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Modelling plant disease epidemics

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Abstract

An epidemic is the progress of disease in time and space. Each epidemic has a structure whose temporal dynamics and spatial patterns are jointly determined by the pathosystem characteristics and environmental conditions. One of the important objectives in epidemiology is to understand such spatio-temporal dynamics via mathematical and statistical modelling. In this paper, we outline common methodologies that are used to quantify and model spatio-temporal dynamics of plant diseases, with emphasis on developing temporal forecast models and on quantifying spatial patterns. Several examples of epidemiological models in cereal crops are described, including one for Fusarium head blight.

Introduction

Mathematical modelling of crop disease is a rapidly expanding discipline within plant pathology. The first models of the temporal development of epidemics were developed by Van der Plank (1960; 1963), and have since formed the basis for disease modelling (Campbell and Madden, 1990; McCartney, 1997). In 1969, Waggoner and Horsfall published their model of potato early blight epidemics caused by *Alternaria solani*. Subsequently, various models have been developed (Jeger and Tamsett, 1983). A model is a simplification of reality and attempts to summarise the main processes, to put forward hypotheses and to verify their coherence and consequences. It also represents a trial to determine the minimal hypotheses which would allow minimal mathematical representation of real processes. In epidemiology, modelling aims to understand the main determinants of epidemic development in order to develop sustainable strategies for strategic and tactical management of diseases.

Epidemiological models can be classified in several ways. For convenience, Kranz and Royle (1978) classified them into three types – descriptive, predictive and conceptual – according to their main objective. Descriptive models provide hypotheses or generalise experimental results, but they do not usually reveal the mechanisms underlying the processes. Predictive models, which are also descriptive, allow the prediction of the occurrence and the severity of epidemics. Both descriptive and predictive models use mathematical tools, such as simple or complex functions, regression and differential equations, or simple decision models. The conceptual models, also known as explanatory or analytical models, allow the identification of problems by distinguishing cause from effect and quantify the effects of specific events on epidemic development. They are constructed as representations of underlying biological and ecological processes. These models may eventually lead to the development of complex simulation models. It should be pointed out that models can be disease-specific, but can also be very general. A descriptive model is often concerned with understanding and predicting development of specific diseases, and thus is generally used for assisting growers in making tactical decisions in managing diseases. A conceptual model is often concerned with the theoretical understanding of generic features of epidemic development and thus is used more for making policy and strategic decisions.

Recent advances in computer tools have made mathematical/statistical modelling more accessible and

have led to the development of more complex models for many diseases. In this paper, we attempt to provide a brief introduction to the framework for developing epidemiological models. For this purpose, various modelling processes, such as problem specification, are reviewed. Finally, several models for cereal diseases are briefly presented. We have focused on two areas: forecasting temporal disease dynamics and quantifying spatial patterns. We intentionally do not include any discussion of the more theoretical aspects of epidemiological modelling, nor recent approaches which attempt to combine temporal models with spatial models.

Problem specification

Since models are most useful when directed at a clearly specified and well-defined problem, the crucial first step in modelling is to precisely define the question(s) to be addressed. The nature of the problem is likely to determine what are the most appropriate modelling techniques to use (Kranz and Royle, 1978; Sutherst, 1993). In fungal epidemiology, possible objectives for modelling include (Norton et al., 1993):

1. predicting the timing of an event, such as when disease infection is likely to occur;
2. predicting the scale of an event, such as the severity of disease infection or crop loss;
3. estimating the frequency or the probability of an event, such as monocyclic or polycyclic epidemics;
4. assessing and comparing the performance of different management strategies.

Biological characteristics of a fungal pathosystem

Once the objective of a model is clearly established, the second step is usually to determine the variables that are needed for developing the model. These variables generally represent key features in the development of epidemics, including initial inoculum, progeny/parent ratio and the length of latent period (Van der Plank, 1982) as well as major external factors. Recently, de Vallavieille-Pope et al. (2000) assessed the use of various epidemiological parameters in modelling. They identified spore production, latent period, the number of infection cycles, host resistance and external factors as key components of disease development. The importance of the interactions between pathogen and host population dynamics has long been underestimated (Gilligan, 1985) and is now receiving due attention (Webb et al., 2000).

Fungal pathogens

Inoculum

Fungal inoculum is of prime concern; its source, density and type will greatly influence the design of the forecasting scheme. Without inoculum, there is no epidemic. A fungal population consists of individuals at various stages of their life-cycle. The population can be described by the proportion or absolute quantity of individuals at each stage, i.e. age structure of the population (Shaw, 1998). For example, for modelling purposes, *Blumeria graminis* on cereals may consist of three components: (1) spores, each potentially capable of infection, (2) mycelia, and (3) cleistothecia (Shaw, 1998). Passage from one stage to the next can be very fast, usually depending on environmental conditions. Inoculum may also be simply divided into primary and secondary; such division has been found to be very useful in modelling cereal soil-borne pathogens (Gilligan, 1985). van Maanen and Gourbiere (2000) showed the importance of the life-cycle stages in the control of fungal dynamics, by adapting a patch-occupancy model previously developed by Gourbiere et al. (1999).

Fungal populations are difficult to study under field conditions because individual mycelia and spores cannot generally be easily quantified (Siefert, 1981). The assessment of the amount of the pathogen on and in the leaves is notoriously difficult. For example, sporulating mycelia of *Stagonospora nodorum* or *Mycosphaerella graminicola* of wheat, causal agents of leaf blotching and spotting infection, are hidden within the leaves. Visual assessment of the percentage leaf area covered by lesions can be rapid, but is quite subjective, and hence may not be a reliable assessment of inoculum (de Vallavieille-Pope et al., 2000). Numbers of colonies or pustules can be counted accurately on a small scale, but this is not practical on a large scale. To simplify the matter, a pathogen population is often measured indirectly as disease incidence or severity. Other methods used to quantify pathogen populations include determination of the fungal biomass using enzyme-linked immunosorbent assay (ELISA) (Newton and McGurk, 1991), ergosterol and/or chitin content measured using high performance liquid chromatography (HPLC) (Johnson and McGill, 1990; Barajas-Aceves

et al., 2002) and competitive PCR methods (Nicholson et al., 2002). Quantifying inoculum of soil-borne pathogens is generally more problematic (Jeger, 2000).

Inoculum dispersal

Inoculum dispersal fulfils essentially three functions: (1) population survival, (2) colonisation of new habitats and (3) reproduction (Ingold, 1971). Dispersal can occur either by mycelium or spores. Spore dispersal comprises three phases; liberation, which can be passive or active (Dix and Webster, 1995), transport, and deposition (Ingold, 1978). The dispersal scale depends on inoculum properties as well as the transport vector, and may range from a few metres through rainsplash (Madden, 1992), to 100–10 000 m for airborne spores, such as those of powdery mildew of cereals (Andrivon and Limpert, 1992). Often, spore dispersal can be described by either an exponential or a power function (Fitt et al., 1987).

Disease models differ in the method by which the various steps in the disease-cycle are formulated, but each model includes the equivalent of a primary deposition function (McCartney and Fitt, 1998). The primary deposition function plays an important role in both the shape of the disease gradients and the expansion rate of the disease focus. Ferrandino (1993) derived a function to account for loss of spores both by escape from the canopy and by deposition. The model generated disease gradients that became shallower as the epidemics progressed. Such a pattern has been observed in diseased crops, e.g. potato late blight (Minogue and Fry, 1983b) and *A. linicola* on linseed (Vloutoglou et al., 1995). The efficiency of spore dispersal affects the density of new infections (de Vallavieille-Pope et al., 2000). Gourbiere et al. (1999) considered dispersal as the main parameter which determines the number of newly colonised units in their model, which has been further extended to simulate the distribution and frequency of new infections along weather gradients (van Maanen et al., 2000).

