CHAPTER 7

Epidemics of Plant Diseases: Mechanisms, Dynamics and Management

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7.1 BOTANICAL EPIDEMIOLOGY

Botanical epidemiology deals with interacting populations of host plants and plant pathogens [71]. This field of science is at the interface of microbiology, plant physiology, ecology, environmental physics, genetics, statistics, economics, and applied mathematics. The purpose of this section is to provide the reader with an overview of concepts, methods, and definitions that shape both research and applications in botanical epidemiology, as a discipline. In particular, the field emphasizes host–pathogen interactions at the populational level; these interactions depend on the physical and biological environment; and much of these, at least in agroecosystems, depend on man-made management practices. Figure 7.1 thus illustrates the paradigm where modern botanical epidemiology evolves [91]: a pathogen population (B), a plant host population (P), their environment (E), and man’s actions (M). This figure provides the context where the field of botanical epidemiology evolves, and also why it leads to applications: man’s actions imply management, or mismanagement, of what has been called a pathosystem [56]. This framework of thinking invites consideration of populations, rather than individuals, of interactions, rather than main effects, and of the dynamics of a system, rather than its status at a given point of time. All these are best addressed through different kinds of modeling methods.

A classical example of man’s influence on plant diseases is that of nitrogen fertilization on rice diseases [52]. Although nitrogen is one important input to achieve higher attainable yield, high nitrogen application favors profuse growth of a darker, more humid canopy, and so favors some diseases, such as blast and sheath blight (both caused by fungi, Magnaporthe grisea and Rhizoctonia solani, respectively). However, lack of nitrogen, inducing poor growth and lower attainable crop yields, often favors other diseases, including brown spot (Cochliobolus miyabeanus). Contrary to a common misconception, there are diseases for poorly or well-tended crop stands, for healthy or unhealthy crops, and for rich or poor farmers [88].

One important remark must be made here: botanical epidemiology was borne and exists as a discipline because of the effects epidemics may have on cultivated plant communities, that is, fields, and for the need to understand, and so, manage epidemics in crops where they can cause disastrous losses [39]. This is briefly addressed later on in this chapter. However, botanical epidemiology also does address epidemics in spontaneous plant communities (see, e.g. [4, 17, 28, 41]). A key difference between the two types of plant communities is that a crop typically consists of plants of the same age, that is, cohorts of individuals. When dealing with cultivated crops, therefore, botanical epidemiology profoundly differs from medical or animal epidemiology, as well as from epidemiology in spontaneous plant populations, where the host population generally consists of several age cohorts.

Pathogens are major drivers of the diversity of species, including plants, in the biosphere (see, e.g. [3, 76]). They also contribute to the diversity of crop management and practices in the agricultural world [58]. Although botanical epidemiology primarily deals with practical, and important, issues that pertain to food security and food safety, it rests on scientific principles. Many of these have been derived from studies in cultivated plant stands, where experiments involving artificial infection, spatio-temporal analysis of disease progress, and the study of the diseased population performances have been comparatively easy to conduct.
7.2 PHENOMENOLOGY OF BOTANICAL EPIDEMICS

Botanical epidemics may take a number of shapes over time [31], although this diversity may be explained by only a few components: the amount of primary inoculum of the pathogen, the existence and number of successive cycles during the life cycle of the host plant population, and the existence of a limit to disease progress. Figure 7.2 illustrates typical shapes of epidemics. Epidemics of the type in Figure 7.2a are seldom found: rarely are epidemics unlimited in their spread, unless the host plant cycle is short enough, and successive, concatenated disease cycles are numerous enough in a short lapse of time. More frequent is the type in Figure 7.2b, where no secondary spread of the disease occurs during the host’s cycle. Figure 7.2c exemplifies the prototype of epidemics that have been subject to the bulk of epidemiological research, with successive, concatenated cycles multiplying over time the disease from an initial amount of primary infections up to a limit set by the carrying capacity of the host population. Much of the early literature has been grounded on these shapes, especially the typical polycyclic epidemics of type 2c, providing the basis of the seminal work by Van der Plank [71, 72]. Shapes may be misleading, however, and Pfender [55] showed that phenomena must not be disconnected from their underlying mechanisms. In particular, many epidemics of type 2b, conventionally termed “monocyclic,” may in reality hide a number of secondary spread events. Such is the case of, for example, many soilborne diseases, whose epidemic shapes reflect a restricted access of the pathogen to host tissues, and environmental effects.

