The Study of Plant Disease Epidemics
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Introduction

Chemical industry and plant breeders have forged fine tactical weapons; but only epidemiology sets the strategy.

J. E. Vanderplank

1.1 Plant Disease Epidemics

Plants are susceptible to many pathogens, such as fungi, oomycetes, bacteria, viruses, viroids, and nematodes (Agrios, 2005). The book by Farr et al. (1989) lists over 13,000 fungal and oomycete pathogens of plants in the United States alone, with more than 75,000 listed diseases (because some pathogens can infect many species of plants). A given crop or forest tree species may be affected by over 200 distinct pathogens world-wide; however, in a given region, there are generally 5–20 serious diseases to control on an annual basis (Madden and Wheelis, 2003).

The consequences of plant diseases can be high, especially in terms of reductions in yield and costs of control (James et al., 1991; Teng and Ohima, 1983). Tremendous advances have been made over the last 100 years in the management of plant diseases, especially in the development of disease-resistant cultivars, development of many chemical and biological-control compounds, as well as the use of many cultural methods (Fry, 1982; Strange, 1993). Nevertheless, many plant diseases remain difficult to control using affordable methods, both in the developed and developing world.

Although infection of a single high-value plant may sometimes be of importance, such as a mature elm tree on a city street infected by *Ophiostoma ulmi* (the cause of Dutch elm disease), infection of an individual plant is generally not of any significance. Rather, plant diseases usually matter when many plants or plant parts (such as fruits or leaves) in a field, forest, or region are infected. Unlike the situation with several highly contagious animal diseases, very few plant diseases have been eradicated (Madden and Wheelis, 2003; NRC, 2003); thus, some inoculum of the pathogen can usually be found in a given area for the important diseases of a given crop. Therefore, the typical goal of disease management is to maintain the intensity of disease to a *low* (but not necessarily zero) level, or as stated by Merrill (1977), "preventing an intolerable build-up of disease within a population." Just what constitutes intolerable will depend on the value of the crop and the impact of the disease.

The issue can also be addressed in terms of the pathogen population and not just the population of diseased plants or plant parts (such as leaves). Vanderplank wrote in the first paragraph of the first chapter of his 1963 book: "In order to control rust in wheat fields (caused by *Puccinia graminis* f.sp. *tritici*) one must stop, or at least retard, the growth of millions or billions of rust pustules on thousands or millions of wheat plants. The fate of a single wheat plant and the growth of a single pustule are relatively trivial details in a large picture." It can be reasonably argued that one must study plant diseases in populations in order to understand diseases at this scale, and also to predict their outbreaks, quantify their impact on yield, and, ultimately, to better control them (Jones, 1998b). This book deals with quantifying diseases in populations.

1.2 Some Concepts

1.2.1 Epidemics

Epidemiology can be considered, quite simply, the study of epidemics. There are several possible definitions of an epidemic, and the one we prefer is

> *EPIDEMIC: Change in disease intensity in a host population over time and space.*

We generally do not overly emphasize the nuances of definitions in this book, but it is helpful here for instructional purposes to consider the components of this definition of an epidemic. First of all, the use of "change" emphasizes that epidemics are *dynamic* processes, and many aspects of their study involve characterizing *rates* of change. The use of "disease" emphasizes that epidemics specifically involve diseases, and not just pathogens or hosts. "Intensity" is a general term that characterizes the magnitude of disease or the infection, and will be defined and explained in the next chapter. The use of "host population" emphasizes that an epidemic is primarily a *population phenomenon*, involving in this case, a population of plants susceptible to one or more species of pathogen. Finally, "time" and "space"

are indications of the two physical dimensions over which the dynamic process of an epidemic occurs. Specifically, disease intensity varies from time to time and from location to location during epidemics.

There are variations of this definition. Kranz (1990b), for instance, succinctly states that "An epidemic is the progress of disease in time and space." Often, "space" is omitted from the definition. As will be clear from Chapters 7 to 9, diseases do not exist at the same intensity everywhere at any given time. Thus, the spatial dimension would then simply be implicit in any definition when space is not mentioned. Commonly, "increase" is used instead of "change". An epidemic could then be considered an increase in disease intensity in a host population over time (with an implied spatial component). This can be, in fact, a very useful way of looking at an epidemic, because it is often of interest (both theoretically and practically) to know what factors (or combination of factors) lead to a rapid increase in disease intensity and what factors (or combination of factors) prevent an increase in disease intensity or slow the rate of increase to a tolerable level. This approach underlies much of Chapters 4 and 5.

However, we prefer to maintain the term of "change" in the general definition instead of "increase" to better reflect the full range of situations that can be studied. For instance, consider a foliar disease of a perennial plant. Although the intensity of disease may, in general, increase during the growing season, there will eventually be a decrease as defoliation occurs at the end of the season. Moreover, there may be a decline in the proportion of plant tissue affected by disease if the host is producing new foliage faster than infections are occurring. In addition, for the spatial component of epidemic, the concept of monotonic increase (or decrease) in disease is not always easy to apply, as will be seen in Chapters 7 and 9 and elsewhere.

Sometimes our concept of an epidemic becomes more restrictive in this book, depending on the specific topic being addressed. For instance, in Chapter 5 we formally address the conditions necessary for disease to *increase* in time for diseases that spread from plant to plant, leaf to leaf, root to root, and so on.

It should be emphasized that the everyday concept of an epidemic, as well as the technical definition used by some non-epidemiologists, is different from the concept and definition espoused here. For instance, the Merriam-Webster definitions of epidemic include: "affecting or tending to affect a disproportionately large number of individuals within a population," or "excessively prevalent." A Dictionary of Plant Pathology (Holliday, 1989) defines an epidemic as "a widespread increase, usually limited in time, in the incidence of an infectious disease." In contrast, the operational definition of epidemic of this book makes no mention of large numbers, excessive prevalence, or widespread incidence. The reason can be seen with an example.