Latent and infectious periods

In epidemiology, the latent period is the interval between the onset of spore germination and the appearance of the next spore generation. The rate of epidemic development is largely influenced by the length of latent period, which determines the number of potential infection cycles that can be completed during a growing season (de Vallavieille-Pope et al., 2000). The shorter the latent period, the more reproduction cycles the fungus can have per season. In contrast to polycyclic diseases, monocyclic diseases have only one reproductive cycle throughout a single season.

The importance of the latent period and spore deposition frequency in modelling has been emphasised by Gumpert et al. (1987). Latent periods have been reported to depend on inoculum and lesion density (de Vallavieille-Pope et al., 2000), but are mainly influenced by temperature (e.g. Beresford and Royle, 1988; Xu, 1999; Xu and Robinson, 2000; 2001). They may also vary with the level of host susceptibility and with host growth stages, features that emphasise the importance of studying both pathogen and host dynamics. Another key factor influencing the development of an epidemic is the length of the infectious period (i.e. the length of time during which a single colony continues to produce spores), as this determines the quantity of spores that a single colony is likely to produce during its lifetime.

Pathogen dynamics regulation

Fleming (1980) argued that the role of predation and parasitism in pathogen regulation might be greater than previously recognised. Burgess and Hepworth (1996) recognised various hyperparasites attacking sclerotia of *Sclerotinia* spp. Brasier (1990) showed that the transfer of virus pathogens of fungi would occur more efficiently when the fungal population is at a high density. Another natural regulation comes from pathogen population competition, e.g. the competition between the eyespot pathogens *Tapesia yallundae*, *T. acuformis* and the sharp eyespot pathogen *Rhizoctonia cerealis* (Bateman et al., 1995). This aspect of microbial community interaction (symbiosis or competition) is, in general, poorly understood for fungal pathogens; it is now becoming gradually more important with the present moves to more integrated disease management strategies.

Host dynamics

For a long time, epidemic modelling has emphasised pathogen activity, ignoring effects of the host on pathogen development. Particular interests are changes in susceptibility, and the contribution of resistance, e.g., to the length of the latent period (Kranz and

Royle, 1978). Another reason why host dynamics should be included in epidemiological models arises from the fact that pathogen population dynamics are linked to host dynamics, and pathogens may affect growth and reproduction of their hosts (Anderson and May, 1979). Simple models can be developed to capture the essential features of host–pathogen interactions, though more complex models are usually necessary.

Host susceptibility and resistance

Among the host factors which need to be taken into account are the levels of intrinsic host resistance and age-related resistance associated with specific host tissues (Shtienberg, 2000). Some cultivars display increased tolerance or partial resistance (Walters and Hardwick, 2000). The nature of host resistance will affect the rate of disease development and must therefore be taken into consideration in modelling (Shtienberg, 2000). Theoretical models have been developed on the effects of cultivar mixtures or crop heterogeneity on epidemic development based on the gene-for-gene relationship (Barrett, 1978; Jeger et al., 1981a,b; Xu and Ridout, 2000). Host resistance and/or pathogen infectivity/aggressiveness may also depend on the age of host tissues. For example, studies have shown that rose tissue become resistant to infection by *Sphaerotheca pannosa* as leaves age (Rogers, 1959; Mence and Hildebrandt, 1966).

Multiple hosts and crop rotation

Paramount among farm practices is crop rotation, which has conventionally been adopted to reduce the carry-over of pathogens from one crop to another (Walters and Hardwick, 2000). However, crop rotations are also likely to favour certain pathogens when multiple hosts are available. *Sclerotinia sclerotiorum*, causal agent of stem rot in spring-sown oilseed rape, has a wide host range of about 400 species, including many weeds (Bolland and Hall, 1994). Maize rotating with wheat is known to be one of main reasons for recent severe epidemics of Fusarium head blight (Obst et al., 1997). The importance of crop rotation has been included in a forecasting system for Sclerotinia stem rot (Twengstrom et al., 1998). Crop rotation emphases the importance of the infection time-length, and as consequence, the survival of inoculum. *Polymyxa betae* produces resting spores which survive in the soil between successive crops. Survival of sclerotia for up to 5 or 10 years has been reported (Ben-Yephed et al., 1993).

Environmental factors

It is generally agreed that the environment is the driving force in the development of epidemics (Rabbinge and Bastiaans, 1989; Hardwick, 1998). This includes major climatic variables, such as rain, temperature and humidity. Wind and rain are essential for pathogen dispersal; rain provides free water on host surfaces for most pathogens to infect and sporulate and sun provides favourable temperatures for disease development (Ingold, 1971; Lacey, 1996; Francl and Panigrahi, 1997; de Vallavieille-Pope et al., 2000; Walters and Hardwick, 2000). The duration of each event as well as its timing is also important. For example, for apple scab, night rain results only in a much smaller proportion of mature ascospore being discharged compared to daytime rain (MacHardy, 1996).

Moisture, particularly the duration of wetness, is the dominant factor for most pathogens (Huber and Gillespie, 1992). Free water or near saturation moisture on the host surface is essential for germination and penetration of the host for many pathogens. Thus, not surprisingly, a single parameter indicating water availability is used in several forecasting systems. Prediction of actual wetness duration is preferable to prediction of occurrence because many pathogens cause more damage as the duration of wetness increases (Hosford et al., 1987; Francl and Panigrahi, 1997). Artificial neural network models have been developed to predict wetness on wheat flag leaves from both dew and rain (Francl et al., 1995; Francl and Panigrahi, 1997).

The role of temperature has been studied for many pathosystems, mostly for its influence on initial germination, infection, and the length of incubation, latent and infectious periods. For example, Newton (1989) showed that the infection efficiency of barley powdery mildew was reduced by over 50% at 7 °C compared to 20 °C. The different stages of the disease-cycle of *P. betae*, the fungal vector of beet necrotic yellow vein virus, are sensitive to soil temperature, which appeared to be a principal factor in influencing the occurrence and severity of the sugar beet disease rhizomania (Blunt et al., 1992).

The relationships between disease development and environmental factors are the key component and often

the only component of disease forecasting systems. Both past and future weather forecasts can be used in these systems for predicting epidemic development. Forecasting systems provide an indication or quantification of disease development, especially when the disease is likely to exceed an economic-injury threshold and thus warrants a treatment.

Mathematical representation of epidemic development

Overall, modelling can be divided into three steps: model development, model analysis and hypothesis testing. When developing a model, biological characteristics of the pathosystem are expressed as mathematical relationships. In model analysis, epidemic dynamics are investigated in relation to the parameters of interest (or variables, which may be formulated as functions of external factors such as rain and temperature). Finally in hypothesis testing, the results from model analysis are used to test or verify whether or under what conditions the hypothesis (i.e. the specified problem/question) is valid. Mathematical expression of biological features of the system is critical, since it will, more or less, determine the techniques to be used in model analysis and hypothesis testing. Here, we briefly introduce several common mathematical tools used to analyse plant disease epidemics.