One important field where botanical epidemiologists have been investing time and efforts is the measurement of disease in a host population. This cannot be discussed here with any detail, and the reader is referred to James and Teng [22], Kranz [32], and Nutter et al. [51] for further information. At the center of disease assessment is the concept of lesion, which enables the pathogen to multiply and spread. It thus may be an individual corn plant infected by the maize streak virus, a tomato root infected by a soilborne Oomycete such as *Pythium* sp., or an individual pustule on an oat leaf caused by *Puccinia coronata*. Being the source of possible further spread of disease, lesions are the epidemiological basis for measuring the amount of disease in a host population.

Several models have been developed to describe the temporal structure of epidemics. Most express the speed of epidemics, with the shape [36]:

$$\frac{dx}{dt} = g f(x)h(x), \quad \text{dimension [T}^{-1}]$$

where $x$ is the amount of disease in the host population (commonly, the fraction of host tissue diseased, i.e., disease severity, dimension [1]), $t$ is the time (dimension [T]), $g$ (often, a constant) is the apparent rate of disease (severity) increase, $f(x)$ represents the re-mobilization of inoculum from infected tissues, and $h(x)$ represents the consequences of the disease being present in the host population, especially in terms of tissues that are still healthy, and thus available to new infections.

Three main types of equations have conventionally been used, which correspond to the disease time–progress curves of Figure 7.2. A fourth type is indicated, which initiated important developments, from both the scientific and practical standpoints.

<table>
<thead>
<tr>
<th>Kinetic type</th>
<th>$f(x)$</th>
<th>$h(x)$</th>
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<tbody>
<tr>
<td>Exponential</td>
<td>$x$</td>
<td>1</td>
</tr>
<tr>
<td>Monomolecular</td>
<td>1</td>
<td>$1 - x$</td>
</tr>
<tr>
<td>Polycyclic (logistic)</td>
<td>$x$</td>
<td>$1 - x$</td>
</tr>
<tr>
<td>Paralogistic</td>
<td>Infectious tissues at $t$</td>
<td>Healthy tissues at $t$</td>
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These equations have found many applications: they do encapsulate (very simple) hypotheses on the underlying mechanics of disease progress in time, are simple to manipulate, and lead to simple calculation of apparent rates of disease increase:

\[ r = \frac{1}{t} \ln \frac{x}{x_0}, \text{ dimension } [T^{-1}]; \]

\[ r = \frac{1}{t_2 - t_1} (\ln x_2 - \ln x_1), \text{ dimension } [T^{-1}]; \]

and \[ r = \frac{1}{t_2 - t_1} \ln \frac{x_2}{1 - x_2} - \ln \frac{x_1}{1 - x_1}, \text{ dimension } [T^{-1}]; \]

for the exponential, monomolecular, and logistic models, respectively.

Such calculations have proven extremely practical tools in comparing epidemics and measuring the efficiency of disease management, whether through increased host plant resistance, chemical control, or crop management. One widespread application of the logistic model in “poly-cyclic” epidemics has led to the conventional distinction of three phases in the development of diseases in cultivated fields. In a first phase, the correction coefficient for healthy tissue availability, \( h(x) = 1 - x \) is close to 1, and the epidemic may be correctly described by an exponential equation. A typical limit to this phase is reached when \( x \) approaches 0.05. A second phase takes place when 0.05 < \( x \) < 0.5, that is, until the inflection point of disease progress curve is reached. The terminal phase, \( x > 0.5 \), corresponds to disease severity tapering off. In many cases, and for several reasons, disease severity does not reach the carrying capacity, and \( x < 1 \). Distinguishing these three phases may seem oversimplifying the reality and restricting it to a limited number of cases. It nevertheless has proven very useful in the early stages when botanical epidemiology was put into practice, particularly for tactical control of foliar diseases of annual crops: the first phase is a vital one, when chemical action, if necessary, will be the most efficient. Efficiency of chemical control rapidly drops in the second phase, where injury is caused to the crop, which may translate in reduced performances. In the third phase, tactical decisions such as chemical applications are useless. Interestingly, the average (visual) detection level of disease occurring in the field lies between the first and the second phase. When farmers need to rely on tactical decisions to control diseases in their field, the window of time when action may alter the course of an epidemic, and possibly change the outcome of a cropping season, is extremely narrow.

