Epidemiology and Geophytopathology of Selected Seed-borne Diseases

Marlene Diekmann

International Center for Agricultural Research in the Dry Areas (ICARDA)

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Contents

Foreword

This study in ICARDA's Seed Production series deals with seed-borne pathogens and their geographical distribution. It is widely acknowledged that plant diseases are one of the limiting factors in seed production, and particular problems are caused by pathogens which are transmitted from one generation of seed to the next or from one area to another by the seed itself. While some diseases occur wherever the host plant is grown, others are limited in their distribution. Among the reasons for the limited distribution of pests and pathogens, climatic conditions are most important. This study therefore looks at the relation between the distribution of selected seed-borne pathogens and climatic conditions.

Seeds are an excellent vehicle for the pathogens' spread. They may be shipped over great distances, and they remain viable for extended periods of time. Quarantine measures are set up by many countries to limit the risk of introducing new pests and pathogens to areas where they do not occur.

In seed production there are various ways to manage seed-borne diseases. Seed certification schemes prescribe standards for field inspection and laboratory testing, and pesticides may be applied to directly control the pathogens either in the crop or as seed treatment. Another method, which can be more cost-effective, is the selection of seed production sites or seasons with climatic conditions unfavorable for disease development. For example, in chickpea seed production, this is practiced by farmers in the Middle East who plant chickpeas in spring to avoid the winter rains that result in a high risk of *Ascochyta* blight. This study indicates that it is possible to calculate a risk index for any location and any chosen planting time if long-term average data for weather data are available. Examples are given to illustrate how, with the help of statistical models, information with relevance for practical seed production can be gained.

We hope that this study will encourage seed producers and plant pathologists to look further into possibilities for managing seed-borne pathogens in seed production, by utilizing conventional and new methodologies.

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Summary

Seed-borne pathogens may reduce considerably the yield of agricultural crops, particularly cereals and legumes. Infected seeds also serve frequently as a vehicle for the dissemination of pathogens to new areas. The epidemiology of seed-borne pathogens is complex and influenced by weather factors and by the relation between host and pathogen in the different stages of development. Of particular importance are the amount of initial inoculum, the rate of seed transmission (seed to plant), the rate of subsequent disease development in the field and the rate of re-establishment of seed-borne inoculum (plant to seed).

A major practical application of plant disease epidemiology is the disease forecast. To date, little work has been done in the forecasting of seed-borne diseases.

Geophytopathology has two aspects: a descriptive part, which entails mapping of plant diseases, and an analytic part which results in an assessment of the probability of a disease occurring under given climatic conditions. Climate data rather than weather data are used for this appraisal.

In this study the conversion of a weather-based forecast model for the occurrence of yellow rust on winter wheat to a climate-based geophytopathological projection is presented. This approach, however, seems to be more pertinent for seed-borne diseases, because a spread of the inoculum with infected/contaminated seeds is likely. Models were developed for three seed-borne diseases by using stepwise discriminant analysis. For bacterial leaf blight of rice, caused by *Xanthomonas campestris* pv. *oryzae*, mean daily maximum temperature in month 1 of the vegetation (x_1), mean daily minimum temperature in month 2 of the vegetation (x_2), and mean precipitation in month 3 of the vegetation (x_3) were used to calculate a disease index, which can be used for the classification of locations according to the formula

$$y = -14.16 - 0.29\,x_1 + 1.19\,x_2 - 0.01\,x_3.$$

A positive disease index indicates a high disease risk under the given climatic conditions, a negative index signifies a low disease risk, and an index around zero implies a risk for susceptible varieties or certain years with favorable weather only. For Ascochyta blight of chickpea, caused by *Ascochyta rabiei*, the discriminating climatic parameters were mean daily temperature in month 1 of the vegetation (x_1), mean precipitation in month 2 of the vegetation (x_2), average precipitation per rainy day in month 1 of the vegetation (x_3), average precipitation per rainy day in month 2 of the vegetation (x_4), mean number of days with precipitation in month 1 of the vegetation (x_5), and mean number of days with precipitation in month 2 of the vegetation (x_6). The discriminant function (y) was calculated

$$y = -1.11 - 0.22\,x_1 - 0.05\,x_2 - 0.32\,x_3 + 0.91\,x_4 - 0.51\,x_5 + 1.15\,x_6.$$

Locations with and without occurrence of Karnal bunt of wheat could be classified based on the parameters: difference between mean daily maximum and mean daily minimum temperature in the month of planting (x_1), the mean daily maximum temperature in the month of flowering (x_2), and the mean daily minimum temperature in the coldest month of the year (x_3). The discriminant function (y) was found to be

$$y = -16.98 + 2.11\, x_1 - 0.52\, x_2 + 0.28\, x_3.$$

For the three diseases the disease risk for other locations could be estimated based on the above formulas. This approach may be useful in disease risk assessment in seed production areas or quarantine.

Chapter 1. Introduction

About 90% of the world food crops are propagated through seed (Franke 1976). Cereal grains, for example, contribute about 50% of the per capita energy intake at world level and exceed 65% in the Near and Far East (Duffus and Slaughter 1980). Seed of high quality is recognized as an important input in agricultural production. One of the important aspects of seed quality — besides germination and purity — is the freedom from seed-borne pathogens. We have to distinguish between pathogens directly attacking seeds and those that use the seeds only as a vehicle for transmission. In the former group are fungi and bacteria which may digest the seed material, generate off-flavors, cause heating or produce toxins (Duffus and Slaughter 1980). Examples of such pathogens are species of *Aspergillus*, *Penicillium* and *Fusarium*. A number of pathogens, including viruses and viroids, bacteria, fungi, nematodes and parasitic weeds, may be transmitted through seeds. Richardson (1990) listed more than 1200 pathogens attacking over 350 crop species. About 80% of them are fungi. There seems to be hardly any crop which is not attacked by one or more seed-borne pathogens.

Yield losses due to seed-borne diseases are difficult to quantify, particularly since many of the pathogens are not exclusively transmitted by seed, but also by wind, soil, plant debris and other means. Chiarappa and Gambogi (1986) summarized impressive examples, such as the Philippine downy mildew of maize (*Peronosclerospora philippinensis*) or the southern leaf blight of maize (*Drechslera maydis*), which may cause total yield losses. Apart from direct yield losses due to seed-borne diseases, there are reports on effects of pathogens on seed viability, which were summarized by Agarwal and Sinclair (1987).

Dissemination with seeds can be important for the spread of pathogens to new areas. Many pathogens survive in seeds for many years, often as long as the seeds remain viable, as summarized by Agarwal and Sinclair (1987). Storage conditions that maintain seed viability (low temperature and moisture) generally also conserve the pathogen (Neergaard 1977). This is of particular concern for genebanks, which inadvertently may assemble a world collection of pathogens and their various strains. Increased international seed exchange, either in the form of germplasm or as commercial seed, entails the risk of increased pathogen spread. Table 1 gives an impression on the magnitude of seed exchange handled by only one of the International Agricultural Research Centers.

The concept of geophytopathology, which links plant pathology with geography, can help in the planning of plant disease management, such as quarantine, or the forecasting of diseases. Research on this topic has been started by Reichert and Palti (1966). The forecasting aspect was stressed by Weltzien (1967) and his cooperators. In this study a practical approach to the geophytopathology of seed-borne diseases is attempted.

Table 1. International movement of germplasm (wheat, barley, lentils, chickpeas and faba beans) at the International Center for Agricultural Research in the Dry Areas (ICARDA)†.

	No. of shipments‡		No. of countries	
Year	Received	Sent	Received	Sent
1985/86	96	446	31	68
1986/87	99	542	35	69
1987/88	77	421	39	69
1988/89	96	525	38	72
1989/90	111	458	40	73

Source: ICARDA Genetic Resources Unit: Program Reports 1986-1990.
† From ICARDA Genetic Resources Unit, 1986 to 1990.
‡ Shipments consist of different number of lines varying from 1 to 8,400.

Chapter 2. Epidemiology of Seed-borne Diseases

General Considerations

Devastating plant diseases and pests have been known since biblical times, when they were regarded mostly as God's punishment, e.g.. "The Lord will strike you...with scorching heat and drought, with blight and mildew, which will plague you until you perish" (Deuteronomy 28:22), or "When famine or plague comes to the land, or blight or mildew, locusts or grasshoppers...then hear from heaven, your dwelling place" (Kings 8:37), or "Many times I struck your gardens and vineyards, I struck them with blight and mildew. Locusts devoured your fig and olive trees, yet you have not returned to me, declares the Lord" (Amos 4:9).

Although in the 19th century disastrous epidemics struck agricultural production, plant disease epidemiology as a science did not start until van der Plank (in subsequent publications Vanderplank), published his well-known book on "Plant Diseases: Epidemics and Control" in 1963. He defined epidemiology as "the science of disease in populations."

Occasionally, the words 'epiphytotic' or 'epiphytology' are used instead. The question of whether to use epidemic or epiphytotic has been discussed in letters to the editor of scientific journals (Phytopathology (1978) 68:681-683 and Plant Disease (1981) 65:459, 704). The Encyclopaedia Britannica (1973) defined epidemiology as: "Although originally limited to disease in human populations, epidemiology has been applied to the study of disease in animal herds and in plant life. Such usage is justified by the derivation of the word epidemic, which literally translated from the Greek means 'upon the population'. The population may consist of human beings, animals, plants or whatnot." According to Kykkotes' dictionary (1947) the word epidemia means "stay in a place." Barnhart (1988) translated the Greek word "demos" in the sense of "people" or "district." Byrne (1987) interpreted epidemic as "visitation of a disease upon a people or district." Moreover, the word 'epiphytology' is related to the word epiphyte, which describes a plant growing on another plant, usually not as a parasite. Both the origin and usage in international journals, such as Phytopathology or the Annual Review of Phytopathology, offer ample justification for the use of epidemiology rather than epiphytology in this paper.

"Famous" epidemics are those of potato late blight (*Phytophthora infestans*) in Ireland from 1845 to 1849 (Zadoks and Schein 1979), the helminthosporium disease of rice (*Helminthosporium oryzae*) which caused "the great Bengal famine" in 1942/43 (Padmanabhan 1973), the Dutch elm disease (*Ceratocystis ulmi*) in the USA in the 1930s (Klinkowski 1970) and in England in the early 1970s (Brasier and Gibbs 1978; Gibbs 1978), and the southern corn leaf blight (*Helminthosporium maydis*) in the USA in 1970/71 (Ullstrup 1972).

Some scientists hold the opinion that plant disease epidemics are man-made, and that without human interference plants would "exist in a dynamic balance in which they have grown accustomed to each other" (Cowling and Horsfall 1978). Most of the epidemics

cited above would indeed not have happened without human interference, and there are many more examples such as planting 80% of an area with a susceptible variety, which resulted in a wheat leaf rust (*Puccinia recondita*) epidemic in Mexico in 1976/77 (Dubin and Torres 1981). However, epidemics also occur on wild species, as reported from Israel (Dinoor 1974), and in natural forest ecosystems (Schmidt 1978).

Zadoks and Schein (1979) distinguished descriptive, quantitative and comparative epidemiology. All entail the collection of vast amounts of data, particularly those on weather, on disease incidence and severity, or on yield loss. Factors affecting development of an epidemic are: pathogens' life cycles, weather, host plant resistance, availability of susceptible host plants, availability of vectors and management practices including control measures. Dinoor (1974) presented examples for pathogens present in an area before an epidemic developed, which was induced by intensification of agriculture, abandoning crop rotation, overhead irrigation, or cropping under cover. Some of the most devastating epidemics, however, resulted from the introduction of new pathogens, e.g., grape downy mildew (*Plasmopara viticola*) in France in 1878, potato late blight (*P. infestans*) in Ireland in 1842, and, to name a more recent example, tobacco blue mold (*Peronospora tabacina*) in Europe in 1957 (Zadoks 1967). Also the introduction of a new pathogen strain may be responsible for the development of an epidemic. The Dutch elm disease (*C. ulmi*) was well established in England since about 1930, when it had killed about 10-20% of the tree population. In 1970, the period of stabilization was disturbed by an aggressive strain, apparently introduced from North America (Brasier and Gibbs 1978).

Practical applications of plant disease epidemiology include various aspects of disease management, such as sanitation, resistance breeding, crop loss assessment, forecast systems and geophytopathology. Often mathematical models are used as a tool in plant disease epidemiology. Kranz and Royle (1978) published a critical perspective of modelling in epidemiology.

Particular Points on Seed-borne Diseases

The epidemiology of seed-borne diseases follows the same principles as that of other plant diseases. Seed-borne diseases, however, have some additional characteristics in common. Neergaard (1977) listed the following factors as important:

- amount of seed-borne inoculum
- rate of seed transmission (seed to plant)
- rate of subsequent disease development in the field
- rate of re-establishment of seed-borne inoculum (plant to seed).

The importance of any of these factors is largely determined by pathogens, environmental conditions and ultimate use of the crop.

Amount of inoculum

The amount of seed-borne inoculum in a seed sample can either be determined as

percentage of infected seeds, or quantitatively with regard to the pathogen propagules in the seeds. In seed health testing, usually the percentage of infected seeds is established; only in the case of the washing test is the number of spores per gram of seed or per grain counted. Occasionally, seed lots are requested to be free from a certain pathogen, e.g., when zero tolerance is required for quarantine pathogens. This may be desirable in certain cases; however, such statements are unrealistic. All that can be reasonably testified is that the level of infection or infestation is less than a specified percentage. The sensitivity of the method used in seed health testing and the sample size are very important factors. Geng *et al.* (1983) elaborated on the necessary sample size for testing a seed sample directly or indirectly by bioassays. They established that a sample size of 48 000 seeds is required to be 99% sure that a seed lot will be rejected if its infestation rate is greater than an established standard of 0.1%, and have a 95% confidence that a seed lot will not be rejected if its disease rate is less than 0.05%. For faba beans this comes to a sample of approximately 48 kg. If bioassays are used, the probability of detecting one infected seed in a sample of x seeds is important. At a sensitivity of 80%, nine assays of sample units containing 347 seeds are required to be 99% confident of detecting the pathogen if the disease rate is 2% (Geng *et al.* 1983).

Although the importance of quantifying the inoculum per seed and its relation to disease expression has been recognized (Colhoun 1983), there are still considerable difficulties for many pathogens. Few problems are encountered with pathogens that are exclusively surface-borne, such as *Tilletia* spp. on wheat. The spores can be washed off the seeds and counted. A similar method can be employed for bacteria, such as *Xanthomonas campestris*, where internally seed-borne bacteria ooze into sterile saline, which then can be plated on a selective medium in dilutions so that colonies can be counted after incubation (Schaad 1982). Seeds infected with *Ustilago* spp. may be seen as "a simple unit of inoculum potential" (Neergaard 1977). With other pathogens, e.g., *Fusarium* spp. and *Ascochyta* spp., the percentage of infected seeds as a unit of measurement does not do justice to the complicated host-pathogen relationship. A differentiation between superficial and deep-seated inoculum can be made by sterilizing the seed surface before the test.

Little is known about the importance of the surface-borne inoculum in disease epidemiology. Singh *et al.* (1974) studied the relation between seed infection and disease incidence of the emerging plants for *D. maydis* on maize and found a good correlation if the seed infection was recorded in the freezing blotter test with 10 minutes pretreatment with 2% sodium hypochlorite. In some samples a very high incidence of seed infection was found in tests without pretreatment, indicating the presence of surface-borne inoculum, which apparently did not play a role in disease development.

Generally, in tests without surface sterilization of the seeds a higher percentage of pathogens is recorded (Diekmann and Asaad 1989). Such considerations are particularly important when it comes to the establishment of tolerances in quarantine regulations.

While in tests that require incubation only viable inoculum is considered, in washing tests it is difficult to differentiate between spores that are able to germinate and those that are dead, e.g., killed by seed treatment. A spore germination test on water agar could

provide such information; however, such test may be time consuming and particularly difficult if the spore load is small.

Most detection methods in virology — latex agglutination and passive hemagglutination assays, enzyme-linked immunosorbent assay (ELISA), radioimmunoassay (RIA) and immunoelectron microscopy (IEM) — depend on the detection of virus antigens (coat protein), whereas various "blot tests" permit the detection of specific viral nucleic acids by the use of isotope-labelled complementary DNA. With immunosorbent assays, the detection of 1-2% seed infection is possible. A clear disadvantage is the fact that there is no discrimination between infective and noninfective virus particles. Furthermore, it is important to distinguish between viruses located in the embryo, which may cause seedling infection, and those in or on the seed coat, which most likely will not infect the seedling. Thus, serological tests should be conducted on germinated seedlings rather than on seeds (Hamilton 1983).

How important the initial inoculum is depends on the maximum infection rate. Hewett (1978) gave some examples: 0.07/day for *Ascochyta fabae*, 0.11/day for *Pseudomonas phaseolicola*, 0.44/day for *Septoria apiicola*. Obviously, in the latter case a small amount of initial inoculum may lead more easily to an epidemic than in the first two examples. For quarantine diseases, even an otherwise insignificant level of initial inoculum may have drastic effects when the conditions in the new area are favorable for disease development.

Rate of seed transmission

The seed transmission rate can be expressed as the percentage of infected seeds that result in infected seedlings or plants. It varies greatly, depending on pathogen, host and environmental factors. A simple relationship between seed infection and percentage infected plants in the field was found for loose smut of barley (*Ustilago nuda*), where the transmission rate is close to 100% (Neergaard 1977; Smith *et al.* 1988). On the other hand, faba bean seeds infected with *A. fabae* produced only 2 to 15% of seedlings with primary infection (Hewett 1973). A similarly low transmission rate of approximately 10% was reported by Taylor (1970) for *P. phaseolicola*. Taylor *et al.* (1979) found that the transmission rate depends on the severity of seed infection. Seeds showing severe symptoms of bacterial infection (wrinkled, discolored seeds with lesions) had a higher number of bacteria per seed than those with slight to moderate symptoms or those that were symptomless. The seedling infection rates were 69, 44 and 5%, respectively. However, since many of the heavily infected seeds failed to germinate, their contribution to the total disease transmission was relatively low compared with that of moderately infected and symptomless seeds. Contrary to the earlier results, which were obtained under field conditions, in this study a transmission rate of approximately 2:1 (i.e., 50%) was found.