Disease progress curves

Van der Plank (1960) used exponential, monomolecular and logistic models to describe the development of epidemics. He illustrated how polycyclic diseases could be described by logistic models, and monocyclic diseases by monomolecular models. Growth models (monomolecular, Gompertz and logistic models) provide a range of curves that are often similar to disease progress curves. These non-linear curves can be easily fitted to experimental data by any standard statistical package. Use of such growth curves has been described by Campbell and Madden (1990). The important parameters in these models are the initial amount of disease, the apparent rate of disease increase and the level of maximum disease. These parameters can be estimated separately for each individual treatment and their relationships with treatment or environmental factors can then be investigated. Alternatively, the mathematical relationships between the model parameters and treatment/environmental factors can be incorporated into the growth curve models and fitted to the observed data directly. More complex model fitting procedures are required for the latter approach.

Most analyses of disease progress data rely on time as the independent factor. This may not be appropriate when data are collected in different years, seasons, locations, etc. A measure of heat-sum or degree-days provides an alternative method. This assumes that temperature is the most important factor driving growth rate of the host, the pathogen and the disease. This method was used to model various epidemics, such as the cotton-Verticillium wilt system (Gutierrez et al., 1983), powdery mildew on tomato (Correll et al., 1988), and more recently, take-all on wheat (Brassett and Gilligan, 1989; Cohlbach et al., 1997). Many other modifications to standard growth models are possible. For example, to take into account the temporal variability of host susceptibility to pathogens, Lalancette and Hickey (1986) modelled disease progress as a function of host growth, which was used to represent biological time. Van der Plank (1963) introduced a correction factor for the rate of disease increase based on the exponential change in mass of susceptible host tissue. This correction factor was studied by Kranz (1975), and Waggoner (1986), and reviewed by Campbell (1998).

Area under disease progress curve

Not all disease progress curves are well or easily described by a growth curve model. Alternative methods to quantify epidemic development include the area under the disease progress curve (AUDPC). Van der Plank (1963) related area under the stem rust progress curve to the yield loss in wheat. Jeger and Viljanen-Rollinson (2001) listed a range of publications using AUDPC to assess quantitative resistance to disease. The resulting AUDPC values can be used as a measure of epidemic development and used in further analysis and hypothesis testing, such as regression and in variance analyses.

Linked differential equations

One of most commonly used mathematical techniques in modelling epidemics is the linked differential

equation (LDE), which is usually used to investigate theoretical questions concerning the dynamics of plant disease in relation to host, environment and human interventions. Van der Plank (1963) demonstrated how analytical models written as differential equations could be integrated and used to quantify the various parameters associated with disease progress. The LDE models are of the susceptible, exposed, infectious and removed (SEIR) type, which is the standard modelling approach in human disease epidemiology, and is also widely used in plant disease epidemiology (Jeger et al., 1998; Jeger, 2000).

In this approach, the host population is usually divided into several non-overlapping categories, such as healthy susceptible, latently infected, infectious and removed (post-infectious). When an individual plant becomes infected, the pathogen moves through the latent stage to become infectious at a rate which is the inverse of mean latent period. Infected plants lose infectiousness and proceed into the removed or post-infectious stage at a rate which is the inverse of mean infectious period (Segarra et al., 2001). Plant populations may be constant, but may also assume increase or decrease to model host growth or senescence processes. Depending on the hypothesis to be tested, the number of these categories used varies greatly. For example, when modelling the effect of induced resistance, an extra category, healthy resistant, may also be required.

Linked differential equation models are specified for each defined plant category, written generically as

$$\frac{\mathrm{d}P}{\mathrm{d}t} = B(P) - D(P),$$

where $B(P)$ and $D(P)$ are functions describing the increase and decrease of the host population of category P. $B(P)$ and $D(P)$ are jointly determined by host growth functions, pathogen attributes, pathogen transmission/dispersal characteristics and disease management. Some density-dependent population regulation can also be included.

Linked differential equation models are usually evaluated analytically to determine the key dynamic features of the system, and then numerically to explore the dynamics in the important conditions identified. Typical questions to be asked include (1) what are the values and stability of equilibria? (2) how are the values and stability of equilibria affected by model parameters/variables? (3) how does the epidemic approach the equilibria? (4) what are the conditions necessary for the epidemic to persist? and (5) how sensitive are the system dynamics to model parameters/variables?

Computer simulation

Many computer simulation models have been developed in the past decades. Computer simulation is in general a natural extension of LDE modelling. In computer simulation, model parameters in LDE are often assumed to be functions of external factors such as temperature and humidity. These functions can either be of simple linear type or complex non-linear type. Computer simulation can be used to study both theoretical and applied problems. Using a stochastic simulation model, the relationships of spatio-temporal statistics with underlying biological, physical and biological factors have been successfully studied (Xu and Ridout, 1998; 2000; 2001). One of the earliest spatio-temporal simulation models was EPIMUL (Kampmeijer and Zadoks, 1974), which laid the foundations for further developments (Minogue and Fry, 1983a,b; Van den Bosch et al., 1988; Zawolek and Zadoks, 1992; Ferrandino, 1993; Maddison et al., 1996). Jeger (1986) discussed the advantages and disadvantages of the simulation approach in comparison to the analytical approach.

Examples of forecasting models

Rust development of epidemics

Rust development of epidemics (RustDEp) is a dynamic simulator of the daily progress of brown rust severity on wheat (Rossi et al., 1997). It takes into account (1) the proportion of spores able to establish new infections influenced by temperature and leaf wetness (de Vallavieille-Pope et al., 1995), (2) the fact that the latent period depends on temperature (Johnson, 1980), and (3) the fact that the infectious period depends on temperature and host growth stage (Tomerlin et al., 1983). In the RustDEp model, the inputs of meteorological data are recorded by a weather station, allowing more accurate simulation of the disease progress (Rossi et al., 1997).

Sclerotinia stem rot forecasting systems

Several approaches have been used to develop forecasting methods for *Sclerotinia* stem rot, caused by

S. sclerotiorum, including checklists (Thomas, 1984), risk point tables (Ahlers, 1989), serological tests (Jamaux and Spire, 1994; Lefol and Morrall, 1996) and petal infestation assessments (Turkinson and Morrall, 1993). Nevertheless, their practical use has not been adequately evaluated and their accuracy was sometimes not satisfactory. Twengstrom et al. (1998) improved an existing risk point system using logistic regressions. The factors that affect *Sclerotinia* infection were given points with regard to the risk of heavy infestations. The choice of risk factors and risk points in the improved model was made on the basis of statistical and biological considerations. New threshold values for spraying recommendations were determined. Mild *Sclerotinia* epidemics were predicted for <40 points and spraying was not recommended. If the risk point was ≥ 50, spraying was recommended. This system is a type of decision model, which has several advantages, including ease of use and distribution.

Disease forecasting system for Fusarium head blight in the USA

A series of severe Fusarium head blight epidemics experienced in the USA (McMullen et al., 1997), led to a collaborative project between the States of Ohio, Dakota, Minnesota and Manitoba to create a forecasting model. De Wolf et al. (2000) used weather data (temperature, RH, rainfall) and mean disease levels collected in Ohio from 1982 to 1999 to identify critical environmental periods. Correlation analysis was used to identify weather variables that were potentially associated with epidemic development and then to use these variables to develop a logistic regression. The resulting risk model had an 84% level of accuracy in predicting epidemics. The model is currently under evaluation.