<table>
<thead>
<tr>
<th>Kinetic</th>
<th>Equation</th>
<th>( f(x) )</th>
<th>( h(x) )</th>
<th>Linearized form</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gompertz</td>
<td>( dx/dt = r x (\ln(1) - \ln(x)) )</td>
<td>( x )</td>
<td>(( \ln(1) - \ln(x) ) – ( -\ln(-\ln(x)) ) = ( -\ln(B) + rt )</td>
<td>Initial epidemic faster than the conventional logistic. Inflexion point at ( x = 1/e ).</td>
<td></td>
</tr>
<tr>
<td>Richards</td>
<td>( dx/dt = r x/(1 - m) )</td>
<td>( x/(1 - m) )</td>
<td>(( 1 - x^1 - m ))</td>
<td>( \ln(1/X^{1 - m} - 1) ) = ( -\ln(B) + rt )</td>
<td>( m ) is a shape parameter. For ( m = 0 ), this amounts to the monomolecular model. When ( m ) tends to 1, this amounts to Gompertz. When ( m = 2 ), this amounts to the conventional logistic.</td>
</tr>
<tr>
<td>Weibull</td>
<td>( dx/dt = (c/b) ) [( \ln[1/[1 - x)]^{c - 1/c} )</td>
<td>( c/b )</td>
<td>(( c/b ) [( \ln[1/[1 - x)]^{c - 1/c} ) - ( 1 - x )</td>
<td>( \ln[1/[1 - x)]^{c - 1/c} )</td>
<td>( a/b )</td>
</tr>
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The simplicity of these models reflects a number of hypotheses, including (1) that two types of host tissues only are considered, healthy or diseased, (2) that $r$ is constant over the course of an epidemic, (3) that all healthy tissues are equally exposed to infection, and (4) that the host population size does not vary during the course of an epidemic. A large amount of epidemiological research has dealt with these issues. Hypothesis (1) was addressed at an early stage by Van der Plank [71], and will briefly be discussed below. Hypothesis (2) refers to much of the idiosyncrasies of epidemics: each epidemic is unique, partly because of the biological and environmental (soil, climate) context where it develops; any change in host plant resistance (e.g., through aging), any variation in climate inevitably must translate in changes of $r$ values. Hypothesis (3) links these equations with the spatial dimension of botanical epidemics: the probability of a healthy site of becoming diseased is not isotropically distributed; on the contrary, it depends on the spatial distribution of the host tissues, the spatial distribution of the attributes of the physical environment, and the spatial pattern of dispersal of the pathogen. This is a field of extremely active research today, and the reader is referred to several texts [6, 48, 60, 74]. Hypothesis (4) refers to change in host population size [6], especially host growth, and possibly host population reduction (e.g., disease-induced defoliation). This latter hypothesis has particular relevance in the context of cohorts of individuals constituting the host population, as is the case in (most) cultivated field populations.

Many botanical epidemics are polyetic, that is, the result of the concatenation of individual epidemic components occurring during successive cycles of a plant host population. Polyetic epidemics are commonplace in soilborne plant diseases, diseases in spontaneous or natural plant stands, whether annual or perennial, and diseases of perennial crops including fruit crops. One is then dealing with a higher level of integration, where (1) individual epidemic processes depend, to a varying extent, on the previous epidemic process in terms of primary inoculum ($x_0$), (2) these epidemic processes also depend on previous epidemic processes which may have affected the host population (size, growth, and structure), and (3) successive epidemic processes progressively shape the biological composition of both the host and pathogen populations (which is retained, possibly with a bottleneck effect in the primary inoculum generating each successive epidemic process). Polyetics are ancient phenomena, both in natural and agricultural ecosystems. They are comparatively new to botanical epidemiology, and represent a massive and exciting scientific challenge. Initial thoughts on this topic, with epidemics of order 0, 1, and 2, initiated as initial infection points leading to local disease foci (zero-order epidemic), to individual epidemic processes at the population scale, for example, a cultivated field (first-order epidemic), and concatenated epidemic processes, spreading from field to field and to continental scales in polyetic epidemics (second-order epidemics) may be found in Heesterbeek and Zadoks [20].

### 7.3 PROCESSES IN BOTANICAL EPIDEMICS

The terms “processes” and “concatenation” have been used several times in the previous section. They are used again here, but in a different context. Epidemics represent the usual level of integration [10] of interest for botanical epidemiologists. Below it are several levels: (i) the disease cycle (e.g., for a soilborne disease: inoculum mobilization, contact with the host tissue, infection, development of the pathogen within or onto the root system, secondary infections from root-to-root contacts, production of survival structures, and survival in the soil); (ii) processes governing individual stages of the cycle (e.g., for the germination of a fungal spore onto a leaf surface: leaf moisture and temperature, leaf age, phylloflora composition); (iii) the various substages which may be defined for each of these individual stages (e.g., the successive morphological steps of the germination of a rust urediniospore); down to (iv) processes governing individual substages at the finest level of biological integration, including, for example, the molecular dialogue between a susceptible potato root and a virulent cyst nematode, Globodera rostochiensis.