The effect of the host was demonstrated for several viruses. The transmission rate for bean yellow mosaic virus (BYMV) is 0.1-2% in faba beans (Hamilton 1983) and 6% in lupins (Bos 1970). Pea seed-borne mosaic virus (PSbMV) is seed transmitted in pea at a rate of up to 90% (Hampton and Mink 1975), but not in faba bean (Cockbain 1983).

Monocyclic pathogens, i.e., pathogens with only one generation per year, cause "simple interest diseases" (van der Plank 1963). Examples are *Tilletia caries* or *U. nuda.* The initial or seed-borne inoculum is closely correlated with disease incidence. Sanitation either by using seed treated with an eradicative fungicide or by preventing seed infection is an effective way of disease control. Hewett (1978) found this confirmed in practice for *U. nuda.* In countries where cereal seed is regularly treated, loose smut and common bunt of wheat almost disappeared.

However, there may be significant differences between monocyclic pathogens. Hewett (1978) gave the example of *U. nuda* which, although it has an extremely high seed to plant transmission rate of approximately 100%, failed to maintain the infection level in seed lots resown for several years without control measures. In contrast, *Drechslera graminea,* with a transmission rate of less than 15%, maintained the initial level of 30-90% fairly well over several years.

The rate of transmission from seed to plant, along with the first of Neergaard's factors — the amount of seed-borne inoculum — are important in quarantine. The critical, although somewhat theoretical, question is: can a single spore on, in, or with the seed cause a disease? The answer is clearly no if the spore is not viable, but it may not be easy to verify that. The answer is also negative if seeds are planted in an area with adverse environmental conditions; it is unlikely that dwarf bunt would be established in a country like Egypt, since *Tilletia contraversa* spores require a temperature of about 5°C for several weeks (Lowther 1948). The susceptibility of the host is also important. Heald (1921) found that in a susceptible variety, 104 spores of *Tilletia tritici* per grain resulted in bunted heads, whereas a resistant variety remained healthy even with 542 spores per grain. Van der Plank (1963) proved for *Puccinia graminis tritici* that two different cases are possible: a synergic infection at higher spore concentrations with more infection points per 100 spores, and the opposite case of spores acting independently, resulting in single spore infections. This independent action of spores is the basis of many of van der Plank's equations. Van der Plank (1963) also challenged Heald's (1921) interpretation of an interaction between spores of common bunt. More evidence on the independent action of fungus spores was provided by van der Plank (1975). He argued that there is no evidence for the theory of a numerical threshold of infection as postulated by Heald (1921), Gäumann (1951) and others. All his disease-inoculum curves pass through the origin, which proves that one spore can cause an infection. The same is true for a virus infection. Although there is a dilution endpoint, "the greatest dilution of inoculum compatible with being reasonably sure of getting infection" (van der Plank 1975), which for tobacco mosaic virus is about one million particles, the actual infection is by a single particle.

Zadoks and Schein (1979) confirmed that one "dispersal unit" can establish one "infection unit", and indicated that the infection efficiency is generally lower, for example in the range of 10%.

Rate of subsequent disease development in the field

The more life cycles a pathogen completes in a season, the greater the potential for

causing an epidemic. In the case of such "compound interest diseases", as van der Plank (1963) called them (alluding to the world of money lending and interest earning), the relationship between initial inoculum and disease development in the field is complicated and affected by many factors, particularly those related to the environment. Here the amount of initial inoculum may be very small and yet cause an epidemic, because there are chances to multiply throughout the year.

Good examples are bacteria or viruses. For *X. campestris*, as little as 0.05% infected cabbage seedlings, that is 5 in 10 000, resulted in a considerable incidence of black rot (Schaad *et al.* 1980). Trigalet and Bidaud (1978) established a tolerance level of 1 in 20 000 for *P. phaseolicola*, causing halo blight of beans. Effective control of lettuce mosaic virus in California required that the seed infection had to be less than 0.003% (Baker and Smith 1966). An example for fungi was provided by Gaunt and Liew (1981), who found in New Zealand 15% diseased faba bean plants resulting from an initial inoculum of 0.2%.

Kranz (1978) stressed that, other factors being equal, seed-borne pathogens will develop at more rapid rates than other types of pathogens. The reason is that the initial inoculum, if seed-borne, is more evenly distributed throughout the field, so that the number of infections or cycles required to cover a given crop area is less than with a disease that develops from a localized primary focus. Seed-borne inoculum also makes an early start of disease development and a higher initial disease intensity more likely. Moreover, we can assume that the cultivar is susceptible to the pathogen race, because otherwise there would be no seed-borne inoculum.

In the case of pathogens with several generations per year it is, however, difficult to verify whether the disease incidence results from seed-borne inoculum that has been multiplied, or whether other sources of inoculum, such as from neighboring fields, also contributed to the disease.

Kaack (1983) compared the disease incidence in plots that had been planted with chickpea seeds infected with *A. rabiei* at a rate of 7.7% and those inoculated with infected plant debris. He found that in both cases the disease spread in the main wind direction. The infected seeds resulted in 31% infected plants, almost randomly distributed in the field. Within 6 weeks the disease incidence had reached 100%. In the plots infected with plant debris the first symptoms were observed more than 3 weeks after those resulting from infected seeds. Originating from only one focus, the final disease incidence was 16%.

Another factor determining the epidemic potential of a pathogen is whether or not there are other means of transmission. If a pathogen is exclusively transmitted by seed, such as *U. nuda*, or barley stripe mosaic virus, measures such as production of "pathogen-free" seed, or seed treatment may control the disease satisfactorily. However, additional means of transmission, such as the soil-borne inoculum of a *Tilletia* species, are generally much more difficult to control.

Rate of re-establishment of seed-borne inoculum

This point is particularly important if the harvested crop is to be used as seed. Jellis *et al.* (1985) reported for *A. fabae* a strong correlation of seed infection with both foliar and pod infection. In many cases seed infection depends largely on weather conditions, particularly rainfall. For successful infection of faba bean seed with *A. fabae* there must be rain early in the development of a crop so that the pathogen is carried sufficiently high in the leaf canopy to re-infect the pods and seeds as they develop (Hewett 1973). Lockwood *et al.* (1985) reported for the same host and pathogen that short-strawed faba bean cultivars showed a higher incidence of pod infection. Steiner and Lamprecht (1983) and Lamprecht and Steiner (1984) also found that faba bean samples from areas with consistently dry climatic conditions showed a lower infection rate of *A. fabae*. For three rice pathogens, *Trichoconis padwickii*, *Drechslera oryzae* and *Pyricularia oryzae*, Aulakh *et al.* (1974) found that seeds harvested from the relatively dry region of Ludhiana generally showed a low pathogen incidence in seeds, even if a moderate infection (in the case of *D. oryzae*) was recorded in the field. Generally, weather conditions favorable for a low seed infection do not boost the yield. This limits the advantage of dry areas in seed production.

Shortt *et al.* (1981) found the highest incidence of Phomopsis seed decay of soybean in wet years and in counties bordering major waterways. The distribution of the disease was primarily influenced by the rainfall patterns during the maturation period of the crop. Correlation between disease incidence and temperature was low.

Reinfection of loose smut of wheat and barley (*U. nuda*, *Ustilago tritici*) is greatly influenced by weather. Since spore transfer occurs only during the few weeks of flowering, the pathogen is particularly vulnerable during this relatively short period of time. If moisture conditions are favorable, the rate of increase could be 10 to 20 times (Hewett 1978). For this monocyclic disease, isolation of a crop grown from clean seed from sources of inoculum is a valuable control measure. Hewett (1978) found that the amount of reinfection in a plot was largely determined by the level of inoculum in this plot. Isolation in time also may be effective.

Cultural practices also may affect seed infection. Sprinkler irrigation, for example, is to be avoided where cereal seed production may be affected by the bacterial stripe disease caused by *Xanthomonas campestris* pv. *translucens*. Many pathogens, such as *P. phaseolicola* on beans or *Cercospora beticola* on beets, do not infect seeds when planting of the crops can be timed so that flowering and maturity phases are in a dry season (Palti 1981).

For faba bean viruses, infection after flowering often does not result in seed transmission (Vorra-Urai and Cockbain 1977; Jones 1978). The time of harvesting also may be important, with an early harvest preventing high seed infection rates with *A. fabae* (Hewett 1973).

Applied Epidemiology: Forecasting of Plant Diseases

Forecasting or the prediction of disease outbreaks, or, in the terminology of Zadoks (1984) "disease warning", is a major application of epidemiology. Ideally, a forecast enables the farmer to decide whether a treatment is economic, and to determine the optimal time of application. As Shrum (1978) pointed out, there is an interesting difference in the usage of 'forecasting' in the English literature, and 'prognosis' or 'Prognose' in German. Prognosis comes from the Greek 'progignoskein', meaning to know before, whereas forecast, according to Webster's Dictionary, implies 'to estimate or calculate in advance; predict or seek to predict'. Why in German the term 'Prognose' is preferred over the German equivalent to forecast 'Vorhersage' is subject to speculation; perhaps because the latter has a connotation of unscientific prophecy. In any case, the term forecast or 'Vorhersage' seems more appropriate, since the disease development is estimated rather than known.

Bourke (1970) summarized the requirements for a forecast of practical value:

- economic importance of the disease concerned
- appreciable impact of weather factors on disease variability
- availability of control measures
- availability of information on the nature of the weather dependence.

Coakley (1988) pointed out that most of the approaches to the prediction of plant diseases use daily or hourly weather data; rarely are climatic data (i.e., meteorological data of at least 10 years) employed.

While in earlier approaches a forecast was either based on inoculum potential or on weather, assuming that the other factor was always favorable, the support from powerful computers now allows extensive modeling, taking into account host (stages of development), pathogen (initial inoculum, spore survival), weather (favorable or adverse conditions), time (rate of disease spread), and space (spread of the disease to so far unaffected areas). Weather, however, remains the most important ingredient in a warning system (Zadoks 1984). It is one of the factors in the classical disease triangle that determine the development of an epidemic, the others being a susceptible host and a virulent pathogen. The effects of weather on plant diseases were the subject of many studies. Comprehensive reviews were published by Schrödter (1987), Friesland and Schrödter (1988) and Coakley (1989).

Coakley (1988) defined weather as "the current and predictable meteorological state of the atmosphere," which includes, among various factors, temperature, precipitation and barometric pressure. Climate is seen as the average of the complete set of atmospheric, hydrospheric and cryospheric variables over a specified period of time (Coakley 1989).

Obviously, there are wide variations in weather at any given location. "Exceptional" droughts, floods, or periods of heat or freezing always make good news stories. Besides plant growth and crop yields, they also affect the development of plant pests and pathogens. There is no doubt, however, that the climate is changing as described, for

instance, in the proceedings of a symposium on "Recent Climatic Change and Food Production" (Takahashi and Yoshino 1978), or by Bolin *et al.* (1986). A journal entitled "Climatic Change" began publication in 1977. Changes in climate, however, are not a new phenomenon. A number of Greek and Roman authors mentioned climatic changes, e.g., Aristotle in his *Meteorologia*: "...places that formerly enjoyed a good climate deteriorate and grow dry. This has happened in Greece to the land about Argos and Mycenae...." (Neumann 1985).

Farmers are generally more concerned about changes in weather than in climate, and an accurate short-term forecast helps them make decisions about whether or not to apply frost protection or irrigation, and in the timing of sowing, harvesting, or plant protection measures. Climatic changes, depending on magnitude, may affect the economy of states. Continued desertification will interfere with many African countries' efforts to become self-sufficient in basic foods. A worldwide increase in temperature might allow the USSR to extend the area it devotes to wheat production and simultaneously decrease wheat production in the USA.

Some plant diseases are more affected by weather conditions than others. Soil-borne pathogens, for example, are little influenced by weather; their occurrence depends more on climate than on weather and epidemics are relatively rare (Rotem 1978). Pathogens invading the aerial parts of a plant are more subject to weather conditions. In favorable conditions an epidemic can develop from a minimum amount of inoculum. Unfavorable conditions such as hot and dry weather can prevent the further development of an epidemic by killing spores. Particularly sensitive to the effect of adverse weather conditions between sporulation and infection are the spores of downy mildew fungi, e.g., *P. viticola* on grape. They do not survive more than 6 hours at a temperature of 30°C, regardless of relative humidity (Blaeser 1978; Blaeser and Weltzien 1979).

Interesting relations between accumulated precipitation or average temperature and the severity of cereal diseases were described by Saur and Löcher (1989). By evaluating 10 years of trials they found significant correlations for the following diseases and weather data: *Pseudocercosporella herpotrichoides* in winter wheat and the total precipitation from November to June, *Erysiphe graminis* in winter wheat and the total precipitation from March to June, *Leptosphaeria nodorum* in winter wheat and the total precipitation from January to June, *Pyrenophora teres* in winter barley and the total precipitation from February to June, and *P. recondita* in winter wheat and the average mean daily temperature from April to June.

Weather data can be obtained from meteorological stations found in any country, at least at airports. Other networks are the NOAA (National Oceanic and Atmospheric Administration) with about 9000 stations in the USA (Coakley 1988) or the "Deutscher Wetterdienst" with 358 stations in West Germany (Anonymous 1990). These data are very reliable, although they may differ greatly from those in the crops. While there is no doubt that microclimate is largely determined by macroclimate, it is also significantly affected by the crop itself. Particularly in canopy-forming crops, such as fruit trees, grapes, legumes, etc., the temperature under the canopy is usually higher than measured at a meteorological station. Wind speed is greatly affected by turbulence in the crop and

could be either higher or lower than indicated by the station's anemometer. Nevertheless, macroclimate and weather set the frame within which the disease development takes place (Schrödter 1987).

Rainfall and relative humidity data influence disease development only indirectly. Effects of rain are spore dispersal in splashes, washing down of spores from atmosphere or from leaves, and leaf wetness. Relative humidity varies considerably within a canopy, and is affected by rainfall, air movement, radiation, density of the canopy, and respiratory activity of the plants. Dew is generally not recorded by meteorological stations. Pedro and Gillespie (1982) published a method to estimate dew duration based on air temperature, dew point temperature, wind speed, and cloud cover. Leaf wetness duration, which may be caused by dew or rainfall, is crucial for infection with many pathogens, such as downy mildews (Blaeser and Weltzien 1977, 1978). Leaf wetness duration is affected largely by the same factors as the relative humidity, and even measurements in the canopy can give only a vague idea, because of the variation within the canopy. In grapes, the leaves inside the canopy remain wet for up to 4 hours longer than those directly exposed to wind and radiation (Diekmann, unpublished data). Electronic equipment developed in recent years for recording weather data makes localized data collection easier and more reliable (Sutton *et al.* 1988), but many measuring points are necessary to obtain data that are representative for a crop as a whole. Of all the factors mentioned, moisture and temperature are most important in plant disease epidemiology. Colhoun (1973) gave a detailed overview of temperature effect on plant disease development.

Early forecasts were empirical in nature and mostly based on a combination of meteorological parameters favorable for disease development (e.g., Schrödter 1987). While frequently successful under specific conditions, they sometimes failed under different conditions. Later on, statistical techniques helped to take into account the complex nature of plant disease epidemics. Complex models such as EPIVEN (for *Venturia inaequalis*, Kranz *et al.* 1973), or EPIGRAM (for *E. graminis* on barley, Aust *et al.* 1983) simulated time of infection and progress of an epidemic. More comprehensive is the integrated model EPIPRE (EPIdemic PREvention, Rabbinge and Rijsdijk 1983). It was developed for winter wheat and includes weeds and economic factors as well as diseases.

In Table 2 the weather parameters used in the forecasting of important plant diseases are summarized. Most of them are related to temperature and/ or precipitation. Little has been accomplished in the forecasting of seed-borne diseases, probably because of their rather complex epidemiology.

Table 2. Weather parameters used for the forecast of plant diseases.

Disease/pathogen	Weather parameters	Reference
Grape downy mildew (*Plasmopara viticola*)	Leaf wetness, temperature	Blaeser and Weltzien 1979
Grape downy mildew (*P. viticola*)	Rainfall during oospore maturation	Tran Manh Sung *et al.* 1990
Hop downy mildew (*Pseudoperonospora humuli*)	Leaf wetness caused by rain	Kremheller and Diercks 1983
Apple scab (*Venturia inaequalis*)	Leaf wetness, temperature	MacHardy and Gadoury 1989
Barley powdery mildew (*Erysiphe graminis*)	Temperature, sun, windspeed	Polley and Clarkson 1978
Fireblight (*Erwinia amylovora*)	Temperature, rainfall	Billing 1978
Early blight of potato (*Alternaria solani*)	Temperature, hours of RH >90%, minimum RH, precipitation	Shtienberg *et al.* 1989
Late blight of potato (*Phytophthora infestans*)	Temperature, hours of RH >90%, precipitation	Krause *et al.* 1975
Botrytis leaf blight of onion (*Botrytis squamosa*)	Temperature, RH, precipitation probability	Vincelli and Lorbeer 1989
Stripe rust of wheat (*Puccinia striiformis*)	Temperature >25°C, precipitation frequency, mean maximum temperature, days with temperature <0°C	Coakley *et al.* 1988a, 1988b

Chapter 3. Geophytopathology

Distribution of Plant Diseases

There are numerous examples in the literature indicating that host plants and their pests or pathogens have the same center of origin. Leppik (1970) cited examples of potato, wheat, *Cucumis*, sunflowers, corn and many other cultivated plants that have a center of origin identical to their major pests and pathogens. He also pointed out that large numbers of physiological races frequently are found in these areas, and he warned collectors not to import new races of pathogens with material collected from gene centers.