Another model, which incorporates rainfall, temperature and wheat heading date has been developed to produce a risk map for the production of the *Fusarium*-associated mycotoxin deoxynivalenol (DON). These maps forecast the amount of DON (in ppm) that would accumulate if the wheat was at Zadoks growth stage 59 (75% of the heads completely emerged from the flag leaf). The model was improved using data from 399 farm fields across Ontario from 1996 to 2000, using both forecast and actual weather data. DON predictions are meant to serve as a guide; a high DON level may warrant a timely application of fungicide. The DON level maps are available on the Ontario Weather Network website (www.ownweb.ca).

Quantifying spatial patterns

Recently, there has been increased interest in the statistical description and theoretical modelling of the spatio-temporal dynamics of epidemics, especially for disease incidence data, where individual plants or plant-parts are classified as diseased or healthy (Hughes and Madden, 1992; 1993; Madden and Hughes, 1995; Yang, 1995; Hughes et al., 1996; 1997; Xu and Ridout, 1998). The individuals often occur in groups that may arise naturally (e.g. leaves on a shoot) or artificially (e.g. plants in a quadrat). Many different methods have been used to characterise spatial aggregation for disease incidence data, including fitting of the beta-binomial distribution (Hughes and Madden, 1993; Madden and Hughes, 1995), variance–mean relationships (Hughes and Madden, 1992; Madden and Hughes, 1995), spatial autocorrelation (Campbell and Madden, 1990; Gottwald, 1995; Madden and Hughes, 1995), geostatistical methods (Chellemi et al., 1988; Stein et al., 1994; Gottwald et al., 1996), and distance class methods (Gray et al., 1986; Nelson et al., 1992; Gottwald, 1995; Nelson, 1995; Ferrandino, 1996; 1998). In this paper, only the most common and recent statistical methods for quantifying spatial heterogeneity of disease incidence data are briefly introduced.

To assess spatial heterogeneity of plant disease, researchers usually collect data in two ways. First, disease incidence is assessed within a sampling unit comprising a group of plants at a particular spatial point (often called a quadrat); a number of such quadrats would be randomly taken over space or time. Second, disease is assessed on all individual plants or on a subset of plants; in this case, spatial location is recorded as well as disease incidence. Sometimes, a hybrid of these two methods can also be used, i.e. disease is assessed within a quadrat but a number of such quadrats is selected on a set of predefined spatial locations. In the former method, the heterogeneity can be investigated within the quadrat. In the latter case, the spatial dependence of disease can be studied between sampling units. Usually, the information extracted from the data is positively related to the amount of effort put into the disease assessment.

When the spatial dynamics of plant diseases are studied, the following questions can be considered: (1) is

the disease aggregated? (2) how does the degree of aggregation vary with time? (3) is there any spatial dependence in disease development? (4) can such spatial dependence be quantified? (5) what is the rate of disease spread, disease gradient or spore dispersal gradient? (6) what is the impact of disease aggregation on disease management? and (7) what factors have caused the observed spatial pattern. To understand many spatial analytical tools, there is a need to first understand the expected variance of a number of infected plants within a sampling quadrat.

Disease incidence and its variance

Disease incidence is an example of a *binary* variable, one that can take only two possible values, i.e. diseased or not. Thus if X denotes the disease assessment on a particular plant (i.e., $X = 0$ for a healthy plant; $X = 1$ for a diseased plant) and π denotes the probability that the plant is diseased, then $\Pr(X = 0) = 1 - \pi$ and the value of π determines the distribution of X completely. In particular, the mean and variance are $E(X) = \pi$ and $\text{var}(X) = \pi(1 - \pi)$, respectively. This distribution, known as the Bernoulli distribution, is the only possible probability distribution for a binary random variable.

Suppose that a rectangular quadrat is sampled, with n number of plants in the quadrat, and a binary random variable X_i indicates the disease status of the ith plant in the quadrat. The total number of diseased plants (Y) in the quadrat is therefore $Y = \sum_i X_i$. If π denotes the probability that a randomly selected plant from the population is infected, then $E(X_i) = \pi$ and hence $E(Y) = n\pi$. Using the standard formula for the variance of a sum of random variables, the variance of Y is then calculated as:

$$\text{var}(Y) = n\pi(1 - \pi)[1 + (n - 1)\rho] \quad (1)$$

where

$$\rho = \frac{1}{n(n-1)/2} \sum_{i<j} \text{corr}(X_i, X_j) \quad (2)$$

Thus, the variance has two components: the variance of a binomial distribution, $\{n\pi(1 - \pi)\}$, and an additional component due to pairwise correlation in disease status between plants within a quadrat, $\{n(n - 1)\pi(1 - \pi)\rho\}$. The parameter ρ is often called the intracluster correlation (Ridout et al., 1999). In terms of spatial pattern, positive values of ρ imply spatial aggregation or clustering, whereas $\rho = 0$ implies a random pattern of disease incidence. A negative value of ρ implies some sort of regularity in the pattern, whereby the presence of a diseased plant reduces the likelihood of neighbouring plants being diseased. Thus, the degree of spatial aggregation can be assessed by determining the magnitude and significance of ρ, either directly or indirectly.

In assessing spatial heterogeneity, either incidence (i.e. proportion or percentage infected plants within a sampling unit) or counts data (number of infected plants within a quadrat) can be used. Madden and Hughes (1995) discussed the relative merits of both approaches. Here, if not specified otherwise, incidence is assumed.

Beta-binomial distribution

It follows from equation (1) above that if the incidence data can be satisfactorily fitted by a binomial distribution then this implies that intracluster correlation is expected not to be significantly different from zero. Therefore, there is no spatial aggregation of diseased plants. Typically, for an aggregated data set, there are more observed units in either the upper or lower disease incidences and correspondingly less in the mid-level of incidence than that predicted by the binomial distribution at a given level of overall incidence.

If the binomial distribution fails to describe the observed data satisfactorily, other types of distributions may be fitted to the data. One commonly used distribution is beta-binomial (Hughes and Madden, 1993; Madden and Hughes, 1995). This distribution can be derived by assuming that the incidence of disease varies from quadrat to quadrat in a random way, for example due to spatial variability in environmental factors, which gives rise to a *mixture* distribution for Y. The binomial parameter π is replaced by a random variable P, with $\text{E}(P) = \pi$ and $\text{var}(P) = \rho\pi(1 - \pi)$. Unconditionally the variance of Y is given by equation (1) above. Commonly, P is assumed to follow a beta distribution as this gives a flexible range of distributions for P, and hence for Y (Hughes and Madden, 1993; Madden and Hughes, 1995). The resulting mixture distribution is thus called beta-binomial distribution with two parameters: p and θ. Parameter p is the expected or average value of π, i.e. overall disease incidence; θ is an index of aggregation, ranging from 0 to ∞, although typically less than 1. This distribution has been successfully fitted to many observed data sets (Hughes and Madden, 1993;

Madden and Hughes, 1995; Xu et al., 2001). If the beta-binomial distribution fits observed incidence data satisfactorily, θ can then be used to indicate the degree of aggregation. In general, beta-binomial distribution may not be sensitive enough to detect aggregation for small sample sizes.

Corresponding to the incidence, Poisson distribution is equivalent to binomial distribution for counts data in assessing the randomness of the observed data. Negative-binomial distribution is often used to describe aggregated counts data; one of its parameters, k, is also used to indicate the degree of aggregation. This parameter has been incorporated into temporal epidemic models to investigate the effects of spatial aggregations on temporal disease development. Other distributions for describing aggregated counts data are also possible.

