

An institution, made up of scientists engaged in a common pursuit, has an inescapable desire to "look over the shoulders" and to see where it came from and how far it has progressed.

J. C. ZADOKS AND L. M. KOSTER, 1976

## 2.1 HISTORICAL EPIDEMICS OF PLANT DISEASE

The epidemics of historical (and modern) times and the development of botanical epidemiology are separate but related topics. Epidemics of plant diseases have changed the course of human history in localized and widespread geographical areas on several occasions. These epidemics or, perhaps more appropriately, the results of these epidemics have called attention to plant diseases and have directly contributed to the establishment and development of plant pathology as a science. Thus, before we examine the development of plant disease epidemiology, it is worthwhile to examine some of the major epidemics of plant diseases that have influenced recent human history.

### 2.1.1 Ergot and Ergotism

Ergot of rye is caused by *Claviceps purpurea*. The fungus invades the grain and produces a large purple-black sclerotium, which is also called an ergot. This survival structure contains large amounts of many alkaloids, including nearly pure LSD, a hallucinogenic drug. When ergot is ground into flour with rye grain, the alkaloids are baked into the bread. Thus the problem begins.

Symptoms that result from consuming the contaminated rye bread vary with the amount of bread ingested and the amount of alkaloids present in the particular sclerotia. Small amounts of the alkaloids can induce abortion

in humans and cattle. (Even smaller amounts have been used as an aid in childbirth by midwives over many years.) If larger amounts are ingested, fingers and toes tingle, a high fever develops, and if the fever persists, mental derangement or death can result. Hallucinations may accompany the whole process and even gangrene of the extremities can occur.

In some years, because of environmental conditions and availability of inoculum, the ergot disease is more severe—these can be problem years. The first recorded epidemic of ergotism was in AD 857, when thousands died in the Rhine Valley in Europe. Because of its symptoms, the disease in humans was called *sacer ignis*—the holy fire. When the monks of the order of St. Anthony were able to relieve many of the symptoms of the disease during the epidemic of AD 1039 in France, the disease became known as St. Anthony's fire. Whether it was the spiritual ministrations of the monks or the ergot-free wheat bread that the monks fed their patients that relieved the symptoms of ergotism, one can only speculate. Ergotism continued to plague the rye-growing regions in France and Germany during the eleventh, twelfth, and thirteenth centuries and was a significant contributing factor in the defeat of Peter the Great of Russia in 1722 as he sought to gain control of certain warm-water ports on the Black Sea (Carefoot and Sprott, 1967). There is also some evidence that an epidemic of ergot of rye may have led to the accusations of witchcraft in Salem Village, Massachusetts (and several adjacent towns) and Fairfield County, Connecticut in 1692—47 years after the last upsurge of witch persecution in England (Caporeal, 1976; Matossian, 1982).

Epidemics of ergot on rye have occurred in recent times as well. In 1951, the growing season was unusually moist and ergot occurred on rye in locations in southern France. In the fall of 1951, through the combination of the ergot on the rye and some unscrupulous business practices of a farmer, a miller, and a baker who sold, ground, and baked the ergot into what should have been pure wheat bread, 200 cases of severe and damaging illness, 32 cases of insanity, and four deaths occurred in Pont-St.-Esprit in Provence, France (Carefoot and Sprott, 1967; Fuller, 1968). As recently as 1977–1978, when drought resulted in the loss of most domesticated grains in Ethiopia and the people were forced to collect and eat wild ergot-infected grains to survive, ergotism was a significant factor in making the already bad situation worse (Demeke et al., 1979).

### 2.1.2 Late Blight of Potato

Late blight of potato is caused by *Phytophthora infestans*. The disease is favored by cool, wet weather. It is a disease that influenced the history of Europe and the United States on several occasions and probably contributed at least as much as any other single disease to the development of the science of plant pathology.

In the nineteenth century, potatoes formed the foundation of the daily

diet of Irish farmers, except for the relatively few who could afford bread and bacon. Grain crops and pigs were raised by most Irish farmers but these went to the landlord to pay the rent. What remained was the potato, and the typical Irishman consumed 8–14 lbs (3.6–6.4 kg) of potato each day!

The spring and summer of the growing season in Ireland in 1845 were warm, but the fall turned cool and damp. Cold rains persisted and some of the potatoes were blighted late in the season. When the tubers were stored, many rotted in the winter of 1845–1846, and some hunger was felt by Irish families. It was not until the spring of 1846, however, that the full effects of the 1845 epidemic were noticed. Few sound tubers remained for planting; many were discarded and left in the traditional cull piles. Those that were planted did not thrive. Late blight was rampant, the potato crop of 1846 failed, and many Irish starved.