Botanical epidemiology, just as medical epidemiology does, attempts to explain an epidemic not because of its shape as a phenomenon but because of the mechanisms that build it. Being used as the reference level of integration, an epidemic is seen as a system of its own (Fig. 7.1) and is explained from the level of integration immediately beneath it, the individual stages of the infection cycle.

Gaëtán [16] coined the concept of “infection chain” (Fig. 7.3) to represent the basis for understanding the underlying mechanics of botanical epidemics [30]. The concatenation of these elementary processes result in a cycle, a monocycle, which itself is the elementary component of an epidemic. Some (actually, few) epidemics consist of only one monocycle, and many others consist of few or several, concatenated monocycles. Figure 7.3 uses terms that pertain to a disease of the foliage caused by a fungus; other terms would have to be used for other diseases caused by quite different plant pathogens. Numerous environmental factors influencing individual stages could be linked to the chain (see section below), and their respective weight on the course of epidemics, indicated. Whichever the disease, each of its specific biological links and their particular behavior to environmental factors, the resulting diagram, an “ethograph” (C.A.J. Putter, personal communication) depicting the successive stages of an infectious cycle would retain the same overall shape. Drawing ethnographs of plant diseases perhaps is mere basic biology. For botanical epidemiologists, it however serves the two important purposes of identifying where the chain might be broken, and so, an epidemic stopped, and of locating where environmental factors may play an important role in epidemics.
7.4 FACTORS INFLUENCING EPIDEMICS

The body of literature dealing with factors influencing epidemics is so large that only a very brief overview can be offered here. Analyzing environmental effects on botanical epidemics serves several purposes. One is to analyze and quantify how components of the monocycle react to changes in the environment, and so perhaps predict the behavior of epidemics [30]. Another is to determine whether an epidemic caused by a given pathogen, in a given host population, in a given environment, may occur [73]. A third is to determine whether the environment might be altered so that epidemics would be reduced; in cultivated host populations, crop management may then translate in disease management [53]. A fourth is the identification of specific intervention points in disease cycles [67] where targeted control action, including pesticide application, will have the strongest disease-reducing effect. Historically, the latter objective has attracted much effort.

The environment of epidemics is simultaneously physical, chemical, and biological, with two broad components in the latter, the physiological-genetic status of the host, and its microbiological environment. One convenient way of addressing the diversity of factors is to consider an electronic analogy [87], whereby the performance of a given stage in the monocycle (e.g., number of new pathogen propagules produced, number of successful virus transmissions per vector and per time unit, or latency period duration) is seen as responses to environmental stimuli [91]. Two groups of components of the monocycle may be considered with respect to the effects of the environment, a nondispersive and a dispersive phase ([34]; see also the last section of this chapter with an example of strategic management of a polyetic epidemic). The effects of environmental factors on the nondispersive and dispersive phases are thus briefly discussed here.

Soybean (Glycine max) is a major agricultural product of North America, where producers have to deal with a broad range of diseases caused by several viruses, airborne fungi, and soilborne pathogens. The above points are briefly illustrated here using this example. For many years, soybean leaf spot (Cercospora kikuchii) has been the main focus of research for its management. Stimulus-response studies have dealt with the ability of the causal fungus to grow and sporulate at varying light and temperature [7,8], infect the host at different development stage of plants [33], and its ability to survive and establish infections on leaf tissues that alternately are favorable (wet) or unfavorable (dry [61]). Web blight, caused by the soilborne fungus Rhizoctonia solani, is also an important soybean disease where summers are warm and humid, and crop canopies are dense and often wet. Spread of this type of disease results in conspicuous patches [80, 81], whose appearance and extension (several cm per day) strongly depend on canopy wetness, and thus, on crop density and management. Quantitative stimulus-response information was also gathered on another fungal disease, soybean rust (Phakopsora pachyrhizi),
which has long been feared in Northern America [82],
including reaction types between different cultivars of the host
and different isolates of the pathogen [45] and variation in
host susceptibility with plant age [46]. Soybean rust may be
dispersed over long distances by wind currents, and by late
2004, the disease was first observed in the southern part of the
United States. Close surveillance is underway to monitor its
overwintering and deploy different management options [83].

Dispersal of pathogens, that is, the dispersive phase of the
monocycle, represents a field of its own in botanical epi-
demiology. Dispersal of pathogens, which may result in (but
should not be confused with [25]) disease spread, depends on
a large number of environmental factors, including the cli-
matic environment of the host population stand (which
affects the efficiency of the sources of inoculum and capture
of inoculum by target host tissues [57]), the nature and struc-
ture of the inoculum source, and the physical characteristics
of the host plant population (including the root or foliage
system spatial structure and density). Introduction to this field
may be found in Aylor [1], Zadoks and Schein [91], Jeger
[25], and Campbell and Madden [6].