Index of dispersion

Poisson distribution fits counts data if the counts data are randomly distributed. For counts data, the variance-to-mean (VM) ratio is usually used to measure aggregation. This is essentially a variance-to-variance (observed-to-expected) ratio since the mean is the expected variance for a Poisson distribution. Thus, for a random distribution of observed data VM is expected to be 1. This VM ratio can be generalised to give the definition of index of dispersion (D) as the ratio of the observed to theoretical variance. $D = 1$ for random data, $D > 1$ for over-dispersed data and $D < 1$ for under-dispersed data. For binary data such as disease incidence:

$$D = \frac{S^2}{np(1-p)} \tag{3}$$

where $np(1-p)$ is the expected variance of incidence data assuming that the binomial distribution fits the data, and S^2 is the observed variance. A chi-square test has been used to determine whether to reject or accept the null hypothesis (randomly distributed) (Pielou, 1977). This is based on the fact that $(n-1)D$ has a chi-square distribution with $n-1$ degree of freedom for a random distribution and a constant p.

Intracluster correlation

Intraclass correlation (ρ) measures the tendency of the plants within a sampling unit (quadrat) to have a similar disease status. Positive values of ρ indicate aggregation of disease. ρ can be calculated directly from the quadrat data as (Fleiss, 1981):

$$\rho = 1 - \frac{1}{p(1-p)NT(T-1)} \sum_{i=1}^{N} Y_i(T - Y_i) \tag{4}$$

where Y_i is the number of infected plants in the ith quadrat, N is the total number of quadrats and T is the number of plants in a quadrat and is the same for all quadrats. ρ can also be calculated from equation 1 directly as $\rho = (D-1)/(n-1)$, where D is the index of dispersion assuming n is a constant. In addition, if the beta-binomial distribution fits the data, ρ is related to θ as $\rho = \theta/(1+\theta)$.

Power–law relationship

It has been shown that for counts data there is usually a linear relationship between logarithm of the observed variance and the observed mean (Taylor, 1961), which can also be interpreted as variance–variance relationship as pointed out above. To estimate this linear relationship, many pairs of variance and mean are required. This empirical power–law has successfully fitted numerous experimental as well as simulated data, though there is much debate about the biological and mathematical basis for this relationship.

Hughes and Madden (1992) have modified this power law for a binary variable, called binary power law, relating the variance of disease incidence to mean incidence, with the general form:

$$\mathrm{var}(Y) = A\pi^b(1-\pi)^c \tag{5}$$

This might be used, e.g., to model the variance–mean relationship at different times during the development of an epidemic or to compare data from different experimental treatments. It has provided a good fit to many data sets (Madden and Hughes, 1995). Frequently the simpler two-parameter model (i.e., the symmetric form) with $c = b$ is adequate. This simpler model implies that the variance is greatest when $\pi = 1/2$. More generally, the maximum variance occurs when $\pi = b/(b+c)$, provided b and c are positive. The θ parameter of the beta-binomial distribution, hence ρ and D, is also mathematically related to the binary power law parameters (Madden and Hughes, 1995).

The interpretation of binary power law parameters can be found in several published papers (Hughes and Madden, 1992; Madden and Hughes, 1995; Ridout and Xu, 2000).

Spatial autocorrelation

Spatial autocorrelation determines the spatial dependence of disease incidence between sampling units over various lags, thus measuring inter-cluster correlation in contrast to ρ. Details can be found in Campbell and Madden (1990). This technique has been successfully used to characterise the spatial dependence of disease. Typically, spatial autocorrelation declines exponentially over distance; the rate of this decline indicates the degree of disease gradient. Spatial autocorrelation is closely related to the semi-variogram in geostatistical methods (Burrough, 1987), which has been used to analyse spatial disease patterns (Chellemi et al., 1988; Stein et al., 1994; Gottwald et al., 1996).

Xu and Ridout (1998) found the following model to be useful in describing the relationship of spatial autocorrelation with mean disease incidence and spatial lag for data generated from computer-simulated epidemics:

$$\gamma(d, \pi) = S\pi^{u}(1-\pi)^{v} \exp[-td(1-\pi)] \qquad (6)$$

where S is a scaling parameter, u and v are power law parameters, d is the distance, and t determines the rate of decrease of correlation with distance at fixed incidence. In this model, the relationship between spatial autocorrelation and incidence at any spatial lag was of a binary power–law form; at any level of disease incidence spatial autocorrelation declined exponentially over distance. Ridout and Xu (2000) further showed that quadrat-based statistics such as index of dispersion, intra-cluster correlation and binary power law can all be derived from a given model that describes the spatial autocorrelation at the level of individual plants.

Concluding remarks

There are other methods that have been developed to detect and quantify spatial patterns of plant diseases, such as distance class analysis (Nelson et al., 1992; Nelson, 1995; Ferrandino, 1998), which requires data that are more intensive than those methods outlined above. This approach is based on the thinking that the joint disease status of two plants may depend not only on the distance between them, but also on their orientation relative to one another, due to the directional effects in pathogen dispersal under field conditions. Spatio-temporal stochastic models have also been fitted directly to experimental data using Markov Chain Monte Carlo Methods (Gibson, 1997). A new method was recently developed, called spatial analysis by distance indices (SADIE) (Perry, 1995; 1998). The key concept behind SADIE is the distance to regularity, i.e. the total effort in terms of distance moved that individuals in the observed sample must expend to move such that the individuals in the samples are spaced as uniformly as possible. The degree of non-randomness within a data set is quantified by comparing the observed spatial pattern with rearrangements in which the sampled counts are randomly redistributed among the sampling units.

The spatio-temporal statistics and models are very useful for describing and summarising observed epidemics. However, to compare them between studies, there is a need to understand their relationships to the underlying biological and physical processes. In a series of papers based on simulation studies, Xu and Ridout (1998; 2000; 2001) demonstrated the importance of initial epidemic conditions, especially the spatial pattern of initially infected plants, and the size and shape of sampling quadrat in relation to prevailing wind in influencing some spatial statistics, as shown in a few experimental studies, e.g. for rice sheath blight. (Savary et al., 1997), as well as the effect of quadrat size on spatio-temporal statistics (Gottwald et al., 1995; Madden et al., 1995).

It is not expected that a single method could capture everything and answer all the questions concerned with a complex spatial pattern that may be a hierarchical and multi-scaled phenomenon (Kotliar and Wiens, 1990). It is thus important for researchers to use appropriate statistical methods either individually or in combinations that are most appropriate to their research objectives. Biological processes are in a constant state of flux and it is unlikely that all eventualities can be considered by even the most complex model. This is an important constraint, for if the model is too complex it may be impractical. Neither should a model cover all these possibilities, since, if it does, it is no longer a ‘model’. A model should be sufficiently complex but no more than necessary to answer the posed question(s). Although several modelling activities have been undertaken in plant disease epidemiology, only one type of model is

likely to be most used by the agriculture industry, that is a disease forecasting system. Often, these systems have been developed, but not properly followed through or supported in their use. One of the challenges facing epidemiologists is to demonstrate convincingly the benefit of using disease forecasting systems, not only in experimental plots but also on a commercial scale, and to ensure the systems are used widely and correctly.