Late blight was not limited to Ireland in the 1840s. The disease was widespread at this time in the northern United States and northern Europe (Bourke, 1964). Reports of the disease were common in local newspapers. The differences in Ireland that made late blight so devastating were the nearly absolute reliance of the Irish people on potatoes as a food source, the generally unfavorable political situation in Ireland, and the growth of the Irish population from 4 million to 8 million between 1800 and 1845. The result of the epidemic was that by 1855, Ireland's population had dropped by 3 million—1 million dead because of starvation and associated maladies, and 2 million who had emigrated to the United States, Canada, and other countries (Carefoot and Sprott, 1967).

Epidemics of late blight of potato also played a role in the outcome of World War I. The weather in Germany during the growing season of 1916 resembled that in northern Europe (including Ireland) in 1846—cool and wet. Late blight was a problem. Bordeaux mixture had been discovered in France in 1882, and this mixture of copper sulfate and lime was effective in controlling late blight. But because of war needs, the military leaders would not release the copper needed to make Bordeaux mixture. With most of the grain and potatoes being provided as supplies for the army in 1916 and 1917, the soldiers were not hungry, but their families were starving. Thus morale of the troops declined, undoubtedly contributing to the military collapse of Germany in 1918.

### 2.1.3 Coffee Rust

Coffee rust, caused by *Hemileia vastatrix*, was a devastating disease in Ceylon (now Sri Lanka) between 1870 and 1889, and is currently of major concern in coffee-growing regions of Central and South America. The rise and fall of coffee production in Ceylon illustrate the dangers of planting large areas with genetically uniform crops and the rapidity with which plant disease epidemics can destroy a crop and, as a result, change a social custom (Large, 1940; Carefoot and Sprott, 1967).

In 1835, the British in Ceylon were growing only about 200 ha of coffee. By 1870, nearly 200,000 ha of coffee was in production, exports were 50 million kg of coffee beans per year, the Oriental Bank flourished, and the British were a nation of coffee drinkers. But as early as 1869, the Reverend M. J. Berkeley had described and named the fungus *Hemileia vastatrix* from coffee trees in Ceylon, where it was associated with the premature fall of leaves in an area of about 1 ha. Berkeley suggested immediate application of sulfur because the disease, with the fungal mycelium in the leaves and the sporulation on the lower leaf surfaces, would be difficult to combat once it had been allowed to spread. However, neither the planters nor the government responded and by 1874 the "coffee-leaf disease" had spread to every plantation on the island. The disease did not kill the trees, but left them weakened and unproductive. By 1878, yields were down by 55%. In 1880, Henry Marshall Ward arrived in Ceylon and began to unravel the life history of the coffee rust fungus. He collected spore samples on sticky, glass slides hung on the coffee trees, made careful observations, described the life cycle of the fungus, and even demonstrated the effectiveness of spraying the diseased trees with a lime-sulfur mixture (Ward, 1882). But the ideas came too late and were too costly for the nearly bankrupt planters. The Oriental Bank closed its doors and the bankrupt and nearly bankrupt planters planted tea bushes. Tea had been somewhat popular in Britain (one could make about 300 cups from each pound of leaves, but only 30–40 cups from a pound of coffee), and with the demise of coffee in Ceylon, tea became even more popular. Coffee rust also ravaged plantations all over Southeast Asia and India. In only one decade, the coffee industry of an entire continent was destroyed.

Coffee production shifted to the New World. Extensive plantations of coffee are grown in Central and South America. For a time, the coffee trees were free of rust. Today, however, epidemics of coffee rust occur wherever coffee is grown throughout the Americas. Modern control strategies and a better understanding of the epidemiology of coffee rust are helping to solve the problem, but coffee rust remains as the most important threat to coffee production.

#### 2.1.4 Chestnut Blight

The destruction of the American chestnut as a major forest species by chestnut blight (caused by *Cryphonectria parasitica*, formerly *Endothia parasitica*) is perhaps the best known epidemic of a plant disease in the United States (Hepting, 1974). Of more than 100 commercial hardwood species, chestnut, at one time, made up over one-fourth of hardwood timber cut in the southern Appalachian region. The nuts provided a good food source for humans and wildlife. The wood was used in furniture, homes, and fences, as firewood, as decay-resistant poles for telegraph and telephone lines, and

as railroad ties. The chestnut was also the major source of tannin for a highly successful leather tanning industry.