Many important crops were introduced into new areas, e.g., potatoes, tobacco and maize from South America to Europe, and wheat from Europe to North America. Sometimes, their pests and pathogens were introduced simultaneously. In other cases, they followed many years, even centuries later, as in the case of tobacco blue mold, when an agrochemical company introduced the pathogen into England for research purposes, from where it spread all over Europe (Zadoks 1967). Occasionally, introduced crops still may be free from some pests that occur in their center of origin.

There is also a chance that introduced crops are attacked by a pest or pathogen that, until then, had been unimportant or even unknown. An example is the introduction of potatoes into North America where they were attacked by the Colorado beetle. This insect had been feeding on native weeds, but then spread rapidly with the potatoes (Gauthier *et al.* 1981). Such pests or pathogens may then be introduced into the crop's country of origin.

While new pests and pathogens can be devastating to a crop, new races or *formae speciales* of already well-established pests and pathogens also may be a threat. Zadoks (1967) cited examples for *Puccinia striiformis* where an until-then resistant variety was attacked, or for *U. tritici*, where a new race was introduced from England to France with imported wheat seed. At ICARDA in Aleppo, Syria, six physiologic races of *A. rabiei* were identified. The less virulent ones, namely races 1, 2 and 3, were prevalent in farmers' fields, whereas the more virulent races 4, 5 and 6 were found in experimental sites only (ICARDA 1984). This leads to the conclusion that races 4 to 6 have been introduced with seeds from abroad. ICARDA (1985) reported that most of the lines which were resistent to race 3 were found susceptible to the newly identified race 6. None of the genotypes tested were resistant to race 5 (ICARDA 1986).

Systematic information on the geographical distribution of plant pests and diseases is scarce. The Commonwealth Agricultural Bureaux started publishing "Distribution Maps of Plants Diseases" in 1942. By 1990 this loose-leaf collection contained maps for 625 different pathogens. Each year about 10 to 15 new maps are added and about 30 to 40 maps are updated. The information included comes mostly from articles summarized in the Review of Plant Pathology, formerly Review of Applied Mycology. A list of countries

with the references is also provided. However, an unlisted country is not necessarily free from a particular disease; or, as the saying goes, "absence of evidence is not evidence of absence". In Syria, for example, faba beans are greatly affected by *A. fabae*, *Uromyces fabae* and *Botrytis fabae* (Hawtin and Stewart 1979), yet this country is not listed in the respective maps (Commonwealth Agricultural Bureaux 1976a, 1977a, 1981). It was added, however, to the second edition of the map on *A. fabae* (Commonwealth Agricultural Bureaux 1989).

Collection of disease data requires frequent field surveys by experienced personnel. Precise surveys are frequently lacking, or are not published. Some diseases, such as common bunt (*T. caries* and *Tilletia foetida*) or even moreso, Karnal bunt (*Tilletia indica*), are not easily detected in the field. A bread wheat field in Libya was found to be infected at an incidence of 90% with common bunt, and yet the disease had not been reported to occur in this country (A.H. Kamel, ICARDA, pers. comm.). In general, it is difficult to detect diseases that occur at a low incidence. Bourke (1970) emphasized that usually three to five generations of a fungus precede the first visual appearance of a disease.

Usually only those diseases are carefully monitored that may cause epidemics resulting in economic damage. In Germany, for example, grape diseases are reported in great detail by the extension service and the results are published along with data on crop development and weather (e.g., Englert and Holz 1989). Detailed observations are often made on potentially devastating diseases recently introduced into a country or an area, such as fireblight (*Erwinia amylovora*) in Europe (Zeller 1974)) or citrus canker (*Xanthomonas citri*) in Florida (Schoulties *et al.* 1987). On the other hand, it is difficult to find reports in the literature on diseases occurring only sporadically. Yet, this group may be quite interesting, because epidemics could easily develop if, for example, agronomic practices (increase of field size, irrigation, zero tillage practices, shorter rotations, etc.) change.

In addition to obtaining disease data by field surveys, there are also indirect methods. Leppik (1964) complemented the information available in the current CMI Distribution Map for *Plasmopara halstedii* (Commonwealth Agricultural Bureaux 1954) with results of his seed health tests on sunflower seeds originating from various parts of the world, and thus more than doubled the number of host countries. Maps published subsequently incorporated this information (Commonwealth Agricultural Bureaux 1977b). This approach, however, may yield misleading results if seeds have been reshipped from the country of origin, as happened in the case of *T. indica* tests carried out in India (Diekmann 1987).

Another indirect way to monitor diseases could be by collecting the released spores. Young *et al.* (1978) provided an overview for various methods — from slides coated with sticky material to sophisticated samplers. CIMMYT, the International Center for the Improvement of Maize and Wheat, monitored cereal diseases by distributing their Regional Disease Trap Nursery (RDTN), consisting of about 50 important cultivars, to various locations in a region (Young *et al.* 1978).

Many valuable data are not published at all, or only in annual reports and other publications not easily accessible to the scientific community.

The Concept of Geophytopathology

The idea of applying geography to botany or human diseases started as early as 1792 and was reviewed by Weltzien (1972). Reichert (1958) studied the distribution of *Sclerotinia sclerotiorum* by considering geographical, ecological, paleohistorical and migratory aspects. He called this approach "mycogeography" or "phytopathogeography." The paleohistorical aspect of his study revealed that all genera in the family Sclerotiniaceae originated in North America. Practical implications are the possible avoidance of growing susceptible crops in areas that meet the pathogen's ecological requirements, as well as the possibility of locating sources for resistance to the pathogen in its center of origin.

Reichert and Palti (1966) included in their "patho-geographical approach"

- geographical distribution of the pathogen
- ecological characteristics of the pathogen
- climatic conditions during the presence of the host
- limitations of pathogenicity due to host-parasite factors
- cultural conditions, e.g., soil type, irrigation, etc.

Whereas data on the first three subjects may be scattered in the literature, a profound knowledge of an area will most likely be necessary for the last two. Nevertheless, Reichert and Palti (1966) arrived at a prediction of occurrence for eight plant diseases. In other words, they presented an analysis of areas where the occurrence of those diseases is highly unlikely.

Zadoks (1967) cited the examples of potato late blight before the great epidemic in 1845, grape downy mildew before its introduction into France in the 1870s, or tobacco blue mold before its introduction into Europe in 1958.

Weltzien (1967) created the term "geophytopathology." He suggested starting an investigation with a study of the distribution of the host plant. He also considered it necessary to include data on frequency of disease occurrence, its intensity and the extent of damage, and suggested a differentiation in

- "areas of main damage", where epidemics with considerable economic losses occur frequently
- "areas of marginal damage", where epidemics with considerable economic losses occur occasionally
- "areas of sporadic attack", where the disease is reported, but does not usually cause significant damage.

Maps following these criteria were published subsequently by Drandarevski (1969) for *Erysiphe betae* and Bleiholder and Weltzien (1972) for *C. beticola*.

Geophytopathology has two different aspects: (1) the geographical aspect which

concentrates on mapping of the occurrence of host, alternate host(s) and disease, or of disease frequency or intensity, and (2) the climatological aspect which takes into consideration the environmental conditions required for the pathogen's development. An example of how the first aspect might have prevented a serious disease problem was given by Zadoks and Schein (1979): pine blister rust caused by *Cronartium ribicola* is heteroecious, i.e., it occurs only where the two hosts *Pinus* and *Ribes* are found together. The pathogen originated in the northeastern USSR and in the Alps and spread from there only after the introduction of susceptible *Pinus* species from the USA, causing severe damage on pines, currants and gooseberries. In 1909 the fungus was introduced to North America with infected trees from Europe and soon spread across the continent.

Considering the climatological aspect, geophytopathology makes use of the relation between disease development and meteorological factors in a manner similar to plant disease forecasting. A major difference lies in the fact that a forecast depends on weather data to predict disease development in a limited geographical area (e.g., a vineyard), and geophytopathology employs climate data to anticipate disease occurrence on a regional basis (e.g., the area of Burgundy). Geophytopathology can be regarded as a kind of "negative prediction" in the sense of Ullrich and Schrödter (1966), a statement indicating that under the prevalent climatic conditions in an area the occurrence of a disease is not to be expected. This aspect is important when new crops are introduced or with international seed exchange (Weltzien 1988).

Verification of Earlier Geophytopathological Forecasts

Sufficient time has elapsed since prognoses have been published that they now can be examined. Examples 1 to 5 (following) are based on predictions of Reichert and Palti (1966); example 6 verifies Drandarevski's (1969) forecast; example 7 is based on Miller's (1969) publication; and example 8 is the negative forecast by Weltzien (1978).

1. Potato wart disease (*Synchytrium endobioticum*). This is a quarantine disease in many countries. Reichert and Palti (1966) concluded that there will be no disease risk in areas where soil temperatures rise to approximately 30°C, such as in lowland tropics and arid areas. The CMI Distribution Map for this pathogen (Commonwealth Agricultural Bureaux 1983; Fig. 1) listed new records from Sikkim (China), Bhutan, Central and South China, Nepal, Montenegro (Yugoslavia) and Southern New Zealand. As far as could be confirmed from the original sources, these areas were all at high altitude or located in a temperate climate (New Zealand 45° latitude). No reports could be found for new introductions in subsequent years.

2. Seedling blight (*Sclerotium rolfsii*). The same authors stated that possible occurrence of this pathogen is in climates where average temperatures of 20-30°C coincided with considerable rainfall, or with irrigated summer crops in arid or semi-arid regions. While the recent literature contains numerous reports on this pathogen, most refer to occurrence in more tropical crops, such as peanut, rice, pigeonpea, sweet potato, etc. Therefore, it seems that although *S. rolfsii* is widespread on a wide range of host plants, it develops mainly on plants grown under the conditions cited above (Fig. 2).

Fig. 1. Distribution map of potato wart disease (*Synchytrium endobioticum*), after Commonwealth Agricultural Bureaux (1983).

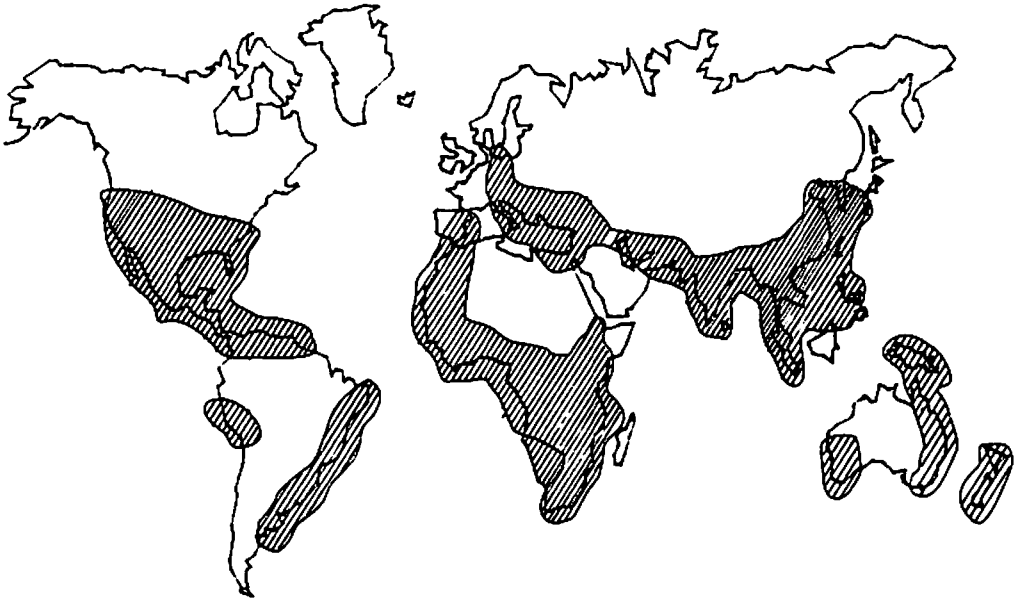

Fig. 2. Distribution map of seedling blight (*Sclerotium rolfsii*), after Commonwealth Agricultural Bureaux (1969).

3. Powdery mildew (*Leveillula taurica*). This powdery mildew occurring on various hosts — such as cotton, or potato and other Solanaceae — was predicted to cause substantial losses in areas with monthly means of 15-25°C and low or no rainfall (for potato, tomato and alfalfa) or a wide range of humidity conditions (for chili and eggplant). According to the CMI Distribution Map for *L. taurica* (Commonwealth Agricultural Bureaux 1984; Fig. 3) the pathogen is absent from the wet tropics and cold climate areas.

Fig. 3. Distribution map of powdery mildew (*Leveillula taurica*), after Commonwealth Agricultural Bureaux (1984a).

4. Blast and blackpit disease of citrus (*Pseudomonas syringae*) pv. *syringae*. This disease was predicted as unlikely to occur on citrus in Florida as well as East and West Africa. Although other pathovars of *P. syringae* occur on many different crops in other parts of the world (Commonwealth Agricultural Bureaux 1979), a literature search proved that the blast and blackpit disease caused by the pathovar *syringae* remained confined to locations in the Mediterranean area, California, South Africa, Japan and Australia (Fig. 4).

Fig. 4. Distribution map of blast and blackpit of citrus (*Pseudomonas syringae* pv. *syringae*).

5. Citrus canker (*Xanthomonas campestris* pv. *citri*). The pathogen was referred to as *Phytomonas citri* Hasse by Reichert and Palti (1966); it was renamed later as *Xanthomonas citri* (Hasse) Dowson, and yet later as *Xanthomonas campestris* pv. *citri* (Hasse) Dowson. The prediction was remarkably precise: a potential threat in tropical Africa and countries with a similar climate; no risk in areas without summer rains, such as California and the Mediterranean countries. The CMI Distribution Map for *X. citri* (Commonwealth Agricultural Bureaux 1978; Fig. 5) showed occurrence in some African countries, in southeast Asia (where according to Gonzalez (1978) the disease originated) and southeast America, but not in the Mediterranean area or the western USA. Citrus canker had been eradicated from Florida between 1914 and 1933, and also from South Africa, Australia and New Zealand (Stall and Seymour 1983). It appeared again in a Florida nursery in 1984 (Schoulties and Miller 1985), and two forms, "nursery" and "Asiatic," subsequently spread in Florida (Schoulties *et al.* 1987). Whereas the eradication of canker in Florida is still continuing (Timmer *et al.* 1991), and citrus growers as well as authorities in California were very concerned about the possible spread into their state (Anonymous 1984), the disease has not been observed in California to date.

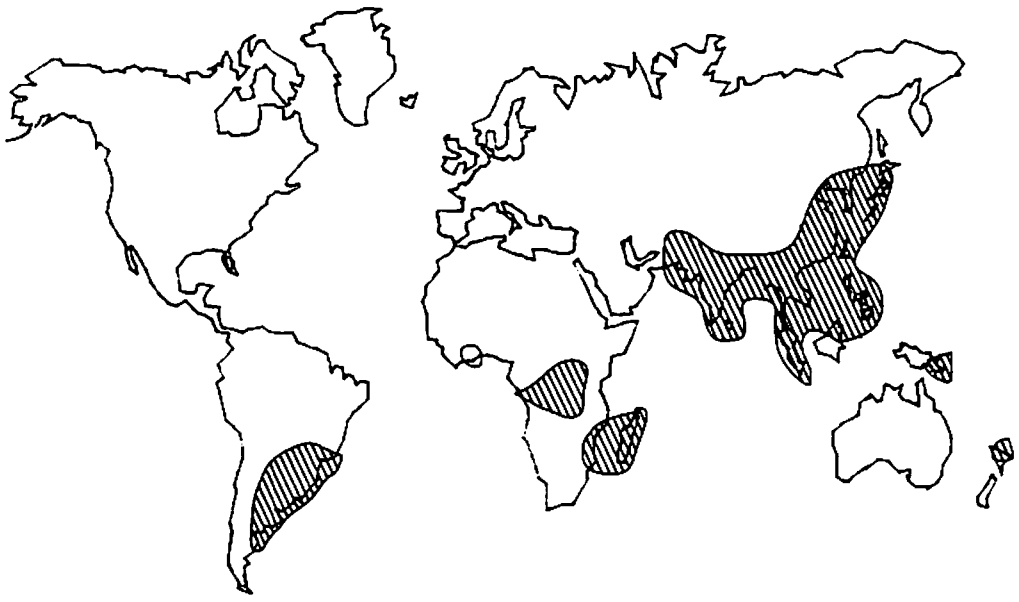

Fig. 5. Distribution map of citrus canker (*Xanthomonas campestris* pv. *citri*), after Commonwealth Agricultural Bureaux (1978).

6. Tobacco blue mold (*Peronospora tabacina*). Without naming his research "geophytopathology," Miller (1969) came to an interesting conclusion. In 1950, he had concluded from his studies on forecasting the incidence of tobacco blue mold (caused by *P. tabacina*) in the United States that the pathogen would cause a serious disease if it were imported to Europe. His prediction was proven accurate by the blue mold epidemics in Europe (Klinkowski 1970; Fig. 6).

7. Powdery mildew of sugar beet (*Erysiphe betae*). Drandarevski (1969) found that although there were no reports in the literature of powdery mildew on sugar beets in Finland, Bulgaria, Yugoslavia, Albania, Greece, the Iberian Peninsula, North Africa, Southeast Asia, South America and Australia, the climatic conditions were such that epidemics could be expected in some of these regions. Moreover, for the United States, where the disease occurred but was not reported to be of importance, increased importance was suspected in the future. A literature review covering the years 1968 to 1990 revealed reports on the occurrence of the pathogen in Bulgaria (V"rbanov 1978), Egypt (El-Kazzaz *et al.* 1977) and Portugal (Lucas *et al.* 1979). Amano (1986) listed the following additional host countries: Yugoslavia, Spain, Morocco and Japan, unfortunately all without direct reference to the original source.