References

Ahlers D (1989) Integrated plant protection for fungus diseases in winter oilseed rape. Gesunde Pflanzen 41: 306–311

Anderson RM and May RM (1979) Population biology of infectious diseases: part I. Nature 280: 361–367

Andrivon D and Limpert E (1992) Origin and proportions of the components of composite populations of *Erysiphe graminis* f.sp. *hordei*. Journal of Phytopathology 135: 6–19

Barajas-Aceves M, Hassan M, Tinoco R and Vazquez-Duhalt R (2002) Effect of pollutants on the ergosterol content as indicator of fungal biomass. Journal of Microbiological Methods 50: 227–236

Barrett JA (1978) A model of epidemic development in variety mixtures. In: Scott PR and Bainbridge A (eds) Plant Disease Epidemiology (pp 129–137) Blackwell Scientific Publications, Oxford, London, Edinburgh, Melbourne

Bateman GL, Dyer PS and Manzhula L (1995) Development of apothecia of *Tapesia yallundae* in contrasting populations selected by fungicides. European Journal of Plant Pathology 101: 695–699

Ben-Yephed Y, Genizi A and Siti E (1993) Sclerotial survival and apothecial production by *Sclerotinia sclerotiorum* following outbreaks of lettuce drop. Phytopathology 83: 509–513

Beresford RM and Royle DJ (1988) Relationships between leaf emergence and latent period of leaf rust (*Puccinia hordei*) on spring barley, and their significance for disease monitoring. Zeitschrift fur Pflanzenkrankheiten und Pflanzenschutz 95: 361–371

Blunt SJ, Asher MJC and Gilligan CA (1992) The effect of sowing date on infection of sugar beet by *Polymyxa betae*. Plant Pathology 41: 148–153

Bolland GJ and Hall R (1994) Index of plant hosts of *Sclerotinia sclerotiorum*. Canadian Journal of Plant Pathology 16: 93–108

Brasier CM (1990) The unexpected element: Mycovirus involvement in the outcome of two recent pandemics, Dutch elm disease and chestnut blight. In: Burdon JJ and Leather SR (eds) Pests, Pathogens and Plant Communities (pp 289–307) Blackwell, Oxford

Brasset PR and Gilligan CA (1989) Fitting of simple models for field disease progress data for the take-all fungus. Plant Pathology 38: 397–407

Burgess DR and Hepworth G (1996) Biocontrol of Sclerotinia stem rot (*Sclerotinia minor*) in sunflower by seed treatment with *Gliocladium virens*. Plant Pathology 45: 583–592

Burrough PA (1987) Spatial aspects of ecological data. In: Jongman RHG, Braak CFG and van Tongeren OFR (eds) Data Analysis in Community and Landscape Ecology (pp 213–251) PUDOC, Wageningen, the Netherlands

Campbell CL (1998) Disease progress in time: Modelling and data analysis. In: Gareth Jones D (ed) The Epidemiology of Plant Diseases (pp 181–206) Kluwer Academic Publishers, Dordrecht, the Netherlands

Campbell CL and Madden LV (1990) Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York

Chellemi DO, Rohrbach KG, Yost RS and Sonoda RM (1988) Analysis of the spatial pattern of plant pathogen and diseased plants using geostatistics. Phytopathology 78: 221–226

Cohlbach N, Lucas P and Meynard JM (1997) Influence of crop management on take-all development and disease cycles in winter wheat. Phytopathology 87: 26–32

Correll JC, Gordon TR and Elliott VJ (1988) Powdery mildew on tomato: The effect of planting date and triadimefon on disease onset, progress, incidence, and severity. Phytopathology 78: 512–519

de Vallavieille-Pope C, Giosue S, Munk L, Newton AC, Niks RE, Ostergard H, Pons-Kuhnemann J, Rossi V and Sache I (2000) Assessment of epidemiological parameters and their use in epidemiological and forecasting models of cereal airborne diseases. Agronomie 20: 715–727

de Vallavieille-Pope C, Huber L, Leconte M and Goyeau H (1995) Comparative effects of temperature and interrupted wet periods on germination, penetration and infection of *Puccinia recondita* f.sp. *tritici* and *P. striiformis* urediniospores on wheat seedlings. Phytopathology 85: 409–415

De Wolf ED, Madden LV and Lipps PE (2000) Risk assessment models for wheat Fusarium head blight. Phytopathology 90: S19 (abstract)

Dix NJ and Webster J (1995) Fungal Ecology. Chapman and Hall, London, New York, USA

Ferrandino FJ (1993) Dispersive epidemic waves. I. Focus expansion in a linear planting. Phytopathology 83: 795–802

Ferrandino FJ (1996) Length scale of disease spread: Fact or artefact of experimental geometry. Phytopathology 86: 806–811

Ferrandino FJ (1998) Past non-randomness and aggregation to spatial correlation: 2DCORR, a new approach for discrete data. Phytopathology 88: 84–91

Fitt BDL, Gregory PH, Todd AD, McCartney HA and MacDonald OC (1987) Spore dispersal and plant disease gradients: A comparison between two empirical models. Journal of Phytopathology 118: 227–242

Fleiss JL (1981) Statistical Methods for Rates and Proportions, 2nd edn. Wiley, New York, USA

Fleming RA (1980) The potential for control of cereal rust by natural enemies. Theoretical Population Biology 18: 374–395

Francl LJ and Panigrahi S (1997) Artificial neural network models of wheat leaf wetness. Agricultural and Forest Meteorology 88: 57–65

Francl LJ, Panigrahi S, Pahdi T, Gillespie TJ and Barr A (1995) Neural network models that predict leaf wetness. Phytopathology 85: 1128

Gibson GJ (1997) Markov Chain Monte Carlo methods for fitting spatiotemporal stochastic models in plant epidemiology. Applied Statistics 46: 215–233

Gilligan CA (1985) Mathematical modelling of crop disease. In: Gilligan CA (ed) Advances in Plant Pathology, Vol 3 (p 255) Academic Press, Inc, New York

Gottwald TR (1995) Spatio-temporal analysis and isopath dynamics of citrus scab in nursery plots. Phytopathology 85: 1082–1092

Gottwald TR, Avinent L, Llacer G, Hermoso de Mendoza A and Cambra M (1995) Analysis of spatial spread of sharka (plum pox virus) in apricot and peach orchards in eastern Spain. Plant Disease 79: 266–278

Gottwald TR, Cambra M, Moreno P, Camarasa E and Piquer J (1996) Spatial and temporal analysis of citrus tristeza virus in eastern Spain. Phytopathology 86: 45–55

Gourbiere F, Gourbiere S, van Maanen A, Vallet G and Auger P (1999) Proportion of needles colonised by one fungal species in coniferous litter: The dispersal hypothesis. Mycological Research 103: 353–359

Gray SM, Moyer JW and Bloomfield P (1986) Two-dimensional distance class model for quantitative description of virus-infected plant distribution lattices. Phytopathology 76: 243–248

Gumpert F-M, Geiger HH and Stahle U (1987) A mathematical model of the epidemics in homogeneous and heterogeneous host stands. Zeitschrift fuer Pflanzenkrankheiten und Pflanzenschutz 94: 206–215

Gutierrez AP, DeVay JE and Frieverthauser GE (1983) A model of Verticillium wilt in relation to cotton growth and development. Phytopathology 73: 89–95

Hardwick NV (1998) Disease forecasting. In: Gareth Jones D (ed) The Epidemiology of Plant Diseases (pp 207–230) Kluwer Academic Publishers, Dordrecht, the Netherlands