In 1904, H. W. Merkel noted that the chestnut trees of the Bronx Zoological Park in New York City were dying. The cause was an exotic fungus, then called *Endothia parasitica*, which caused a severe blight on American chestnut. Somehow the fungus had been brought into the United States. Oriental chestnut trees were resistant to the disease because they had evolved in the presence of the disease. To the American chestnut, however, the pathogen was a newcomer, and the stands of chestnuts in the United States were completely susceptible to the pathogen. By 1911, the blight had spread over New Jersey and parts of New York, Connecticut, Massachusetts, Rhode Island, Delaware, Virginia, and West Virginia, and the pathogen continued to spread (see Fig. 10.11). Chestnut blight eventually reduced the once mighty American chestnut tree to a relatively insignificant species that occurs rarely, and then primarily as stump sprouts.

As a result of chestnut blight, entire communities in the Appalachians were forced to disband or turn to other enterprises as the chestnut tree disappeared as a source of tannin. The loss in lumber has been conservatively estimated to be 30 billion board ft (Carefoot and Sprott, 1967). Huge creosoting plants were built to inject creosote into rot-susceptible pine for railroad ties and poles. And fuel-intensive, synthetic agents are now used for tanning leather.

### 2.1.5 Southern Corn Leaf Blight

In the summer of 1970, an unprecedented epidemic of leaf blight occurred throughout most of the corn-producing areas of the eastern United States. In February 1970, southern corn leaf blight (SCLB) was found in Florida on hybrids that had exhibited resistance to *Bipolaris maydis* in previous years. Symptoms included a serious leaf blight and stalk rot, as well as an unexpected ear rot. The affected corn lines possessed several different genes for resistance to *B. maydis*, but all were hybrids that had been produced using a cytoplasmic male sterility technique. The observation of SCLB on these corn hybrids that had all been produced using the gene for Texas cytoplasmic male sterility, *Tcms*, was alarming because as much as 85% of the total acreage in the United States in 1970 was to be planted to corn lines with *Tcms*.

The first observation on the hypersusceptibility of corn possessing *Tcms* to infection by *B. maydis* was made in the Philippines in 1961 (Mercado and Lantican, 1961). Prior to 1969, SCLB was not considered to be of economic importance in the United States. In 1969, however, the extreme susceptibility of corn with *Tcms* was noted in August and September in Iowa, Illinois, Indiana, and Minnesota (Ullstrup, 1970). A new race of *B. maydis*—race T—had invaded the corn belt. Isolates of race T were highly virulent on corn with *Tcms* but were generally mildly virulent on corn with normal

cytoplasm. Later, a survey of a large world collection of *B. maydis* made between 1955 and 1966 would show that race T had been present in many parts of the world, but mainly on gramineous hosts, not on corn.

By May 1970, SCLB was well established in the southern United States. Weather conditions favorable for the northward movement of inoculum occurred at least six times between June and August 1970, and a tropical storm moved air parcels from the Gulf of Mexico into the Midwest in July. Inoculum of race T arrived in the heart of the corn belt where weather conditions were favorable not only for infection but also for pathogen reproduction. Because as much as 85% of the corn crop was susceptible, a large-scale epidemic developed. Losses ranged from 100% in some southern fields to an average of 20–30% in Indiana and Illinois. In some northern and eastern areas losses were negligible. Although it is an educated guess, it is estimated that about 15% of the U.S. corn crop, or about 20 million metric tons of corn worth about \$1 billion, was lost (Horsfall, 1972).

Following the devastating epidemics of 1970, seed companies took measures to provide as much corn seed without *Tcms* as possible for 1971. In the spring of 1971, enough normal cytoplasm seed was available to plant about 25% of the corn acreage and enough of blends of normal and *Tcms* seed to plant another 40%. Although isolates of race T of *B. maydis* overwintered in the southern United States and weather was conducive to disease development early in the growing season, in most areas little disease developed. Weather systems did not favor the rapid, northward dispersal of inoculum and the weather was unusually cool in July and August. The widespread, devastating epidemic of 1970 was not repeated in 1971; however, as a result of the 1970 epidemic, questions remained about the genetic vulnerability of our major crops to plant diseases.