   In addition, an epidemic of sugar beet powdery mildew occurred in 1974 in the USA (Ruppel *et al.* 1975), with severe attacks in California, Utah, Idaho, Oregon, Washington, northwest Kansas and Nebraska and moderate disease severity in most

Fig. 6. Distribution map of tobacco blue mold (*Peronospora tabacina*), after Commonwealth Agricultural Bureaux (1976b).

of the Great Plains. Drandarevski's map projected areas of significant economic importance in California, Utah and Idaho, and areas of occasional significant importance for the other Western beet-producing states. In 1975, the pathogen was observed for the first time in Michigan, although it did not cause economic losses (Schneider and Hogaboam 1977). Drandarevski (1969) had projected Michigan as an area where the pathogen could occur without economic importance. Also in 1975, powdery mildew on sugar beet was reported for the first time in Canada (Harper and Bergen 1976). The authors supposed that there was little danger that the mycelium could overwinter in Canada, and no reports on the occurrence of powdery mildew on sugar beet in Canada could be found in the literature between 1976 and 1990.

8. Leaf spot and powdery mildew of sugar beet (*Cercospora beticola* and *Erysiphe betae*). A "negative prognosis" was made by Weltzien (1978) by combining the data of Drandarevski (1969) and Bleiholder and Weltzien (1972) with the distribution of sugar beet (Fig. 7). He concluded that the northern coastal areas of Europe, the East Coast of the USA and an area in northeast Argentina were not likely to suffer from either *C. beticola* or *E. betae*. No reports for *E. betae* in these areas could be found, and for leaf spot the search yielded only one report mentioning a *C. beticola* isolate from Maryland (Whitney and Lewellen 1976). Presumably, therefore, even though the pathogens might occur in the areas mentioned by Weltzien (1978), they are not considered important enough to warrant conducting research.

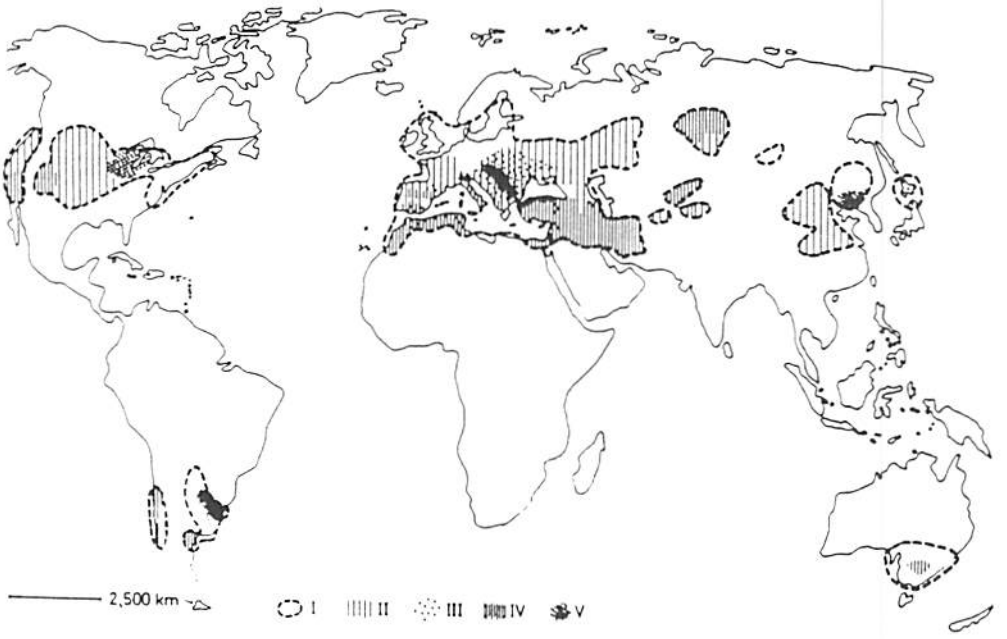

Fig. 7. World map of sugar beet areas with the three zones of disease intensity for leaf spot (*Cercospora beticola*) and powdery mildew (*Erysiphe betae*) (from Weltzien 1978). I. Distribution of the crop and areas of sporadic attack for both diseases; II. Areas of occasional powdery mildew epidemics; III. Areas of occasional leaf spot epidemics; IV. Areas of regular powdery mildew epidemics; V. Areas of regular leaf spot epidemics.

Chapter 4. Geophytopathological Approach to Predicting the Occurrence of Selected Diseases under Specific Climatic Conditions

Adaptation of a Weather-based Model: Stripe Rust of Wheat (*Puccinia striiformis* Westend)

The weather-based forecast models are helpful to predict the occurrence or severity of a disease in a given season and location. For an estimation on whether or not a disease is likely to occur in an area, the use of climatic data is appropriate. Since no forecast models have been developed as yet for seed-borne diseases, the adaptation of a well-documented prediction is attempted below.

Coakley (1978) studied the relation between stripe rust (*P. striiformis*) and meteorological data over 40 years. She found that the more frequent occurrence of stripe rust epidemics in the Pacific Northwest from 1960 on was correlated with an average temperature increase of 2°C in January and February and an average temperature decrease in April of 1.2°C. Increased winter temperatures favored the pathogen by shortening its incubation period, thus accelerating its spread and ensuring survival of more fall-infected leaves. Lower spring temperatures were unfavorable for the host because they delayed the stage of adult-plant resistance.

Similar results were obtained by Coakley and Line (1981) for a different location in the Pacific Northwest, and by correlating "degree days" with the disease index. Degree days were calculated by subtracting 7 from the daily average temperature, because 7°C is the optimum temperature for urediospore germination and infection. Negative values, obtained when the daily average temperature was below 7°C, were recorded as negative degree days (NDD), others as positive degree days (PDD). The authors concluded from the results that stripe rust does not play an important role in the central USA because of lower winter temperatures, or in the southeastern USA because of higher spring temperatures. In addition to the above-mentioned factors, temperature in June also showed a significant negative correlation with disease intensity. However, no precipitation parameter was sufficiently correlated with the disease index to be useful in the prediction of the disease.

Based on these findings, Coakley *et al.* (1982) developed a model that allowed the prediction of stripe rust on winter wheat in the Pacific Northwest of the USA. The degree-day data were standardized by using the formula

$$NDDZ = (x_i - \bar{x})/s$$

with x_i being the yearly NDD (negative degree days), $\bar{x}$ the long-term NDD, and s the standard deviation of the mean. By using this method of standardization, the authors could accurately predict the disease intensity for various locations.

The model was further improved by including the "Julian day of spring" (JDS), that is, the first day which is followed by a PDD (positive degree value) of at least 40 within 14 days. Example: if the first 14-day period with a PDD value (= sum of daily mean temperatures - 7°C) of >40 falls between 24 April and 8 May, the JDS is 114 = 24 April. This is considered a reasonably accurate way of identifying the time when plant growth and rust development start at a given site. Including positive degree days (PDD) as well improved the accuracy (Coakley *et al.* 1983, 1984). The forecast of a severe epidemic in 1981 led to an emergency registration of the fungicide Bayleton, which prevented serious losses (Line 1983). A validation of the models resulted in 80% of the predictions being within the standard error limits for model I which included NDDZ and JDS, and all predictions within the limits for model II, which also included PDD in addition to the above. The advantage of model I over model II is that it can be applied earlier in the season (Coakley and Line 1984).

The authors have developed their models as specifically as possible, i.e., with different equations for susceptible and resistant varieties, and have incorporated weather data (NDD, JDS, PDD) as well as long-term averages (in the NDDZ). The hypothesis is that by generalizing their data, i.e., averaging the data for the varieties, and using climatic (long-term average) data instead of annual weather data, an indication of the risk of stripe rust occurring under specific climatic conditions can be obtained. The calculations described below are based on published data (Coakley 1978; Coakley and Line 1981; Coakley *et al.* 1982, 1983, 1984). Since these data were selected and computed with a different objective, namely an accurate prediction of stripe rust in a given location at an early stage, the accuracy of the prediction of a risk in geographical locations can possibly be improved by going back to the original data.

From the equations for the two wheat varieties Gaines and Omar (Coakley *et al.* 1983), the following overall formula for a "risk index $\hat{r}$" can be calculated

$$\hat{r} = 4.233 - 1.448\ \text{NDDZ} + 0.037\ \text{JDS} - 0.006\ \text{PDD}.$$

The weather-related NDDZ has to be replaced by the climate value NDD, using the regression equations for the same varieties (Coakley and Line 1981; Coakley *et al.* 1982)

$$\hat{y} = 11.112 - 0.011\ \text{NDD}$$
$$\hat{y} = 5.205 - 1.690\ \text{NDDZ}.$$

Because the prediction $\hat{y}$ is the same (Coakley *et al.* 1982), we can conclude

$$11.112 - 0.011\ \text{NDD} = 5.205 - 1.690\ \text{NDDZ}$$
or
$$\text{NDDZ} = 0.007\ \text{NDD} - 3.497$$

and arrive at the equation

$$\hat{r} = 9.296 - 0.010\ \text{NDD} + 0.037\ \text{JDS} - 0.006\ \text{PDD}.$$

This equation is too specific for the prediction of a risk of stripe rust occurring in a given area, based only on meteorological data and regardless of varieties. The formula is therefore simplified and adapted to monthly averages of mean daily temperatures, which are more easily accessible than long-term daily averages.

$$RI = 9.3 - 0.6 \cdot (7 - x_1) + \frac{x_2 \cdot \text{month no.}}{10} - \frac{x_3 - 7}{2}$$

RI = risk index
x_1 = average mean daily temperature December and January
x_2 = average mean daily temperature of the months with avg. below 10°C and above 10°C
x_3 = average mean daily temperature of the three first months where the temperature exceeds 10°C.

The input data and the calculated risk index for several locations are given in Table 3. The lower the risk index, the lower the risk of stripe rust occurring epidemically in these locations. The calculated risk index agrees well with the distribution of yellow rust indicated in Figure 8. The locations in the western USA have a higher disease index than those in the east. In Europe, the risk is calculated as high in the coastal areas (Brest) and lower in more southern locations (Hof). For Aleppo, a relatively high risk is predicted, which coincides with the actual situation in Syria. This approach is restricted to climatic conditions related to those for which the models of Coakley and Line (1981) and Coakley *et al.* (1982) were developed. Because the key value is the 7°C limit for development of the pathogen *P. striiformis*, a risk assessment with this model is not possible for areas where temperature is not a limiting factor, i.e., where the daily average temperature is above 7°C throughout the wheat-growing season, such as southern Australia or Egypt. The parameters limiting yellow rust development in these climates need to be investigated.

Coakley *et al.* (1988a) improved their stripe rust model by applying a method used for *Septoria tritici* blotch (Coakley *et al.* 1985). Depending on the variety, positive degree days, negative degree days, and the Julian day of spring were replaced by the "total days maximum temperature greater than 25°C, DG25C" (for all varieties), "precipitation frequency", or "total days average temperature less than 0°C", or "mean maximum temperature". Whereas the inclusion of DG25C from 23 April to 27 June increased the accuracy of the model, the results are obtained too late for use in decisions whether or not a fungicide application is warranted (Coakley *et al.* 1988b). The authors stressed the necessity to include at least 8-10 years of meteorological data in any such model.

Because the improved model is even more variety specific than the previous one, it will not be used here for risk prediction. However, the calculations show that various climatic parameters can be applied to predict disease severity.

Table 3. Mean daily temperature during wheat vegetation (from Müller, 1982) and risk index (RI) for stripe rust (Puccinia striiformis) in different locations; x_1 = average mean daily temperature December and January, x_2 = average mean daily temperature of the months with average below 10°C and above 10°C, x_3 = average mean daily temperature of the three first months where the temperature exceeds 10°C.

Location	Mean daily temperature (°C)											RI
	Dec.	Jan.	Feb.	Mar.	Apr.	May	June	July	x_1	x_2	x_3	
Spokane, WA	−0.5	−2.5	−0.3	4.4	9.5	13.6	17.3	21.1	−1.5	11.6	17.3	4.7
Detroit, MI	−1.2	−2.8	−2.7	1.6	8.4	14.7	20.7	23.3	−2.0	11.6	19.6	3.2
Omaha, KA	−2.1	−5.4	−3.1	2.7	10.9	17.2	22.8	25.8	−3.8	6.8	17.0	0.3
Minneapolis, MN	−7.9	−10.9	−8.9	−2.4	7.1	14.2	19.6	22.8	−9.4	10.7	18.9	−1.1
Des Moines, IO	−3.3	−6.1	−4.4	2.5	10.3	16.2	21.4	24.2	−4.7	6.4	16.0	0.1
Bozeman, MT	−1.7	−4.9	−3.5	0.7	7.5	13.2	17.6	22.9	−3.3	10.4	17.9	2.7
Brest, France	7.0	6.1	5.8	7.8	9.2	11.6	14.4	15.6	6.6	10.4	13.9	10.8
Hof, Germany	−1.8	−3.4	−2.5	1.3	5.7	10.5	13.9	15.6	−2.6	8.1	13.3	4.2
Aleppo, Syria	7.8	5.6	8.1	10.9	16.4	21.4	25.9	26.7	6.7	9.5	16.2	7.4

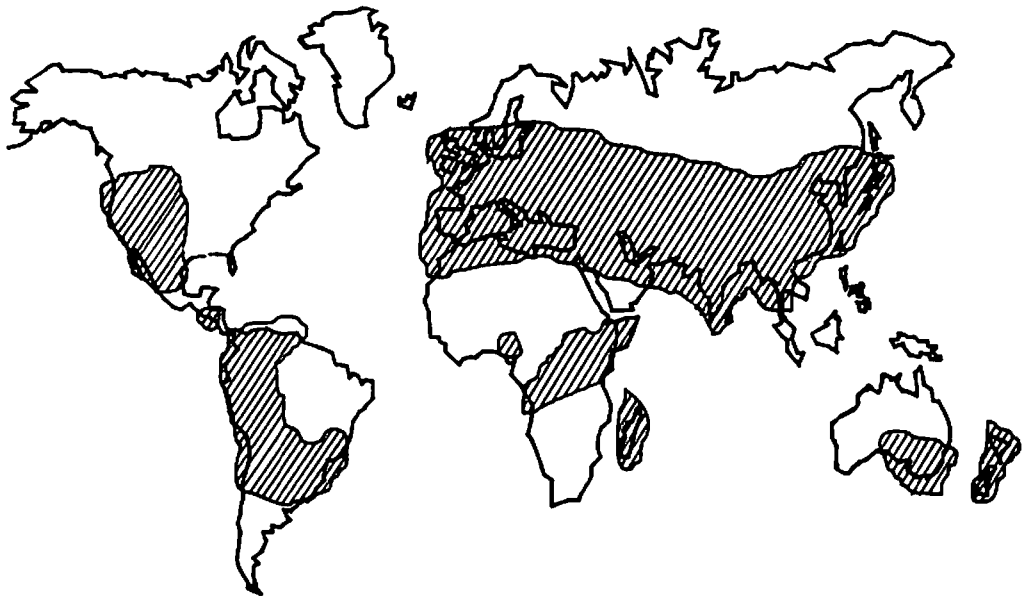

Fig. 8. Distribution map of yellow rust of wheat (*Puccinia striiformis*), after Commonwealth Agricultural Bureaux (1984b).

Discriminant Analysis of Climatic Data for Selected Seed-borne Diseases

Literature survey on distribution and epidemiology of the pathogens

Bacterial leaf blight of rice. The pathogen [*Xanthomonas campestris* pv. *oryzae* (Ishiyama) Dye] is widely distributed (Table 4, Fig. 9); however, it has not been reported from Madagascar or Brazil, which both have extensive rice-growing areas (Commonwealth Agricultural Bureaux 1987; FAO 1988). According to Ou (1985), the disease was reported from Madagascar without a positive identification of the pathogen. Lozano (1977) concluded that occurrence of the disease in the Caribbean and South America during the 1970s was probably due to indiscriminate importation of rice seed with few quarantine precautions. He reported light to moderate disease severity, probably due to relatively unfavorable climatic conditions, i.e., no typhoons and moderate temperatures during the rainy season in most of the rice-growing areas. A recent publication reported the occurrence of the pathogen in the states of Texas and Louisiana (Jones *et al.* 1989).

In culture, the bacteria have a slimy yellow appearance. The critical temperatures are: minimum 5-10°C, optimum 26-30°C, maximum 40°C (Ou 1985). Hsieh and Buddenhagen (1975) found that, under low relative humidity and low temperature, the pathogen survives for long periods in diseased leaves in soil. Under typical wet tropics conditions, the pathogen would require living host tissue.

Table 4. Geographical distribution of *Xanthomonas campestris* pv. *oryzae* (Ishiyama) Dye (Commonwealth Agricultural Bureaux 1987; * from Awoderu 1983; ** from Mew 1987; *** from Jones *et al.* 1989).

Asia	**North America**
Bangladesh	Mexico
Burma	USA (Texas, Louisiana)***
China	
India	**Central America and West Indies**
Indonesia	Costa Rica
Japan	Honduras
Kampuchea	Panama
Korea	El Salvador
Laos	
Malaysia	
Nepal	**South America**
Pakistan	Bolivia
Philippines	Colombia
Sri Lanka	Ecuador
Taiwan	Venezuela
Thailand	
Vietnam	**Africa**
	Burkina Faso**
	Cameroon
Australasia and Oceania	Ivory Coast*
Australia	Mali
Togo	Niger
	Nigeria*
	Senegal

Srivastava (1972) summarized the effects of weather on the disease: no symptoms were observed below 20°C, and optimum development seems to be above 25°C. Furthermore, rainy weather and strong winds are considered favorable, whereas sunlight and dry air kill the inoculum. Kainth and Mehra (1984) found disease incidence to be correlated with high rainfall (500-900 mm) and the number of cloudy days (10-30) during the crop period between June and September. When comparing a "normal" with a "bacterial leaf blight epidemic" year, Muralidharan and Venkata Rao (1979) reported differences in the number of rainy days (9 vs. 13) and total rainfall (120 vs. 384 mm) in July and August, the months of crop transplanting. Later in the epidemic year, three cyclones damaged the crop, and heavy rains alternated with bright sunny days. Premalatha Dath *et al.* (1979) also compared the effect of different weather on disease development during the 15 days between inoculation and evaluation and found the differences summarized in Table 5. They considered temperature to be most decisive, with a mean minimum temperature of 16.7°C and a mean maximum temperature of 37.4°C being the limits for optimum growth of lesions.