Hosford RM Jr, Larez CR and Hammond JJ (1987) Interactions of wet periods and temperature on *Pyrenophora tritici-repentis* infection and development in wheats of different resistance. Phytopathology 77: 1021–1027

Huber L and Gillespie TJ (1992) Modelling leaf wetness in relation to plant disease epidemiology. Annual Review of Phytopathology 30: 553–577

Hughes G and Madden LV (1992) Aggregation and incidence of disease. Plant Pathology 41: 657–660

Hughes G and Madden LV (1993) Using the beta-binomial distribution to describe aggregated patterns of plant disease incidence. Phytopathology 83: 759–763

Hughes G, Madden LV and Munkvold GP (1996) Cluster sampling for disease incidence data. Phytopathology 86: 132–137

Hughes G, McRoberts N, Madden LV and Nelson SC (1997) Validating mathematical models of plant-disease progress in space and time. IMA Journal of Mathematics Applied in Medicine and Biology 14: 85–112

Ingold CT (1971) Fungus Spores: Their Liberation and Dispersal. Clarendon Press, Oxford

Ingold CT (1978) Role of mucilage in dispersal of certain fungi. Transactions of the British Mycological Society 70: 137–140

Jamaux I and Spire D (1994) Development of a polyclonal antibody-based immunoassay for the early detection of *Sclerotinia sclerotiorum* in rapeseed petals. Plant Pathology 43: 847–862

Jeger MJ (1986) The potential of analytic compared with simulation approaches to modelling in plant disease epidemiology. In: Leonard K and Fry W (eds) Plant Disease Epidemiology, Population Dynamics and Management, Vol 1 (pp 255–281) Macmillan, New York, USA

Jeger MJ (2000) Theory and plant epidemiology. Plant Pathology 49: 651–658

Jeger MJ and Tamsett J (1983) The status of models in crop protection: An analysis using data base systems. WPRS Bulletin IV-2: 57–76

Jeger MJ and Viljanen-Rollinson SLH (2001) The use of the area under the disease-progress curve (AUDPC) to assess quantitative disease resistance in crop cultivars. Theoretical and Applied Genetics 102: 32–40

Jeger MJ, Griffiths E and Jones DG (1981a) Effect of cereal cultivar mixtures on disease epidemics caused by splash-dispersed pathogens. In: Jenkyn JF and Plumb RT (eds) Strategies for the Control of Cereal Disease (pp 81–88) Blackwell Scientific Publications, Oxford, UK

Jeger MJ, Jones DG and Griffiths E (1981b) Disease progress of non-specialised fungal pathogens in intraspecific mixed stands of cereal cultivars. II. Field experiments. Annals of Applied Biology 98: 199–210

Jeger MJ, VanDenBosch F, Madden LV and Holt J (1998) A model for analysing plant-virus transmission characteristics and epidemic development. IMA Journal of Mathematics Applied in Medicine and Biology 15: 1–18

Johnson DA (1980) Effect of low temperature on the latent period of slow and fast rusting winter wheat genotypes. Plant Disease 64: 1006–1008

Johnson BN and McGill WB (1990) Comparison of ergosterol and chitin as quantitative estimates of mycorrhizal infection and Pinus contorta seedlings response to inoculation. Canadian Journal of Forest Research 20: 1125–1131

Kampmeijer P and Zadocks JC (1974) A simulator of foci and epidemics in mixtures, multilines and mosaics of resistant and susceptible plants. Simulation Monograph (p 50) Pudoc, Wageningen, the Netherlands

Kotliar N and Wiens J (1990) Multiple scales of patchiness and patch structure: A hierarchical framework for the study of heterogeneity. Oikos 59(2): 253–260

Kranz J (1975) Beziehungen zwischen Blattmasse und Befallsentwicklung bei Blatt Krankheiten. Zeitschrift fur Planzenkrankeiten und Pflanzenschutz 82: 641–654

Kranz J and Royle DJ (1978) Perspectives in mathematical modelling of plant disease epidemics. In: Scott PR and Bainbridge A (eds) Plant Disease Epidemiology (pp 111–120) Blackwell Scientific Publications, Oxford, London, Edinburgh, Melbourne

Lacey J (1996) Spore dispersal – its role in ecology and disease: The British contribution to fungal aerobiology. Mycological Research 100: 641–660

Lalancette N and Hickey KD (1986) Disease progression as a function of plant growth. Phytopathology 76: 1171–1175

Lefol C and Morrall RAA (1996) Immunofluorescent staining of *Sclerotinia* ascospores on canola petals. Canadian Journal of Plant Pathology 18: 237–241

MacHardy WE (1996) Apple Scab: Biology, Epidemiology, and Management. American Phytopathological Society, St. Paul, MN, USA