Consider late blight of potato, caused by the oomycete *Phytophthora infestans*, which may be the most infectious disease known of any crop based on rates of increase (Vanderplank, 1963). Assume there is a potato field, of a susceptible cultivar, with 4 million (4×10^6) plants, and that the epidemic starts with one lesion on a single plant. The density of lesions per field would be $1/(4 \times 10^6)$. The source of the lesion could be spores (sporangia) produced on an infected tuber in or near the field of interest. According to van der Zaag (1956), one late blight lesion covers 0.1% of the foliage, on average (i.e., 0.001 = 1/1000 of a plant with visible disease symptoms). The relation between lesion number and foliage coverage would likely depend on the plant cultivar and pathogen strain (i.e., some cultivars will have higher leaf area than others, and some strains may result in larger lesions than others), but we can accept this equivalence for the exercise. The proportion of foliage in the field covered by lesions is our measure here of disease intensity, which we denote as y. In the next chapter, we will provide a detailed discussion of different types of disease intensity measurements.

We refer to time (in days here) as t, and indicate the start of the epidemic as $t = 0$. With one lesion per field, intensity for the field is $y = 0.001/(4 \times 10^6) = 2.5 \times 10^{-10}$. If the number of lesions doubles every 2 days, there will be an average of one lesion per plant (or 4×10^6 total lesions per field) after about 44 days. Although doubling of disease intensity this often may seem exaggerated, it is a typical value for late blight epidemics with susceptible cultivars grown under favorable environmental conditions (Fry, 1978). So, $y = 0.001$ at $t = 44$ days. If disease intensity continues to increase in the same manner, except for the slowing effect of loss of disease-free foliage (new spores are more and more likely to land on lesions rather than on disease-free foliage), $y \approx 0.5$ at $t = 64$ (i.e., half the foliage with lesions), $y \approx 0.90$ at $t = 70$, $y \approx 0.99$ at $t = 77$, and $y \approx 0.999$ at $t = 83$ days.

A plot of y versus t, known as a *disease progress curve* or epidemic growth curve, exhibits an S-shaped pattern in this example (Fig. 1.1), typical for diseases that spread from plant to plant or leaf to leaf (see Chapter 4). Note that at one lesion per plant ($y = 0.001$), the curve is barely above the zero axis. Interestingly, one lesion per plant may be about the level of intensity at which a person (e.g., grower, field scout) would first detect the disease in the field under many circumstances. That is, with an average of one lesion per plant, some plants will have multiple lesions, and many plants will have no lesions. So, unless a person is observing many plants, there is a good chance that lesions will not be found until disease intensity reaches 0.001 or so. (See Chapter 9 for a presentation on patterns of disease and Chapter 10 for a presentation on sampling for disease.)

There is a 1000-fold increase in disease intensity from day 44 to day 84. With the common layperson definition of an epidemic, 100% coverage of foliage with lesions would certainly be considered an epidemic, but 0.1% (i.e., $y = 0.001$) or less foliar coverage would probably not be considered an epidemic, given that a casual

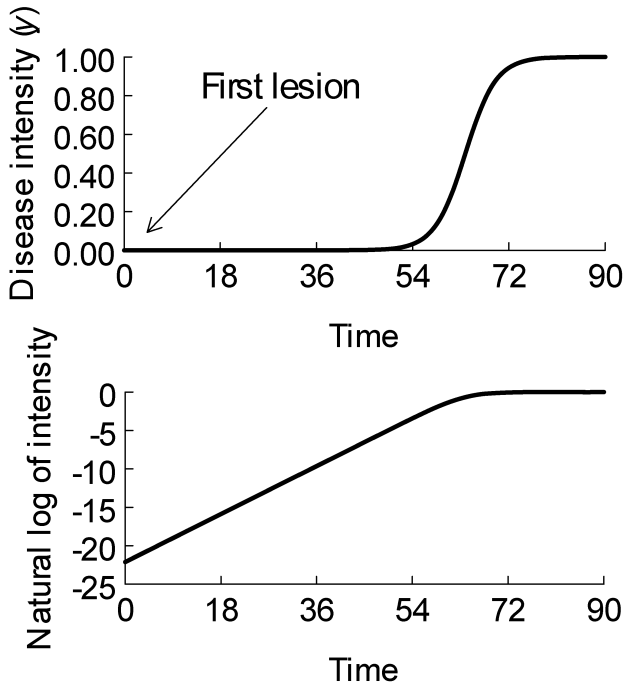

FIG. 1.1. Hypothetical epidemic growth curve (i.e., a disease progress curve) for potato late blight that started with a single lesion in a field of 4 million plants. Upper frame: proportion of foliage with lesions (a measure of disease intensity). Lower frame: natural logarithm (log) of the proportion with lesions.

observer of the field may not even notice any disease at all in the latter case. However, between days 0 and 44, there was a 4-million-fold increase in disease intensity (from one lesion to 4 million lesions per field, or from $y = 2.5 \times 10^{-10}$ to $y = 0.001$). Clearly, there was a much greater *increase* in disease during the first 40 or so days than in the subsequent 40 days. From a population biology perspective, there is no difference in the increase in disease during the first part of the time span than in the later part, and as will be shown in Chapter 4, a single model with a constant rate parameter for disease increase can represent the entire epidemic process from the formation of the first lesion until all foliage is diseased. In fact, the curve in Fig. 1.1 was drawn based on a model known to accurately represent disease increase for potato late blight (Fry, 1978).

There is no practical way to draw a line separating epidemic from non-epidemic phases in this example; rather, there is a continuum of disease increase over the given time period. In fact, when one plots the log of y versus t, a straight line is produced over most of the time period (Fig. 1.1, lower frame). Only late in the epidemic does the log line flatten, because of the limitation to disease increase due to depletion of disease-free foliage available for infection. Other transformations can produce a straight line over the entire time period for many epidemics, a subject addressed later. For practical necessity, plant pathologists mostly study epidemics where y increases from about 0.001 to 1.0, but this increase is really just an extension of the increase in disease that is often not observed because of sampling limitations.