### 2.1.6 Historical Epidemics in Perspective

The five plant diseases highlighted above are only selected examples of the effects of epidemics caused by plant pathogens (Table 2.1). Large (1940), Carefoot and Sprott (1967), Klinkowski (1970), and Horfall and Cowling (1978a) have chronicled other important, historical epidemics of plant diseases. In some cases, humans have set the stage for major epidemics either

TABLE 2.1 Some Major Epidemics of Plant Diseases

Year(s)	Epidemic and Consequence	Reference
857	First recorded epidemics of ergotism: thousands died in the Rhine Valley	Carefoot and Sprott (1967)
1039	Ergotism in France: monks of the Order of St. Anthony relieved many symptoms	Carefoot and Sprott (1967)

(continued)

TABLE 2.1 Some Major Epidemics of Plant Diseases (continued)

Year(s)	Epidemic and Consequence	Reference
1722	Ergotism at Astrakhan: aided in defeat of Peter the Great of Russia	Carefoot and Sprott (1967)
1845-1846	Late blight of potato: Irish potato famine; 1 million Irishmen died of starvation and related maladies, another 2 million emigrated	Bourke (1964), Carefoot and Sprott (1967), Woodham-Smith (1962)
1845-1860	Powdery mildew of grape in England and France: financial loss and importation of <i>Phylloxera</i> aphid from North America	Large (1940), Carefoot and Sprott (1967)
1882-1885	Downy mildew of grape in France: financial loss and discovery of Bordeaux mixture	Large (1940), Carefoot and Sprott (1967)
1870-1880	Coffee rust in Ceylon: financial ruin for planters; English people became primarily tea drinkers	Large (1940), Carefoot and Sprott (1967)
1904-present	Chestnut blight in the United States: destruction of American chestnut as a forest tree species in eastern United States; financial loss	Hepting (1974)
1913	Leaf spot on banana cultivar Gros Michaels in the Sigatoka Valley in Fiji: financial loss	Carefoot and Sprott (1967)
1915-1923 and 1930-1935	Panama disease of bananas in Costa Rica, Panama, Colombia, and Guatemala: financial loss	Carefoot and Sprott (1967)
1916-1917	Late blight of potato in Germany: food shortages in civilian population; contributing factor to demoralization of German troops in World War I	Carefoot and Sprott (1967)
1930-present	Dutch elm disease in the United States: loss of American elm as shade tree species in many areas; loss of property value	Carefoot and Sprott (1967)
1942-1943	Leaf blight of rice in Bengal: great Bengal famine—nearly 2 million people died from starvation	Padmanabhan (1973)
1951	Ergotism at Pont-St.-Esprit, France: 4 deaths, 32 cases of insanity, numerous cases of hallucinations	Fuller (1968)
1970	Southern maize leaf blight in the United States: 15% of U.S. maize crop lost	Horsfall (1972)
1977-1978	Ergotism in Ethiopia: hallucinations, human suffering, and death	Demeke et al. (1979)
1979-1980	Blue mold of tobacco in eastern United States and Canada: financial loss	Lucas (1980)

through our ignorance, as in the cases of late blight in Ireland and coffee rust in Ceylon, or by our inability to learn from past examples, as in the case of maize leaf blight (caused by *Bipolaris maydis*) in 1970. In other instances, we have been rather innocent victims, as in the case of ergot and ergotism, or at least unknowing participants as in the case of chestnut blight. In addition to the historical and sociological lessons that can be derived from these examples of devastating epidemics of the past, the understanding of the multitude of biological and environmental factors that contributed to these epidemics should provide us with information and opportunities that will allow us to avert future epidemics. The principles and practices derived from this understanding should help us to manage plant diseases better at the field, county, state, national, and international levels. This understanding is what plant disease epidemiology is all about.

## 2.2 DEVELOPMENT OF PLANT DISEASE EPIDEMIOLOGY

Plant disease epidemiology was not a recognized discipline of plant pathology until the 1960s. Many factors led to the development of botanical epidemiology, but it was the appearance of J. E. Vanderplank's *Plant Diseases: Epidemics and Control* in 1963 that served as the catalyst for the development of the discipline. It was the right book appearing at the right time and served as a focal point of the new science. Many other important contributions to the development of epidemiology are listed in Table 2.2. Before we continue with the recent history of plant disease epidemiology, however, it is worthwhile to review briefly some of the earlier steps toward plant disease epidemiology as we know it today. The publication of Zadoks and Koster (1976) is the key reference in this area.

### 2.2.1 Terminology

"Epidemiology is the science of disease in populations" (Vanderplank, 1968). The populations of concern here are populations of plants, although parallel disciplines in medicine and veterinary sciences deal with disease in populations of humans and animals, respectively. Much of our terminology in botanical epidemiology is borrowed from these parallel sciences.