Fig. 9. Distribution map of bacterial leaf blight (BLB) of rice (*Xanthomonas campestris* pv. *oryzae*). For details, see Table 4.

Table 5. Effect of weather factors two weeks after inoculation on the development of bacterial leaf blight of rice (*Xanthomonas campestris* pv. *oryzae*, after Premalatha Dath *et al.* 1979).

Weather factor	Favorable	Unfavorable
Relative humidity	83.4%	69.0%
Mean maximum temperature	31.4°C	25.5°C
Mean minimum temperature	24.3°C	14.8°C
Rainfall	68.2 mm	47.0 mm
Sunshine hours/day	6.5	7.8
Number of rainy days	6	2

Ascochyta blight of chickpea. The disease, caused by *Ascochyta rabiei* (Pass.) Lab., has been reported from the countries listed in Table 6. It attacks stems, leaves, pods and seeds of chickpea. Characteristic are the black pycnidia in lesions, which release millions

of pycnospores. According to K.B. Singh *et al.* (1990), Ascochyta blight is extremely important in areas between 31° and 45° latitude, and occasionally important between 26° and 30° (Fig. 10). In India, the disease occurs in the states of Punjab, Bihar, Haryana, Uttar Pradesh and Himachal Pradesh, but was not or only occasionally reported from Madhya Pradesh and Andhra Pradesh (Sandhu *et al.* 1984; Kotasthane *et al.* 1985; G. Singh *et al.* 1988; Dahiya *et al.* 1988; Tripathi *et al.* 1988; Kalia and Dawa 1988; Pundir *et al.* 1989).

Table 6. Geographical distribution of *Ascochyta rabiei* (Commonwealth Agricultural Bureaux 1986; * from Nene 1982; ** from Abdel Monem *et al.* 1984; * from Kovics *et al.* 1986; **** from Nene and Reddy 1987).**

Asia	**Africa**
Bangladesh*	Algeria
India	Egypt**
Iran	Ethiopia
Iraq	Morocco
Israel	Tanzania
Jordan*	Tunisia*
Lebanon	
Pakistan	**Europe**
Syria	Bulgaria
Turkey	Cyprus
USSR	France
	Greece
Australasia and Oceania	Hungary***
Australia	Italy
	Portugal
North and Central America	Romania
Canada	Spain
USA	
Mexico****	

In the early 1980s, the pathogen was introduced in Washington and Idaho in the USA, where it caused severe epidemics. It was, however, not mentioned in an extensive survey of chickpea diseases in California (Kaiser and Muehlbauer 1984; Kaiser and Muehlbauer 1988; Buddenhagen *et al.* 1988). In Ethiopia, Ascochyta blight has been observed only when chickpeas are planted in July instead of September (Bejiga 1984). No reports on

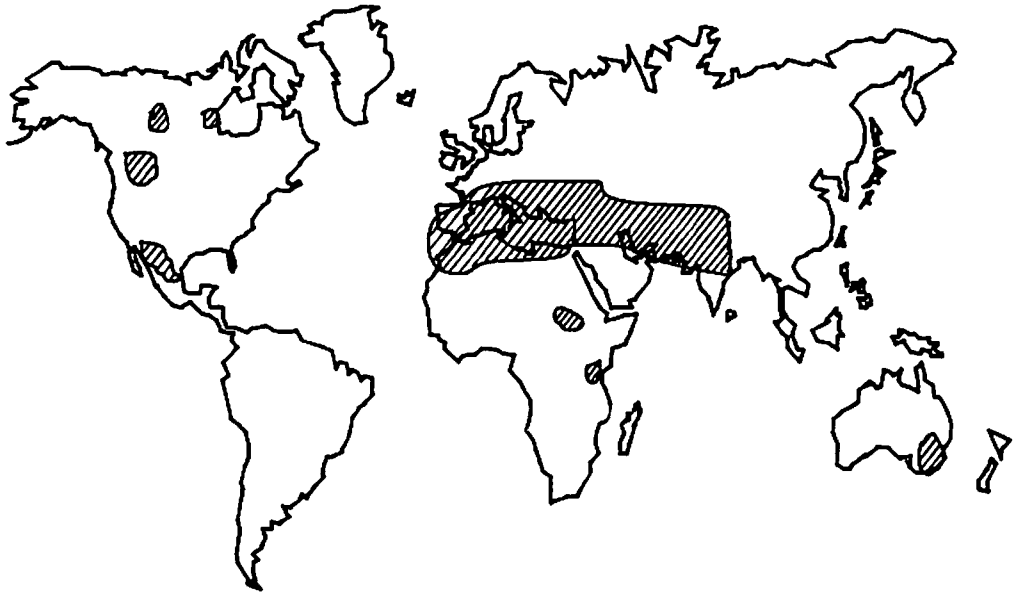

Fig. 10. Distribution map of Ascochyta blight of chickpea (*Ascochyta rabiei*). For details, see Table 6.

the occurrence of the disease could be found from Burma, Nepal, Argentina, Bolivia, Chile, Colombia, Peru, Libya, Malawi, Zambia, Sudan, Uganda and Yugoslavia, which all grow considerable areas of chickpea.

The minimum temperature for infection with *A. rabiei* in the field was found to be 10°C, whereas the optimum is in the range of 20-25°C and the maximum is 30°C (Zachos *et al.* 1963; Chauhan and Sinha 1973). Kaack (1983) found in laboratory experiments that between 9 and 27°C a leaf wetness duration of 6 hours is required for infection. Disease severity increased with increasing leaf wetness duration throughout the tested temperature range of 3 to 30°C. At marginal temperatures the disease severity was so low that these temperature/leaf wetness duration combinations are not relevant for field conditions. Kaack (1983) concluded that under field conditions the minimum temperature for infection is about 6°C. A high relative humidity favors disease development (Chauhan and Sinha 1973).

Karnal bunt or partial bunt of wheat. The pathogen (*Tilletia indica* Mitra syn. *Neovossia indica*) was first described in Karnal, Punjab, India by Mitra (1931). The first record in Mexico was provided by Duran (1972), who concluded that the fungus very likely had been introduced from India on infected wheat seed. The CMI Distribution Map (Commonwealth Agricultural Bureaux 1974) listed in addition Iraq, Pakistan and Afghanistan. Zhang *et al.* (1984) reported that the disease occurred in Nepal. The

Fig. 11. Distribution map of Karnal bunt of wheat (*Tilletia indica*). For details, see Table 7.

pathogen also has been intercepted with seeds from Lebanon, Syria, Turkey and Sweden (Lambat *et al.* 1983). The disease, however, has not been observed in Syria, and the Syrian germplasm samples intercepted were most likely reforwarded (Dickmann 1987). Likewise, no reports on the occurrence of Karnal bunt in Lebanon, Turkey and Sweden could be found. Since all consignments concerned were sent by research institutes or genebanks, it is possible that in these cases the seeds originated in one of the countries listed in Table 7. The confirmed distribution of Karnal bunt is shown in Figure 11.

In India, Karnal bunt was regarded as a disease of minor significance until its epidemic occurrence in 1974/75, mainly in the northern part of the country. After its spread to new areas in West Bengal, Gujarat and Maharashtra in 1979/80 it is now reported from most Indian states, except Karnataka, Maharashtra, Andhra Pradesh, Bengal, Orissa, Kerala and Tamil Nadu. The highest incidence was found in the state of Punjab (D.V. Singh *et al.* 1980, 1985). In Pakistan until 1987, the provinces of Sind and Baluchistan were free from Karnal bunt, whereas the Northwest Frontier Province and Punjab are affected to various degrees (Begum and Mathur 1989).

Unlike with common bunt, caused by *T. caries* or *T. foetida*, the typical symptom of Karnal bunt is a partial infection of wheat seeds. Only a severe infection may result in the same type of completely bunted seeds which are typical of common bunt.

Table 7. Geographical distribution of *Tilletia indica* (Commonwealth Agricultural Bureaux 1974; * Zhang *et al.* 1984).

Asia	North and Central America
India	Mexico
Pakistan	
Afghanistan	
Nepal *	
Iraq	

This disease is important because it is, unlike common bunt, extremely difficult to control by either seed or foliar treatment (Smilanick *et al.* 1987). The teliospores are larger than those of *T. caries* or *T. foetida* and have a thick, dark brown to black, reticulated wall.

Joshi *et al.* (1983) reported that the disease incidence depended on the weather conditions at the time of anthesis and gave the following data as crucial: maximum temperature 19-23°C, minimum temperature 8-10°C, and light showers to provide high atmospheric humidity. The relative humidity during wheat flowering should be at least 70% to have a high intensity of Karnal bunt.

High relative humidity and low temperature during flowering combined with excess fertilizer and lodging are considered ideal conditions for disease development (Zhang *et al.* 1984). These authors found that the optimum temperature for spore germination on water agar was between 15 and 22°C, with the maximum germination percentage reached after 3 weeks. Lower temperature delayed germination, and no germination was observed at 35°C. An irreversible inhibition of germination occurred only after exposure of up to 12 weeks at 35°C or -18°C. A 5-year-old sample showed good germination even after being exposed to 1 week at -18°C. Smilanick *et al.* (1985) reported similar cardinal temperatures for teliospore germination. They also found that teliospores that were covered with 2 mm of soil or agar failed to produce promycelia or sporidia at the surface. This implies that spores which are not at or very near the soil surface do not contribute to the infection. Warham (1988) found that the length of time between promycelium emergence (approximately after 7 days of incubation) and production of sporidia varied considerably.

Under field conditions the primary and secondary sporidia produced by germinating teliospores are wind dispersed and infect the spikelets by directly penetrating glumes and ovary wall (Wiese 1987). According to Royer and Rytter (1985), cultivars with tight glumes may be more resistant than those with loose glumes, and artificial inoculation by injecting the sporidia suspension into the boot resulted in a higher disease level than the inoculation of emerged spikes. Singh and Krishna (1982) found that the awn-emerging

stage is the most susceptible. The latest infection, although only at a low percentage, occurred at flowering. According to Dhaliwal *et al.* (1983) the disease spreads from the site of primary infection to other florets within the spikelet, and from there to adjacent spikelets; it is not a systemic infection.

Materials and methods

Conventional planting and harvesting times in areas growing the crops were identified from the literature. If a crop was planted in two seasons, e.g., rice, both were considered. Inevitably there are inaccuracies, because varietal differences and other factors can not be taken into account. Also, planting may extend over more than one month. Information on rice seasons is according to Mohr (1969), Matsuo (1975), Buddenhagen and Persley (1978), Mikkelsen and De Datta (1980), IRRI (1980, 1983) and TARC (1987). Chickpea cropping patterns are according to Saxena and Singh (1984, 1987) and ICRISAT (1990). Planting and flowering times of wheat were taken from Peterson (1965), Martin *et al.* (1976), Hanson *et al.* (1982), CIMMYT (1985), ICARDA (1988, 1989) and FAO (1990). The areas were then classified in two groups: areas where the respective disease is known to occur regularly ("disease" or "risk" group) and areas where it has not been reported ("non-disease" or "non-risk" group, Tables 8, 9 and 10).

Monthly data for mean daily maximum and mean daily minimum temperature, mean precipitation, mean number of days with precipitation, and mean windspeed were selected from standard stations (Müller 1982) in the areas where the respective crops are grown. In addition to the original data, simple transformations such as the product of rain and wind, the difference between maximum and minimum temperature, the quotient of rain and number of rainy days, or the quotient of rain and minimum temperature were included. Such transformed data may be more meaningful for the epidemiology of a disease than the original data. Because of the significance of weather conditions just before and during flowering for the infection of wheat with *T. indica*, in addition to the climatic data for the first five months of the vegetation, those for the month of flowering were analyzed.

Stepwise discriminant analysis (SDA, Afifi and Clark 1984; Jennrich and Sampson 1985) was applied to the data sets. This technique can be used to classify an individual (here: location) into one of two alternative groups (here: climatic conditions favorable or unfavorable for disease development) based on a set of measurements (here: climatic parameters). At each step the variable that adds most to the discrimination between the groups is entered into the discriminant function. A classification criterion is developed, using a measure of generalized square distance (Mahalanobis D^2), based on the pooled covariance matrix. The D^2 is related to the R^2 in a multiple regression analysis, taking into account also the correlation between variables. When this value is larger for a group other than where it was originally classified, a misclassification occurs. The "posterior probability" of a case belonging to a particular group indicates the likelihood of a case belonging to one group. The resulting linear function (Fisher discriminant function) can be used to classify new units (here: locations) not used in the development of the function (Afifi and Clark 1984). For each disease approximately 100 different functions were tried.

Stepwise discriminant analysis has been widely used in taxonomy, e.g., by Hindorf (1973), to separate different species of *Colletotrichum* on coffee or in plant genetics, e.g., by Erskine *et al.* (1989). The technique was used in the development of a prediction model for *S. sclerotiorum* on winter rapeseed by Ahlers and Hindorf (1987). Madden and Ellis (1988) employed discriminant analysis to predict mild or severe levels of infection with maize dwarf mosaic in Ohio.

Results

Bacterial leaf blight of rice. The climatic factors that were contributing most to the discrimination between disease and non-disease locations were: mean daily maximum temperature in month 1 of the vegetation, mean daily minimum temperature in month 2 of the vegetation, and mean precipitation in month 3 of the vegetation. The resulting canonical correlation (Jennrich and Sampson 1985) is 0.7467. F-values are given in Table 11.

The long-term average data for these climatic parameters are listed in Table 12 for all locations analyzed. In Table 13 Mahalanobis D^2 values, which indicate the distance between the point representing an individual location and the point representing the estimated group mean, are given. If Mahalanobis D^2 is relatively large in the group in which a location is classified, the individual location is relatively distant from the group mean. We have this situation in the case of Montgomery, originally classified in the "non-risk" group, with a Mahalanobis D^2 of 3.0 from the "non-risk" group mean, and 1.0 from the "risk" group mean. This indicates that the location is closer to the mean of the "risk" group and belongs to the "risk" group rather than to the "non-risk" group. Consequently, the probability of belonging to the "non-risk" group is low: 0.274, which means the probability that the location belongs to the "non-risk" group is only 27.4% and the probability of belonging to the "risk" group is 72.6%.

We find the same situation with the location Charleston. Mahalanobis D^2 from the "non-risk" group mean is 6.4, whereas from the "risk" group mean it is only 1.7, indicating that this location should be classified in the "risk" group. The probabilities are 8.4% for the "non-risk" group and 91.6% for the "risk" group. The location Wuhan, in contrast, has only a probability of 11.5% of belonging into the "risk" group where it was originally classified.

A critical evaluation of other posterior probabilities in Table 13 shows that relatively low probabilities (0.6-0.8) of correct classification were calculated for Atlanta, Georgia in the "non-risk" group, as well as for Chengdu, China, for the first season at Colombo, Sri Lanka, and for Bobo Dioulasso, Burkina Faso in the "risk" group. These locations were correctly classified because Mahalanobis D^2 is smaller in the group they were originally classified in; however, the probability of belonging to this group is only 60-80%, whereas it is 100% for example for Dagoretti, Kenya, in the "non-risk" group and for Karachi, Pakistan, in the "risk" group.

Table 8. Meteorological stations, rice-growing seasons, and occurrence of bacterial leaf blight (BLB, *Xanthomonas campestris* pv. *oryzae*).

Location of station	Season analyzed	BLB†
Majunga, Madagascar	November - March	–
Tamatave, Madagascar	November - March	–
Dagoretti, Kenya	May - October	–
Freetown, Sierra Leone	July - October	–
Monrovia, Liberia	August - November	–
Bouake, Ivory Coast	April - July	+
Bobo Dioulasso, Burkina Faso	August - November	+
Niamey, Niger	August - November	+
Veraval, India	July - November	+
Kanpur, India	July - November	+
New Delhi, India	June - October	+
Hyderabad I, India	April - August	+
Hyderabad II, India	August - January	+
Patna, India	June - September	+
Ahmadabad, India	June - September	+
Karachi, Pakistan	June - September	+
Wuhan, China	April - July	+
Chengdu, China	June - September	+
Djakarta I, Indonesia	January - April	+
Djakarta II, Indonesia	April - August	+
Niigata, Japan	June - October	+
Colombo I, Sri Lanka	March - August	+
Colombo II, Sri Lanka	Sept. - February	+
Manila, Philippines	March - July	+
Batdambang, Kampuchea	Sept. - January	+
Phnum Penh, Kampuchea	Sept. - January	+
Nakhon Ratchasima, Thailand	May - October	+
Bangkok, Thailand	May - October	+
Houston, USA	April - August	+
San Antonio, USA	May - August	+
New Orleans, USA	April - August	+
Charleston, USA	May - August	–
Atlanta, USA	May - August	–
Montgomery, USA	May - August	–
Belo Horizonte, Brazil	October - February	–
Porto Alegre, Brazil	October - February	–
Caetite, Brazil	November - February	–

† +: Bacterial leaf blight reported to occur; –: bacterial leaf blight not reported to occur.

Table 9. Meteorological stations, chickpea-growing seasons and occurrence of Ascochyta blight (*Ascochyta rabiei*).