Thus, the magnitude of disease intensity is not an indication of whether an epidemic has occurred or not. Depending on the initial level of disease and the rate of increase, the disease intensity can be either low or high at a given time during an epidemic. Of course, this does not mean that all levels of disease intensity are equally important, either in affecting crop yield or in the need to impose controls. Chapter 11 covers some topics related to predicting whether or not a control is needed based on sampling for disease intensity, and Chapter 12 covers the relationship between crop yield and disease epidemics.

There is sometimes a need to refer specifically to situations with widespread occurrence of high disease intensity. The term *pandemic* can be used to label epidemics that occur over large areas with high levels of disease. The late blight epidemics in Western Europe in 1845 and 1846 and the southern corn leaf blight epidemics in the United States in 1970 are example pandemics that were also of high consequence (Horsfall and Cowling, 1978a).

1.2.2 Epidemiology

As stated above, *epidemiology is the study of epidemics*. For those who prefer a more detailed definition, Diekmann and Heesterbeek (2000) define epidemiology as "the study of the spread of diseases, in space and time, with the objective to trace factors that are responsible for, or contribute to, their occurrence." More specifically to plant diseases, Kranz (1990b) refers to epidemiology as "the science of populations of pathogens in populations of host plants, and the diseases resulting therefrom under the influence of the environment and human interferences." Because of the emphasis on populations, it is sometimes useful to think of epidemiology as the ecology of disease.

Clearly, this science involves the classic disease triangle of host, pathogen, and environment (Stevens, 1960), often taught in beginning plant pathology courses, but at the population and not the individual level. For pedagogical purposes, the triangle is sometimes expanded to a square, pyramid, or other multi-dimensional structure to either emphasize the effects of time and/or space on disease, or to reflect the influence of humans on diseases (Francl, 2001). To be complete, the environment can be partitioned, when relevant, into abiotic (e.g., weather, soil moisture) and biotic (e.g., insect vectors) components.

Populations comprise individuals, so the study of epidemics often involves characterizing or summarizing the disease status of individuals in order to understand the dynamics of disease in populations. For example, the sporulation of fungal pathogens on individual plants is one of the determinants of the rate of disease intensity increase in a population.

1.2.3 Epidemic versus epiphytotic

Some authors, such as Unger (1833), Whetzel (1929), and Ryan and Birch (1978), have argued in favor of the word "epiphytotic" instead of epidemic to describe plant diseases in populations. The argument centers on the derivation of the word epidemic from the ancient Greek adjective, which is most often translated as *what is among the people* or *upon the people*. However, as pointed out by Millar (1978), the Greek adjective can also be correctly translated as *upon the population*, with the population consisting of humans, other animals, or plants.

The term epidemic apparently was first used by Hippocrates (ca. 400 BC), and the word has received increasing use over time. As pointed out by Zadoks and Koster (1976), there is also early precedence for the use of epidemic over epiphytotic to describe plant diseases in populations. Duhamel de Monceau (1728), von Martius (1842), Kühn (1858), and Ward (1901), among others, used the term epidemic in their extensive writings on plant diseases in populations. Perhaps an even stronger argument for the use of epidemic is based on population biology and dynamics. In particular, many of the population-dynamic processes are the same for diseases of humans, other animals and plants, and some of the useful analytical approaches for characterizing and modeling plant disease epidemics originated in medical epidemiology (as will become evident in later chapters) or were first motivated by medical problems.

Thus, although it is not wrong to use epiphytotic for diseases of plants, there is no reason to do so. Incidentally, for those who prefer to use epiphytotic, the unusual term *epiphytotiology* should be used to describe the science of plant diseases in populations. This term has had virtually no use in plant pathology. When it is necessary to refer specifically to the study of plant disease epidemics, *botanical epidemiology* is a very useful label. We use botanical epidemiology, plant disease epidemiology, and plant epidemiology as synonyms in this book.

1.3 Some Historical Developments

1.3.1 Up to 1963

There are several excellent articles on the history of plant disease epidemiology. Zadoks and Koster (1976) present an especially thorough review of the early history of the study of plant diseases in populations, with some interesting translations of passages of some early texts. Cowling and Horsfall (1978), Zadoks (2001), and Waggoner and Aylor (2000) all provide valuable information. We present here only a very limited history of some developments in the field.

Hippocrates (Jones, 1972) wrote 2,400 years ago about epidemics of humans, such as mumps and malaria, and indicated the importance of the environment on disease outbreaks. Theophrastus (372–287 BC) and Pliny (23–79 AD) commented on plant diseases and also emphasized the role of the environment in epidemic occurrences. In a publication far ahead of its time, but ignored for centuries, Duhamel de Monceau (1728) wrote about epidemics of saffron crocus, caused by the soil-borne fungus *Rhizoctonia violacea*, and provided control methods based on his knowledge of the epidemics. Moreover, he even gave evidence that the disease was caused by a "biological entity." Zadoks and Koster (1976) believe that this is the first documentation of a plant epidemiological study.

The role of the environment (especially weather) in disease outbreaks in crops, especially for rust of wheat, was addressed by various authors in the 18th and 19th centuries, including Fontana and Tozzetti (Zadoks and Koster, 1976). In possibly the first textbook of plant pathology, Kühn (1858) wrote clearly about the importance of epidemics of disease in crops, and compared them to epidemics of disease in human and other animal populations. At this stage, epidemics were still mostly considered to be the widespread occurrence of high disease intensity. A more ecological approach to understanding epidemics was taken by Ward in his influential 1901 textbook *Diseases in Plants*. He states "When we come to enquire into what circumstances bring about those severe and apparently sudden attacks on our crops, orchards, gardens, and forests … we soon discover the existence of a series of complex problems of intertwined relationships between one organism and another, and between both and the non-living environment which fully justify the caution already given against concluding that any cause of disease can be a single agent working alone."