The term *epidemic* was used by Hippocrates (460–380 BC), the revered physician of the Aegean isle of Cos (Jones, 1972). As an adjective in Greek, it means literally "what is among the people." The term "epidemic" was used in the title of a book related to plant disease by Ramazini in 1691 and in a publication by Duhamel in 1728. In 1833, Unger introduced the German term *Epiphytozie* for epidemics on plants. As translated into English by Zadoks and Koster (1976), Unger wrote: "The same behavior in the world of disease among humans leads to epidemics, among animals to epizootics, and among plants to epiphytotics. . ." Von Martius (1842) used the term

TABLE 2.2 Selected Landmarks in the Development of Plant Disease Epidemiology

Year	Event
1728	H. L. Duhamel de Monceau publishes paper describing contagious epidemics of the Death in saffron crocus; paper goes unnoticed
1833	Franz Unger uses the term "Epiphytose" for epidemics of diseases on plants
1858	Julius Kühn compares plant disease epidemics to those among people and animals in the first textbook of plant pathology
1901	Henry Marshall Ward's <i>Disease in Plants</i> is published with chapters "Spreading Disease and Epidemics" and "The Factors of an Epidemic"
1913	Lewis Ralph Jones emphasizes the importance of environment in plant disease development
1946	Ernst Gäumann's <i>Pflanzliche Infektionslehre</i> , the first comprehensive work in plant pathology to emphasize botanical epidemiology, is published and the "factors of an epidemic" are enumerated; the concept of infection chains is presented
1960	Jonathan Edward Vanderplank's chapter "Analysis of Epidemics" is published in <i>Plant Pathology</i> , Vol. 3 (edited by J. G. Horsfall and A. E. Dimond). Other significant chapters on disease forecasting, inoculum potential, and spore dispersal also appear in this volume
1961	Phillip Harries Gregory's <i>Microbiology of the Atmosphere</i> is published
1963	NATO Advanced Study Institute, Epidemiology of Fungal Pathogens, is held in Pau, France
1963	Vanderplank's <i>Plant Diseases: Epidemics and Control</i> is published and serves as the cornerstone of modern plant disease epidemiology
1966	First epidemiology symposium, "Plant Disease Epidemics—Analysis and Implications," is held at a meeting of the American Phytopathological Society
1968	Epidemiology section (with six sessions) is included as part of the First International Congress of Plant Pathology in London
1969	Paul E. Waggoner and James G. Horsfall publish <i>EPIDEM: A Simulator of Plant Disease Written for a Computer</i>
1974	Jürgen Kranz's <i>Epidemics of Plant Diseases: Mathematical Analysis and Modelling</i> is published
1976	Raoul Robinson's <i>Plant Pathosystems</i> is published.
1979	Jan C. Zadok and Richard D. Schein's textbook <i>Epidemiology and Plant Disease Management</i> is published
1989 (anticipated)	Michael J. Jeger's <i>The Theory of Plant Disease Epidemics</i> is published

"epidemics" in the title of his book on potato dry rot caused by *Fusarium* spp. The term also appears with varying degrees of emphasis in textbooks, such as those by Kühn (1858), Von Tubeuf (1895), and Ward (1901). In Ward's classic text, he includes a chapter on the factors of an epidemic. In the 1900s, the term "epidemic" appears with increasing frequency.

The term "epidemiology" is a neologism composed of three parts: *epi* (upon) + *demio* (people) + *logy* (treatise). Zadoks and Koster (1976) have found the term used in medicine at least since 1873 (Parkin, 1873). Whetzel (1929) maintained that epiphytotic (i.e., the change in disease intensity in a

population of plants over time and space), as proposed by Unger, should have precedence in phytopathology, and thus the study of diseases in plant populations should be *epiphytology*. This term has not received general acceptance (Ryan and Birch, 1978; Millar, 1978) among plant pathologists and, to be correct, should be *epiphytotiology* to follow the parallel derivation of epidemiology from epidemic. We believe that epidemic and epidemiology are completely appropriate terms and should be considered in their current sense as proposed by Vanderplank (1963).

## 2.2.2 Trends, Events, People, and Publications

Phytopathology did not exist as a discipline until the late 1800s, and the trends that would lead to epidemiology were not really evident until the twentieth century. The work of Duhamel from the eighteenth century, however, deserves mention, even though it had no discernible influence on the development of phytopathology or epidemiology. None of the early workers on plant disease mention the work. In fact, J. C. Zadoks (Zadoks and Koster, 1976) was the first to bring this work to the attention of modern plant pathologists.

