Review

Network epidemiology and plant trade networks

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Abstract. Models of epidemics in complex networks are improving our predictive understanding of infectious disease outbreaks. Nonetheless, applying network theory to plant pathology is still a challenge. This overview summarizes some key developments in network epidemiology that are likely to facilitate its application in the study and management of plant diseases. Recent surveys have provided much-needed datasets on contact patterns and human mobility in social networks, but plant trade networks are still understudied. Human (and plant) mobility levels across the planet are unprecedented—there is thus much potential in the use of network theory by plant health authorities and researchers. Given the directed and hierarchical nature of plant trade networks, there is a need for plant epidemiologists to further develop models based on undirected and homogeneous networks. More realistic plant health scenarios would also be obtained by developing epidemic models in dynamic, rather than static, networks. For plant diseases spread by the horticultural and ornamental trade, there is the challenge of developing spatio-temporal epidemic simulations integrating network data. The use of network theory in plant epidemiology is a promising avenue and could contribute to anticipating and preventing plant health emergencies such as European ash dieback.

Keywords: Complex networks; epidemic threshold; global change; Hymenoscyphus pseudoalbidus; infectious diseases; information diffusion; network structure; Phytophthora ramorum; scale-free; small-world.

Introduction

Networks are sets of nodes connected by links (Table 1). They provide a flexible framework to study emerging properties of systems composed of discrete units in the biological (Jacobi and Jonsson 2011; Latty et al. 2011), medical (Grassly and Fraser 2008; Gleeson et al. 2012), social (Newman and Park 2003; Derzsi et al. 2011) and technological sciences (Schweitzer et al. 2009; Vespignani 2012). Networks are increasingly used as models to study the spread of infectious diseases through host populations (reviewed in Kretzschmar 2000; Newman 2002; Keeling 2005; Danon et al. 2011; House and Keeling 2011; Robinson et al. 2012; Fig. 1), thus adding realism to models previously assuming that the susceptible individuals of a population were equally likely to be in contact with infectious individuals.

One of the main results of network epidemiological studies is that network structure can have a strong influence on epidemic dynamics (Morris and Kretzschmar 1995; Christley and French 2003; Shirley and Rushton 2005; Bansal et al. 2007; Drewe et al. 2011; Smilkov and Kocarev 2012). Depending on the way links connect nodes, various network types can be distinguished (e.g. local, small-world, random and scale-free). Local networks show neighbouring connectivity, small-world networks add some rewiring of local links into long-distance connections, random networks have nodes connected with a certain probability, and scale-free networks are characterized...
by the presence of hubs (super-connected nodes) (Watts and Strogatz 1998; Keeling and Eames 2005; May 2006; House 2012). The presence of hubs, other things being equal, drastically reduces the epidemic threshold compared with non-scale-free network structures.

Network analyses have delivered various key insights to improve our understanding and management of epidemics (Table 2). There is a growing proportion of publications on epidemics using network approaches (Fig. 1). There is thus the need for an update, an overview of the main achievements and a summary of outstanding challenges. The aims of the present contribution are therefore to (i) selectively review key recent studies in network epidemiology, (ii) provide an overview of the main issues in this research area, and (iii) identify some major challenges for further progress in the field of plant epidemiology. The review does not aim to be comprehensive, as it attempts to draw lessons for plant epidemiology from the body of literature on diseases spreading through

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**Table 1.** A selection of key terms in network epidemiology (see Newman 2003 and the references in the table for further definitions and details).