Historically, the emphasis generally was on describing or understanding the epidemics with high disease intensity (e.g., the "severe and apparently sudden attacks" in Ward, 1901). A broader view began to emerge by the middle of the 20th century, where the diversity of possible epidemics (e.g., those with high and low disease intensity) was appreciated. The classic treatise by Gäumann, *Pflanzliche Infektionslehre* (Principles of Plant Infection) in 1946, with an English translation 1950, is especially relevant in this regard. Gäumann states "… an epidemic is delimited in two ways; it has a beginning and an end, both in time and space", and explicitly writes about the specific course followed by each epidemic. Moreover, he developed a clear listing of the conditions necessary for an epidemic, such as the presence of a susceptible host, high reproductive capacity and efficient dispersal ability of the pathogen, and optimal weather conditions, and presented the initial ideas to compare epidemics, at least in a qualitative sense.

The period from about 1945 to 1963 was of fundamental importance in the transition of botanical epidemiology from a mostly qualitative to a mostly quantitative discipline. Large (1952) showed the value of disease progress curves, such as the one in Fig. 1.1, for understanding and comparing epidemics, as well as for determining yield losses due to disease. P. H. Gregory performed elegant

research on the dispersal of pathogen spores, and utilized detailed physical models to describe the results. His book on *The Microbiology of the Atmosphere* developed an integrated theory of spore movement and had a great influence on many plant pathologists who were concerned with the spread of disease (Gregory, 1961). The second edition of his book (Gregory, 1973) should still be required reading for all those interested in studying microbes in the atmosphere. Focusing just on books (for now), an important event was the publication of the last volume of a three-volume treatise on plant pathology. The treatise editors, J. G. Horsfall and A. E. Dimond, took the bold and visionary step of dedicating the entire third volume to plant diseases in populations (Horsfall and Dimond, 1960). Finally, the modern concepts of epidemics had started to take hold, and the quantitative aspects of the discipline were on display. Populations were emphasized in chapters that dealt with, among other things, disease forecasting or prediction, physics of spore dispersal, inoculum density, and mathematical modeling. Of special note is the chapter entitled "Analysis of Epidemics", by a little-known potato breeder from South Africa, J. E. Vanderplank (who used to spell his last name as van der Plank). Vanderplank (1960) had a radical idea (at least for most plant pathologists at the time) that epidemics could actually be modeled, and that analysis based on models was essential for understanding the population process of disease progress and for determining control strategies.

Writing that chapter for the 1960 book inspired Vanderplank to write several other books on epidemiology, host resistance, and plant-pathogen interactions (see discussion in Cowling and Horsfall, 1978). Of great significance was his first book, *Plant Diseases: Epidemics and Control*, published in 1963. The book expanded on the ideas in the 1960 article and developed a theory of plant disease epidemics as dynamic population processes that was firmly rooted on empirical data found in the literature (Vanderplank, 1963). In particular, he developed a thorough rationale for the characterization of epidemics using rates of change that could be expressed in the form of nonlinear mathematical models (see Chapter 3 and later chapters of this book). Moreover, the linkage between epidemiology and control was firmly established, with a great deal of emphasis on disease resistance, and chemical control, as well as inoculum reduction. Here, the concepts of vertical and horizontal resistance were developed, which were later expanded upon in his 1968 book (Vanderplank, 1968), updated in 1984 (Vanderplank, 1984), and argued about for decades.

In many ways, Vanderplank's 1963 book was the birth of modern botanical epidemiology. Rarely has a single publication had such a large effect on a discipline. Within a few years, epidemiology was considered to be as important as mycology, virology, and so on, in plant pathology departments (Campbell and Madden, 1990; Cowling and Horsfall, 1978; Zadoks, 1974). Of course, the mathematical treatment was too much for many pathologists to comprehend at the time, but the basic concepts underlying the mathematics were accepted by many. By the 1970s, graduate education included more statistics, mathematics, and computer programming than in the past, and many quantitative epidemiologists entered the profession and began to make important contributions (Campbell, 1998). As Cowling and Horsfall wrote in 1978, Vanderplank is "the man who 'pulled the bung on the epidemiological barrel'. At present new knowledge is spilling out rapidly in ever-widening pools of understanding. With his stimulation, quantitative thinking has spread like an epidemic across the field of plant pathology and it appears that the field will never again be the same."

Vanderplank was certainly not the first to use mathematics or statistics in plant pathology, nor the first to quantify disease progress curves. But it was his elegant exposition of the topic, and innovative integration of biology and mathematics, that convinced many others to change directions. Before him, several researchers calculated disease progress curves (see Campbell, 1998, for brief review), and some, such as Fracker (1936) and Large (1952), used some statistical methods to quantify rates of increase in time. For the spatial component of epidemics, various authors, especially Gregory and Read (1949) and Gregory (1961), characterized gradients of disease in relation to inoculum sources. Vanderplank (1960) considered some spatial aspects of epidemics in 1960, and even made an early contribution to the quantification of spatial patterns of disease in 1946 (Vanderplank, 1946). A significant major early study on spatial patterns of disease was by Bald (1937), which was the basis for some detailed statistical modeling by Cochran (1936).

There had been many important developments in the study of human disease epidemics, including some very elaborate and complicated modeling by Kermack and McKendrick (1927) which still serves as the foundation for theoretical research in quantitative epidemiology (see Chapter 5). Even earlier, the physician Ross (1911) developed a model for malaria epidemics that incorporated dynamics of the vector as well as the infected human population in the form of differential equations. Ross was also quite prophetic in stating:

> *As a matter of fact all epidemiology, concerned as it is with variation of disease from time to time or from place to place, must be considered mathematically, however many variables are implicated, if it is to be considered scientifically at all. … And the mathematical method of treatment is really nothing but the application of careful reasoning to the problems at hand.*

Outside of epidemiology, there is also a long history of important quantitative work on the population growth of organisms, especially humans and other animals

(see Renshaw, 1991). Good examples include the development of the logistic model for representing the increase in numbers of a population (Pearl and Reed, 1920; Verhulst, 1838).

