PLANT DISEASE EPIDEMIOLOGY

When a pathogen spreads to and affects many individuals within a population over a relatively large area and within a relatively short time, the phenomenon is called an **epidemic**. An epidemic has been defined as any increase of disease in a population. The study of epidemics and of the factors that influence them is called **epidemiology**. Plant disease epidemics, sometimes called **epiphytotics**, occur annually on most crops in many parts of the world. Most epidemics are more or less localized and cause minor to moderate losses. Some epidemics are kept in check naturally, for example, by changes in weather conditions. Others are kept in check by chemical sprays and other control measures. Occasionally, however, some epidemics appear suddenly, go out of control, and become extremely widespread or severe on a particular plant species. Some plant disease epidemics, for example, wheat rusts, southern corn leaf blight (Figure 8-1), and grape downy mildew, have caused tremendous losses of produce over rather large areas. Others, for example, chestnut blight (Figure 1-8), Dutch elm disease, and coffee rust, have threatened to eliminate certain plant species from entire continents. Still others have caused untold suffering to humans. The Irish potato famine of 1845–1846 was caused by the *Phytophthora* late blight epidemic of potato, and the Bengal famine of 1943 was caused by the *Cochliobolus* (*Helminthosporium*) brown spot epidemic of rice.

THE ELEMENTS OF AN EPIDEMIC

Plant disease epidemics develop as a result of the time combination of the same elements that result in plant dis ease: susceptible host plants, a virulent pathogen, and fa vorable environmental conditions over a fairly long pe riod of time. Humans may unwittingly help to initiat and develop epidemics through some of their activitie for example, by topping or pruning plants in wet weath er. More frequently, humans may stop the initiation an development of epidemics by using appropriate contro measures under situations in which epidemics would a most certainly occur without human intervention. Thus the chance of an epidemic increases when the susceptibi ity of the host and virulence of the pathogen are greate as the environmental conditions approach the optimum level for pathogen growth, reproduction, and spread and as the duration of all favorable combinations is pro longed or repeated.

To describe the interaction of the components of plan disease epidemics, the disease triangle, which is discussed in Chapter 2 and describes the interaction of the com ponents of plant disease, can be expanded to include time and humans. Indeed, the amount of each of the three components of plant disease and their interaction in the development of disease are affected by a fourth component: time. Both the specific point in time at which

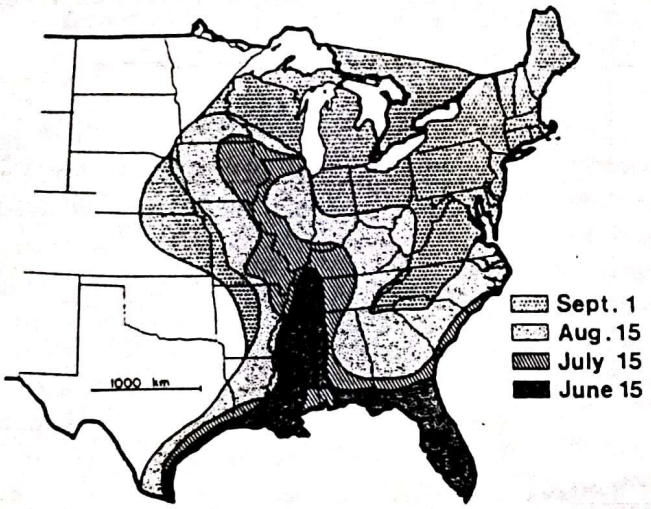

FIGURE 8-1 Development and northward spread of the southern corn leaf blight epidemic, caused by *Cochliobolus heterostrophus* (*Helminthosporium maydis*), in the United States, from June 15 to September 1, 1970. (From Zadoks and Schein, 1979).

a particular event in disease development occurs and the length of time during which the event takes place affect the amount of disease. The interaction of the four components can be visualized as a tetrahedron, or pyramid, in which each plane represents one of the components. This figure is referred to as the disease tetrahedron or disease pyramid (Figure 8-2). The effect of time on disease development becomes apparent when one considers the importance of the time of year (i.e., the climatic conditions and stage of growth when host and pathogen may coexist), the duration and frequency of favorable temperature and rains, the time of appearance of the vector, the duration of the infection cycle of a particular disease, etc. If the four components of the disease tetrahedron could be quantified, the volume of the tetrahedron would be proportional to the amount of disease on a plant or in a plant population.