<table>
<thead>
<tr>
<th>Term</th>
<th>Explanation</th>
<th>Example of reference(s)</th>
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<tbody>
<tr>
<td>Adjacency matrix</td>
<td>A table summarizing the presence or absence of a link between nodes in a network</td>
<td>Hummon and Doreian (1990)</td>
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<tr>
<td>Basic reproduction number ($R_0$)</td>
<td>The average number of individuals infected by an infectious individual in a completely susceptible population. Typically, $R_0 = 1$ is the epidemic threshold in a homogenous population, but this result is not valid in the presence of heterogeneity in the network contact structure</td>
<td>Heesterbeek (2002), May (2006)</td>
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<tr>
<td>Clustering of a network</td>
<td>The extent to which nodes connected to any node $x$ are connected to each other</td>
<td>Boccaletti et al. (2006)</td>
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<tr>
<td>Connectance</td>
<td>The number of links present in a network divided by the maximum potential number of links in that network (i.e. the squared number of nodes)</td>
<td>Borrett and Patten (2003)</td>
</tr>
<tr>
<td>Contact network</td>
<td>The network of interactions among individuals. A contact is a realized link. Contact structures are equivalent to network structures</td>
<td>Rohani et al. (2010)</td>
</tr>
<tr>
<td>Degree distribution</td>
<td>Frequency distribution of the number of links per node. In scale-free networks, the degree distribution is well described by a power law (due to the presence of super-connected nodes). Power-law degree distributions can be caused by preferential attachment mechanisms (new nodes becoming connected to nodes that already have more links than others)</td>
<td>May (2006)</td>
</tr>
<tr>
<td>Epidemic threshold</td>
<td>The boundary (e.g. in the number of contacts or the probability of infection transmission) between no epidemic and epidemic development</td>
<td>Brés (1986)</td>
</tr>
<tr>
<td>Meta-population</td>
<td>A network of connected populations, rather than of individuals</td>
<td>Hess (1996)</td>
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<td>Modularity</td>
<td>The degree of correlation between the probability of having a link joining nodes $x$ and $y$ and whether nodes $x$ and $y$ belong to the same community</td>
<td>Newman (2006)</td>
</tr>
<tr>
<td>Network</td>
<td>A set of individuals connected by links. If the links are asymmetric, the network is directed</td>
<td>Danon et al. (2011)</td>
</tr>
<tr>
<td>Shortest path length</td>
<td>The shortest number of steps (links between nodes) needed to move from node $x$ to node $y$</td>
<td>Frank (1969)</td>
</tr>
<tr>
<td>Transmission trees</td>
<td>A reconstruction of the transmission events between hosts</td>
<td>Ypma et al. (2013)</td>
</tr>
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</table>

**Figure 1.** Temporal trend in the proportion of epidemiological publications mentioning networks (obtained by dividing the number of papers retrieved each year searching at the same time for the keywords ‘networks’ and ‘epidemic’ by the number of papers retrieved that year with the keyword ‘epidemic’), in Google Scholar and Web of Science (1991–2010, as abstracts are searched in Web of Science starting from 1991 only; some papers published after 2010 may still need to be indexed).
human networks. There is much less material on diseases spreading in networks for horticulture, grassland and forestry compared with human medicine, despite the potential of the approach in the plant sciences (Jeger et al. 2007; Moslonka-Lefebvre et al. 2011; Garrett 2012). Network applications are relatively underused in animal epidemiology too (Woolhouse et al. 2005; Craft and Caillaud 2011; Dubé et al. 2011), but less so than for plant diseases.

The review discusses five major types of challenges relevant to

(i) defining contact networks related to epidemics,
(ii) assessing the consequences for diseases of modern mobility,
(iii) studying disease spread and control in directed, hierarchical networks,
(iv) moving from static to dynamic network models, and
(v) developing spatio-temporal network simulations of diseases.

The review concludes by pointing out challenges due to the interdisciplinarity of the field and the need to ensure long-term funding for research and review in the network epidemiology of plant diseases.

Contact Patterns

Network structures are a key factor in epidemics (Eames and Keeling 2002; Christensen et al. 2010). Much effort is thus being invested to improve our understanding of real-world contacts between human beings (Ames et al. 2011; Potter et al. 2011; Oliveira and Gama 2012). Contacts are

Table 2. A selection of key network theory insights about disease management.