Duhamel (1728) discussed the epidemiology of a disease of saffron crocus called the Death. He showed that the causal agent is a biological entity, now known as *Rhizoctonia violacea*, recognized epidemics on plants, compared these epidemics on plants to epidemics of animals, and provided control recommendations for the disease. He wrote (as translated by J. C. Zadoks):

And I have been surprised by the damages which this disease causes in the places that have the misfortune to be affected with it. And who would fail to see that a plant attacked by the disease becomes murderous to others of this species? Has anyone until now observed contagious epidemics in plants? That which attacks the bulb of saffron is none the less of that nature, because like the pest of animals, it spoils the neighboring bulbs. . . .

This is the first known published work that could reasonably be called plant disease epidemiology (Zadoks and Koster, 1976). It apparently received little attention in the scientific world and thus had no discernible influence on what was to be a nearly century-long debate on the parasitic origins of plant diseases.

The concept of an epidemic in the 1850s, at the dawn of the science of phytopathology, is exemplified in the writings of Julius Kühn (1858) in the first "textbook" on plant diseases: "As the epidemics among people and animals appear suddenly and unexpectedly, spread destruction over entire regions for a time, and then slowly disappears, so also do plant epidemics." The concept was one of suddenly appearing disease over a large area, much as was observed in the potato late blight epidemics in northwest Europe

(including Ireland) in 1845 and 1846. The writings of H. Marshall Ward in his 1901 textbook *Diseases in Plants* reflect a shift in perception concerning epidemics. The ecological approach to studying diseases is indicated in the chapters "Spreading of Disease and Epidemics" and "The Factors of an Epidemic." He wrote:

When we come to enquire into what circumstances bring about those severe and apparently sudden attacks on our crops, orchards, gardens, and forests by hosts of some particular parasite, bringing about all the dreaded features of an epidemic disease, we soon discover the existence of a series of complex problems of intertwined relationships between one organisms and another, and between both 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.

Another trend in the developing science of epidemiology was the emphasis on the role of environment in development of plant diseases. L. R. Jones of the University of Wisconsin, a proponent of understanding influence of environment on plant diseases, wrote in 1913: "The relation of environment to the pre-disposition of the host, as well as to the virulence of the parasite cannot be over-emphasized." Jones's energy and vigor for pursuing environmental effects provided many tangible results and his enthusiasm was infectious. This enthusiasm and productivity in research led E. J. Butler to conclude in 1926 (in Keitt and Rand, 1946):

There are three phases in the history of plant pathology: First, the period of DeBary in which the fungus held first place; second, the period in which the host received the most attention; and finally, the present period in which disease is considered as an interaction of both under the conditioning influence of the environment. The leader in this is Jones.

The emphasis on environment occurred at a time when breeding for resistance to plant diseases was beginning to have a significant impact on agriculture. In the decade following the rediscovery of Gregor Mendel's work, resistant varieties to many plant pathogens were developed. The environmental investigations and resistance breeding efforts were part of a larger trend of "relevant" or practical research in plant pathology as a whole. The view that epidemiological studies could provide vital information to support the practical, control-oriented research is reflected in a section of a paper by Blunck (1929): "During recent years, people from the most diverse backgrounds have pointed with increasing emphasis to the epidemiological gaps in the science of plant protection and, in particular, an increase in our knowledge concerning the outbreak, course, and decline of disease-causing factors must be obtained."

The first comprehensive work in plant pathology to concentrate on the

specifics of botanical epidemiology was *Pflanzliche Infektionslehre* by Ernst Gäumann of Switzerland. In this 1946 book and the 1950 English translation of it (*Principles of Plant Infection*), Gäumann emphasized the uniqueness of each plant disease epidemic. He wrote: "Every epidemic develops according to its own rules, changes its character, expands and becomes malignant, decreases and becomes milder; it has an appearance of its own, its own morphology, its own *genius epidemius*."

This *genius epidemius* identifies the typical characteristics that distinguish one epidemic caused by a specific pathogen on a host from other epidemics caused by that same pathogen on the same host. As a means for determining these characteristics and differences, Gäumann (1946) enumerated the nine conditions that must occur *simultaneously* if an epidemic is to develop. These conditions for the establishment of an epidemic are as follows:

. . . On the part of the host, an abundant supply of susceptible individuals produced by (a) an accumulation of susceptible individuals. . . ; (b) heightened disease proneness of the hosts. . . ; (c) the presence of appropriate alternate hosts. . . ; on the part of the pathogen, the possession of high infective capacity, i.e., a high epidemic potential, conditioned by (d) the presence of an aggressive pathogen. . . ; (e) high reproductive capacity. . . ; (f) efficient dispersal. . . ; (g) unexacting growth requirements. . . ; on the part of environment, (h) optimal weather conditions for the development of the pathogen. . . .