Disease development in cultivated plants is also greatly influenced by a fifth component: humans. Humans affect the kind of plants grown in a given area, the degree of plant resistance, the numbers planted, time of planting, and density of the plants. By the resistance of the particular plants they cultivate, humans also determine which pathogens and pathogen races will predominate. By their cultural practices, and by the chemical and biological controls they may use, humans affect the amount of primary and secondary inoculum available to attack plants. They also modify the effect of environment on disease development by delaying or speeding up planting or harvesting, by planting in raised beds or in more widely spaced beds, by protecting plant surfaces with chemicals before rains, by regulating the humidity in produce storage areas, etc. The timing of human activities in growing and protecting plants may affect various combinations of these components to a considerable degree, thereby greatly affecting the amount of disease in individual plants and in plant populations. The human component has sometimes been used in place of the component "time" in the disease tetrahedron, but it should be considered a distinct fifth component that directly and indirectly influences the development of plant disease.

In Figure 8-3, host, pathogen, and environment are each represented by one of the sides of the triangle, time is represented as the perpendicular line arising from the center of the triangle and humans as the peak of the tetrahedron whose base is the triangle and height is the length of time. In this way, humans interact with and influence each of the other four components of an epidemic and thereby increase or decrease the magnitude of the epidemic. Sometimes, of course, humans themselves can be affected to a greater or lesser extent by plant disease epidemics.

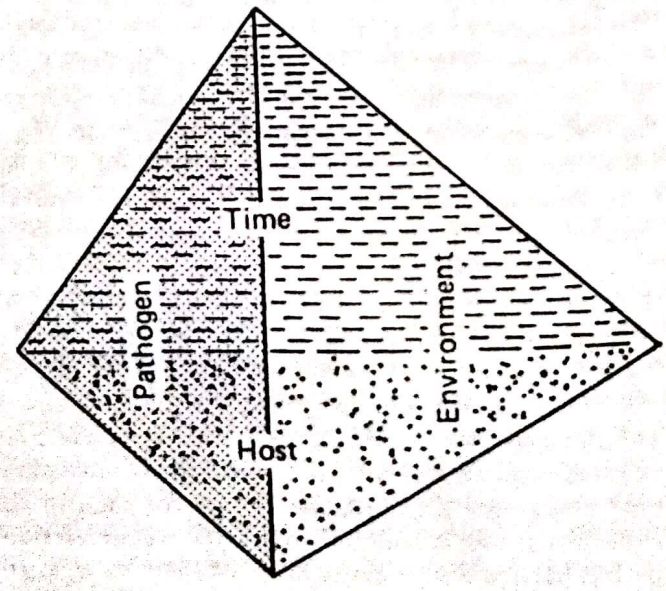

FIGURE 8-2 The disease tetrahedron.

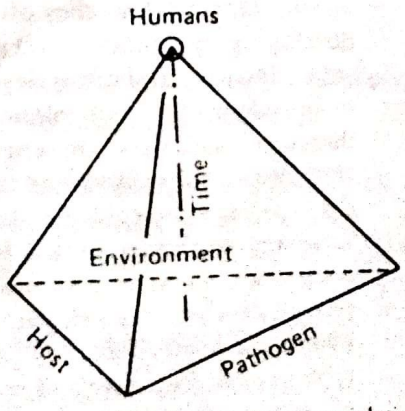

FIGURE 8-3 Schematic diagram of the interrelationships of the factors involved in plant disease epidemics.