<table>
<thead>
<tr>
<th>Insight</th>
<th>Explanation</th>
<th>Reference</th>
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<tbody>
<tr>
<td>No epidemic threshold in scale-free networks of infinite size</td>
<td>In infinite-size networks with super-connected nodes, pathogens are able to spread no matter how low their infectivity is, because the low transmission rate is counteracted by the efficiency of the scale-free structure</td>
<td>Pastor-Satorras and Vespignani (2001)</td>
</tr>
<tr>
<td>Resistance and vulnerability of scale-free networks</td>
<td>Disease spread in scale-free networks is generally resistant to random removal of nodes, but vulnerable to targeted control of hubs</td>
<td>May (2006)</td>
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<td>Acquaintance immunization</td>
<td>Super-connected nodes can be identified by randomly choosing individuals and tracing their contacts because, by definition, hubs have more connections than other nodes</td>
<td>Cohen et al. (2003)</td>
</tr>
<tr>
<td>Network topology—epidemic spread</td>
<td>Epidemics are affected by network structure, but also tend to modify the patterns of contacts from infected to susceptible individuals</td>
<td>Kamp (2010a)</td>
</tr>
<tr>
<td>Host behavioural changes can amplify epidemic cycles</td>
<td>If mild epidemics are followed the next year by low vaccination rates, and vice versa for severe epidemics, there is the potential for an amplification of natural epidemic cycles</td>
<td>Cornforth et al. (2011)</td>
</tr>
<tr>
<td>Vaccination makes sense if connectivity is local</td>
<td>For an outbreak to be contained by voluntary vaccination only, infection transmission has to happen through close contacts only</td>
<td>Perisic and Bauch (2009)</td>
</tr>
<tr>
<td>Epidemics are more likely in a small-world network</td>
<td>Heterogeneity in commuting distances between individuals reduces the critical threshold for epidemics</td>
<td>Balcan and Vespignani (2012)</td>
</tr>
<tr>
<td>A static network structure can sometimes be realistic</td>
<td>If demographic and social changes do not result in substantial changes to the network (at least compared with the rate of spread of the disease), then a fixed network structure can be a realistic first approximation</td>
<td>Christensen et al. (2010)</td>
</tr>
<tr>
<td>The identity of moving individuals matters</td>
<td>Meta-population epidemic models that keep track of the identity of moving individuals result in spatial spread of disease reduced by ~20%</td>
<td>Keeling et al. (2010)</td>
</tr>
<tr>
<td>Lower epidemic threshold in dynamic small-world networks</td>
<td>Small-world networks with dynamic long-distance links show a higher probability of disease spread (equivalent to adding 20% more short-cuts in a static small-world network)</td>
<td>Ochab and Gora (2011)</td>
</tr>
<tr>
<td>Real-world small-world networks are also slow</td>
<td>Community structure and the temporal dynamics of links slow down the spread of diseases in real-world small-world networks</td>
<td>Karsai et al. (2011)</td>
</tr>
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</table>
interactions between individuals which create a network (Table 1). Methods used to gather these data and key challenges in their application in modelling infectious diseases have been reviewed by Barrat et al. (2014).

For example, a diary-based survey of close interactions of a sample of the human population in different European countries showed a high degree of age assortativity, i.e. the tendency of individuals of a given age range to interact with other individuals of the same age range (Mossong et al. 2008). Not taking into account age assortativity in epidemic models of pertussis has been shown to lead to misleading policy recommendations (Rohani et al. 2010). High-resolution contact patterns were recently obtained from a French school using sensing technology over 2 days (with an average of 323 contacts per day per participant; Stehle et al. 2011) and from an Italian paediatrics hospital ward during a 1-week period (with a median of 20 contacts per day per participant; Isella et al. 2011).

Heterogeneities in the number of links are fundamental not just within a population, but also among different population subgroups (Conlan et al. 2011). Also the duration of a contact matters, for example because an infected individual will not be able to infect a new individual if it keeps staying in contact with those that have already been infected (Miller et al. 2012). A postal and online survey of the contacts of 5000 citizens in England, Scotland and Wales found high levels of clustering (the extent to which contacts are transitive; Table 1), which increased with the frequency and duration of contacts (Danon et al. 2013). Data on the frequency and duration of individual-to-individual interactions were recorded at scientific conferences, with a tendency for most interactions to be ephemeral (Zhao et al. 2011). In the European diary-based survey, the majority of daily contacts lasted an hour or more, but this length was reduced to <15 min for the majority of contacts with previously unknown people (Mossong et al. 2008). This implies that contacts tend to be longer within clusters of human individuals than across them.

