PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Twenty-Five Years of Botanical Epidemiology [and Discussion]

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Twenty-five years of botanical epidemiology

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Epidemiological models in plant pathology usually belong to the family of logistic equations, describing the increase in disease intensity with time. Expansion and refinement are possible by applying dynamic simulation techniques on digital computers. Among these are models of disease increase in time and two-dimensional (horizontal) space, and spore dispersal in and over a crop in two-dimensional (vertical) space. Recently, an analytical model was developed by Van den Bosch and co-workers to describe focus expansion in time and two-dimensional (horizontal) space as a function of three biological parameters that were relatively easy to measure: gross reproduction, time kernel, and contact distribution. The model was tested using data from previous experiments not designed for this purpose and from a new experiment specifically designed for validation. The model treats focus expansion as a process with a constant radial velocity and seems valid on a small scale of a few metres and, after rescaling, on a large scale of hundreds of kilometres.

Introduction

Botanical epidemiology deals with epidemics among plants. It shares its general, holistic outlook with medical and veterinary epidemiology, but it has some distinctive characteristics.

In botanical epidemiology the susceptibles are plants, which are usually immobile as they are rooted in the soil. So, the pathogen must move to cause an epidemic. Plant pathogens are well adapted for dispersal, by wind, rain-splash, or vectors.

Another distinctive characteristic of botanical epidemiology is the occurrence of susceptible hosts in discrete cohorts. For example, barley in the U.K. appears in spring as a cohort of about 8×10^{12} susceptibles (*ca.* 4 million stems ha^{-1} on 2 Mha per year). Seasonal discontinuity is typical for botanical epidemiology, even in perennials such as apple trees which shed their leaves each autumn.

Fungi are highly specialized in the production of structures for dispersal and for survival during the 'off' season which is the inter-cohort period when growth conditions are unfavourable. Some fungi have as many as five radically different spore forms (e.g. *Puccinia graminis*, see Zadoks & Schein (1979)). Many plant-pathogenic fungi have sexual stages that are adjusted to the seasons, utilizing their sexual structures not only for genetic recombination but also for surviving the off season. Asexual structures then enable rapid multiplication during the growing season. Population ecologists might speak of a dual life strategy: a sexual *K*-strategy combined with an asexual *r*-strategy (Zadoks & Schein 1979). The variety and number of fungal pathogens of plants greatly exceed those of man and animals. Many plant pathogens have only nuisance value as they cause slight damage but some can do great economic damage, and a few cause catastrophic losses.

The third, and most important, distinctive characteristic of botanical epidemiology is the intimate association between the genetics of pathogen and host. The genetic potential of hosts to resist disease is exploited extensively in agriculture and this has profoundly influenced

epidemics among plants. Breeding for resistance in crops has been practised for at least a hundred years. In animals, it is just beginning and hopefully this will not lead to some of the mistakes made with crops and the ensuing problems.

Genetics of resistance

What may be regarded as the 'birth certificate' of botanical epidemiology is the book *Plant diseases: epidemics and control* (Vanderplank 1963) published exactly 25 years ago. In it is registered a 'marriage' between plant pathology and plant genetics, which will continue for a long time.

Two types of resistance are recognized in plants. The first is basic resistance (Heath 1982), a weighty term that hides our lack of understanding of the underlying mechanisms. Basic resistance implies that there must be reasons why, for example, the fungus causing late blight in potatoes (*Phytophthora infestans*) cannot attack wheat or, vice versa, why the fungus causing yellow rust of wheat (*Puccinia striiformis*) cannot infect potato plants. Generally speaking, every plant species is exposed to between several tens and many hundreds of pathogens and parasites in its immediate environment. A large proportion of these harmful agents are fairly specific for a host plant species or group of species. Thus basic resistance protects the plant epidemiologist from absolute despair, and mankind from starvation.