Location of station	Season analyzed	Ascochyta†
Palermo, Italy	January - May	+
Rome, Italy	February - June	+
Sevilla, Spain	March - July	+
Granada, Spain	March - July	+
Casablanca, Morocco	December - April	+
Rabat, Morocco	December - April	+
Oran, Algeria	January - May	+
Algiers, Algeria	January - May	+
Tunis, Tunisia	March - July	+
Alexandria, Egypt	November - March	+
Amman, Jordan	December - April	+
Aleppo, Syria	December - April	+
Erzurum, Turkey	April - August	+
Bursa, Turkey	April - August	+
Ankara, Turkey	March - July	+
Izmir, Turkey	January - May	+
Teheran, Iran	March - July	+
Addis Ababa, Ethiopia	July - November	+
Addis Ababa, Ethiopia	Sept. - January	−
Lusaka, Zambia	February - June	−
Blantyre, Malawi	February - June	−
Peshawar, Pakistan	November - March	+
Katmandu, Nepal	November - March	−
New Delhi, India	November - March	+
Raipur, India	November - March	−
Hyderabad, India	November - March	−
Narayanganj, Bangladesh	November - March	−
Saskatoon, Canada	May - September	+
Spokane, USA	April - August	+
Walla Walla, USA	April - August	+
San Francisco, USA	April - August	−
Fresno, USA	February - June	−
Guaymas, Mexico	December - April	+
Cordoba, Argentina	June - October	−
Valparaiso, Chile	September - January	−
Melbourne, Australia	May - September	+

† +: Ascochyta blight reported to occur; −: Ascochyta blight not reported to occur.

Table 10. Meteorological stations, wheat-growing seasons, and occurrence of Karnal bunt (*Tilletia indica*).

Location	Planting	Flowering	Karnal bunt†
Omaha, USA	September	May	-
Des Moines, USA	September	June	-
Spokane, USA	October	June	-
Dodge City, USA	October	May	-
Oklahoma City, USA	October	May	-
Tulsa, USA	October	May	-
Edmonton, Canada	September	June	-
The Pas, Canada	September	July	-
Winnipeg, Canada	September	June	-
Calgary, Canada	September	July	-
Hannover, Germany	October	June	-
Frankfurt, Germany	October	June	-
Erfurt, Germany	September	June	-
Rennes, France	October	May	-
Firenze, Italy	November	May	-
Plymouth, UK	October	July	-
Kijev, USSR	September	June	-
Valparaiso, Chile	June	October	-
Rosario, Argentina	June	September	-
Buenos Aires, Argentina	June	October	-
Guarapuava, Brazil	June	August	-
Porto Alegre, Brazil	June	August	-
Brasilia, Brazil	May	July	-
Adelaide, Australia	May	September	-
Perth, Australia	May	September	-
Shenyang, China	April	June	-
Xuzhou, China	October	May	-
Guangzhou, China	October	January	-
Nanjing, China	October	May	-
Wuhan, China	October	April	-
Bombay, India	November	February	-
Raipur, India	November	February	-
Surat, India	November	February	-
Nagpur, India	November	February	-
Aleppo, Syria	November	April	-
Konya, Turkey	November	February	-
Ankara, Turkey	October	June	-
Tanger, Morocco	December	March	-
Oujda, Morocco	December	April	-

Table 10 (continued).

Location	Planting	Flowering	Karnal bunt†
Cd. Obregon, Mexico	November	March	+
Katmandu, Nepal	November	March	+
Multan, Pakistan	November	March	+
Peshawar, Pakistan	November	March	+
Kabul, Afghanistan	October	May	+
Qandahar, Afghanistan	November	April	+
Ar-Rutbah, Iraq	November	April	+
Jodhpur, India	November	February	+
Indore, India	November	February	+
Kanpur, India	November	February	+
Allahabad, India	November	February	+
Jabalpur, India	November	February	+
New Delhi, India	November	February	+

† +: Karnal bunt reported to occur; −: karnal bunt not reported to occur.

Table 11. Stepwise order of inclusion of variables within the discriminant analysis together with the respective approximate F-statistics and Wilks' Lambda for *Xanthomonas campestris* pv. *oryzae*.

Step no.	Variable	Wilks' Lambda	Approx. F-statistic	df
1	Mean min. temp. month 2	0.6803	16.444	35
2	Mean rainfall month 3	0.4699	19.181	34
3	Mean max. temp. month 1	0.4424	13.863	33

Figure 12 shows climate diagrams for locations with a climate typical for the "risk" group and the "non-risk" group. In Karachi, climatic conditions with high temperature are conducive to disease development, whereas the conditions in Dagoretti/Nairobi with relatively low temperature make disease occurrence unlikely.

For the classification of further locations a discriminant function (y) was calculated from the classification functions:

$$y = -14.16 - 0.29\,x_1 + 1.19\,x_2 - 0.01\,x_3$$

x_1 = mean daily maximum temperature in month 1 of the vegetation
x_2 = mean daily minimum temperature in month 2 of the vegetation
x_3 = mean precipitation in month 3 of the vegetation.

The higher the risk index (y), the higher the probability of the disease occurring under the given climatic conditions. Table 14 gives some examples for this application of the method. The calculated risk index (y) agrees well with the reported distribution of the pathogen (Table 4). In the area of the Turkish Black Sea Coast (Samsun) the disease risk depends on planting time: it is higher with planting in June than in July.

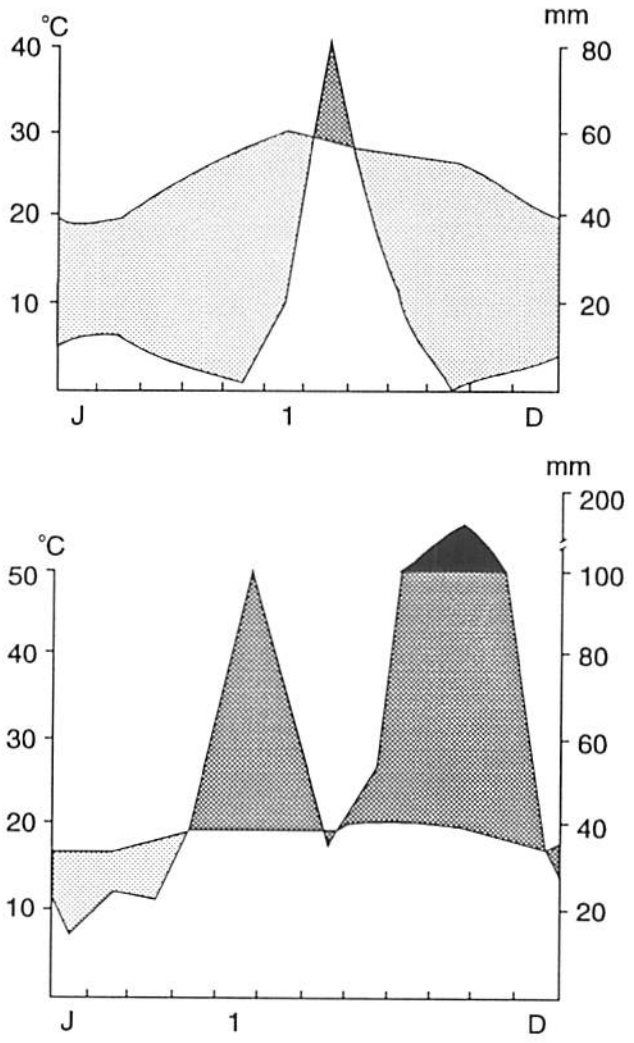

Fig. 12. Climate diagrams for locations conducive and non-conducive for the development of bacterial leaf blight of rice. Top: Karachi, Pakistan (4 m asl); bottom: Dagoretti/Nairobi, Kenya (1675 m asl); the first month of vegetation is marked "1" (after Walter and Lieth 1967).

Table 12. Climatic parameters used for discrimination of locations with and without *Xanthomonas campestris* pv. *oryzae*.

Location	Climatic parameters†		
	x_1	x_2	x_3
Caetite	28.3	17.8	120
Belo Horizonte	27.0	18.0	319
Porto Alegre	26.7	17.8	93
Montgomery	28.3	21.1	119
Atlanta	26.1	19.4	120
Charleston	26.7	22.8	196
Majunga	32.0	24.0	466
Tamatave	29.0	22.0	420
Dagoretti	23.0	11.0	17
Freetown	28.0	23.0	800
Monrovia	29.0	21.0	598
Veraval	28.9	25.6	64
Ahmadabad	38.3	26.1	206
Kanpur	34.4	26.1	142
New Delhi	38.9	27.2	173
Hyderabad I	38.3	26.7	112
Hyderabad II	30.6	22.2	64
Patna	36.1	26.7	333
Karachi	33.9	27.2	41
Wuhan	20.6	17.8	216
Chengdu	28.3	23.3	303
Djakarta I	28.9	23.3	211
Djakarta II	30.6	23.9	97
Niigata	23.9	20.6	107
Colombo I	31.1	24.4	371
Colombo II	29.4	23.9	315
Manila	32.8	22.8	128
Batdambang	31.1	23.3	85
Phnum Penh	31.1	24.4	134
Nakhon Ratchasima	34.7	23.7	120
Bangkok	34.2	24.9	168
New Orleans	28.3	23.3	180
Houston	28.9	22.2	131
San Antonio	30.3	22.2	53
Bouake	23.0	22.0	135
Bobo Dioulasso	29.0	21.0	74
Niamey	32.0	23.0	21

† x_1 = mean daily maximum temperature in month 1 of the vegetation.
x_2 = mean daily minimum temperature in month 2 of the vegetation.
x_3 = mean precipitation in month 3 of the vegetation.

Table 13. *Xanthomonas campestris* pv. *oryzae*: probabilities of locations belonging to the group originally classified in, as well as Mahalanobis D^2 for both groups.

Location	Originally classified†	Probability	Mahalanobis D^2	
			-	+
Caetite	–	0.951	2.1	8.0
Belo Horizonte	–	0.994	1.1	11.3
Porto Alegre	–	0.893	1.8	6.1
Montgomery	–	0.274	3.0	1.0
Atlanta	–	0.603	2.0	2.9
Charleston	–	0.084	6.4	1.7
Majunga	–	0.818	2.6	5.6
Tamatave	–	0.914	1.0	5.7
Dagoretti	–	1.000	13.6	29.7
Freetown	–	0.998	10.8	23.2
Monrovia	–	0.998	4.3	16.4
Veraval	+	0.999	18.6	4.7
Ahmadabad	+	0.941	9.5	4.0
Kanpur	+	0.992	10.6	1.0
New Delhi	+	0.987	12.7	4.0
Hyderabad I	+	0.992	13.2	3.6
Hyderabad II	+	0.915	5.3	0.6
Patna	+	0.914	7.4	2.7
Karachi	+	1.000	19.4	4.1
Wuhan	+	0.115	4.3	8.4
Chengdu	+	0.736	3.7	1.7
Djakarta I	+	0.894	4.8	0.5
Djakarta II	+	0.981	8.2	0.3
Niigata	+	0.863	7.0	3.3
Colombo I	+	0.638	3.3	2.2
Colombo II	+	0.777	3.9	1.4
Manila	+	0.825	4.3	1.2
Batdambang	+	0.962	6.7	0.2
Phnum Penh	+	0.980	8.0	0.2
Nakhon Ratchasima	+	0.898	6.2	1.9
Bangkok	+	0.956	6.8	0.7
New Orleans	+	0.939	6.2	0.7
Houston	+	0.874	4.3	0.4
San Antonio	+	0.932	5.8	0.6
Bouake	+	0.967	12.5	5.8
Bobo Dioulasso	+	0.782	3.7	1.1
Niamey	+	0.971	8.0	0.9

† –: *Xanthomonas campestris* pv. *oryzae* not reported to occur; +: *Xanthomonas campestris* pv. *oryzae* reported to occur.

Table 14. Examples for the prediction of bacterial leaf blight on rice (*Xanthomonas campestris* pv. *oryzae*) risk in various locations or planting time.

Area	Month of planting	Climatic parameters†			Risk index‡
		x_1	x_2	x_3	
Nagasaki, Japan	June	25.6	22.8	189	3.66
Hanoi, Vietnam	May	32.2	25.6	76	6.21
Pusan, South Korea	June	23.9	21.7	156	3.17
Makurdi, Nigeria	July	29.4	22.2	279	0.94
Eala, Zaire	June	30.6	17.8	178	−3.63
Venice, Italy	May	21.0	17.1	52	−0.42
Samsun, Turkey	July	26.1	18.3	61	−0.56
Samsun, Turkey	June	23.3	18.7	33	1.01

† x_1 = mean daily maximum temperature in month 1 of the vegetation, x_2 = mean daily minimum temperature in month 2 of the vegetation, x_3 = mean precipitation in month 3 of the vegetation.
‡ $y = -14.16 - 0.29\,x_1 + 1.19\,x_2 - 0.01\,x3$.

Ascochyta blight of chickpea. The climatic factors that contributed most to the discrimination between disease and non-disease locations were: mean daily temperature in month 1 of the vegetation, mean precipitation in month 2 of the vegetation, average precipitation per rainy day in month 1 of the vegetation, average precipitation per rainy day in month 2 of the vegetation, mean number of days with precipitation in month 1 of the vegetation, mean number of days with precipitation in month 2 of the vegetation. The canonical correlation is 0.7556. F-values are given in Table 15.

The climatological data for all locations analyzed are listed in Table 16. Table 17 shows that some locations were initially misclassified, namely Fresno, California, USA which should be in the disease group, and Guaymas, Mexico, which was wrongly placed in the disease group. Consequently, these locations also have very low posterior probabilities (0.062 and 0.072). A critical evaluation of other posterior probabilities (Table 17) shows that relatively low probabilities between 0.587 and 0.715 were calculated for Blantyre, Malawi; Cordoba, Argentina, and Valparaiso, Chile in the "non-risk" group, and for Palermo, Italy in the "risk" group. These locations were correctly classified; however, the probability of a location belonging to this group is only 58-71%, whereas it is 100% for example for Narayanganj, Bangladesh, in the "non-risk" group and 99.9% for Melbourne, Australia in the "risk" group. Figure 13 shows climate diagrams for climatic conditions conducive to disease development (Melbourne, with a moderately humid climate during chickpea vegetation) and conditions that make disease occurrence unlikely (Narayanganj,

with an arid type of climate in the months after chickpea planting).

From the classification functions the following discriminant function was computed:

$$y = -1.11 - 0.22\,x_1 - 0.05\,x_2 - 0.32\,x_3 + 0.91\,x_4 - 0.51\,x_5 + 1.15\,x_6$$

x_1 = mean daily temperature in month 1 of the vegetation
x_2 = mean precipitation in month 2 of the vegetation
x_3 = average precipitation per rainy day in month 1 of the vegetation
x_4 = average precipitation per rainy day in month 2 of the vegetation
x_5 = mean number of rainy days in month 1 of the vegetation
x_6 = mean number of rainy days in month 2 of the vegetation.

In Table 18 some examples for the application of this method are given: for the areas of Plovdiv (Bulgaria), Bogota (Colombia) and Neustadt (Germany) the computed score indicates a disease risk, whereas for the Kenyan highlands no serious outbreaks of Ascochyta blight are to be expected. While in Bulgaria a high incidence of Ascochyta blight is reported (Nene 1982), the disease has not been reported from Colombia or Germany.

The disease risk depending on planting time in a location can also be assessed. In the area of Aleppo in northern Syria the disease occurs if chickpeas are planted in December (Table 9). If planting is delayed to March, the risk index of 0.76 is still positive, but lower than for December planting (3.05). A further delay of planting to April would result in a considerably decreased disease risk with an index of -3.46. Spring planting, although it reduces crop yields, is in fact a common practice of farmers in this area in order to avoid outbreaks of Ascochyta blight. In extraordinarily wet years, March plantings are affected by Ascochyta blight (K.B. Singh, pers. comm.).

Table 15. Stepwise order of inclusion of variables within the discriminant analysis together with the respective approximate F-statistics and Wilks' Lambda for *Ascochyta rabiei*.

Step no.	Variable	Wilks' Lambda	Approximate F-statistic	df
1	Mean temp. month 1	0.7546	11.059	34
2	Mean rainfall month 2	0.6333	9.553	33
3	Avg. rainfall/rainy day month 1	0.5782	7.780	32
4	Avg. rainfall/rainy day month 2	0.5348	6.740	31
5	Mean no. of rainy days month 1	0.4600	7.044	30
6	Mean no. of rainy days month 2	0.4290	6.432	29

Table 16. Climatic parameters used for discrimination of locations affected and not affected by Ascochyta blight.

	Climatic parameters†					
Location	x_1	x_2	x_3	x_4	x_5	x_6
Palermo, Italy	10.2	43	5.92	5.38	12	8
Rome, Italy	7.7	77	14.67	9.63	6	8
Sevilla, Spain	14.6	59	6.33	8.43	9	7
Granada, Spain	10.9	53	6.20	5.30	10	10
Casablanca, Morocco	13.5	80	10.42	6.67	12	12
Rabat, Morocco	13.6	66	8.60	7.33	10	9
Oran, Algeria	11.7	54	8.75	9.00	8	6
Algiers, Algeria	12.2	76	10.55	8.44	11	9
Tunis, Tunisia	12.6	42	4.78	6.00	9	7
Alexandria, Egypt	20.2	56	4.14	5.60	7	10
Amman, Jordan	10.3	69	9.20	8.63	5	8
Aleppo, Syria	7.8	89	8.40	8.09	10	11
Erzurum, Turkey	5.0	79	6.40	7.18	10	11
Bursa, Turkey	13.1	61	7.63	8.31	8	7
Ankara, Turkey	5.0	33	4.71	4.71	7	7
Izmir, Turkey	8.3	84	11.20	10.50	10	8
Teheran, Iran	9.4	36	9.20	12.00	5	3
Addis Ababa, Ethiopia I	15.0	294	20.14	18.38	14	16
Addis Ababa, Ethiopia II	15.6	21	14.77	7.00	13	3
Lusaka, Zambia	21.7	106	12.25	10.60	16	10
Blantyre, Malawi	24.0	125	14.92	11.36	12	11
Peshawar, Pakistan	16.4	18	8.00	9.00	1	2
Katmandu, Nepal	15.3	1	6.00	1.00	1	1
New Delhi, India	20.0	10	3.00	10.00	1	1
Raipur, India	22.5	5	13.00	5.00	1	1
Hyderabad, India	23.1	8	14.00	8.00	2	1
Narayanganj, Bangladesh	23.9	5	25.00	5.66	1	1
Saskatoon, Canada	11.2	58	4.86	5.80	7	10
Spokane, USA	9.5	33	3.11	3.67	9	9
Walla Walla, USA	12.1	38	4.00	4.22	9	9
San Francisco, USA	13.2	16	6.17	4.00	6	4
Fresno, USA	9.9	50	8.00	7.14	7	7
Guaymas, Mexico	19.4	12	8.50	4.00	2	3
Cordoba, Argentina	11.0	8	3.33	4.00	3	2
Valparaiso, Chile	12.9	16	15.00	8.00	2	2
Melbourne, Australia	12.5	54	3.80	3.25	14	16

† x_1 = mean daily maximum temperature in month 1 of the vegetation, x_2 = mean precipitation in month 2 of the vegetation, x_3 = average precipitation per rainy day in month 1 of the vegetation, x_4 = average precipitation per rainy day in month 2 of the vegetation, x_5 = mean number of days with precipitation in month 1 of the vegetation, x_6 = mean number of days with precipitation in month 2 of the vegetation.