It is now accepted that the number of different contacts (the degree of a node) is one of the key indicators of the risk to acquire and further spread a disease (Salathé et al. 2010), but other network metrics, e.g. repeated exposure, can be just as important (Iozzi et al. 2010). A major challenge remains in inferring network properties from sampling data obtained from a limited part of contact networks (Stumpf et al. 2005; Annibale and Coolen 2011; Nickbakhsh et al. 2011). Variation in sampling effort among studies mapping networks is a source of uncertainty also outside epidemiology, e.g. in anthropology, food web ecology and geography (Dormann et al. 2009). Different types of contacts (e.g. family, healthcare, sexual, social, travel, virtual contacts) will tend to be associated with different probabilities of disease transmission across these contacts for different types of disease (Rothenberg and Narramore 1996; Smith and Christakis 2008; Smieszek 2010; Bolton et al. 2012).

Different types of contacts are found in plant trade networks too, if only because of the diversity of traded plant species and types (cut flowers, plants for planting, bare-rooted saplings, ornamental twigs, fruits, etc.) (Dehnen-Schmutz et al. 2010). Inferring the properties of a whole network when only sampling parts of it is an issue for plant trade networks—just as for human social networks—because of the arbitrariness of social and trade network boundaries, and also due to the general lack of standardized data on plant trade networks. It is likely that contacts among plant traders (e.g. nurseries, garden centres and supermarkets) are more long-lasting than contacts among researchers at a conference, but collaboration of researchers with plant health authorities and traders is needed to prove that this is so.

Plant epidemiologists can only aspire to the data currently gathered on human contact patterns. For reasons of commercial sensitivity, plant trade networks are indeed still poorly characterized. This lack of knowledge on the structure of horticultural and ornamental trade networks makes it difficult to prevent the inadvertent dispersal of plant pathogens and pests together with the traded plant material (Helfer 2014; Schoebel et al. 2014). Regional and international surveys are needed to achieve reconstructions of the networks of plant shipments among plant nurseries and their customers, as well as the networks of informal plant and seed exchanges among farmers, gardeners or urban dwellers.

To assess how likely each commodity is to spread various plant diseases, we then need to overlap the trade network with interception data, which are already recorded (although not always made publicly available) at international borders. Very helpful from a plant health risk assessment perspective would be the systematic inspection of intercepted consignments, e.g. at EU borders, so as to gather data on the proportion of plant material infested by a particular pest or pathogen. But these would be costly measures and border inspectors are already overwhelmed by the increasing amounts of traded plant material (van der Graaf and Khoury 2010).

Of concern is also the lack of studies on the networks of plants and plant commodities traded on the internet (Giltrap et al. 2009; Pautasso et al. 2012; Shirey et al. 2013). We need to map the presence of heterogeneities in contact numbers among internet plant traders, so as to establish whether super-connected nodes are present and, in that case, targeting control at them, because this approach has been shown, for livestock diseases and
vertebrate invasive species, to be more efficient and effective than other control strategies (Natale et al. 2009; Florance et al. 2011).

**Human (and Plant) Mobility**

Current human mobility is massive, unprecedented, still increasing and has important consequences for the potential and actual spread of diseases over multiple scales. Not only people, but a range of associated goods, ideas and organisms are moving around the planet along modern transportation and communication networks (see reviews in Brockmann 2010; Kaluza et al. 2010; Woolley-Meza et al. 2011; Fisher et al. 2012).

One of the challenges of including human mobility data in epidemic models is given by the multiple types of human movements. Although regional commuting flows (e.g. train, car, bus and cycling journeys) do not make the planet a small-world (i.e. a network with a low average number of steps between two randomly chosen individuals) on their own, they are about one order of magnitude larger than airline flows. Thus, taking into account regional commuters in epidemic models leads to increased synchronization of epidemics in different parts of the planet (Balcan et al. 2009). This synchronization may in turn decrease opportunities for health officers, policy makers and the public in regions where a pathogen is still unreported to learn from epidemic developments in regions first hit by that pathogen.