Host Factors That Affect the Development of Epidemics

Several internal and external factors of particular host plants play an important role in the development of epidemics involving those hosts.

Levels of Genetic Resistance or Susceptibility of the Host

Obviously, host plants carrying vertical resistance do not allow a pathogen to become established in them, and thus no epidemic can develop (Figure 8-4). Host plants carrying horizontal resistance will probably become infected, but the rate at which the disease and the epidemic will develop depends on the level of resistance and the environmental conditions. Susceptible host plants lacking genes for resistance against the pathogen provide the ideal substrate for establishment and development of new infections. Therefore, in the presence of a virulent pathogen and favorable environment, susceptible hosts favor the development of disease epidemics.

Degree of Genetic Uniformity of Host Plants

When genetically uniform host plants, particularly with regard to the genes associated with disease resistance, are grown over large areas, a greater likelihood exists that a new pathogen race will appear that can attack their genome and result in an epidemic. This phenomenon has been observed repeatedly, for example, in the *Cochliobolus* (*Helminthosporium*) blight on Victoria oats and in southern corn leaf blight (Figure 8-1) on corn carrying Texas male-sterile cytoplasm. For similar reasons of genetic uniformity, the highest rates of epidemic development generally occur in vegetatively propagated crops, intermediate rates in self-pollinated crops, and the lowest rates in cross-pollinated crops. This explains why most epidemics develop rather slowly in natural populations, where plants of varying genetic makeup are intermingled.

Type of Crop

In diseases of annual crops, such as corn, vegetables, rice, and cotton, and in foliar, blossom, or fruit diseases of trees and vines, epidemics generally develop much more rapidly (usually in a few weeks) than they do in diseases of branches and stems of perennial woody crops such as fruit and forest trees. Some epidemics of fruit and forest trees, for example, tristeza in citrus, pear decline, Dutch elm disease, and chestnut blight, take years to develop.

Age of Host Plants

Plants change in their susceptibility to disease with age. In some plant–pathogen combinations, for example, *Pythium* damping-off and root rots, downy mildews, peach leaf curl, systemic smuts, rusts, bacterial blights, and viral infections, the hosts (or their parts) are susceptible only during the growth period and become resistant during the adult period (adult resistance) (Figure 8-5, Ia,b). With several diseases, such as rusts and viral infections, plant parts are actually quite resistant to infection while still very young, become more susceptible later in their growth, and then become resistant again before they are fully expanded (Figures 8-5, pattern Ib, and 8-6). In other diseases, such as infections of blossoms or fruit by *Botrytis*, *Penicillium*, *Monilinia*, and