These components of an epidemic provide a rational basis for examining and classifying epidemics of plant diseases. Gäumann also discussed the concept of an "infection chain" in plant disease that provides the foundation for analyzing and understanding the components of an epidemic.

With the stimulus of Gäumann's book, the development of modern computers, and a growing awareness on the part of plant pathologists of mathematics, statistics, and ecology in the 1950s, the epidemiology of plant disease as a quantitative discipline began to take shape. The "birth" of modern, botanical epidemiology occurred in 1960 with the publication of a chapter entitled "Analysis of Epidemics" by J. E. Vanderplank of South Africa in *Plant Pathology*, Vol. 3, edited by J. G. Horsfall and A. E. Dimond. In this chapter, the quantitative approach was taken and the logistic equation was first systematically applied to plant disease epidemics (but see Fracker, 1936). This volume is significant for the many chapters on plant disease as a population process and the use of mathematics to describe the process. Other chapters discussed spore dispersal (Ingold, 1960; Schrödter, 1960), inoculum potential (Garrett, 1960), and disease forecasting (Waggoner, 1960). J. C. Zadoks (1961) of The Netherlands applied the logistic equation in a study of yellow rust (caused by *Puccinia striiformis*) on wheat and introduced a graphical method to correct for variable length of the latent period. In 1961, P. H. Gregory of Great Britain published his landmark book

in aerobiology, *The Microbiology of the Atmosphere*, which would have a great impact on epidemiology, especially on studies of spore dispersal.

Other pioneering studies appeared before 1960, but these are seldom cited and apparently had little or no influence on the development of quantitative epidemiology. Gilligan (1985b) discusses some of these papers. He also outlines major advances in other fields (e.g., population dynamics) that have been of great value to epidemiologists.

The cornerstone publication of quantitative epidemiology was Vanderplank's 1963 book *Plant Diseases: Epidemics and Control*. This book provided the first comprehensive treatment of the description and quantification of plant disease epidemics. The applications of the logistic equation were expanded beyond those of the 1960 work, the exponential and monomolecular models were applied to certain types of epidemics, and a theoretical framework for epidemic analysis was provided. In 1965, P. H. Gregory, in a review of Vanderplank's book, wrote: "This book is a landmark in the history of Plant Pathology, giving us for the first time a coherent and developed theory of plant epidemiology, a notable intellectual achievement."

Although *Plant Diseases: Epidemics and Control* served as a focus for the development of botanical epidemiology, other important events and publications provided the fodder for continued development and growth of the new discipline. In Pau, France in 1963, a NATO Advanced Study Institute, "Epidemiology of Fungal Pathogens," was held as a part of the Third International Congress of Biometeorology. The institute, organized by R. D. Schein and J. M. Hirst with assistance from A. J. P. Oort and J. C. Zadoks, brought together some 40 invited participants from 14 countries and stimulated thinking and research in epidemiology (Hirst, 1964). This series of institutes and workshops continued in 1971 with a second NATO Advanced Study Institute, "Epidemiology of Plant Diseases," organized by Zadoks, Schein, Hirst, and H. D. Frinking, in Wageningen, The Netherlands. Some 74 participants from 24 countries and five continents took part in this invitational meeting. A third independent international workshop was organized in 1979 at The Pennsylvania State University, University Park, and the Plant Disease Research Laboratory, Frederick, Maryland by S. P. Penny-packer and C. H. Kingsolver. Some 63 scientists from eight countries participated and a proceedings was published in a special issue of *Protection Ecology* (Vol. 2, No. 3, 1980). The fourth international workshop was held in 1983 in Raleigh, North Carolina with 92 participants from six countries. This workshop was hosted by the Department of Plant Pathology, North Carolina State University, and coordinated by C. L. Campbell and R. I. Bruck. The fifth international workshop was held in 1986 in Jerusalem, Israel, with 85 participants from 12 countries. The workshop, hosted by the Institute of Plant Protection, at the Volcani Centre, Bet Dagan, Israel, was organized by J. Rotem and J. Palti. From their inception, these workshops have provided a forum for lively and in-depth discussions about current topics and the frontiers of botanical epidemiology. In addition, symposia,