Although Vanderplank was well aware of the early use of the logistic model in ecology, there is no apparent evidence that he was directly influenced by the advances in medical epidemiology, such as the work of Ross and of Kermack and McKendrick in the first part of the 20th century. However, later work showed the strong linkage between the early medical models and those utilized and advocated by Vanderplank and others in botanical epidemiology (see, for example, Jeger, 1986b; Jeger and van den Bosch, 1994a; Segarra et al., 2001).

1.3.2 After 1963

As mentioned above, studying the dynamics of disease in populations, and using the obtained information in evaluating disease controls, certainly became fashionable in the years after Vanderplank's article in 1960 and book in 1963. Of special importance are the long-term contributions by three individuals, Jan C. Zadoks of Wageningen University in the Netherlands; Jürgen Kranz of Giessen University, Germany, and Paul E. Waggoner of the Connecticut Agricultural Experiment Station, USA, as well as the early contributions of the Department of Plant Pathology at the Pennsylvania State University in the United States. The three listed individuals and their colleagues made pioneering contributions over more than three decades to the quantitative aspects of botanical epidemiology. Through their roles as university professors, both Zadoks and Kranz trained numerous students in botanical epidemiology, and directed research projects in many developing countries on the temporal and spatial dynamics of disease and prediction of disease outbreaks. Both scientists produced important books on the subject (Kranz, 1974a, 1990a, 2003; Zadoks and Schein, 1979) that influenced many aspiring epidemiologists over the years.

The Department of Plant Pathology at Penn State was established in 1963, right around the publication of Vanderplank's book. Many of the faculty members of this fledgling department fully recognized the value of epidemiology in the study and control of plant diseases, and built a new curriculum that focused heavily on this population-based discipline. Many of the faculty members and their graduate students made major contributions to quantitative epidemiology in the 1960s and 1970s, and some of the graduates from this department went on to train many of the next generation of epidemiologists in the United States (and elsewhere). Because Waggoner was based at a research station, he was less involved in graduate education, but his research contributions were also highly influential, especially the development of models of epidemics (see, for instance, reviews in Waggoner, 1974, 1981).

With the ubiquitous nature of computers today, readers may be surprised to know that the modeling and analysis performed by Vanderplank (1963), and other early researchers, was done without the use of computers. There was, however, a perceived limit in the number of details of the host–pathogen–environment system that could be incorporated into a model that was being evaluated with just "pen and paper." Waggoner and colleagues made a breakthrough by developing so called computer simulation models that represented the components of disease cycles (e.g., infection, sporulation) in relation to the weather and simulated the increase in disease intensity in populations (Waggoner and Horsfall, 1969; Waggoner et al., 1972). This led to a major activity in developing computer simulation models for many diseases, utilizing the principles of systems analysis (Hau, 1988; Teng, 1981).

The computer-simulation-model results showed that plant disease epidemics can be treated in a quantitative manner, and that collections of equations can be utilized to describe disease development, and to understand and compare epidemics. The systems-analysis emphasis of botanical epidemiology, however, generally declined towards the end of the 20th century as researchers realized that elaborate computer simulation was not necessarily needed to answer many fundamental questions about epidemics. In fact, as summarized by Jeger (1986a) and Jeger and van den Bosch (1994b), a few properly constructed differential equations may be more useful to answer general questions about thresholds for epidemics and final level of disease in epidemics. Waggoner (1990) shows several ways of modeling epidemics with and without the use of computer simulation. Of course, computers remain indispensable tools for quickly determining solutions to model equations over the time and space dimensions, for graphing results, and for statistically fitting models to data and comparing the fit of models for multiple epidemics. We do not take a formal systems-analysis approach to the quantification of epidemics in this book, but, as will be discussed later (e.g., Chapters 5 and 8), there are situations where simulation modeling is clearly of benefit in epidemiology.

1.3.3 Some conferences and books, starting in 1963

A scientific discipline can be judged, in part, by the professional conferences and publications dedicated to the subject. For botanical epidemiology, 1963 was a key year because of the publication of *Plant Diseases: Epidemics and Control*, and *also* because it was the year of the first international meeting dedicated to the topic. The meeting, which was sponsored as a NATO Advanced Study Institute, was held in Pau, France, and was organized by R. D. Schein, J. M. Hirst, with assistance by A. J. P. Oort and J. C. Zadoks. All sessions were informal, and there were no proceedings (Zadoks, 2001). Forty participants from 14 countries contributed. The next dedicated meeting was held

TABLE 1.1. Some text and reference books in botanical epidemiology published since 1970.