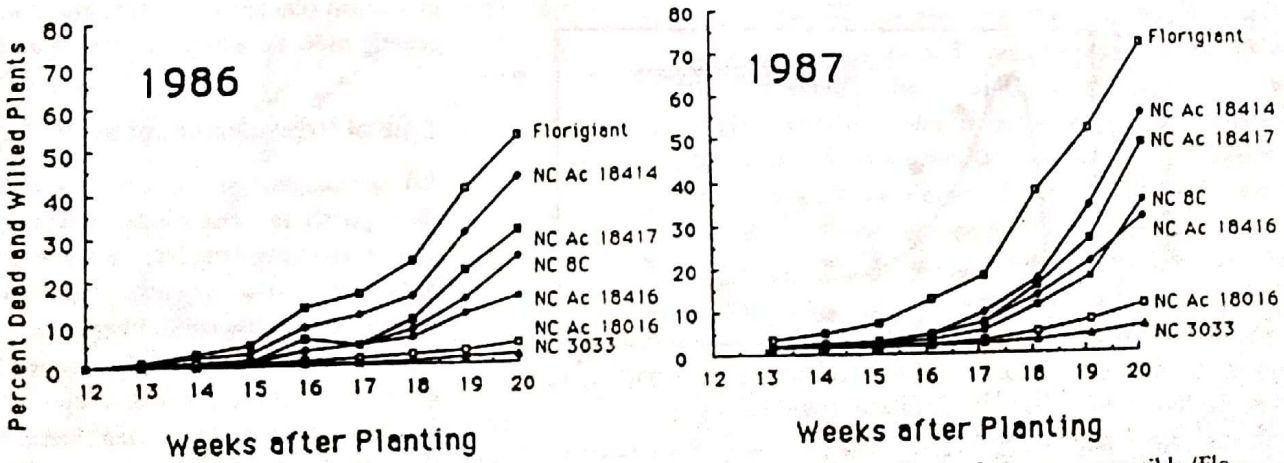

FIGURE 8-4 Development of *Cylindrocladium* black rot, caused by the fungus *C. crotalariae*, on susceptible (Florigiant), resistant (NC3033), and intermediate peanut varieties. The various genotypes maintain their resistance rankings in both years (1986, 1987) and at all inoculum density levels tested. [From Culbreath, Beute, and Campbell (1991), *Phytopathology* 81, 144–150.]

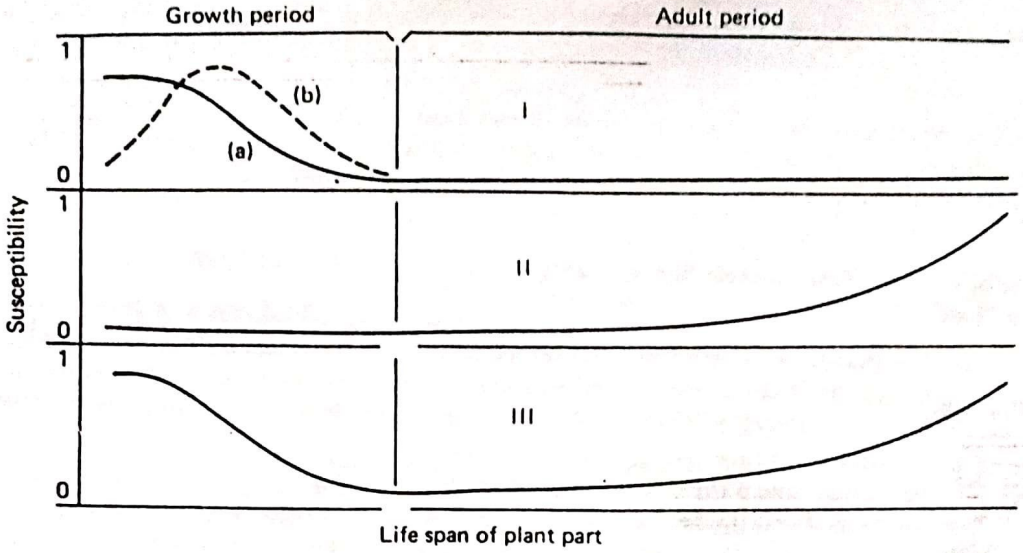

FIGURE 8-5 Change of susceptibility of plant parts with age. In pattern I, plants are susceptible only in the stages of maximum growth (Ia) or in the earliest stages of growth (Ib). In pattern II, plants are susceptible only after they reach maturity, and susceptibility increases with senescence. In pattern III, plants are susceptible while very young and again after they reach maturity. [After Populer (1978), *In* "Plant Disease" (J. G. Horsfall and E. B. Cowling, eds.), Academic Press, New York.]

Glomerella, and in all postharvest infections, plant parts are resistant during growth and the early adult period but become susceptible near ripening (Figure 8-5, II). In still other diseases, such as potato late blight (caused by *Phytopthora infestans*) and tomato early blight (caused by *Alternaria solani*), a stage of juvenile susceptibility during the growth period of the plant is followed by a period of relative resistance in the early adult stage and then susceptibility after maturity (Figure 8-5, III).

Apparently then, depending on the particular plant–pathogen combination, the age of the host plant at the time of arrival of the pathogen may affect considerably the development of infection and of an epidemic.