Human mobility data are being obtained from various sources, from consecutive sightings of banknotes to mobile phone records. While banknote sightings have been useful in establishing the predictability of movement patterns on broader scales, mobile phone trajectories operating in space–time provide detailed insights on individual mobility patterns (González et al. 2008).

A key implication of current human mobility patterns is that epidemics of infectious diseases will spread more rapidly than in previous centuries or decades, other things being equal (Merler and Ajelli 2010a). However, for many human diseases other things are not equal: hygiene levels have steadily improved, and people’s awareness of infectious diseases, their causes and what can be done to prevent and cure them is not what it was in the past (Smith 2007; Ashenburg 2008; Armelagos 2009). At the same time, there are factors other than globalization facilitating the emergence of new diseases. For example, many more people inhabit the planet (and its urbanized areas) than at any time in the past, antibiotic resistance has arisen repeatedly and the climate has started to warm (McMichael and Beaghole 2000; May 2007; Jones et al. 2008; Harper and Armelagos 2010; Conraths and Mettenleiter 2011).

Each of these developments has consequences for epidemic development: for example, many pathogens tend to reach urban areas earlier than rural ones (Weiss and McMichael 2004; Woolhouse and Gaunt 2007; Pongsiri et al. 2009; Merler and Ajelli 2010b), so that a more urbanized planet will tend to have less time to respond to new epidemics of human diseases. A counteracting factor here is that care tends to be more accessible in urbanized regions compared with rural areas (Rabinowitz et al. 1999). A key research issue is how the heterogeneity of mobility (and disease management) networks can be influenced in a timely (and long-term) manner to reduce the risk posed by epidemics (Miller et al. 2009; Draief and Ganesh 2011).

Increasing mobility is a key issue in the network epidemiology of plant diseases, because ideally information about how to handle a new invasive plant pathogen needs to reach farmers and plant health authorities before the pathogen is introduced (Rebaudo and Dangles 2011; Garrett 2012). Often this is not the case. For example, the concurrent emergence of the epidemic of Phytophthora ramorum in the west coast of the USA and in Europe took place when both regions lacked epidemiological knowledge on this aggressive, generalist and new plant pathogen (Brasier and Webber 2010; Pautasso 2013; Redlin et al. 2014).

Plant passporting schemes and their associated trace-forward and trace-back data hold much potential to reconstruct plant mobility patterns, but this potential still waits to be exploited. Also in the case of plant health, molecular advances and international regulations are developing tools to counterbalance the risks posed by globalization, but there is a need to improve the uptake of network approaches by plant health authorities (Brasier 2008; MacLeod et al. 2010; Jeger et al. 2012; Hantula et al. 2014). Research is needed to test whether there is heterogeneity in the average commuting distances of plants-for-planting of different species and in various regions. A potentially confounding factor in such studies is the variation among rural and urbanized regions (as well as among industrialized and developing countries) in resources to track and fight plant epidemics. Even if new plant pathogens tend to be recorded first in urbanized regions (Yemshanov et al. 2013), the bulk of the cultivated area is located in rural areas, so that there might be a longer lag between first report and epidemic, thus providing some more time for rural economies to prepare for new plant health risks.

Taken together, these changes in the global health landscape imply that there is a need for further integration of human and plant mobility data in plant disease epidemic models. The growing complexity of mobility data can potentially enable more detailed epidemiological
models, but these may also be more difficult to interpret and generalize, so that a compromise between realism and parsimoniaiousness is needed.

**Directed and Hierarchical Networks**

One key assumption that is often taken in models of epidemic spread in networks is the symmetrical nature of contacts (Bianconi et al. 2008). This greatly simplifies simulations of epidemics in networks, but may or may not be appropriate depending on the system (reviewed in Newman et al. 2001; Meyers et al. 2006). For example, while disease transmission on transportation networks is often asymmetric, airborne infection can be thought of as symmetric without loss of generality (although asymmetry can be present for airborne dispersal depending on the prevailing wind direction and speed).