The second level of resistance is due to specific genes that, like the ancient god Janus, have a dual effect. These genes in the host are usually called 'R-genes' (R for resistance). This is a misnomer because an R-gene causes specific susceptibility to a compatible genotype in the pathogen, and resistance to the other genotypes of the pathogen. The interrelation between the genotypes of plant host and pathogen was termed the 'gene-for-gene relationship' (Flor 1954). Many host species have R-genes, which the plant breeder can manipulate in crop improvement. But many pathogen species are, unfortunately, able to respond by producing genes for virulence compatible with these R-genes, thus thwarting the plant breeder's efforts.

In principle, a single resistance gene with the dual properties of the Janus-type suffices to protect a country's wheat crop completely, but only until a new fungal genotype emerges that matches the Janus-gene. This then shows its other face by conditioning susceptibility to the new fungal genotype and a devastating epidemic may follow. The plant breeder meanwhile responds by the introduction of a new variety with a second Janus-gene, to which the pathogen may adapt again, and so on.

Such interactions between host and pathogen genetics have caused farmers serious problems. Instead of gaining better control of events, farmers lose control entirely. The phenomenon is termed the 'boom and bust cycle', and is a 'vicious cycle' (Zadoks & Schein 1979) from which there is no easy escape. Great economic losses may occur.

The role of genetically determined resistance is not as well established for humans (Weatherall, this symposium), but it is being investigated and even exploited in animal husbandry. Extreme forms of such resistance could well lead to disappointments similar to those experienced in the plant kingdom. Although Vanderplank did not introduce the concept of gene-for-gene relations, he was the first to discuss their epidemiological implications and to offer suggestions for alternatives, which are now being explored intensively.

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Let us now turn our attention from genetically determined, qualitative epidemiology to quantitative epidemiology, of which – again – we are only just beginning to understand the genetic basis, at least in the host plant (Parlevliet 1979).

Basic mathematical concepts

Whereas quantitative medical epidemiology usually begins with the integro-differential equation first explored by Kermack & McKendrick (1927) and the system of differential equations derived from it, quantitative botanical epidemiology began with the logistic equation. Again, Vanderplank led the way and explored many characteristics of equations of this type. Surprisingly, he did so without reference to earlier developments in ecology.

Vanderplank (1963, 1968, 1975) developed a series of deductions and principles such as those relating to 'simple interest' disease, 'compound interest' disease, the 'threshold theorem', the 'equivalence theorem' and 'sanitation' effects. His pioneering approach is still viable and additional characteristics are still being found.

When computers came into general use, numerical solutions became possible where previously they were intractable, as for example with Vanderplank's equation for 'horizontal resistance'. Analytical methods are satisfactory when one-dimensional systems are studied, such as the increase in the number of diseased individuals not only in time (Waggoner & Horsfall 1969; Zadoks 1971; Teng 1985) but also in space (Kampmeijer & Zadoks 1977).

The logistic growth of the non-spatial model for plant disease epidemics was shown to be an over-simplification, as Vanderplank himself knew quite well. The term 'para-logistic growth' was introduced. One typical result of the spatial model was that, other things being equal, a focus of disease starting with one infected individual in a homogeneous population of susceptibles expands with constant radial speed.

Throughout my professional career I have been fascinated by these foci, which can be initiated by a single fungus spore (Zadoks 1961) or a single infective vector.

New developments

Mathematicians like to play with epidemiological models. Working independently, two mathematicians developed a theorem of constant radial expansion of foci, starting from the classical Kermack & McKendrick model but adding a spatial component (Thieme 1977, 1979; Diekmann 1978, 1979). Various characteristics were derived for the frontal wave of an epidemic proceeding in a homogeneous population of susceptible plants. Meanwhile, the plant pathologists Minogue & Fry (1983*a*, *b*) explored experimentally the one-dimensional wave front with potato late blight as their test system. Tseng Zhi-Mai (1986) studied two-dimensional dispersal, whereas Van den Bosch *et al.* (1988*a–c*) explored the two-dimensional, circular wave front with yellow rust of wheat and downy mildew of spinach respectively as test systems. Here we begin to see the advantage of botanical epidemiology compared with medical epidemiology as theories can more easily be tested experimentally.

Van den Bosch *et al.* (1988*a–c*) made further progress and from general theory derived measurable parameters with a clear biological interpretation. The radial velocity of the

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epidemic front was calculated from the following items, each easily measurable in plant pathology:

(i) the gross reproduction;
(ii) the time kernel;
(iii) the contact distribution.