Table 17. *Ascochyta rabiei*: probabilities of locations belonging to the group originally classified in and Mahalanobis D^2 for both groups.

Location	Originally classified†	Probability	Mahalanobis D^2 +	Mahalanobis D^2 −
Palermo, Italy	+	0.661	5.6	4.3
Rome, Italy	+	0.975	13.5	6.2
Sevilla, Spain	+	0.876	5.5	1.6
Granada, Spain	+	0.960	7.3	1.0
Casablanca, Morocco	+	0.921	7.6	2.7
Rabat, Morocco	+	0.868	4.3	0.6
Oran, Algeria	+	0.873	5.2	1.3
Algiers, Algeria	+	0.828	4.4	1.3
Tunis, Tunisia	+	0.818	4.7	1.7
Alexandria, Egypt	+	0.970	13.8	6.9
Amman, Jordan	+	0.992	12.2	2.5
Aleppo, Syria	+	0.994	11.8	1.6
Erzurum, Turkey	+	0.997	15.0	3.0
Bursa, Turkey	+	0.926	5.6	0.5
Ankara, Turkey	+	0.969	10.2	3.3
Izmir, Turkey	+	0.955	8.9	2.8
Teheran, Iran	+	0.982	16.3	8.3
Addis Ababa, Ethiopia I	+	0.939	32.4	26.9
Addis Ababa, Ethiopia II	−	0.999	13.0	27.1
Lusaka, Zambia	−	0.874	9.3	13.1
Blantyre, Malawi	−	0.587	8.3	9.0
Peshawar, Pakistan	+	0.868	8.5	4.8
Katmandu, Nepal	−	0.992	10.3	20.0
New Delhi, India	+	0.946	18.3	12.5
Raipur, India	−	0.995	2.6	13.1
Hyderabad, India	−	0.973	3.4	10.6
Narayanganj, Bangladesh	−	1.000	16.7	35.5
Saskatoon, Canada	+	0.995	12.8	2.3
Spokane, USA	+	0.965	8.6	2.0
Walla Walla, USA	+	0.939	7.0	1.5
San Francisco, USA	−	0.824	2.4	5.5
Fresno, USA	−	0.062	5.9	0.5
Guaymas, Mexico	+	0.072	2.7	7.8
Cordoba, Argentina	−	0.630	6.7	7.8
Valparaiso, Chile	−	0.715	5.4	7.3
Melbourne, Australia	+	0.999	25.7	12.4

† −: *Ascochyta rabiei* not known to occur, +: *Ascochyta rabiei* known to occur

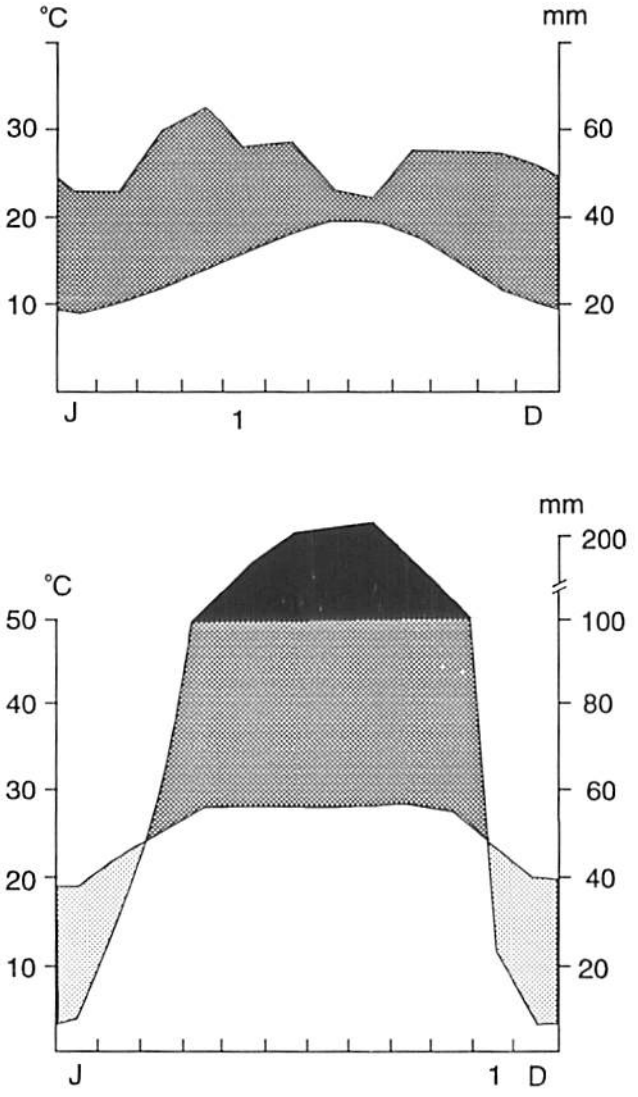

Fig. 13. Climate diagrams for locations conducive and non-conducive for the development of Ascochyta blight of chickpea. Top: Melbourne, Australia (38 m asl); bottom: Narayanganj, Bangladesh (8 m asl). The first month of vegetation is marked "1" (after Walter and Lieth 1967).

Table 18. Examples for the prediction of Ascochyta blight (*Ascochyta rabiei*) risk on chickpea in different locations or growth seasons.

Area	Month of planting	Climatic parameters†						Risk index‡
		x_1	x_2	x_3	x_4	x_5	x_6	
Plovdiv, Bulgaria	April	12.2	55	6.2	6.1	7	9	3.84
Kenyan highlands	June	15.7	17	5.8	3.4	5	5	-0.98
Bogota, Colombia	April	13.7	105	5.3	5.0	19	21	7.94
Neustadt, Germany	April	10.0	50	3.4	3.8	14	13	4.37
Aleppo, Syria	March	10.9	28	5.4	7.0	7	4	0.76
Aleppo, Syria	April	16.4	8	7.0	4.0	4	2	-3.46

† x_1 = mean daily maximum temperature in month 1 of the vegetation, x_2 = mean precipitation in month 2 of the vegetation, x_3 = average precipitation per rainy day in month 1 of the vegetation, x_4 = average precipitation per rainy day in month 2 of the vegetation, x_5 = mean number of days with precipitation in month 1 of the vegetation, x_6 = mean number of days with precipitation in month 2 of the vegetation.
‡ $y = -1.11 - 0.22\,x_1 - 0.05\,x_2 - 0.32\,x_3 + 0.91\,x_4 - 0.51\,x_5 + 1.15\,x_6$.

Karnal bunt of wheat. In the case of Karnal bunt the climatic factors that contribute most to the discrimination between disease and non-disease locations were all temperature related: the difference between mean daily maximum and mean daily minimum temperature in the month of planting, the mean daily maximum temperature in the month of flowering, and the mean daily minimum temperature in the coldest month of the year. The canonical correlation is 0.8419. F-values are given in Table 19.

The climatic data for these parameters are listed in Table 20 for all locations analyzed. Table 21 shows that all locations were correctly classified. An evaluation of the posterior probabilities indicates that for most locations the probability of belonging to the group they were classified in is very high. Relatively low probabilities of 0.587 and 0.708 were calculated for Konya, Turkey and Dodge City, USA in the "non-risk" group, and for Ar-Rutbah, Iraq (0.712) in the "risk" group. These locations were correctly classified; however, the probability of belonging to their respective group is only 58 and 71%. Typical climates favorable (high difference in daily minimum and maximum temperature in the month of planting and relatively high yearly temperature) and unfavorable for disease development are shown in Figure 14.

Table 19. Stepwise order of inclusion of variables within the discriminant analysis together with the respective approximate F-statistics and Wilks' Lambda for *Tilletia indica*.

Step no.	Variable	Wilks' Lambda	Approximate F-statistic	df
1	Diff. max. and min. temp. month 1	0.4123	71.285	50
2	Mean daily min. temp. in coldest month	0.3080	55.053	49
3	Mean daily max. temp. in flowering	0.2742	42.351	48

Table 20. Climatic parameters used for discrimination of locations affected and not affected by Karnal bunt (*Tilletia indica*).

	Climatic parameters†		
Location	x_1	x_2	x_3
Omaha, USA	10.5	22.2	-10.6
Des Moines, USA	11.6	26.7	-11.1
Spokane, USA	12.3	23.9	- 5.6
Dodge City, USA	14.5	23.9	- 8.3
Oklahoma City, USA	11.7	25.6	- 2.2
Tulsa, USA	4.7	25.7	- 2.2
Edmonton, Canada	13.4	21.1	-20.0
The Pas, Canada	12.3	23.9	-27.8
Winnipeg, Canada	12.2	23.3	-25.0
Calgary, Canada	15.0	24.4	-16.7
Hannover, Germany	7.7	20.7	- 2.5
Frankfurt, Germany	8.7	22.6	- 3.0
Erfurt, Germany	10.0	21.4	- 3.7
Rennes, France	8.5	18.4	1.8
Firenze, Italy	7.0	23.2	1.9
Plymouth, UK	5.4	19.0	3.5
Kijev, USSR	10.0	23.7	-10.2
Valparaiso, Chile	6.7	18.3	8.3
Rosario, Argentina	10.6	20.6	5.0
Buenos Aires, Argentina	8.9	20.6	5.0
Guarapuava, Brazil	10.0	21.1	7.8
Porto Alegre, Brazil	9.5	20.0	9.4
Brasilia, Brazil	12.0	26.5	12.5
Adelaide, Australia	8.9	18.9	7.2
Perth, Australia	8.9	19.4	8.9
Shenyang, China	13.3	28.9	-18.3

Table 20 (continued).

Location	Climatic parameters†		
	x_1	x_2	x_3
Xuzhou, China	14.5	27.2	- 6.1
Guangzhou, China	8.9	17.2	9.4
Nanjing, China	8.9	25.6	- 1.7
Wuhan, China	7.2	20.6	1.1
Bombay, India	8.9	28.3	19.4
Raipur, India	12.8	26.7	- 5.0
Surat, India	13.4	33.3	15.0
Nagpur, India	6.1	29.4	21.7
Aleppo, Syria	12.2	23.9	1.1
Konya, Turkey	15.0	26.7	- 5.0
Ankara, Turkey	13.9	25.6	- 4.4
Tanger, Morocco	7.0	18.0	9.0
Oujda, Morocco	10.0	25.0	5.0
Cd. Obregon, Mexico	17.5	27.1	12.8
Katmandu, Nepal	16.1	25.0	1.7
Multan, Pakistan	17.7	30.0	6.1
Peshawar, Pakistan	17.2	23.9	3.9
Kabul, Afghanistan	18.8	24.2	-13.6
Qandahar, Afghanistan	18.3	27.9	- 0.6
Ar-Rutbah, Iraq	14.5	25.6	1.1
Jodhpur, India	18.3	26.7	8.9
Indore, India	16.1	28.3	10.0
Kanpur, India	16.1	25.6	8.3
Allahabad, India	16.1	26.1	8.3
Jabalpur, India	16.1	27.8	7.8
New Delhi, India	17.8	23.9	6.7

† x_1 = difference between mean daily maximum and mean daily minimum temperature in the month of planting, x_2 = mean daily maximum temperature in the month of flowering, x_3 = mean daily minimum temperature in the coldest month of the year.

The discriminant function that was computed from the classification functions is:

$$y = -16.98 + 2.11\, x_1 - 0.52\, x_2 + 0.28\, x_3$$

x_1 = difference between mean daily maximum and mean daily minimum temperature in the month of planting

x_2 = mean daily maximum temperature in the month of flowering

x_3 = mean daily minimum temperature in the coldest month of the year.

Table 21. *Tilletia indica*: probabilities of locations belonging to the group originally classified in and Mahalanobis D^2 for both groups.

Location	Originally classified	Probability	Mahalanobis D^2	
			+	–
Omaha, USA	–	1.000	1.2	19.5
Des Moines, USA	–	1.000	2.6	21.2
Spokane, USA	–	0.992	0.6	10.1
Dodge City, USA	–	0.708	3.6	5.3
Oklahoma City, USA	–	0.997	0.4	12.4
Tulsa, USA	–	0.975	1.0	8.4
Edmonton, Canada	–	0.994	4.6	14.8
The Pas, Canada	–	1.000	7.7	29.9
Winnipeg, Canada	–	1.000	5.9	26.3
Calgary, Canada	–	0.923	4.3	9.3
Hannover, Germany	–	1.000	2.2	26.1
Frankfurt, Germany	–	1.000	1.3	23.3
Erfurt, Germany	–	1.000	0.5	16.0
Rennes, France	–	1.000	2.3	17.9
Firenze, Italy	–	1.000	3.6	30.6
Plymouth, UK	–	1.000	5.3	33.7
Kijev, USSR	–	1.000	1.9	23.6
Valparaiso, Chile	–	1.000	3.5	22.9
Rosario, Argentina	–	0.974	2.1	9.3
Buenos Aires, Argentina	–	0.999	1.0	15.4
Guarapuava, Brazil	–	0.987	1.7	10.4
Porto Alegre, Brazil	–	0.988	2.6	11.4
Brasilia, Brazil	–	0.831	3.4	6.6
Adelaide, Australia	–	0.997	2.7	14.1
Perth, Australia	–	0.996	2.6	13.5
Shenyang, China	–	1.000	6.3	24.0
Xuzhou, China	–	0.879	2.9	6.9
Guangzhou, China	–	0.985	5.5	13.9
Nanjing, China	–	1.000	2.7	26.2
Wuhan, China	–	1.000	2.3	26.2
Bombay, India	–	0.999	5.8	20.1
Raipur, India	–	0.838	5.8	9.1

Table 21 (continued).

Location	Originally classified	Probability	Mahalanobis D^2 +	Mahalanobis D^2 −
Surat, India	−	0.813	10.6	13.6
Nagpur, India	−	1.000	13.0	38.9
Aleppo, Syria	−	0.956	1.0	7.2
Konya, Turkey	−	0.587	3.8	4.5
Ankara, Turkey	−	0.873	2.2	6.0
Tanger, Morocco	−	1.000	3.6	21.1
Oujda, Morocco	−	0.999	0.5	14.9
Cd. Obregon, Mexico	+	1.000	20.7	1.1
Katmandu, Nepal	+	0.992	10.0	0.4
Multan, Pakistan	+	0.999	14.9	1.3
Peshawar, Pakistan	+	1.000	17.5	0.9
Kabul, Afghanistan	+	0.999	16.5	3.4
Qandahar, Afghanistan	+	0.999	15.0	0.5
Ar-Rutbah, Iraq	+	0.712	4.4	2.6
Jodhpur, India	+	1.000	22.3	1.2
Indore, India	+	0.996	11.7	0.9
Kanpur, India	+	0.998	12.9	0.2
Allahabad, India	+	0.998	12.3	0.2
Jabalpur, India	+	0.994	10.8	0.7
New Delhi, India	+	1.000	22.6	1.9

† -: *Tilletia indica* not reported to occur; +: *Tilletia indica* reported to occur.

A risk assessment was done for several wheat-growing areas not included in the analysis. The results are given in Table 22. Karnal bunt was not reported from the areas for which no disease risk is indicated (negative values). The disease, however, was not yet observed at either location in Pakistan (Begum and Mathur 1989) or in Ethiopia or Australia (Table 7). After the recent spread within India (D.V. Singh *et al.* 1985), a similar expansion of the infested area in Pakistan is not unlikely. The fact that areas in Ethiopia and Australia seem to have a climate suitable for the development of Karnal bunt warrants attention with respect to quarantine for infected/contaminated seed.

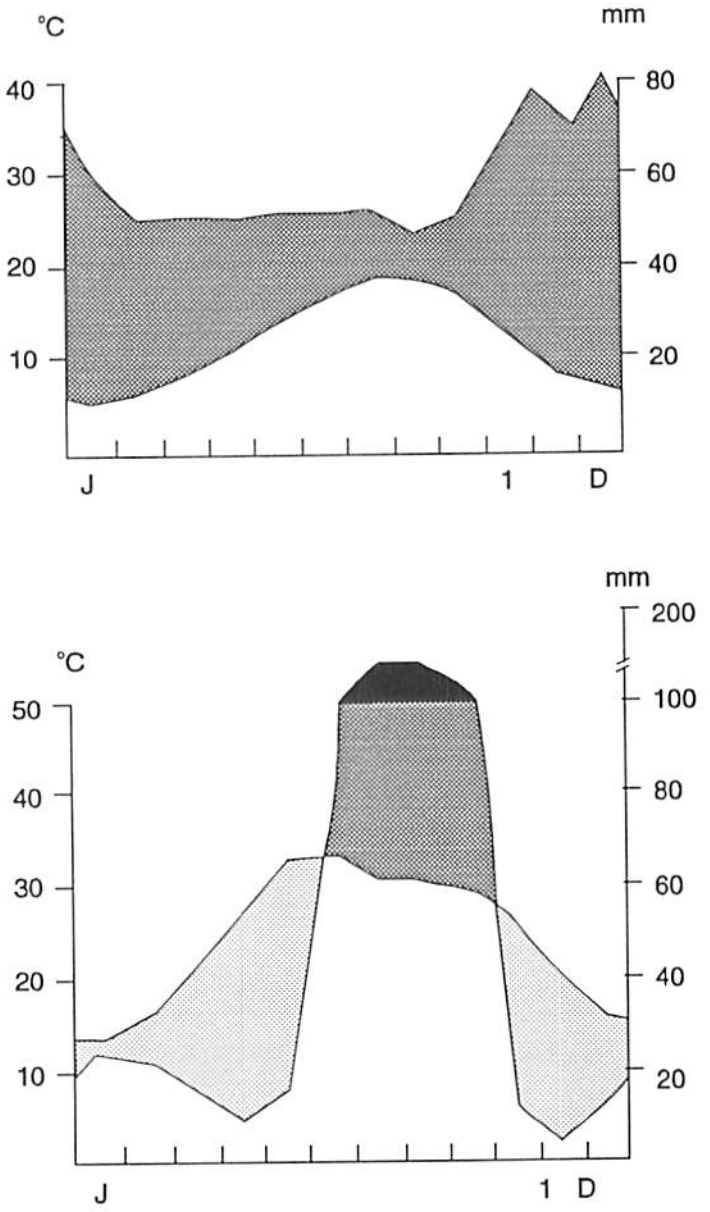

Fig. 14. Climate diagrams for locations conducive and non-conducive for the development of Karnal bunt of wheat. Top: Rennes, France (60 m asl); bottom: New Delhi, India (217 m asl). The first month of vegetation is marked "1" (after Walter and Lieth 1967).