Year	*Title*	*Authors (a) or Editors (e)*
1974	*Epidemics of Plant Diseases: Mathematical Analysis and Modeling*	J. Kranz (e)
1978	*Plant Disease: An Advanced Treatise, Vol. 1: How Disease Develops in Populations*	J. G. Horsfall and E. B. Cowling (e)
1978	*Plant Disease Epidemiology*	P. R. Scott and A. Bainbridge (e)
1979	*Epidemiology and Plant Disease Management*	J. C. Zadoks and R. D. Schein (a)
1980	*Comparative Epidemiology: A Tool for Better Disease Management*	J. Palti and J. Kranz (e)
1983	*Plant Virus Epidemiology: The Spread of Insect-borne Viruses*	R. T. Plumb and J. M. Thresh (e)
1985	*Advances in Plant Pathology, Vol. 3: Mathematical Modelling of Crop Disease*	C. A. Gilligan (e)
1986	*Plant Virus Epidemiology: Monitoring, Modelling, and Predicting Outbreaks*	G. D. McLean, R. G. Garrett, and W. G. Ruesink (e)
1986	*Plant Disease Epidemiology, Vol. 1: Population Dynamics and Management*	K. J. Leonard and W. E. Fry (e)
1986	[*Handbook of Plant Disease Epidemiology*], in Chinese	Zeng Shimai (a)
1987	*Crop Loss Assessment and Pest Management*	P. S. Teng (e)
1988	*Experimental Techniques in Plant Disease Epidemiology*	J. Kranz and J. Rotem (e)
1989	*Plant Disease Epidemiology, Vol. 2: Genetics, Resistance, and Management*	K. J. Leonard and W. E. Fry (e)
1989	*The Spatial Components of Plant Disease Epidemics*	M. J. Jeger (e)
1990	*Epidemics of Plant Diseases: Mathematical Analysis and Modeling, 2nd edition.*	J. Kranz (e)
1990	*Introduction to Plant Disease Epidemiology*	C. L. Campbell and L. V. Madden (a)
1991	*L'Épidémiologie en Pathologie Végétale*	F. Rapilly (a)
1994	*Epidemiology and Management of Root Diseases*	C. L. Campbell and D. M. Benson (e)
1996	*Doenças de Planta Tropicais: Epidemiologia e Controle Econômico*	A. Bergamin Filho and L. Amorim (a)
1996	*Epidemiologie der Pflanzenkrankheiten*	J. Kranz (a)
1997	*Exercises in Plant Disease Epidemiology*	L. J. Francl and D. A. Neher (e)
1998	*The Epidemiology of Plant Diseases*	D. Gareth Jones (e)
2003	*Comparative Epidemiology of Plant Diseases*	J. Kranz (a)
2006	*The Epidemiology of Plant Diseases, 2nd edition*	B. M. Cooke, D. Gareth Jones, and B. Kayle (e)

at Wageningen, the Netherlands, in 1971, also as a NATO Advanced Study Institute session. This meeting was organized by Zadoks, Schein, Hirst, and H. D. Frinking. There were 74 participants from 24 countries.

The next international workshop was held at the Pennsylvania State University in 1979. The fourth workshop was held in 1983 at North Carolina State University, and the fifth international workshop was held in Jerusalem in 1986. The next three international workshops were held in Giessen, Germany, in 1990; Papendal, the Netherlands, in 1994; and Ouro Preto, Brazil, in 2001. The ninth international workshop was held in Landerneau, France, in 2005. These meetings generally have been fairly informal, with considerable discussion of various topics of interest. Attendance was limited to about 100 in order to facilitate discussion. Until recently, there was no official structure linking these workshops, except for the common subject matter. Then, the decision was made during the workshop in 1994 to place these meetings under the umbrella of the Epidemiology Committee of the International Society of Plant Pathology (ISPP). Although the meetings have continued to be operated locally, the selection of the venue, date, and organizer is now done by the ISPP committee.

Of course, epidemiology has been, and continues to be, the subject of numerous other symposia, colloquia, and workshops, often organized as parts of larger general conferences, such as national and international congresses of plant pathology. Other meetings focus on particular crops or diseases. However, the international workshops in epidemiology provide some continuity for the discipline and serve as an avenue for the epidemiologists to exchange ideas.

In addition to the many journal articles published, many of which are cited throughout this book, several valuable text and reference books have been written on botanical epidemiology since 1963. Some of the books are listed in Table 1.1 in chronological order. Zadoks (2001) lists additional books of relevance. Some of these books focused on groups of pathogens (e.g., viruses, soilborne pathogens), and others focused on themes, such as

comparative epidemiology or epidemic analysis and modeling. In terms of the latter, the multi-author book edited by Kranz in 1974 was especially important historically because it synthesized many of the mathematical and statistical approaches to the study of epidemics that were introduced in the decade after Vanderplank's first book. This work further set the stage for many of the quantitative advances in the 1970s and 1980s in epidemiology.

The first textbook of botanical epidemiology was written by Zadoks and Schein and published in 1979. The authors of the book did an excellent job of conveying much of the excitement of the field, showing the need for mathematics in understanding epidemics, and clearly demonstrating how management of disease is linked to epidemiology. The book dealt with diseases caused by fungi. The textbook of Campbell and Madden (1990) expanded the coverage to diseases caused by all pathogen groups and gave detailed instructions on the quantification, statistical analysis, and modeling of plant disease epidemics. The "laboratory manual" edited by Francl and Neher (1997) expanded on the Campbell and Madden (1990) textbook and gave more details on many forms of epidemic quantification and analysis. Other textbooks have been written in Chinese, French, and Portuguese (see Table 1.1).

1.3.4 Final thoughts on the review of historical developments

The above sections addressed only *some* of the major developments in botanical epidemiology, with an emphasis on the progression toward quantification in the field. The tremendous growth in the discipline within plant pathology during the 1960s, 1970s, and 1980s has been eclipsed somewhat in recent decades by the growth in the larger discipline of molecular biology. However, until molecular biology (or more traditional breeding) results in durable resistance to all plant pathogens on all crops, coupled with the acceptance of the new cultivars by growers and the public, there will be plant disease epidemics, and many of these will have a substantial impact on growers, consumers, and society as a whole. Thus, botanical epidemiology continues to be of utmost importance in understanding when and how epidemics occur, and in giving a sound theoretical and practical basis for disease management decisions.

In addition to the individuals mentioned in the previous sections, *many* other scientists have made important contributions to the field of botanical epidemiology, both recently and in the past. The problem is that we cannot adequately explain the historical importance of many scientific contributions until the contributions are first explained in some detail. Thus, comments on historical developments in botanical epidemiology are presented, where appropriate, throughout this book. It is not our goal to provide a thorough review of the history of the discipline, but we feel it is of value for the reader to gain some understanding of major early contributions in epidemiology.