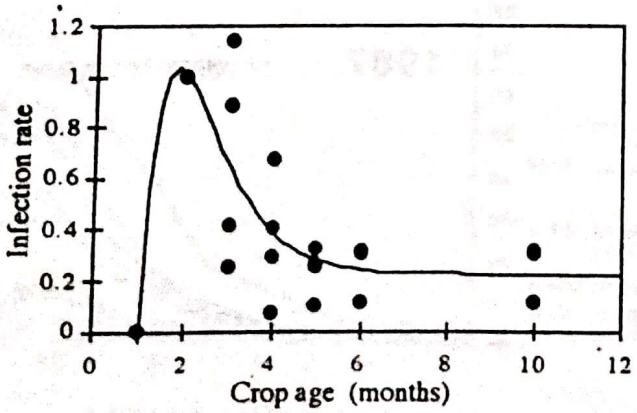

FIGURE 8-6 Effect of crop age on rate of infection. Cassava plantings of different ages exposed to the whitefly-transmitted African cassava mosaic geminivirus show increased resistance to infection as they age. [From Fargette and Vie (1994), *Phytopathology* 84, 378–382.]

Pathogen Factors That Affect Development of Epidemics

Levels of Virulence

Virulent pathogens capable of rapidly infecting the host ensure faster production of larger amounts of inoculum, and, thereby, disease, than pathogens of lesser virulence.

Quantity of Inoculum near Hosts

The greater the number of pathogen propagules (bacteria, fungal spores and sclerotia, nematode eggs, virus-infected plants, etc.) within or near fields of host plants, the more inoculum reaches the hosts and at an earlier time, thereby greatly increasing the chances of an epidemic (Figure 8-7).

Type of Reproduction of the Pathogen

All pathogens produce many offspring, but some of them, such as most fungi, bacteria, and viruses, produce a great many more offspring than others. A few fungi, all nematodes, and parasitic plants produce relatively small numbers of offspring. Even more important is the fact that some pathogens (most fungi, bacteria, and viruses) have short reproduction cycles and therefore can produce many generations in a single growing season. These are the polycyclic pathogens that usually cause rusts, mildews, and leaf spots, and they are responsible for most of the sudden, catastrophic plant-disease epidemics