Again, only a very brief overview can be provided here,
where emphasis is given to the very large body of empirical
work that has been conducted. In an early review, Gregory
[19] discussed the epidemiological implications of disease
gradients whether caused by localized sources of inoculum or
by environmental heterogeneity, using an empirical approach.
The approach was later on reviewed by Fitt et al. [15], who
analyzed 325 sets of observations describing the decrease
with distance in deposition of airborne or splashborne fungal
spores, or pollen, or in amounts of symptoms caused by fungi,
bacteria or viruses. Two empirical models were compared:

The power law model \( y = ax^{-b} \), or \( \ln(y) = \ln(a) - b \ln(x) \)
and the exponential law model \( y = e^{c - d x} \), or \( \ln(y) = \ln(c) - dx \) where \( y \) is the amount of disease (or deposition)
and \( x \) is the distance from the source [19], and \( a, b, c, \) and \( d \)
are parameters.

There is generally little difference between the two mod-
els in the goodness of fit to the data, although deposition gra-
dients for spores borne in splash droplets are fitted better by
exponential equations and gradients for fungi with airborne
spores less than 10 m in diameter are fitted better by power
law equations. The exponential model has the property that
the modeled variable decreases by half as the distance from
the source increases by a constant increment (the half-distance);
this provides a measure of the gradient that is easier to visu-
alyze than the exponent of the power law model. According to
the analytical review conducted by Fitt et al. [15], half-distances
increase from 0.004–0.02 m for soilborne fungal pathogens,
to 0.06–0.15 for splashborne bacteria and fungi, to 0.30–30
m for insectborne virus, bacteria, and fungi (with an extreme
value of 4331 m for beet mosaic virus), and up to 0.7–130 m
for airborne fungal spores (with an extreme value of 1.2 10^7 m
for Puccinia graminis, the stem rust pathogen). The exponential
model is easier to incorporate into models of disease devel-
oment than the power law model because the boundary
condition at the source (the predicted amounts of propagules
or of disease at the source) is finite rather than infinite.

Although both models can empirically be used to describe
patterns of pathogen dispersal or of disease spread, they do
not refer to similar underlying processes [15]. The exponen-
tial model implicitly refers to the extinction of a signal, with
parameter \( b \), away from the source of inoculum, whereas
the power law model applies to dilution from turbulent diffusion
as a cloud of particles moves away from the inoculum source.
Both processes actually may occur simultaneously in a host
population stand, and both models therefore may aptly be
used to describe gradients in the same epidemic.

One important difference between the two models, how-
ever, is that the exponential equation predicts a much steeper
gradient than the power law equation does at the tail of the
gradient, with very important consequences on the spatial
pattern of epidemics. This difference may be linked to cur-
rent research directions in botanical epidemiology pertaining
to disease spread [60], which may be outlined as follows. On
the one hand, focal epidemics [93] occur when a homoge-
neous plant host population (e.g., a field crop) is exposed to
inoculum (aerially) dispersed from a source in an isotropic
way. In this case, the concept of traveling wave applies: focal
expansion results from the radial expansion of a front away
from the source with constant velocity, which results from
the gradient having a negative exponential shape. On the
other hand, the theory of turbulent diffusion suggests that
spread should become more efficient as the disease area
expands [60]. The concept of dispersive wave [13, 63] then
applies, that is, a wave of disease progress in space with
increasing frontal velocity, which has an algebraic (e.g.,
power) gradient. This concept leads to predicting disease
gradients that become shallower as epidemics progress [60],
consistent with the early work of Gregory [19].

Environmental factors may strongly affect the parameters
of dispersal gradients [19], one of them being the density of
the host plant population [5], cultivated either in pure (e.g.,
[57]), or in mixed or heterogeneous stands [34].

### 7.5 Some Simple Models in Botanical Epidemiology

The models discussed above are primarily meant to describe,
rather than explain, epidemics as processes. One of the most
important contributions to theoretical epidemiology in plant
populations was the differential–difference equation of Van
der Plank ([71], equation 8.3, p. 100):

\[
\frac{dx(t)}{dt} = R_e (x(t-p) - x(t-p-i)) (1-x(t)) \quad \text{(dimension: [T^{-1}])}
\]

where \( x \) is the proportion of disease (dimension [1]), \( t \)
the (current) time ([T]), \( p \) is the latency period duration ([T]),
i is the infectious period ([T]), and \( R_e \) is the basic infection
rate corrected for removals ([T⁻¹]), that is, the amount of new disease generated per (infectious) disease fraction per unit time. This equation introduces two delays, p and i, and thus states that the increase of disease depends on lesions that are not latent any more, and not yet removed from the epidemiological process. Note that the product $R_i$ is the equivalent of the gross reproduction rate, $R_o$, commonly used in medical or animal epidemiology.