Madden LV (1992) Rainfall and dispersal of fungal spores. Advances in Plant Pathology 8: 29–79
Madden LV and Hughes G (1995) Plant disease incidence: Distributions, heterogeneity, and temporal analysis. Annual Review of Phytopathology 33: 529–564
Madden LV, Nault LR, Murral DJ and Apelt MR (1995) Spatial pattern analysis of the incidence of aster yellows disease in lettuce. Researches on Population Ecology 37: 279–289
Maddison AC, Holt J and Jeger MJ (1996) Spatial dynamics of a monocyclic disease in a perennial crop. Ecological Modelling 88: 45–52
McCartney HA (1997) The influence of environment on the development and control of disease. In: Rechcigl J (ed) Environmentally Safe Approaches to Crop Disease Control (pp 3–31) CRC Press, Bocan Ratan, Florida, USA
McCartney HA and Fitt BDL (1998) Disease spread: Modelling development of loci. In: Gareth Jones D (ed) The Epidemiology of Plant Diseases (pp 137–160) Kluwer Academic Publishers, Dordrecht, the Netherlands
McMullen M, Jones R and Gallenburg D (1997) Scab of wheat and barley: A re-emerging disease of devastating impact. Plant Disease 81: 1340–1348
Mence MJ and Hildebrandt AC (1966) Resistance to powdery mildew in rose. Annals of Applied Biology 58: 309–320
Minogue KP and Fry WE (1983a) Models for the spread of disease: Model description. Phytopathology 73: 1168–1172
Minogue KP and Fry WE (1983b) Models for the spread of disease: Some experimental results. Phytopathology 73: 1173–1176
Nelson SC (1995) Spatiotemporal distance class analysis of plant disease epidemics. Phytopathology 85: 37–43
Nelson SC, Marsh PL and Campbell CL (1992) 2DCLASS, a two-dimensional distance class analysis software for the personal computer. Plant Disease 76: 427–432
Newton AC (1989) Measuring the sterol content of barley leaves infected with powdery mildew as a means of assessing partial resistance to *Erysiphe graminis* f.sp. *hordei*. Plant Pathology 38: 534–540
Newton AC and McGurk L (1991) Recurrent selection for adaptation of *Erysiphe graminis* f.sp. *hordei* to partial resistance and the effect on expression of partial resistance of barley. Journal of Phytopathology 132: 328–338
Nicholson P, Turner AS, Edwards SG, Bateman GL, Morgan LW, Parry DW, Marshall J and Nuttall M (2002) Development of stem-base pathogens on different cultivars of winter wheat determined by quantitative PCR. European Journal of Plant Pathology 108: 163–177
Norton GA, Holt J and Mumford JD (1993) Introduction to pest models. In: Norton GA and Mumford JD (eds) Decision Tools for Pest Management (pp 89–99) CAB International, Cambridge, UK
Obst A, Lepschy-von Gleissenthall J and Beck R (1997) On the aetiology of Fusarium head blight of wheat in South Germany – preceding crops, weather conditions for inoculum production and head infection, proneness of the crop to infection and mycotoxin production. Cereal Research Communications 25: 699–703
Perry JN (1995) Spatial-analysis by distance indexes. Journal of Animal Ecology 64: 303–314
Perry JN (1998) Measures of spatial pattern for counts. Ecology 79: 1008–1017
Pielou EC (1977) Mathematical Ecology. Wiley, New York
Rabbinge R and Bastiaans L (1989) Combination models, crop growth and pests and diseases. In: Rabbinge R, Ward SA and van Laar HH (eds) Simulation and System Management in Crop Protection (pp 217–240) Pudoc, Wageningen, the Netherlands
Ridout MS and Xu X-M (2000) Relationships between several quadrat-based statistical measures used to characterise spatial aspects of data on disease incidence. Phytopathology 90: 568–575
Ridout MS, Demétrio CGB and Firth D (1999) Estimating intraclass correlation for binary data. Biometrics 55: 137–148
Rogers MN (1959) Some effects of moisture and host plant susceptibility on the development of powdery mildew of roses caused by *Sphaerotheca pannosa* var. *rosae*. Memoir of the Cornell University Agricultural Experiment Station, 363, 38 pp
Rossi V, Racca P, Giosue S, Pancaldi D and Alberti I (1997) A simulation model for the development of brown rust epidemics in winter wheat. European Journal of Plant Pathology 103: 453–465
Savary S, Willocquet L and Teng P (1997) Modelling sheath blight epidemics in rice tillers. Agricultural Systems 55: 359–384
Segarra J, Jeger M and van den Bosch F (2001) Epidemic patterns and dynamics of plant disease. Phytopathology 91: 1001–1010
Shaw MW (1998) Pathogen population dynamics. In: Jones DG (ed) The Epidemiology of Plant Diseases (pp 161–180) Kluwer Academic Publishers, Dordrecht, the Netherlands
Shtienberg D (2000) Modelling: The basis for rationale disease management. Crop Protection 19: 747–752
Siefert RP (1981) Applications of a mycological database to principles and concepts of population and community ecology. In: Wicklow DT and Carroll GC (eds) The Fungal Community, its Organisation and Role in the Ecosystem Mycological Series, Vol 2 (pp 11–23) Marcel Dekker, Inc., New York, USA
Stein A, Kocks CG, Zadoks JC, Frinking HD, Ruissen MA and Myers DE (1994) A geostatistical analysis of the spatio-temporal development of downy mildew epidemics in cabbage. Phytopathology 84: 1227–1239
Sutherst RW (1993) Role of modelling in sustainable pest management. In: Corey S, Dall D and Milne W (eds) Pest Control and Sustainable Agriculture (pp 66–71) CSIRO, Australia
Taylor LR (1961) Aggregation, variance and the mean. Nature 189: 732–735
Thomas P (1984) Sclerotinia stem rot checklist. In: Canola Council of Canada (ed) Canola Growers Manual (pp 1053–1055) Minipeg, USA
Tomerlin JR, Eversmeyer MG, Kramer CL and Browder LE (1983) Temperature and host effects on latent and infectious periods and on urediniospore production of *Puccinia recondita* f.sp. *tritici*. Phytopathology 73: 414–419
Turkinson TK and Morrall RAA (1993) Use of petal infestation to forecast Sclerotinia stem rot of canola: The influence of inoculum variation over the flowering period and canopy density. Phytopathology 83: 682–689
Twengstrom E, Sigvald R, Svensson C and Yuen J (1998) Forecasting Sclerotinia stem rot in spring sown oilseed rape. Crop Protection 17: 405–411

Van den Bosch F, Zadoks JC and Metz JAJ (1988) Focus expansion in plant disease. I: The constant rate focus expansion. Phytopathology 78: 55–58

Van der Plank JE (1960) Analysis of epidemics. In: Horsfall JG and Cowling EB (eds) Plant Pathology: An Advance Treatise, Vol 3 (pp 229–289) Academic Press, New York, USA

Van der Plank JE (1963) Plant Diseases: Epidemics and Control (p 344) Academic Press, New York, London

Van der Plank JE (1982) Host-Pathogen Interactions in Plant Disease. Academic Press, New York, USA

van Maanen A and Gourbiere F (2000) Balance between colonisation and fructification in fungal dynamics control: A case study of *Lophodermium pinastri* on *Pinus sylvestris* needles. Mycological Research 104: 587–594

van Maanen A, Debouzie D and Gourbiere F (2000) Distribution of three fungi colonising fallen *Pinus sylvestris* needles along altitudinal transects. Mycological Research 104: 1133–1138

Vloutoglou I, Fitt BDL and Lucas JA (1995) Periodicity and gradients in dispersal of *Alternaria linicola* in linseed crops. European Journal of Plant Pathology 101: 639–653

Waggoner PE (1986) Progress curves of foliar diseases: Their interpretation and use. In: Leonard KJ and Fry WE (eds) (pp 3–37) MacMillan, New York, USA

Waggoner PE and Horsfall JG (1969) E.P.I.D.E.M.: A simulator of plant disease written for a computer. Connecticut Agricultural Experimental Station Bulletin, USA, 698

Walters KFA and Hardwick NV (2000) Principles of pest and disease management in crop protection. In: Alford DV (ed) Pest and Disease Management Handbook (pp 1–18) Blackwell Science, Oxford

Webb CR, Gilligan CA and Asher MJC (2000) Modelling the effect of temperature on the development of *Polymyxa betae*. Plant Pathology 49: 600–607

Xu X-M (1999) Effects of temperature on the length of the incubation period of rose powdery mildew (*Sphaerotheca pannosa* var. *rosae*). European Journal of Plant Pathology 105: 13–21

Xu X-M and Ridout MS (1998) Effects of initial epidemic conditions, sporulation rate, and spore dispersal gradient on the spatio-temporal dynamics of plant disease epidemics. Phytopathology 88: 1000–1012

Xu X-M and Ridout MS (2000a) Effects of quadrat size and shape, initial epidemic conditions, and spore dispersal gradient on the spatio-temporal statistics of plant disease. Phytopathology 90: 738–750

Xu X-M and Ridout MS (2000b) Stochastic simulation of the spread of race specific and non-specific aerial fungal pathogens in cultivar mixtures. Plant Pathology 49: 207–218

Xu X-M and Ridout MS (2001) Effects of prevailing wind direction on spatial statistics of plant disease epidemics. Journal of Phytopathology 149: 155–166

Xu X-M and Robinson JD (2000) The effects of temperature on the incubation and latent periods of hawthorn powdery mildew (*Podosphaera clandestina*). Plant Pathology 49: 791–797

Xu X-M and Robinson JD (2001) The effects of temperature on the incubation and the latent periods of the clematis powdery mildew (*Erysiphe polygoni*). Journal of Phytopathology 149: 565–568

Xu X-M, Robinson JD, Berrie AM and Harris DC (2001) Spatio-temporal dynamics of brown rot (*Monilinia fructigena*) on apple and pear. Plant Pathology 50: 569–578

Yang XB (1995) Analysis of variance–mean relationships of plant diseases. Journal of Phytopathology 143: 513–518

Zawolek MW and Zadoks JC (1992) Studies in focus development: An optimum for dual dispersal of plant pathogens. Phytopathology 82: 1288–1297