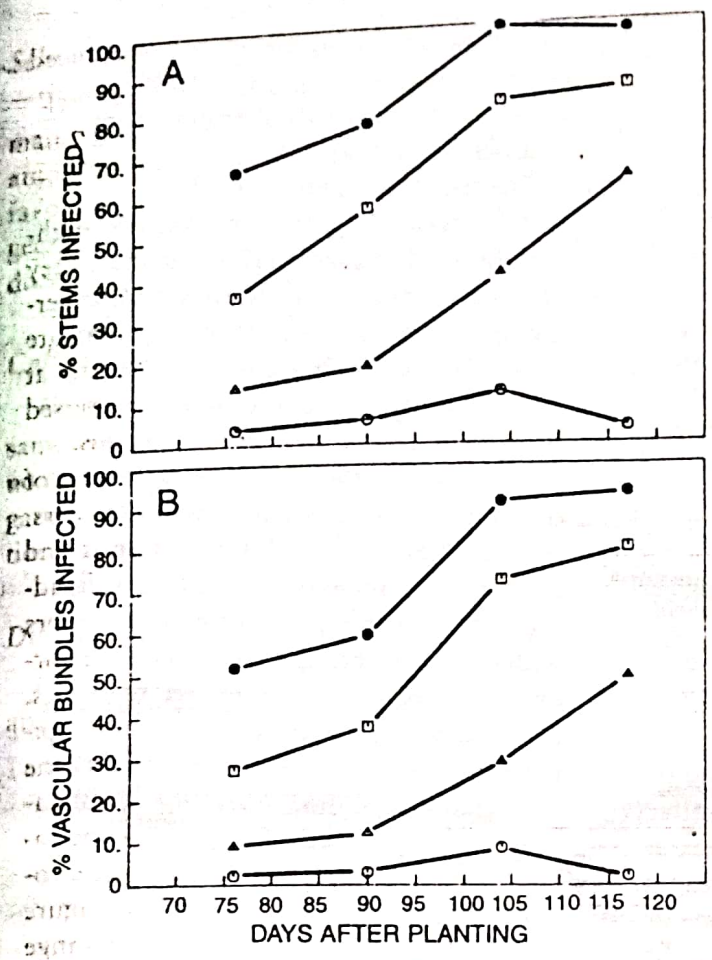

FIGURE 8-7 Effect of amount of soil inoculum of *Verticillium dahliae* on the amount of vascular wilt on potato plants at various dates after planting. Disease is expressed as a percentage of stems (A) and of main vascular bundles (B) infected at the base of the plants. ○, No pathogen detected; ▲, 1–5 propagules per gram (ppg); □, 6–10 ppg; and ●, more than 10 ppg. [From Nicot and Rouse (1987), *Phytopathology* 77, 1346–1355.]

in the world. Some soil fungi, such as *Fusarium* and *Verticillium*, and most nematodes usually have one to a few (up to four) reproductive cycles per growing season. For these latter pathogens, the number of offspring and, especially, the conditions of dispersal limit their potential to cause sudden and widespread epidemics in a single season. Nevertheless, they often cause localized, slower developing epidemics. Several pathogens, such as the smuts and several short-cycle rusts, require an entire year to complete a life cycle (monocyclic pathogens) and can therefore cause only one series of infections per year. In such diseases, the inoculum builds up from one year to the next, and the epidemic develops over several years. Similarly, epidemics caused by pathogens that require more than a year to complete a reproductive cycle are slow to develop. Examples are cedar-apple rust (2 years), white pine blister rust (3–6 years), and dwarf mistletoe (5–6 years). As a result of overlapping of the polyetic generations, however, even such pathogens produce more inoculum and cause a series of infections each year th lead to long-term epidemics.

Ecology of the Pathogen

Some pathogens, such as most fungi and all paras higher plants, produce their inoculum (spores and see respectively) on the surface of the aerial parts of the ho From there, spores and seeds can be dispersed with ea over a range of distances and can cause widespread ep demics. Other pathogens, such as vascular fungi and ba teria, mollicutes, viruses, and protozoa, reproduce insi the plant. In this case, spread of the pathogen is rare impossible without the help of vectors. Therefore, suc pathogens can cause epidemics only when vectors a plentiful and active. Still other pathogens, such as so borne fungi, bacteria, and nematodes, produce their i oculum on infected plant parts in the soil, within whi the inoculum disperses slowly and presents little dang for sudden or widespread epidemics.

Mode of Spread of the Pathogen

The spores of many plant pathogenic fungi, such as tho causing rusts, mildews, and leaf spots, are released in the air and can be dispersed by air breezes or stron winds over distances varying from a few centimeters u to several kilometers. These kinds of fungi are respons ble for the most frequent and most widespread ep demics. In terms of their ability to cause sudden an widespread epidemics, the next most important group pathogens includes those whose inoculum is carried b airborne vectors. Many of the viruses are transmitted b aphids, whiteflies, and some other insects. Mollicut and fastidious bacteria are transmitted by leafhopper planthoppers, or psyllids. Some fungi (such as the cau of Dutch elm disease), bacteria (such as the cause of ba terial wilt of cucurbits), and even nematodes (such as t cause of pine wilt disease) are disseminated primarily b beetles. Pathogens that are transmitted by wind-blow rain (primarily fungi causing diseases like anthracnos and apple scab, and most bacteria) are almost annual responsible for severe but somewhat localized epidemi within a field, a country, or a valley. Pathogens carri with the seed or other vegetative propagative orga (such as tubers or bulbs) are often placed in the midst susceptible plants, but their ability to cause epidemics de pends on the effectiveness of their subsequent transm sion to new plants. Finally, pathogens present in s spreading through the soil, because of the physical re strictions, are generally unable to cause sudden or wi spread epidemics but often cause local, slow-spread diseases of considerable severity.

