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Thirteen challenges in modelling plant diseases

Nik J. Cunniffe[a,*], Britt Koskella[b], C. Jessica E. Metcalf[c], Stephen Parnell[d], Tim R. Gottwald[e], Christopher A. Gilligan[a]

[a] Department of Plant Sciences, University of Cambridge, Cambridge CB2 3EA, UK
[b] BioSciences, University of Exeter, Penryn Campus, Tremough TR10 9EZ, UK
[c] Department of Zoology, Oxford University, Oxford OX1 3PS, UK
[d] Computational and Systems Biology, Rothamsted Research, Harpenden AL5 2JQ, UK
[e] United States Department of Agriculture, Agricultural Research Service, 2001 South Rock Road, Fort Pierce, FL 34945, USA

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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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* Corresponding author. Tel.: +44 01223333900.
E-mail address: njc1001@cam.ac.uk (N.J. Cunniffe).

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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