Even in the case of symmetric contact patterns (i.e., where the probability of transmission is equivalent in both directions), transmission trees (the routes followed by infection) are directed (Welch et al. 2011). Pathogen transmission tends to occur from one node to another, e.g. from the country of first detection to elsewhere in the world (although subsequent re-infection from elsewhere in the world to the country of first detection can well take place). For example, as a likely consequence of its special position as a transatlantic bridge between the USA and continental Europe, the UK was the only European country to experience a summer peak of the H1N1 flu in 2009 (Merler et al. 2011). Epidemic transmission trees are in many cases unknown, but can be reconstructed from phylogenetic information on the pathogen at various locations in the putative contact network (Mascheretti et al. 2009; Leventhal et al. 2012; Xhaard et al. 2012; Ypma et al. 2012).

The directedness of networks results in hierarchical systems where nodes can be divided into main classes. For example, nodes can have (i) more outgoing than incoming connections, (ii) more incoming than outgoing connections, or (iii) a balance between the two (Pautasso et al. 2010c). Networks relevant to disease spread which show hierarchical features in their structure include the world trade web (Ruzzenenti et al. 2010; Barigozzi et al. 2011; De Benedictis and Tajoli 2011) and subsets of it such as the horticultural and ornamental plant trade networks (Dehnen-Schmutz et al. 2010; Bradley et al. 2012; Liebhold et al. 2012). Food webs of producers, consumers and parasites are also inherently asymmetric and hierarchical (Foster et al. 2010), and so are many networks diffusing health information from researchers to physicians, policy makers and patients (Wang and Noe 2010; Zappa 2011).

In directed networks of small size (hundreds of nodes), the presence of super-connected nodes only lowers the epidemic threshold if these nodes have a high number of both incoming and outgoing links (Moslonka-Lefebvre et al. 2009). An open question is how small the size of networks can be for significant differences in the epidemic threshold among network structures to be present (for a given number of network replicates). In hierarchical, directed networks with node status (infectious vs. susceptible) along a continuum, the epidemic final size correlates well with the number of outgoing links of the starting node of the epidemic, regardless of network structure (local, random, small-world and scale-free) and connectance level (Pautasso et al. 2010b). Since we do not know which network structure applies to plant trade networks, the out-degree of a node can thus be a useful indicator of its risk.

A continuum between infectivity and susceptibility makes sense when nodes are composed of multiple units (e.g. pupils in school classes, patients in hospital wards, plants in plant nurseries), but such an approach has been rarely used in network epidemiology (Moslonka-Lefebvre et al. 2012). There is thus a challenge in inferring whether results obtained from models assuming that nodes are binary, i.e. either entirely susceptible or entirely infectious, can be transferred to plant trade networks, as only a proportion of the plants in nurseries and garden centres are typically infected. The same problem applies in plant pathology when using results obtained from epidemic simulations in symmetric networks, given that networks of plant movements are inherently directed. When plants (and their associated organisms) are moved from location x to location y, the reverse movement from location y to location x does not usually occur (Brenn et al. 2008). Information is needed about the frequency and dynamics, in various countries, of subgroups such as plant producers, wholesalers, retailers and end users, because their relative proportion has been shown to be related to the likelihood that an epidemic will spread (Pautasso et al. 2010c).

**Dynamic Networks**

The influence of network topology on epidemic spread is not mono-directional, because, as the epidemic unfolds and individuals become infected or recover from infection, the patterns of contacts from infected to susceptible individuals will be modified (Kamp 2010a, b; House 2012). In addition, dynamic networks can result from the mobility of hosts (Song et al. 2010a; Gargiulo et al. 2012) and active behavioural changes in response to an epidemic (Saadatian-Elahi et al. 2010; Ridenhour et al. 2011). A review focusing on this area is provided by Funk et al. (2010).

Despite the many sources of network dynamics, most epidemiological models have assumed so far that the structure of a network is fixed (Grindrod and Higham 2010). A similar assumption is that demographic processes
(e.g. birth, migration, marriage, death) do not result in substantial changes in the network (at least compared with the rate of spread of the disease; Christensen et al. 2010). However, taking into account the dynamic nature of social networks can lead to different model outcomes or data requirements (Fefferman and Ng 2007; Vernon and Keeling 2009; Volz and Meyers 2009; Fig. 2).