Environmental Factors That Affect Development of Epidemics

The majority of plant diseases occur in most areas where the host is grown but, usually, do not develop into severe and widespread epidemics. The concurrent presence in the same areas of susceptible plants and virulent pathogens does not always guarantee numerous infections, much less the development of an epidemic. This fact dramatizes the controlling influence of the environment on the development of epidemics. The environment may affect the availability, growth stage, succulence, and genetic susceptibility of the host. It may also affect the survival, vigor, rate of multiplication, sporulation, and ease, direction, and distance of dispersal of the pathogen, and the rate of spore germination and penetration. In addition, the environment may affect the number and activity of the vectors of the pathogen. The most important environmental factors that affect the development of plant disease epidemics are moisture, temperature, and the activities of humans in terms of cultural practices and control measures.

Moisture

As discussed in Chapter 7, abundant, prolonged, or repeated high moisture, whether in the form of rain, dew, or high humidity, is the dominant factor in the development of most epidemics of diseases caused by fungi (blights, downy mildews, leaf spots, rusts, and anthracnoses), bacteria (leaf spots, blights, soft rots), and nematodes. Moisture not only promotes new succulent and susceptible growth in the host, but, more importantly, it increases sporulation of fungi (Figures 7-6A and 7-8) and multiplication of bacteria. Moisture facilitates spore release by many fungi (Figures 7-7 and 7-9) and the oozing of bacteria to the host surface, and it enables spores to germinate and zoospores, bacteria, and nematodes to move. The presence of high levels of moisture allows all these events to take place constantly and repeatedly and leads to epidemics. In contrast, the absence of moisture for even a few days prevents all of these events from taking place, so that epidemics are interrupted or completely stopped. Some diseases caused by soilborne pathogens, such as *Fusarium* and *Streptomyces*, are more severe in dry than in wet weather, but such diseases seldom develop into important epidemics. Epidemics caused by viruses and mollicutes are only indirectly affected by moisture, primarily by the effect that higher moisture has on the activity of the vector. Moisture may increase the activity of some vectors, as happens with the fungal and nematode vectors of some viruses, or it may reduce the activity of the vectors, as happens with the aphid, leafhopper, and other insect vectors of some viruses and mollicutes. The activity of these vectors is drastically reduced in rainy weather.

Temperature

Epidemics are sometimes favored by temperatures higher or lower than the optimum for the plant because they reduce the plant's level of horizontal resistance. At certain levels, temperatures may even reduce or eliminate the vertical resistance of host plants. Plants growing at such temperatures become "stressed" and predisposed to disease, provided the pathogen remains vigorous.

Low temperature reduces the amount of inoculum of fungi, bacteria, and nematodes that survives cold winters. High temperature reduces the inoculum of viruses and mollicutes that survives hot summer temperatures. In addition, low temperatures reduce the number of vectors that survive the winter. Low temperatures occurring during the growing season can reduce the activity of vectors.

The most common effect of temperature on epidemics, however, is its effect on the pathogen during the different stages of pathogenesis, that is, spore germination (Figures 7-8 and 7-9) or egg hatching, host penetration, pathogen growth (Figures 7-3 and 7-4) or reproduction, invasion of the host, and sporulation (Figure 7-5). When temperature stays within a favorable range for each of these stages, a polycyclic pathogen can complete its infection cycle within a very short time (usually in a few days). As a result, polycyclic pathogens can produce many infection cycles within a growing season. Since, with each infection cycle, the amount of inoculum is multiplied manyfold (perhaps 100 times or more), and since some of the new inoculum is likely to spread to new plants, more infection cycles result in more plants becoming infected by more and more pathogens, thus leading to the development of a severe epidemic.