1.4 Prelude to the Rest of the Book

This book is concerned with the study of epidemics. It builds on the contributions of the scientists mentioned above, and many others, and attempts to provide an integrated approach to the quantification of disease development in time and space in host populations. It could be equally stated that this book is about relationships between variables (see Fig. 1.2). For instance, the disease progress curves of Fig. 1.1 and Fig. 1.2A show the relationship between disease intensity and time. A considerable amount of attention is given in Chapters 4, 5, and 6 (and elsewhere) to characterizing the manner in which some disease variable changes over time. Concurrent with the increase (or decrease) in disease over time is the spread of disease, through pathogen dispersal, in space. This is usually expressed as some measure of disease versus distance from an inoculum source, as shown in Fig. 1.2B. Characterization of this relationship is discussed in Chapters 7 and 8. The simultaneous dynamics of disease over time and space are also considered in these chapters.

Often, there are many inoculum sources (e.g., infected weeds, overwintering spores in the soil, infected seeds), possibly at unknown locations, at the start of an epidemic, so there is no unambiguous way to relate disease intensity to distance from the inoculum. This has led to the development of alternate ways of characterizing the spatial component of epidemics, often borrowed from ecology (Pielou, 1977), which are mostly based on statistical methods (Madden, 1989; Madden and Hughes, 1995, 2002). One important approach involves, in part, determining the frequency distribution—and related measures of variability—of diseased individuals per sampling unit (such as diseased leaves per plant or diseased plants per crop row). This is exemplified in Fig. 1.2D for a situation with 10 individuals in each of 194 sampling units. Utilization of a frequency dataset such as this to determine spatial heterogeneity (and, as will be shown, small-scale aggregation) of disease will be explained in Chapter 9. In Chapters 10 and 11, we show how information on disease heterogeneity, or information on the relationship between heterogeneity and magnitude of some measure of disease intensity, is utilized to develop sampling plans. These sampling plans can be for either estimating disease intensity with a desired level of precision (Chapter 10) or for making a decision on whether or not to invoke a control measure, such as a fungicide application (Chapter 11).

When the physical locations of sampling units are known, very extensive and multifaceted quantification of the spatial pattern of disease is possible. Then, instead of simply determining the frequency of diseased individuals,

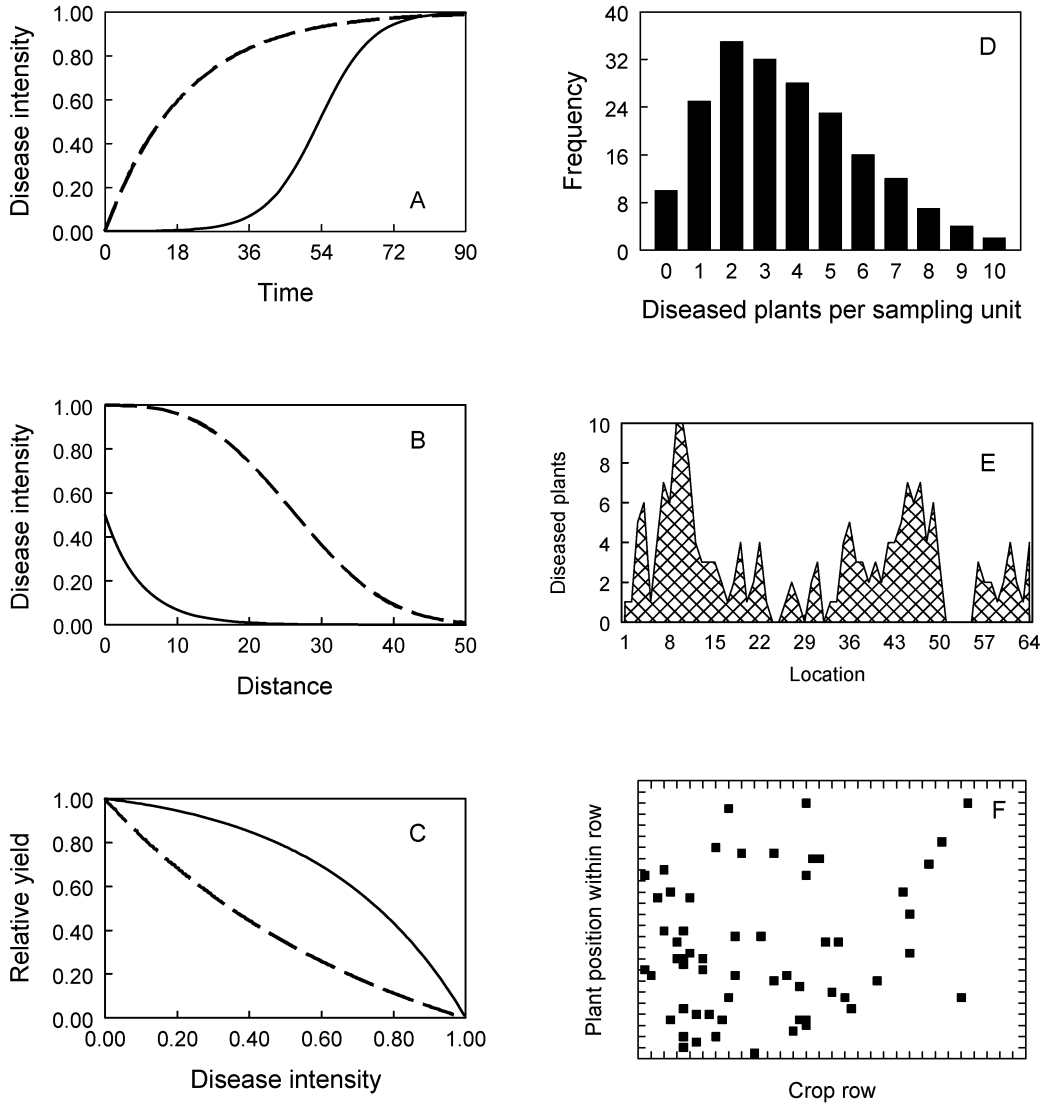

FIG. 1.2. Example relationships of great interest in botanical epidemiology. (**A**) Disease intensity versus time during an epidemic. (**B**) Disease intensity versus distance from an inoculum source. (**C**) Relative yield of a crop versus disease intensity at a particular time during epidemics. (**D**) Frequency of diseased plants per sampling unit (out of 10 possible plants diseased). (**E**) One-dimensional map of diseased plants; that is, number of diseased plants (out of 10) diseased at each of 64 spatially referenced locations in a transect of a field. (**F**) Two-dimensional map of diseased plants in a field. Each solid square represents a diseased plant (with a blank space representing the disease-free plants).