In small-world networks, dynamically rewiring long-distance links during an epidemic leads to a higher probability of disease spread (equivalent to adding 20% more short-cuts; Ochab and Gora 2011). Incidentally, a dynamic small-world network is computationally more tractable than a fixed one, because adjacency matrices are no longer required; all that is needed is the probability for a short-cut to be present (Stone and McKay 2011). This can be a disadvantage though, if the aim is to model adaptive rewiring (the transformation of local connections into long-distance ones in an adaptive way) in response to an epidemic, because with that approach the long-distance connections are chosen randomly (Stone et al. 2010).

Some epidemic models taking into account human mobility are dynamically quenched, which means that if an individual visits a new location, the same individual will be more likely than not to return to that place in the future (Song et al. 2010b). Even if this is the case, the topological distance (the shortest path separating nodes; Table 1) may differ in static vs. temporal networks. Temporal networks are those taking into account changes in the contacts through time. For example, an alternative way to create a dynamic (small-world) network is to simulate disease spread on 365 static (but different) networks, one for each day of the year (Vernon and Keeling 2009). Nodes that appear to be central in static networks may be of limited importance in spreading disease when the temporal fluctuations in contact patterns are considered (Pan and Saramäki 2011). Whether this result also applies to plant trade networks is to the best of our knowledge still to be tested.

For both human and plant diseases, a challenge in modelling disease spread in dynamic networks is the use of metrics that can account for dynamic properties (Natale et al. 2011), given that most network metrics are based on static networks (Bajardi et al. 2011b; Nöremark et al. 2011; Klemm et al. 2012). In addition, it would be interesting to know whether temporal preferential attachment mechanisms (the longer a node is connected to another node, the more the two nodes are likely to be connected in the future; Stehlé et al. 2010) apply also to plant traders.

Nowadays, traded plants and plant parts are no less mobile than human beings, and changes in the behaviour of the industry in response to plant epidemics are observed also in the trade of plants (e.g. switching from susceptible to resistant plant hosts). Research on how long-distance plant trade connections emerge in practice and whether they tend to be maintained through time would be needed. For example, dynamic network models of the epidemic of soybean rust (Phakopsora pachyrhizi) in the USA, with counties as nodes and links weighted in proportion to host cultivated area and wind speed and direction, resulted in a reduction of the resources needed to track and predict the crop epidemic (Sutrave et al. 2012). This is a good example of how network models can be used without an underlying trade network.

**Spatio-temporal Network Simulations**

For disease control to be informed and to benefit from epidemic simulations in networks, these need to be grounded in realistic spatial and temporal settings.
A review of the effects of spatial structure on infectious disease dynamics is provided by Rock et al. (2014).

Networks have traditionally been treated as abstract constructs, given their origin in mathematical topology (Dale and Fortin 2010; Rozhnova et al. 2011). However, where a group of contacts is located and how it evolves in time can make epidemic outcomes differ from what would have been predicted at an abstract network level (Bertuzzo et al. 2010). Similarly, superimposing contact networks on spatial epidemic models can improve overall predictions. For example, the peak in influenza-like incidence reported in Italy in 2009 was shown to have occurred earlier in regions with a higher number of arriving international air-bound passengers (Ajelli et al. 2011).

The key role of space does not just apply to epidemics in networks, because many biological and social networks are also spatially embedded (Bartha 2011; Expert et al. 2011). However, for epidemics, more so than for other systems, there is often the necessity to assess in real time (while the epidemic is ongoing) the potential impact over geographical regions (Birrell et al. 2011). This is difficult also for human diseases given the frequent lack of good data at the beginning of an epidemic (Katriel et al. 2011). Network approaches can help trace back the origin of epidemics. For example, an airline network perspective makes it possible to recover an expanding wave of infection around the airport from which an epidemic started spreading, whereas that origin would not be easily traceable when following the same epidemic on a spatial map of the world (Brockmann and Helbing 2013).