In reality, moisture and temperature must be favorable and act together in the initiation and development of the vast majority of plant diseases and plant disease epidemics.

Effect of Human Cultural Practices and Control Measures

Many activities of humans have a direct or indirect effect on plant disease epidemics, some of them favoring and some reducing the frequency and the rate of epidemics.

Site Selection and Preparation

Low-lying and poorly drained and aerated fields, especially if near other infected fields, tend to favor the appearance and development of epidemics.

Selection of Propagative Material

The use of seed, nursery stock, and other propagative material that carries various pathogens increases the amount of initial inoculum within the crop and greatly favors the development of epidemics. The use of pathogen-free or treated propagative material can greatly reduce the chance of epidemics.

Cultural Practices

Continuous monoculture, large acreages planted to the same variety of crop, high levels of nitrogen fertilization, no-till culture, dense plantings (Figure 8-8), overhead irrigation, injury by herbicide application, and poor sanitation all increase the possibility and severity of epidemics.

Disease Control Measures

Chemical sprays, cultural practices (such as sanitation and crop rotation), biological controls (such as using resistant varieties), and other control measures reduce or eliminate the possibility of an epidemic. Sometimes, however, certain controls, for example, the use of a certain chemical or planting of a certain variety, may lead to selection of virulent strains of the pathogen that either are resistant to the chemical or can attack the resistance of the variety and thus lead to epidemics.

Introduction of New Pathogens

The ease and frequency of worldwide travel have also increased the movement of seeds, tubers, nursery stock, and other agricultural goods. These events increase the possibility of introducing pathogens into areas where the hosts have not had a chance to evolve resistance to these pathogens. Such pathogens frequently lead to severe epidemics. Examples are chestnut blight, Dutch elm disease, and citrus canker caused by the bacterium *Xanthomonas campestris* pv. *citri*.

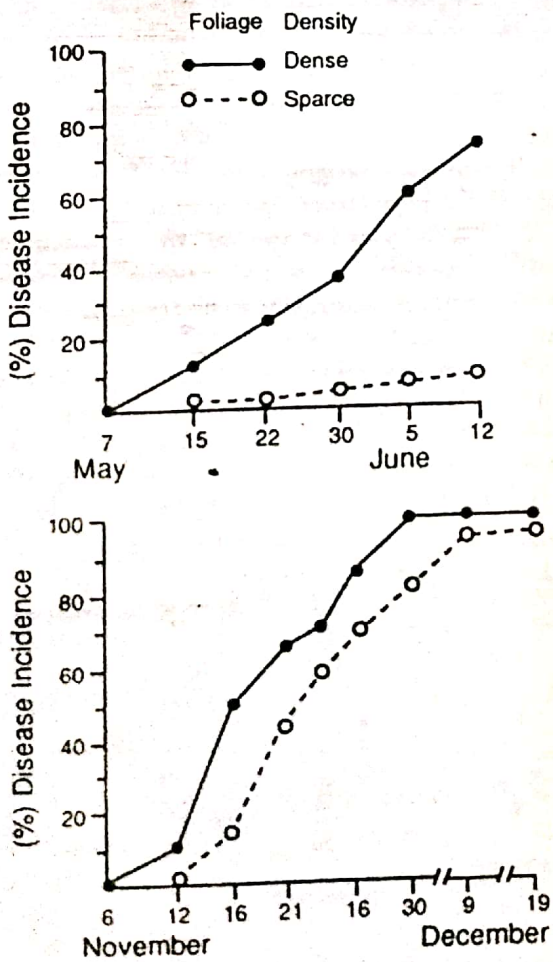

FIGURE 8-8 Effect of foliage density (● = dense, ○ = sparse) on development of *Phytophthora infestans* during a period of partly favorable weather (May–June) and of very favorable weather (November–December). [From Rotem and Ben-Joseph (1970), *Plant Dis. Rep.* 54, 768–771.]