.

9. Beyond a single species: multiple strains, multiple pathogens and evolution

Multiple circulating pathogens (different strains or different species) in a given host population can each influence the spread of the other. This can be directly, e.g. via competition for host resources, or indirectly, e.g. by selecting for shared mechanisms of host resistance to different pathogens (Lozano-Torres et al., 2012). Multiple infections can result in either synergistic or antagonistic interactions among pathogens, with unpredictable consequences. For example, competitive interactions may mean that control of one pathogen may leave a host population exposed to another. Alternatively, infection by one pathogen may increase host vulnerability to infection by another, or may lead to induced resistance and so reduce the chance of infection. *Modelling quantitative disease resistance in populations facing multiple pathogens is a key challenge in predicting disease dynamics and evolutionary responses of pathogens and hosts*.

10. Using models to optimise detection

The recent rise in the number of introduced plant pathogens focuses attention on more effective sampling strategies for detection (Brasier, 2008). *Key questions include: given a surveillance program, what incidence will an epidemic have reached when it is first discovered? How can we optimise the deployment of sampling resources in space and time*? Model-based approaches have begun to be used to address these issues. For example, recent work using a simple epidemic model gives insight into how the dynamics of sampling programs and epidemics relate: the mean incidence an epidemic will have reached when it is first detected can be estimated from the ratio of the rate of epidemic increase to the rate of sampling (Parnell et al., 2012).

There is scope to use realistic spatially-explicit models to optimise sampling programs (e.g. Demon et al., 2011; Parnell et al., 2014). A range of factors still need to be incorporated, including: introduction pathways, pathogen dispersal patterns, cryptic periods, landscape connectivity, and different detection technologies. Specific sampling objectives should always be clearly defined (e.g. early warning versus incidence estimation) and, where management plans have been identified, sampling and control models can be coupled. *Further challenges include optimising sampling programs in plant trade networks, how to sample to detect unknown (or unanticipated) pathogens, and the role of passive surveillance and citizen science*.

11. Optimising dynamic controls in heterogeneous systems

Localised control can be effective, particularly if we "match the scale of control with the intrinsic scale of the epidemic" (Gilligan et al., 2007). The problem, however, is that a general method to quantify the required scale is unavailable, even for simple control strategies (e.g. removal of all hosts within a certain distance of detected infection). *We need to understand how the optimal scale of control depends on the interplay between the epidemiology of the plant-pathogen interaction, the implementation of detection and control, and the current state of the epidemic*. The effectiveness of control can be time-dependent, particularly when controls are applied repeatedly (e.g. chemicals) or are subject to dynamics of their own (e.g. biological control), and this adds complexity. Control also exerts selective pressure on pathogen populations, and although fungicide resistance is well-studied, other human-mediated selection exerted by our controls (e.g. selection for less detectable symptoms when controlling by roguing) has only rarely been modelled (e.g. van den Bosch et al., 2007).

Work for animal pathogens has shown how elaborate control strategies that depend on time and space can reduce the impact of epidemics (e.g. te Beest et al., 2011). *A second challenge is to develop and assess dynamic control strategies based on the risk posed by infection*. An attractive approach would involve "hazard maps", which integrate landscape structure, environmental suitability for infection and dispersal to measure the local impact of a pathogen at each position across a landscape (Meentemeyer et al., 2011), to allow locations where control would be expected to have the most significant effect to be targeted.

12. Accounting for economics: moving optimal control theory to realistic landscapes

Knowing when, where and how to manage disease effectively at the landscape scale, is an area of active research. Progress has been made in establishing a rigorous theoretical framework to link spatio-temporal epidemiological models with economic models in order to identify optimal strategies for chemical and cultural control when resources are limited (cf. Ndeffo Mbah et al., 2010). *Optimal control theory provides an analytical starting point to eliminate some control scenarios that are obviously far from optimal and to identify some options for further investigation*. The quantity to be maximised or minimised is formalised into an objective function.

Common examples include expenditure on control together with a measure of infection or crop loss. Recent work has focused on how to optimise limited resources for monitoring and control of disease in metapopulations when there are insufficient resources to monitor and treat all infected sites (Ndeffo Mbah and Gilligan, 2011). The machinery of optimal control theory is essentially deterministic. The inherent variability in epidemic development can be incorporated using options theory, answering questions such as whether to treat immediately or to delay and continue monitoring in order to make a more informed decision about control (Ndeffo Mbah et al., 2010). *The challenge now is to integrate and test the inferences from these approaches into models for disease spread in realistic landscapes.*

13. Use of models by policy makers and stakeholders

The complexities of disease models are of less concern to stakeholders and policy makers than model validity and usability for practical decision making. Both groups are predominately concerned with avoiding disease, early detection, and control. Regulatory philosophy is to use the latest scientific information that is available (Gottwald et al., 2001). *Models that capture the broad characteristics of a host-pathosystem and that can be used to predict its dynamics are therefore increasingly attractive to policy makers.*

Models can be used to investigate "what if" scenarios to assess and compare the efficacy of controls. Although eradication is often likely to be unachievable, management programmes can delay the full effects of a disease, thereby maintaining trade markets. *Since policy makers are answerable politically to stakeholders to protect agricultural commodities, they require tools to understand the effects of management, particularly if they can be made available in a user-friendly form.* An example is available online at http://www.webidemics.com/.

Regulatory agencies utilise numerous surveillance methods, preferring those that are statistically validated and based on models for disease spread. These models can be used for both early detection and disease monitoring. Invasive diseases are increasingly points of contention between stakeholders nationally and internationally, resulting in quarantines and trade restrictions. Regulatory agencies must balance concerns among stakeholder groups which can lead to litigation and trade restrictions. *Regulatory interventions based on disease models must be scientifically defensible in courts of law and among international regulatory organizations.*

Concluding remarks

Space limitations mean we have not considered models of biosecurity in the plant trade, models of disease for use in precision agriculture, methods to parameterise and test landscape-scale models, making use of population geneticists' expanded molecular toolkit for assessing allelic and genotypic frequency variation in populations to link population models with the "omics" revolution, using insights from soil imaging to improve models for soil-borne pathogens, modelling disease complexes and linking models of control to those from social science. We have also restricted ourselves to plant-oriented challenges. Nevertheless, there is commonality of purpose and approach between models for analysis, prediction and control of plant, human, livestock and wild animal epidemics. Much work during the past two decades has emphasised these similarities, but arguably there is still insufficient "cross-talk" between modelling disciplines. *Building better links between plant disease modellers and the wider epidemic modelling community is a significant challenge of its own.*

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