Considering that a host plant stand consists in a number of sites, which may be considered healthy (vacant to infection), latent, infectious, or removed from the epidemiological process, Zadoks (1971) generated a simple simulation model (Fig. 7.4) enabling to integrate numerically Van der Plank’s differential–difference equation. Both the two models generated considerable advances in the field. The former enabled theories and concepts to be developed and discussed – until today, see, for example, Segarra et al. [64] – such as the threshold for an epidemic to possibly occur: $iR_c \geq 1$, and relationships among the “epidemiological quintuplet” [91]: $x_0$ (the amount of initial disease), $p$ (latency period duration), $i$ (infectious period duration), $N$ (the number of effective propagules per lesion per unit time), and $E$ (infection efficiency, the number of new lesion per effective propagule). The latter (Fig. 7.4) in effect is a Suscept–Exposed–Infectious–Removed (Mollison, 1995) model, which allows to explore, for example, the effects of $p$, $i$, and $R_c = \text{DMFR} = NE$, where DMFR stands for the “daily multiplication factor” [86, 91], the numerical equivalent of $R_c$.

Both models also are useful tools to assess management practices to control epidemics [26]. Figure 7.5, for instance, provides a simulated overview of a “typical” polycyclic epidemic, such as a cereal rust, at reasonable values of parameters $i$, $p$, and $R_c = \text{DMFR}$. The graphs indicate that variation in $p$ has a very strong bearing on epidemic outcomes, variation in $R_c = \text{DMFR}$ also has a very strong effect, but variation in $i$ does not have such strong effects on epidemics. Such results are useful to guide plant protection, in terms of host plant resistance (which may, e.g., increase $p$ or reduce $E$), in terms of chemical control (protectants will decrease $E$), or in terms of crop management, which may affect any of the five components of the quintuplet.

Another approach to modeling disease epidemics is that of Brassett and Gilligan [2], which was developed with respect to soilborne diseases. The model combines properties of the monomolecular and logistic models, and can be written as:

$$\frac{dI}{dt} = (k_1 P + k_2 i)(ZN - I) \text{ (dimension: } \frac{[N\text{}_{\text{les real}]} T^{-1}])}$$

where $I$ is the mean number of infected roots per unit area, $N$ is the mean number of plants per unit area, $P$ is the density of inoculum per unit area, and $Z$ is a parameter for the asymptotic proportion of roots that become infected. $k_1$ and $k_2$ are parameters representing the intrinsic rates of infection from primary and secondary inoculum, respectively. This type of model has undergone a number of developments, including variation of the strength of the inoculum source and host growth over time, some of which are briefly addressed below.

### 7.6 Refinement of Models

Only two types of developments are briefly addressed in this section. One deals with botanical epidemics developing over time and space, and the other with stochasticity in botanical epidemiology.
Epidemics in general, and botanical epidemics in particular, depend on dispersive and nondispersive processes, that is, time and space characteristics. Jeger [23] produced a first bridge between strictly temporal models, including Van der Plank’s and a series of models describing spatial spread, including Gregory’s. This analytical approach is the basis to linking disease variation over both time and space.

Van den Bosch et al. [69] developed a theory of focal expansion based on (1) a time kernel, (2) a contact distribution, and (3) a gross reproduction, where the time kernel describes inoculum production over time and the contact distribution describes inoculum dispersal. Gross reproduction is the total number of victimized (infected) individuals produced by a single infectant (infectious individual) placed in a population consisting of suscepts only (i.e., $R_0$ [21, 35]). This theory (which involves an exponential dispersal gradient) predicts that a focus of disease expands radially at a rate that asymptotically approaches a constant value.

The spatio-temporal structure of botanical epidemics was studied by Kampmeijer and Zadoks [29] and Zadoks and Kampmeijer [90] using the model EPIMUL. Although the approach of Van den Bosch et al. [69] was a mathematical and theoretical one, the approach in EPIMUL is that of a numerical, mechanistic, and spatially explicit simulation model. This model describes the progress of disease from a source at the center of a lattice of cells (which represent host plant entities, each with a given number of sites), where it can spread and infect new cells. Between-cell disease extensification takes place with a Gaussian dispersal function, and within-cell disease intensification follows the earlier model structure by Zadoks (1971; Fig. 7.4). As indicated by its authors, EPIMUL was not designed to depict reality, but as a tool to explore the behavior of botanical epidemics in time and space, including effects of, for example, diversity or change in the pathogen population. EPIMUL was later used to analyze the epidemiological effects of various strategies for the spatial deployment of genetic diversity in the host population [50].