one can show the relationship between disease and location, as demonstrated in Fig. 1.2E for a situation where the number of diseased plants (out of 10) was determined at each of 64 locations in a transect through a field. Two-dimensional maps can also be prepared, as well. An example is shown in Fig. 1.2F, where each sampling unit is a plant (and not a group of plants), so that each disease observation is binary (diseased or not). The relationship between disease and spatial location—or relationship between disease status of neighboring plants—can be characterized with a wide collection of interrelated statistical methods to elucidate small-to-large scale patterns of disease and, hopefully, to gain an understanding of the dispersal of the pathogen causing the measured disease. The statistical methods are discussed in Chapter 9.

A frequent consequence of plant disease epidemics in crops is a reduction in the yield of harvested product. Indeed, this has probably been, historically, the most important driving force for innovation and quantification in botanical epidemiology. Crop losses are often characterized based on the relationship between yield (or yield relative to a disease-free control, or the reduction in yield relative to a control) and a measure of disease intensity, as exemplified in Fig. 1.2C. This topic is addressed in some detail in Chapter 12.

The graphs in Fig. 1.2 demonstrate just some of the most common relationships quantified by botanical epidemiologists. Other relationships are shown throughout the book.

To study disease progress in time and space, or to relate yield to the epidemic, one must first measure

disease. Thus, before addressing key relationships of interest in epidemiology, we discuss in Chapter 2 ways of measuring disease of plants and quantifying the accuracy and reliability of the obtained measurements. Moreover, characterization of relationships, such as those in Fig. 1.2, requires the use of mathematical and statistical models. To help the reader in this regard, we introduce the concepts of modeling in Chapter 3, and show some of the ways of fitting models to data and comparing model-fitting results for different epidemics. Many other aspects of modeling and data analysis are introduced throughout the book, as appropriate, for quantifying aspects of epidemics, sampling, and making disease-management decisions based on disease or other interrelated indicator variables.

There are many facets to the study of epidemics, and no single book can cover all aspects of the topic. Throughout the book we direct readers to journal articles, book chapters, and books that deal with topics that we do not fully address. Two particular topics not covered need to be mentioned here. In order to keep the book to a reasonable size, we decided to mostly deal with epidemics caused by a single pathogen species in a single host species. In crops and forests, of course, diseases caused by several pathogens can generally be found, although one disease may dominate in a growing season in a given year. The study of multiple diseases in populations simultaneously requires expansions of the approaches discussed in this book (e.g., Madden et al., 1987b; Ngugi et al., 2001; Turechek and Madden, 2000), and we provide some relevant citations in various chapters for this general topic.

Ever since Vanderplank's first book in 1963, there has been a strong link between the disciplines of botanical epidemiology and genetics—both in terms of host resistance and pathogen population genetics and evolution (Leonard and Fry, 1989; Robinson, 1976; Vanderplank, 1968, 1975, 1984). Although we acknowledge the great importance of taking an epidemiological approach to studying host-pathogen genetics, and in taking a genetics approach to studying epidemics, we do not give explicit coverage of genetics in this book. The book hopefully does provide enough on the theory and practice of epidemiology to allow interested readers to study epidemics and their control in terms of host and pathogen genetics.

1.5 Possible Course Outlines

This book can be used as either a reference or a textbook. As a textbook, there is sufficient material probably for two courses. However, depending on the objectives of the instructor, the book can be used for a single course by selecting different tracks. We list just a few here.

A purely temporal-analysis course could be based on Chapters 4–6, with the earlier chapters used (as needed) to explain disease variables and types of modeling. Chapter 12 could also be covered in this case to show the consequences of epidemics. A course in theoretical epidemiology could be based on Chapters 5, 6, and 8, with background information in Chapters 3, 4, and 7. A data-analysis, sampling, and decision-making course could be based on Chapters 2–4, 7, and 9–12. A primarily spatial-analysis course could be based on Chapters 7–10, with earlier chapters used to "fill in the blanks." Finally, a standard (classical) course in epidemiology would best be based on parts of *all* of the chapters (with simple avoidance of the more advanced sections or subsections).

1.6 Suggested Readings

At the end of each chapter we list some useful articles, book chapters, or books that deal in more detail with some of the major themes of the chapter. We do not necessarily agree with everything in the suggested readings, but think the citations provide valuable material to assist the reader in understanding one or more topics. For a general background on botanical epidemiology, including historical developments in the field, the following articles are useful.

Cowling, E. B., and Horsfall, J. G. 1978. Prologue: How disease develops in populations. In: *Plant Disease: An Advanced Treatise, Vol. 1: How Disease Develops in Populations* (J. G. Horsfall and E. B. Cowling, editors). Academic Press, NY, pp. 1–15.

Gilligan, C. A. 1985. Introduction. In: *Advances in Plant Pathology, Vol. 3: Mathematical Modelling of Crop Disease* (C. A. Gilligan, editor). Academic Press, London, pp. 1–10.

Jones, D. G. 1998. An introduction to plant disease epidemiology. In: *Plant Disease Epidemiology* (D. G. Jones, editor). Kluwer, London, pp. 3–13.

Zadoks, J. C. 2001. Plant disease epidemiology in the twentieth century: A picture by means of selected controversies. *Plant Dis*. 85: 808–816.