The gross reproduction is the total number of daughter infections produced by a single mother infection in a susceptible population (equivalent to the medical epidemiologists' term 'basic reproduction rate'). The time kernel is the normalized function describing the mean production of infectious units with time due to a single infectious individual. The contact distribution is the normalized function describing the probability per unit area (target area) that an infectious unit will arrive from a given infectious individual (source). Formal definitions are given by Van den Bosch *et al.* (1988*a*). Model predictions are supported by recent experimental evidence (figure 1).

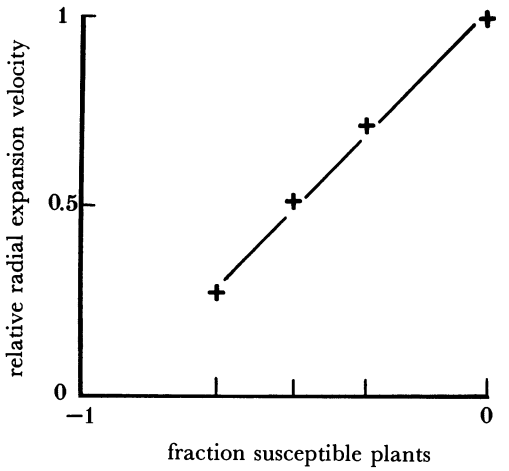

FIGURE 1. Focus expansion in plant disease. The theory of Van den Bosch *et al.* (1988) predicts that in an ideal mixture of resistant and susceptible cultivars the relative radial expansion velocity (equal to the radial expansion velocity of the mixture divided by that of the pure susceptible cultivar) is proportional to the logarithm of the fraction of susceptible plants in the mixture. The graph shows results of an unpublished 1987 experiment with wheat and *Puccinia striiformis*. Data points are averages of three replications. The horizontal axis represents the fraction of susceptible plants in the mixture on a logarithmic scale.

The example highlights an important point: theory preceded experimentation, which is an unusual sequence in botanical epidemiology. Mathematical theory, moulded into shape by competent biologists, provided new, measurable parameters with which further progress could be made by experimentation. Characteristics of parameter-sparse systems can now be studied in theory and experiment. I suggest that medical epidemiologists could profitably use botanical pathosystems (*sensu* Robinson 1976) to test their theories experimentally.

PANDEMICS

The step from the analysis of small-scale foci to the investigation of continent-wide epidemics or pandemics may be possible. It is primarily a matter of rescaling as the old foci become the new individuals. But there are complications. Whereas focus formation is usually a within-

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season and within-field phenomenon that proceeds in a continuum of time and space, a pandemic is a phenomenon that extends over discontinuities of time and space (Heesterbeek & Zadoks 1987). By rescaling once more from foci to fields as basic units, discontinuities of space can be reduced to variations in the density of susceptibles, but the discontinuities in time remain.

In technical terms, focus formation is a zero-order epidemic, developing within a single season and within a single crop field, without discontinuities of time or space. The spread of an epidemic over a large area within a single season is a first-order epidemic, continuous in time but discontinuous in space as fields may be far apart. The pandemic is a second-order epidemic, requiring many seasons and covering large areas, with discontinuities in time and space (table 1).

Empirical evidence again suggests expansion of the pandemic with constant radial velocity, at least over a limited period (figures 2 and 3). This finding may not be a surprise to ecologists studying new invasions (Anderson & May 1986). Conceptually, the two phenomena are