TABLE 2.3 Recent Collective Works in Plant Disease Epidemiology

Year of Publication	Title	Editor(s)
1974	<i>Epidemics of Plant Diseases: Mathematical Analysis and Modeling</i>	J. Kranz
1977	<i>The Genetic Basis of Epidemics in Agriculture</i>	P. R. Day
1978	<i>Plant Disease: An Advanced Treatise, Vol. 2: How Disease Develops in Populations</i>	J. G. Horsfall and E. B. Cowling
1978	<i>Plant Disease Epidemiology</i>	P. R. Scott and A. Bainbridge
1980	<i>Comparative Epidemiology: A Tool for Better Disease Management</i>	J. Palti and J. Kranz
1983	<i>Plant Virus Epidemiology; The Spread of Insect-borne Viruses</i>	R. T. Plumb and J. M. Thresh
1985	<i>Advances in Plant Pathology, Vol. 3: Mathematical Modelling of Crop Disease</i>	C. A. Gilligan
1985	<i>Plant Virus Epidemiology: Monitoring, Modelling, and Predicting Outbreaks</i>	G. D. McLean, R. G. Garrett, and W. G. Ruesink
1986	<i>Plant Disease Epidemiology, Vol. 1: Population Dynamics and Management</i>	K. J. Leonard and W. E. Fry
1987	<i>Populations of Plant Pathogens: Their Dynamics and Genetics</i>	M. S. Wolfe and C. E. Caten
1988	<i>Experimental Techniques in Plant Disease Epidemiology</i>	J. Kranz and J. Rotem
1989	<i>Plant Disease Epidemiology, Vol. 2: Genetics, Resistance, and Management</i>	K. J. Leonard and W. E. Fry
1989	<i>The Spatial Components of Plant Disease Epidemics</i>	M. J. Jeger
1990	<i>Epidemics of Plant Diseases: Mathematical Analysis and Modeling, 2nd ed.</i>	J. Kranz

discussions, and demonstrations on topics in epidemiology have been included in all five of the International Congresses of Plant Pathology (London, 1968; Minneapolis, 1973; Munich, 1978; Sydney, 1983; Kyoto, 1988).

Since 1963, an increasing number of scientists have made significant contributions to plant disease epidemiology. After only about 25 yr, it may be too soon to assess fully the impact and implication of these contributions. Several specific works and publications stand out as being influential or potentially influential in the recent development of epidemiology. The work of Jan C. Zadoks and co-workers (Zadoks, 1961; Zadoks and Rijssdijk, 1972, 1973) on disease progress and spread, forecasting (disease warning), and management strategies has served as a foundation for much of the work on epidemics of foliar diseases. The series of papers and chapters by Ralph

Baker and co-workers (Baker, 1965, 1971; Baker et al., 1967) concerning the relationships between inoculum density of soilborne pathogens and disease were the first quantitative efforts involving root diseases and have stimulated much work and discussion in root disease epidemiology. The work of Jürgen Kranz (1968a-c; 1974b) established comparative epidemiology as an essential component of epidemiology, and subsequent work in his department has pushed back the frontiers of epidemiology in the areas of modeling, systems analysis, and epidemic analysis. The research of Joseph Rotem and colleagues (Rotem and Cohen, 1966; Rotem and Reichert, 1964; Rotem et al., 1971; Rotem, 1978, 1988) on the effects of climate and weather on disease has been essential to the development of this area of epidemiology. The book *Epidemics of Plant Diseases: Mathematical Analysis and Modeling*, edited by Kranz (1974a), provided a needed and logical extension of the initial proposals of Vanderplank and was the first collective volume in plant disease epidemiology after 1963. The first textbook in this discipline, *Epidemiology and Plant Disease Management*, by Zadoks and R. D. Schein (1979) is a landmark in the teaching of epidemiology. The first book dealing exclusively with the theory of plant disease epidemiology, by M. J. Jeger, was published in 1989.

The scope and maturity of modern plant disease epidemiology are indicated by the publication of a number of collective works on this subject in recent years (Table 2.3). In the quarter century since the publication of *Plant Diseases: Epidemics and Control* (Vanderplank, 1963), a new discipline of plant pathology has emerged in its own right. Plant disease epidemiology is now a vital and growing discipline that will continue to play a role in understanding plant diseases and in providing the strategies for successful management of plant diseases.

## 2.3 SUGGESTED READING

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