Another model, involving a spatially explicit model, but with stochastic features, and a contact distribution modeled by a half-Cauchy (i.e., an algebraic, not an exponential) distribution allowed to address similar (initial inoculum, parameters for dispersal, and parameters for disease intensification) and additional (aggregation statistics) issues [77–79].

Spatio-temporal modeling of epidemics may take different shapes, from spatially explicit, to spatially implicit, and from deterministic to probabilistic. This is a field of current and active research.

Within a given host plant stand, botanical epidemics may vary greatly in onset, shape, or speed (see e.g. [43]). Stochasticity is another area of recent developments in botanical epidemiology, where emphasis may concern the inherent variation in disease intensification, the variation of disease onset in a host population during the course of a growing season, and variation in the chance for a host unit becoming infected along a dispersal gradient. Stochasticity may occur at the various levels of integration where an epidemic develops, for example, from spore germination [12], to virus transmission to host plants [14], and to the spatio-temporal development of epidemics (e.g. [42, 65]). Again, this is a field of very active and current research (see, e.g. [18, 62]). A key question is whether stochasticity at one level of integration will lead to divergent behavior of the entire epidemic, seen as a system. This seems to particularly apply when considering the dispersive phase of epidemics, where stochasticity may generate structure at many different scales [62]. In such a context, the gross reproduction parameter ($R_0$) may need to be larger than 1 in order that an epidemic occurs in a population [21].

### 7.7 Disease Management: A Brief Review of Principles

Management of botanical epidemics entails different decisions and actions [91], which may be categorized as tactical (pertaining to a given epidemic in a given growing season),
strategic short term (anticipating a given growing season) or strategic long term (concerning a number of growing seasons, and possibly different scales in agriculture, from an individual farm to a watershed, to a country, and to an entire ecoregion [92]). Typical tactical decisions include, for example, fertilizer management or pesticide applications in a cultivated field, strategic short-term decisions include the choice of a given crop variety to be established in an orchard or a field, while breeding for particular resistance characteristics or a specified ideotype of cultivated plant implies strategic long-term choices.

Botanical epidemiologists continue to address short-term decisions and develop forecasting systems for disease control. Much progress has recently been achieved, bringing compelling arguments for the use of Bayesian decision theory to design real-time disease prediction rules [39]. These rules may be based on (1) disease assessment or measurement of environmental factors which may influence diseases, and (2) predictors, that is, their consequences in terms of epidemic dynamics or losses (using empirical or mechanistic models). The approach derives from processing of diagnosis in medicine, with the additional difficulty that predictions concern epidemics occurring in populations, not disease developing on a particular individual, and therefore has to include an additional level of integration (and of uncertainty). Yuen et al. [84] proposed the use of receiver operating characteristic (ROC) curves as a mean to compare different predictors. The approach centers on the examination of the probability of epidemic occurrence before and after using the predictor [85]. A detailed review of progress in this area is given in Madden [39].

Host plant resistance is a key tool to manage diseases in cultivated ecosystems worldwide. The example of rice, the first global food crop, is a compelling case where plant breeding for host plant resistance has had a major impact in controlling epidemics, in reducing yield losses due to diseases, and improving food security [47]. In Asia, improved crop varieties, including host plant resistance traits, contributed 0.68–0.97% of a total increase in a food crops yield growth rate of 2.11–3.65% per year during 1960–2000 (the estimated average values for all developing countries for the same period are 0.52–0.86 to a total growth rate of 2.19–3.20% [11]). Deployment of host plant resistance and durability of host plant resistance are major areas of research, which cannot possibly be addressed in any detail here; the interested reader is referred to reviews of the field [27, 54]. Recently, Van den Bosch and Gilligan [70] developed a simple epidemiological model to link population dynamics and population genetics. Their approach enabled them to contrast the conventional definition of resistance durability (the delay from the release of a resistant variety to the take-over of a virulent genotype within the pathogen population), to two new ones, (1) the delay from the release of a resistant variety until appearance and invasion of a new virulent genotype within the pathogen population, and (2) the added value, in terms of agricultural production, generated from the release of a resistant variety (measured as the added number of uninfected host growth). They conclude that the conventional definition may not necessarily be the most useful, and that the two other definitions reflect performances of pathosystems which are, too, strongly dependent on the interplay of population genetics and dynamics.