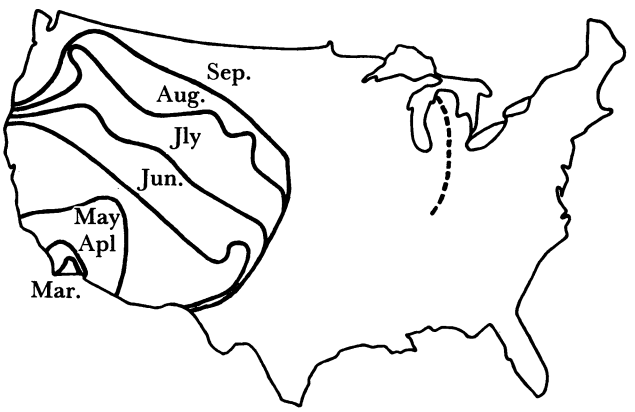

FIGURE 2. Focus expansion in plant disease. An example of a first-order epidemic, powdery mildew of sugar beet (*Erysiphe betae*) in the U.S.A., with a hint of second-order epidemic. Map based on data of Ruppel *et al.* (1975) and Schneider & Hogaboam (1977). Drawn lines indicate the positions of the epidemic fronts in the months March to September 1974, and the broken line the position of the front in October 1975.

TABLE 1. EXAMPLES OF RADIAL EXPANSION VELOCITIES (c_0) OF FOCUS EPIDEMICS (APPROXIMATE VALUES)

(Note that some of the values quoted differ from those in Zadoks & Kampmeijer (1977), because of differences in definition or calculation. Sources: (*a*) Van den Bosch *et al.* (1988*c*); (*b*) J. C. Zadoks, unpublished results; (*c*) Heesterbeek & Zadoks (1987).)

pathogen	host	c_0	source
zero-order epidemics			
Peronospora spinaciae	spinach	2 cm per day	(*a*)
Puccinia recondita	wheat	6 cm per day	(*b*)
Puccinia striiformis	wheat	9 cm per day	(*a*)
first-order epidemics			
Erysiphe betae	sugar beet	53 km per month	(*b*)
Phytophthora infestans	potato	60 km per month	(*c*)
second-order epidemic			
Endothia parasitica	chestnut	4 km per year	(*c*)
Peronospora tabacina	tobacco	130 km per year	(*c*)

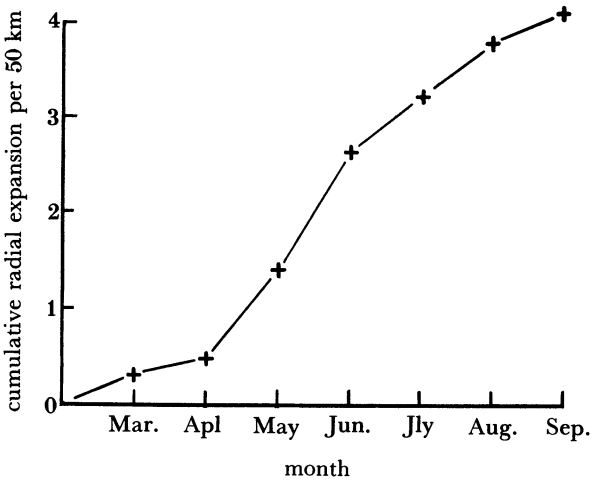

FIGURE 3. Focus expansion in plant disease. Cumulative radial expansion of the first-order epidemic caused by powdery mildew on sugarbeet in the U.S.A., 1974 (figure 2). The derivative of the curve is the radial expansion velocity, which at its highest value (c_0) is approximately constant during two months at least.

identical. The mathematics of pandemics, however, still need to be developed with due consideration of the discontinuities mentioned.

Two problems are of particular importance. The first is that in pandemics there is not always a single means of dispersal of the pathogen. There may be up to three different methods of dispersal, with different scales of distance and different frequencies of occurrence. Such systems can be handled by numerical simulation, but we are not yet sure that they can be handled by analytical methods. The other problem is that botanical pandemics are extremely data-sparse. Solution of these problems is worthwhile, because the theory could be applied when new and potentially dangerous pathogens enter a continent and efforts are made at eradication or containment. In botanical epidemiology, this is a recurring situation.

STOCHASTIC ELEMENTS

Botanical epidemiology differs from medical epidemiology in its relative neglect of the effects of chance or random variation (i.e. stochastic processes). In view of the large numbers involved, simple deterministic laws of mass action may be applied, so that description of average behaviour is adequate. Within these deterministic models there exist mechanistic submodels, where stochasticity is considered. The technical solution usually chosen is to mimic stochasticity by means of numerical simulation techniques. Rarely, real stochastic simulation, which necessitates numerous re-runs of a program, is applied (Eisensmith *et al.* 1985; Legg 1983; Sal 1980).