A step forward would be the development of software for contemporaneous temporal comparison of different policy measures (Maciejewski et al. 2011). There is the need to test the effectiveness of the use of such visual tools by researchers and public health officials in the evaluation of measures to respond to epidemic outbreaks (Afzal et al. 2011). Together with better data at the early epidemic stages, spatio-temporal simulations are needed to make progress in assessing how behavioural changes and the spread of health information affect the resilience of networks (Birrell et al. 2011; Döring et al. 2014). Also control measures tend to differ in space and time, so that an important issue to be explored is the interplay of dynamic disease control measures in network epidemic models (Green et al. 2011).

Epidemic simulations and adaptive disease management modelling in real time are still unrealistic for plant epidemics occurring in trade networks for which there is no information about the network structure, particularly when disease monitoring is patchy and reporting is slower than the epidemic. However, molecular advances (e.g. real-time PCR) and citizen-science approaches (e.g. in the mapping of emerging diseases across whole countries) could make real-time simulations possible for plant epidemics too. A spatio-temporal approach is already inherent in empirical analyses of plant pathogens introduced by trade networks into the wider environment. For example, a study of first records of plant pathogens in the UK (1970–2004) showed that the Netherlands was the origin of introductions (when known) nearly half of the time, and the South-East of England was the region of first report (when known) for 38% of newly recorded pathogens (Jones and Baker 2007). For historical and geographical reasons, the Netherlands and the South-East of England have more trade connections than other European countries and British regions, respectively.

This kind of analysis can provide useful data to parameterize spatial epidemic network models. Similarly, a temporal study of the spatial association in England and Wales (2003–06) between P. ramorum reports in the trade and in the wild found that infected premises (garden centres and nurseries) were significantly associated with infected sites (woodlands and historic gardens) at short distances (<1 km) over the studied period, thus suggesting a role of the trade in spreading the epidemic (Xu et al. 2009). Given that various Phytophthora species are found in plant nurseries, and that some of these species have hybridized to produce new emerging pathogens, plant epidemiology too may benefit from models of the interactions between different pathogen strains spreading in spatial networks (Alexander and Kobes 2011; Dorigatti and Pugliese 2011; Karrer and Newman 2011; Weng et al. 2012).

Conclusions

A major issue for spatially explicit modelling of diseases spreading in networks is the communication of its inherent uncertainty and simplifications to end users (policy makers, health providers, journalists, patients) (Prieto et al. 2012). Despite the growing sophistications, spatial models of epidemics in networks are still simplifications of a complicated system (Reppas et al. 2010). Only rarely is this acknowledged in the discussion of papers reporting results from models of epidemics in spatial networks, so that there is a danger of take-home messages being read without keeping in mind all the various caveats and assumptions of the mathematical models behind policy recommendations (Ferguson et al. 2006; Fraser et al. 2009; Garnett et al. 2011).

Nevertheless, networks provide a flexible framework that can link disciplines and practitioners working in different domains, for example human and plant pathologists, as well as epidemiologists and invasion biologists.
Network theory is a useful addition to the set of tools available to understand and manage (plant) diseases, particularly when pathogens are dispersed by human movements over long distances (Jeger et al. 2007). This review has focused on the use of networks to model the plant trade, but network theory in plant epidemiology can also be applied at the genetic, molecular, meta-population and landscape levels (Moslonka-Lefebvre et al. 2011).

One important research area where network theory is likely to prove fruitful is the integration of plant health models predicting climate change impacts with those assessing the likely effects of globalization on emerging plant diseases (Pautasso et al. 2010a). Long-term funding for plant health modelers, experimenters and practitioners would be needed to develop this research programme. Nevertheless, an improved understanding of the structure and dynamics of plant trade networks can already provide useful insights before the expected climate changes of the coming decades take place. For example, the recent establishment of European ash dieback (caused by Hymenoscyphus pseudoalbidus; Queloz et al. 2011) throughout Europe was facilitated by local and long-distance trade in infected ash saplings, which continued to be planted in forests and plantations for several years despite the growing evidence that tree nurseries were a pathway for the spread of this emerging disease (Gross et al. 2014).

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### Conflicts of Interest Statement

None declared.

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