Policy making and implementation are also examples of long-term decisions for the management of botanical epidemics. Recent results on the rhizomania epidemic of sugar beet in the United Kingdom [66] illustrate this point. A spatially explicit, stochastic simulation model was developed, which considers the polyptic spread of the Beet Necrotic Yellow Vein Virus (BNYVV), which causes rhizomania, a major beet disease. The virus is transmitted by the soilborne myxomycete Polymyxa betae. The model considers disease progress at the plant, field, and regional scales, and addresses epidemics over successive years. Spread of the disease at the individual plant level (i.e., root) is caused by zoospores, but spread at any higher scale is primarily due to movement caused by agricultural equipment is, whether within a field, between fields of the same farm or between farms. The model considers the spatial and temporal dynamics of the disease separately. During a cropping season, the disease intensifies (amplifies) at the plant scale, and does not spread; spread occurs between cropping seasons, when no (virus and soilborne vector) amplification occurs. The model designed by Stacey et al. [66] describes the status of all sugar-beet farms and their relationships in the United Kingdom. Outputs of the model were number of infected (i.e., latent, or infected, but asymptomatic), symptomatic, and disease-free fields and farms. The model incorporates both the intensification (amplification) and extensification (dispersal) phases of the disease. The intensification phase, which may last more than a decade before symptoms are detected, was derived from earlier modeling work [68] including parameters for primary and secondary infection at the plant level. Stochasticity was introduced in the description of disease spread. At the field level, the effect of cultivation on the spatial distribution of inoculum was calculated by convoluting [40] the spatial pattern of inoculum with distribution of the probability that an inoculum particle is displaced on a given distance. Spread within and between farms was modeled using Poisson distributions of potentially “infective” journeys of machinery. Several important results were achieved from this work. First, the local containment policy (revoked in 1984), which does not affect the (asymptomatic) front of disease progress, was shown to be ineffective, since by 2050 it would not reduce the spread of the disease. Simulation further indicated that a small reduction (10%) of between-farm cross infection would strongly reduce disease spread. The authors further concluded that any management action must match the scales of epidemics in time and space. In the case of rhizomania, the time scale (delay between infestation and symptom appearance) is 10–15 years, and the spatial scale is the distance on which the disease may be dispersed during this time (about 20 km).
7.8 CONCLUDING REMARKS

Ultimately, the relevance of botanical epidemiology is measured in terms of its contribution to maintaining and sustaining viable ecosystems, whether cultivated or not. The size of this chapter precludes a discussion on the losses caused by plant pathogens, in spite of the fact that it is one key area where concepts of botanical epidemiology meet with concepts pertaining to environmental and agricultural management.

Plant disease epidemics in agroecosystems cause injuries, which may (or may not) lead to crop losses (or damage, i.e., reductions in yield quantity or quality), which, in turn, may translate into losses (reduction in economic return). This cascade of events [89] is the basis of yet another field of investigation of the discipline. Measurement of crop losses caused by plant pathogens is of course a key to measuring the importance of plant pathogens, the need for management of epidemics, and the efficiency of current management [91]. Too often is the measurement of crop losses considered easy and granted; it actually is a field of experimental and modeling investigations of its own. Crop losses may be seen as a yardstick to measure the harmfulness of epidemics, as well as the efficiency of current disease control [37, 38, 67].

Modeling allows simulating the amount of crop loss caused by disease injuries, which vary over time as an epidemic develops over the course of a cropping season; it also allows measuring the effect of varying man-made environment (which defines the attainable performances of a crop) on crop losses. Further, it allows attention to shift away from crop losses and focus instead on yield gains [9]—benefits derived from better crop management, from better strategies involving short- and long-term vision of plant health. Recent modeling work [75], backed with heavy field experimental support, has enabled to analyze crop losses dynamically caused by a range of diseases, insects, and weeds of rice in tropical Asia [18, 19].

Shifts in paradigms have occurred, which nowadays guide research. One of them derives from the realization that elimination of a plant pathogen is neither possible nor desirable in the immense majority of cases [44, 91]; “control” progressively gives way to “management.” This notion, which derives from ecological research of the middle of the last century, still holds promises of progress today, even in the current context of biological invasions of plant pathogens, which can only be prevented and managed when they occur (see, e.g., [83]). Another is that one host plant population is seldom exposed to injuries caused by one pathogen only, or more generally, by one harmful agent. Farmers are concerned with many problems other than plant protection; when they do, they often have to consider several of them simultaneously; and ecological principles suggest that empty niches seldom exist [44]: multiple pathosystems are facts, whose shape and composition vary with the man-made environment [58]. The characterization, analysis, and modeling of multiple pathosystems is yet another front for future progress [59]. This will probably require novel ways to address botanical epidemiology, where the concepts of epidemiological guilds and guilds of harmful agents might be useful to address and manage syndromes of production and syndromes of disease.

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