THE APPLICATION OF BOTANICAL EPIDEMIOLOGY

The applications of botanical epidemiology are several and include the following: as an explanatory tool; as an incentive to investigation; as an aid in resistance breeding; as a basis for prevention; and as a tool in strategic and tactical plant disease management.

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Epidemiology as an explanatory tool and as an incentive to investigation has been discussed earlier, whereas epidemiology as an aid in resistance breeding has been merely hinted at. When single-gene resistance is used as a means of defence against a fungus, that fungus may be said to 'strike back'. Stated technically, single-gene resistance exerts a selection pressure on the pathogen population that may lead to rapid selection of new virulent strains that can overcome that resistance. A similar phenomenon is known in medical epidemiology, where original wild-type strains of pathogens are replaced by antibiotic-resistant strains. Pesticide resistance in plant pests and pathogens occurs frequently. Epidemiological theory indicates ways to avoid such undesirable events or to alleviate their consequences.

Botanical epidemiology has contributed much to disease prevention in agriculture; quarantine systems, sanitation and plant hygiene are examples. Although plants are usually static, seeds and planting stocks are being moved all around the world. Pests and pathogens travel with them, but it is uncertain where, when and how frequently they arrive.

Plant disease management is needed when a pathogen is established and cannot be eradicated. Management is the rationalized art of 'living with' the pathogen so as to avoid economic loss. In the strategic mode, management is applied before the new season starts and before planting begins; computerized decision-support models are being developed for this purpose. Much of our classical crop husbandry wisdom is primitive strategic disease management. Tactical disease management is needed during the season, to avoid both undue crop loss and undue expense and to decrease environmental pollution. Various sophisticated computer-based systems have been developed, such as EPIPRE (Zadoks 1984, 1988). In The Netherlands and some other countries both modes of disease management can be seen at home, on the television screen, by dialling the appropriate number.

Concluding remarks

In the foregoing, botanical epidemiology was compared with medical and veterinary epidemiology. Some of the similarities and dissimilarities among the three branches of epidemiology were indicated. Some specific results of botanical epidemiology were mentioned. Emphasis was given to the practical application of botanical epidemiology in agriculture. It was suggested that plant pathosystems might be used to test safely theories developed in medical and veterinary epidemiology.

Thanks are due to Mr F. van den Bosch for critical reading of and commenting on the text and to Mr J. A. P. Heesterbeek for additional data.

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Discussion

R. C. RAINEY, F.R.S. (*Elmslea, Old Risborough Road, Stoke Mandeville, U.K.*). Professor Zadoks's very stimulating paper directs attention to the radial expansion shown not only by spore dispersal, over distances of metres, but also by the spread of fungal disease over distances of hundreds of kilometres. Spread of a disease over still greater distances, up to 7000 km, is

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illustrated by the well-documented case of American maize-rust (*Puccinia polysora*), spreading across Africa from its initial appearance in West Africa in 1949, in Sierra Leone, to its arrival in Mozambique and Madagascar in 1953 (Wood & Lipscomb 1956). This spread of the maize-rust showed a remarkably close parallel with that of the locust swarms (*Locusta migratoria migratorioides*) that had appeared in West Africa in 1928, in Guinea and Mali (Batten 1966); counting from this year of first appearance, the swarms had reached Nigeria in the second year, Cameroun in the third, Kenya and Tanzania in the fourth, and Zimbabwe and Mozambique in the fifth; exactly as did the maize-rust in each of these countries (Rainey 1973).

The movements of the locust swarms can now be expected to have been dominated by the large-scale seasonal wind-systems in which they flew, and these winds may accordingly be suggested as likely to have been similarly important in the spread of the rust. On this scale, the spread of the disease could no longer be described as radial; but it may be further suggested that other systems of atmospheric air movement, of the different types characteristic of the different spatial and temporal scales concerned, may perhaps have been involved in the results reported by Professor Zadoks. On the smallest scales, P. H. Gregory's pioneering application (1945) of the findings of Sutton (1932) on atmospheric turbulence demonstrated the importance of this process, with its small and short-lived eddies, in the dispersal of fungus spores. For scales of hundreds of kilometres (and in temperate latitudes), spells of winds from all directions, each spell commonly lasting for periods of between hours and days, might be envisaged as resulting in a net radial spread over successive periods of longer duration.

May I also concur with Professor Zadoks's emphasis on the value of the work of Dr Vanderplank, whose lively exposition, back in the 1940s, of his successful exploitation of the climate of the Karroo for the production of virus-free potato stocks one recalls with particular pleasure.

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J. C. Zadoks. Studying the spread of plant disease we must indeed consider differences in scales of distance and in dispersal mechanisms. Limiting myself to fungal diseases with wind-borne spores, I can only state that radial spread is a reality from small (1 m) to large (1000 km) distances. At the same time, I must state that real life offers us more possibilities than radial spread only. For a zero-order epidemic turbulent diffusion within the canopy is a major mechanism. For a first-order epidemic, turbulent diffusion in the boundary layer above the canopy is – again – a major dispersal mechanism. If, however, a spore cloud is uplifted into upper air-streams (say at the 700 mbar level†, or if it is incorporated into a low-level jet stream) the dispersal becomes directional and very similar to long-distance dispersal of insects. Insects have the facility to take to the air and land as they like. Fungal-spore landing after upper air-stream travel is usually by means of rain. These phenomena follow certain patterns, as with

† 1 mbar = 10^2 Pa.

insects. The second-order epidemic comes about by repeating the first-order in consecutive years.

Besides occasional directional dispersal we know the phenomenon of multiple dispersal. One pathogenic fungus may use several different dispersal mechanisms simultaneously. Each mechanism has its own distance parameter (e.g. mean free path) and its own frequency. Dutch elm disease provides an example; short-distance dispersal with very high probability occurs by means of root contacts among neighbouring trees, medium-distance dispersal with medium probability of success is taken care of by bark beetles, and long-distance dispersal with low probability of success may take place when bark beetles take a lift in a car to the next petrol station. Coming back to your example of maize-rust in Africa, I think that multiple dispersal mechanisms, radial spread and directional dispersal together shaped a pattern that can no longer be sorted out.

D. Mollison (*Department of Actuarial Mathematics and Statistics, Heriot-Watt University, Edinburgh, U.K.*). I found this a very interesting paper: it is a pleasure to see mathematical models applied with such careful attention to the dependence of results on basic biological parameters.

However, deterministic models of spread do have their limitations. For instance, from an initial focus they can only show spread as a single expanding focus, whereas stochastic models can reproduce the mixture of steady (relatively local) progress and great leaps forward that is typical of diseases with light airborne propagules such as fungal spores. A stochastic approach might therefore allow Professor Zadoks and his colleagues to provide a unified treatment of their different scales of spread.

J. C. Zadoks. There is no doubt in my mind as to the limitations of deterministic models. Indeed, stochasticity is an essential characteristic of dispersal processes. We are presently introducing stochastic elements into a powerful mechanistic spore dispersal model intended to simulate focus formation (unpublished work). In plant pathology we deal with large numbers, so that, fortunately, deterministic models can take us quite far.

D. A. J. Tyrrell, F.R.S. (*MRC Common Cold Unit, Harvard Hospital, Salisbury, U.K.*). There have been studies of the spatial spread of virus infections, influenza and measles (Cliff *et al.* 1986), but as they were undertaken by geographers they may not be familiar to many biologists.

Reference

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J. C. Zadoks. Thanks for this useful reference. The models referred to seem to be of a different nature to the models I discussed.

B. D. L. Fitt (*AFRC Institute of Arable Crops Research, Rothamsted Experimental Station, Harpenden, U.K.*). Models of Cliff & Ord (1981), developed for use in geography, are being applied to plant disease epidemiology by Reynolds & Madden (1988). They have been developing spatio-

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temporal autocorrelation models for describing the progress of plant disease epidemics and have specifically applied them to epidemics of strawberry leather rot (*Phytophthora cactorum*).

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J. C. Zadoks. This reference is very helpful, as it points to models of another nature than mine.

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