Table 22. Examples for assessing the risk of Karnal bunt (*Tilletia indica*) occurring on wheat in additional locations.

Area	Planting	Flowering	Climatic parameters†			Risk index‡
			x_1	x_2	x_3	
Rabat, Morocco	November	April	9.0	20.0	9.0	−5.87
Kassel, Germany	October	June	7.9	21.7	−2.4	−12.27
Tunis, Tunisia	November	April	8.0	21.0	7.0	−9.06
Addis Ababa, Ethiopia	June	September	13.0	21.0	4.0	0.65
Quetta, Pakistan	November	April	18.9	23.3	−2.8	9.99
Hyderabad, Pakistan	November	February	16.7	27.2	10.6	7.08
Hay, Australia	May	September	13.3	20.6	3.3	1.30

† x_1 = difference between mean daily maximum and mean daily minimum temperature in the month of planting, x_2 = mean daily maximum temperature in the month of flowering, x_3 = mean daily minimum temperature in the coldest month of the year.

‡ $y = -16.98 + 2.11\ x_1 - 0.52\ x_2 + 0.28\ x_3$.

Discussion

Although the concept of geophytopathology was presented a long time ago (Reichert 1958; Weltzien 1967), relatively little research was done in this field of plant pathology. Recently, however, its potential merits were recognized by Coakley (1988) and Zadoks (1989).

As Rotem (1978) pointed out, occurrence of some pathogens is restricted to specific geographical zones, while others follow the distribution of their host. He quoted the example of potato late blight (*Phytophthora infestans*) which had been restricted to temperate and humid zones and then spread to tropical and semi-arid locations after the introduction of the potato to these areas. There are even cases where alternate hosts need to be present in order to allow disease development, such as *Berberis vulgaris* in the case of wheat stem rust (*Puccinia graminis*). If a new crop is cultivated in an environment only partially suitable for its growth, microclimatic changes are likely to occur, e.g., those created by irrigation in a semi-arid environment.

Plant disease occurrence has changed over the years. Some of these changes can be attributed to changes in climate or microclimate, others are due to other factors such as

1. Introduction of hosts to a new area where the pest or pathogen was already prevalent on indigenous weeds, e.g., introduction of the potato to North America, where it was readily attacked by the Colorado beetle (Gauthier *et al.* 1981).
2. Introduction of a pest/pathogen, e.g., *Phylloxera* to France, followed by *Plasmopara viticola* with *Phylloxera*-resistant rootstocks (Cowling 1978).
3. Introduction of an alternate host, e.g., barberry (*Berberis vulgaris*) to New England, with subsequent epidemics of *P. graminis* (Zadoks and Schein 1979).
4. Narrowed rotations, e.g., for potatoes and sugar beets in Germany and Holland, with the effect that cyst nematodes became a major problem (Palti 1981; Hoffmann and Schmutterer 1983).
5. Planting of susceptible germplasm in large areas, e.g., wheat varieties resistant to *P. graminis* (stem rust) showed little resistance to *P. recondita* (leaf rust), and when new varieties resistant to this disease were released, they proved to be susceptible to stripe rust (*P. striiformis*, Zadoks 1989).
6. Management practices, e.g., soil compaction resulting from traffic and machinery factors can increase the incidence of root diseases (Allmaras *et al.* 1988).
7. Irrigation often promotes diseases, either by pathogen dispersal or by creating conditions conducive for infection (Palti 1981).

In geophytopathological considerations based on climate data it is difficult to include the factors illustrated above. Irrigation in particular will change not only precipitation quantity and frequency, but also microclimatic factors like canopy temperature. Another problem faced in the development of the models was the difficulty of finding data on cropping patterns in a wide range of climates and on the distribution and importance of the diseases.

However, the examples presented in Chapter 3 provide sufficient evidence that valid predictions of disease occurrence in an area can be based on the area's climatic data, the pathogen's ecological requirements, and the cropping patterns of the host plant(s). A precondition is of course the presence of the pathogen. While there are numerous examples of the international dispersal of fungi with wind, water, animals, plants or plant parts and people (Zadoks 1967), seeds seem to be most important in pathogen transfer because of their long viability, the close association of host and pathogen, and their facility for long-distance transport (Baker and Smith 1966). It seems reasonable to assume that owing to the ever-increasing international seed exchange, and regardless of quarantine measures, many seed-borne pathogens will be spread to areas where they did not occur previously. The geophytopathological approach can help to assess the risk of seed-borne pathogens becoming established under certain climatic conditions. Given that other conditions for the occurrence of a disease, namely inoculum and a susceptible plant, are present, weather factors determine whether or not the disease will develop. Several variables and their interaction are involved.

The principle of adapting a weather-based linear regression model developed for disease forecast for a particular year, location and variety to the appraisal of long-term disease risk in an area is demonstrated with the prediction model of yellow rust severity (Coakley *et al.* 1982, 1983, 1984). Similar models are not yet available for seed-borne diseases. The model was modified to accept climatic data instead of weather data. The validation of the model with data from different locations showed that the predicted "risk index" agreed well with the known distribution of the disease.

Models were developed specifically for selected seed-borne diseases by using stepwise discriminant analysis. The comparison of climatological parameters from areas affected by bacterial leaf blight of rice and areas where the disease has not been reported indicated that temperature is the most decisive factor, as reported by Premalatha Dath *et al.* (1979). Numerous locations where the disease was not reported to occur are characterized by high rainfall, e.g., Majunga, Tamatave, Freetown and Monrovia (Table 12). Although humidity is necessary for disease development, excess rainfall may have a detrimental effect on the disease, e.g., by washing off the inoculum. This has been observed in high-rainfall areas in Colombia, where legumes remain largely free from fungal diseases because of this effect (A. van Schoonhoven, ICARDA, pers. comm.). Palti (1981) cited several examples of irrigation reducing air-borne inoculum.

Some locations were not classified into the risk groups on the basis of disease occurrence reported in the literature (Table 13). Wuhan, China is characterized by low daily minimum and maximum temperatures during the first two months of vegetation. For the Montgomery, Alabama and Charleston, South Carolina locations, none of the climatological parameters alone would indicate that there is a disease risk; however, their combination seems to allow for development of the disease. With the understanding that the first report of the disease in the USA was from Texas in 1987, and then from Louisiana in 1988 (Jones *et al.* 1989), the pathogen could be expected to spread, e.g., with infected seeds, to areas with climates that favor disease development, such as Montgomery, Alabama and Charleston, South Carolina. The relatively low posterior probability for Atlanta, Georgia could indicate a disease risk under certain conditions,

such as very susceptible varieties or unusually favorable weather in a particular year. Similarly, the low posterior probabilities for the first season at Colombo, Sri Lanka and Chengdu, China as well as Bobo Dioulasso, Burkina Faso in the "risk" group suggest that conditions for disease development may not be ideal in these locations. These areas are not listed among the most-affected countries by Agarwal *et al.* (1989).

Of the six parameters contributing to the discrimination of locations affected and not affected by Ascochyta blight of chickpea, five are related to rainfall in the first two months of vegetation, and the sixth is the mean daily temperature in the first month after planting. Particularly important are obviously the precipitation parameters in the second month of vegetation, i.e., rainfall, number of days with rainfall, and the average rainfall per rainy day. The last is obtained by a division of the former two, and may be more important for the development of the disease. As Rotem (1978) pointed out, the rainfall pattern is more important for the development of an epidemic than the total amount of rainfall. He gave several examples for the pathogen-dependent effect of rainfall: frequent and evenly distributed rains are expected to be more favorable for foliage epidemics than occasional heavy but short periods of rain; the latter, however, may support epidemics by washing off fungicides or by promoting splash dispersal of spores. Prolonged periods of drizzle will favor pathogens requiring a long wetness duration for infection.

Coincidence of rain and certain growth stages of the crop also may be important, such as in the case of sunflower downy mildew, which needs rain during the first two weeks of the vegetative period, because only then are the seedlings susceptible to systemic infection.

Two coefficients are negative, namely the average precipitation per rainy day and the number of rainy days in month 2 of the vegetation. This indicates that locations with larger values of these variables may be classified more likely as "disease risk." Both parameters result in prolonged periods of leaf wetness, which is favorable for an epidemic of fungi requiring leaf wetness for infection, such as Ascochyta blight. Rainfall in the second month of vegetation (x_2) has a positive coefficient which seems to counteract the effect of the previous two parameters. A comparison of the standardized coefficients ($x_2 = 0.09$, $x_4 = -1.70$, $x_6 = -2.14$) indicates that this is the case only to a relatively small extent. The average precipitation per rainy day and the number of rainy days in month 1 of the vegetation, as well as the mean temperature in the same period, also neutralize the effect of x_4 and x_6: if they are high, the computed score is more likely to be positive and thus the disease risk tends to be lower. The reason for this could be that high temperature and rainfall parameters boost plant growth shortly after planting and that their effect on disease development is relatively low during the period of germination and seedling growth.

Ascochyta blight has not been reported from California (Buddenhagen *et al.* 1988). The climatic data of Fresno, California, however, clearly indicated that there is a disease risk (posterior probability 94% for the risk category). Cool temperature in the first month and high rainfall in the first two months of vegetation account for suitable conditions for disease development. Chickpea is a relatively new crop in the USA and Ascochyta blight was introduced to the states of Washington and Idaho in the early 1980s (Kaiser and

Muehlbauer 1984). Care should be taken to restrict the movement of infected seeds to California.

The other misclassification was Guaymas on the Mexican Pacific coast. High temperatures in the month of planting and low precipitation present unsuitable conditions for disease development. Occurrence of the disease in Mexico is only mentioned by Nene and Reddy (1987), and possibly epidemic development was not observed by other authors.

Karnal bunt of wheat is a disease with a limited distribution. The fact that this disease had not been reported from wheat-growing areas other than those listed in Table 7 could be due to the difficulty in detecting the symptoms in the field. Wiese (1987) described Karnal bunt as not conspicuous because the glumes are not noticeably distorted. Moreover, typically only part of the seed near the embryo is bunted. However, the easy distribution of the pathogen with seeds, e.g., in railway boxcars at the Mexico/USA border (Martin 1989) or in germplasm samples (Lambat *et al.* 1983), renders it more likely that the disease does not occur in many locations because of the pathogen's ecological requirements.

Three temperature parameters were sufficient for a discrimination. Disease-risk locations are characterized by a high difference between daily maximum and minimum temperatures in the month of planting, a relatively low daily maximum temperature in the month of flowering, and mild winters. That moderate maximum temperatures during flowering are conducive to disease development was reported by Joshi *et al.* (1983) and Zhang *et al.* (1984). Very cold winters may affect teliospore survival in the soil, although Zhang *et al.* (1984) found little effect of cold temperature in the laboratory.

All locations were correctly classified in the appropriate group. In the risk group Ar-Rutbah, Iraq was assigned a probability of only 71%, whereas all other locations where the disease had been reported reached a 99 or 100% probability. Information on incidence and severity of the disease in Iraq is scarce; there are no reports other than a mention in the CMI distribution map (Commonwealth Agricultural Bureaux 1974). The low probabilities of Konya, Turkey and Dodge City, Kansas, USA in the disease group indicate that there is a possible disease risk, e.g., in years with particularly favorable weather or with extremely susceptible varieties.

The validation of the three models with additional data sets largely confirmed the distribution of the diseases as indicated in the literature. For bacterial leaf blight of rice the risk on the Turkish Black Sea coast depends on the month of planting. The same result was obtained for Ascochyta blight in Aleppo, Syria. The effect of planting times on diseases is well known among farmers all over the world and was summarized by Palti (1981). A disease risk for Ascochyta blight is indicated for the area of Bogota, Colombia for chickpea planted in April, and care should be taken not to introduce the pathogen. No chickpea is grown in Germany, and a reflection on a disease risk in absence of the crop seems very theoretical. However, farmers could possibly decide that chickpea is a valuable crop in the diversification of farming systems, and in such a case it would be advisable to be prepared for potential disease risks.

In the case of Karnal bunt, the risk index indicates a possible spread of the disease within Pakistan and also a possible occurrence in areas in Ethiopia and Australia. Attention should be devoted to keeping infected or contaminated seeds out of these countries to avoid the establishment of the disease.

The following *caveat* must be considered in the application of the models: if climatic conditions at a location to be evaluated are dramatically different from those that were used in the development of the model, then the result will be misleading. Coakley *et al.* (1988) also pointed out that a correct prediction of stripe rust severity cannot be expected whenever input data exceed the range of the modeled data. In the Ascochyta model, for example, locations where chickpeas are grown with irrigation should be evaluated with caution because the rainfall parameters that largely constitute the model become insignificant.

Undoubtedly disease development depends more on microclimate in a crop and weather in any particular year than on average data. However, although a one-time favorable constellation of inoculum, optimum weather conditions, and presence of a susceptible host plant will result in an infection, it can be assumed the disease will not cause damage or might even remain unnoticed if such conditions do not prevail over a longer period of time. Bourke (1970) emphasized that usually three to five generations of a fungus precede the first visual appearance of a disease; hence, at least three consecutive periods of favorable weather are necessary for establishment of a disease.

Use of climatic, i.e., long-term average data in a discriminant function therefore seems acceptable to classify an area as one of "risk" or "non-risk". A "no disease risk" result, however, does not mean that the disease will never occur in the area; outbreaks may result from favorable weather in a particular year or from planting of new susceptible varieties. However, the risk of the disease developing to epidemic proportions is low in these areas. In particular, those locations that receive a score close to zero should be considered "areas of sporadic attack" *sensu* Weltzien (1972). Because of the initial grouping in the two categories "disease location" and "non-disease location" the classification of locations not included in the analyses was also in two groups "risk" and "non-risk". However, the situation is more complex than that. Weltzien (1978) presented a model (Fig. 15) that can help to interpret the results obtained in this study. According to this model, which considers in addition to the presence of the host only the two factors temperature and wetness, there are three disease zones:

Zone I or main damage area: both factors (temperature and wetness) favorable for the pathogen
Zone II or marginal damage area: one factor in the optimum range, the other not
Zone III or area of sporadic attack: both factors not in the optimum range.

The risk index that was calculated with the classification functions takes into account more factors (between three and six in the analyses described). It ranges between −10 and +10 in the examples given. The locations with high positive scores are equivalent to "main damage areas" (Zone I), those with high negative scores to "areas of sporadic attack" (Zone II), whereas those that score around zero correspond to Weltzien's

"marginal damage areas" (Zone III). Arbitrarily those in the range between −2 and +2 can be considered in this category.

The parameters employed in the discrimination of the locations are not necessarily in a causal relationship with the pathogen development. The pathogens' ability to compensate for unfavorable conditions may account for disease occurrence in areas which do not seem conducive for an epidemic. Rotem (1978) presented three hypotheses of compensation:

1. "A highly favorable state of one factor essential for development of a given phase in the life cycle of a pathogen can compensate for the limitations imposed by the simultaneously unfavorable state of another factor." This is the case, for example, with *Plasmopara viticola*, where a constant temperature sum of 50 degree-hours during the wetness period is required for infection (Blaeser and Weltzien 1979).
2. "A specific 'weakness' in a pathogen can be compensated for by a specific 'strength'." *Phytophthora infestans* requires short wetness periods to infect potatoes and so compensates for the sensitivity to desiccation (Rotem and Palti 1969).
3. "A high frequency of occurrence of one phase in the life cycle of a pathogen can compensate for a low frequency of occurrence of another phase in the life cycle of the same pathogen." Pathogens with a relatively low infection rate, such as loose smut of wheat and barley, compensate by the high number of spores dispersed (Hewett 1978).

Modeling is difficult under such complex situations. An improved model, for example, also should take into account the frequency of events unfavorable for disease development, such as prolonged dry spells. Nevertheless, the validation of the models presented yielded interesting results. The models could serve as a base to work out risk assessments which may be helpful in situations when a crop is introduced to a new area, in quarantine, or in the selection of areas with low disease pressure for seed production.

Although Reichert and Palti (1966) and Weltzien (1967, 1972, 1978, 1983) restricted their reflections to pathogens and diseases, respectively, the same principles also may be applied to insect pests. Blaeser-Diekmann (1982) reported that the occurrence of *Aphis fabae* on faba beans in Egypt was restricted to the Nile Delta area, whereas *Aphis gossypii* was prevalent in the Nile Valley, with a narrow zone in between where both species were found. *Aphis gossypii* requires higher temperatures than *A. fabae* and the temperature in the delta is lower than in more southern parts of Egypt. Noe and Sikora (1990) cited examples for "geophytonematology": the effect of climate on the distribution on plant parasitic nematodes, with the cyst-forming species *Heterodera* and *Globodera* being of greater importance in cooler climates and *Meloidogyne incognita* in subtropical and tropical areas.

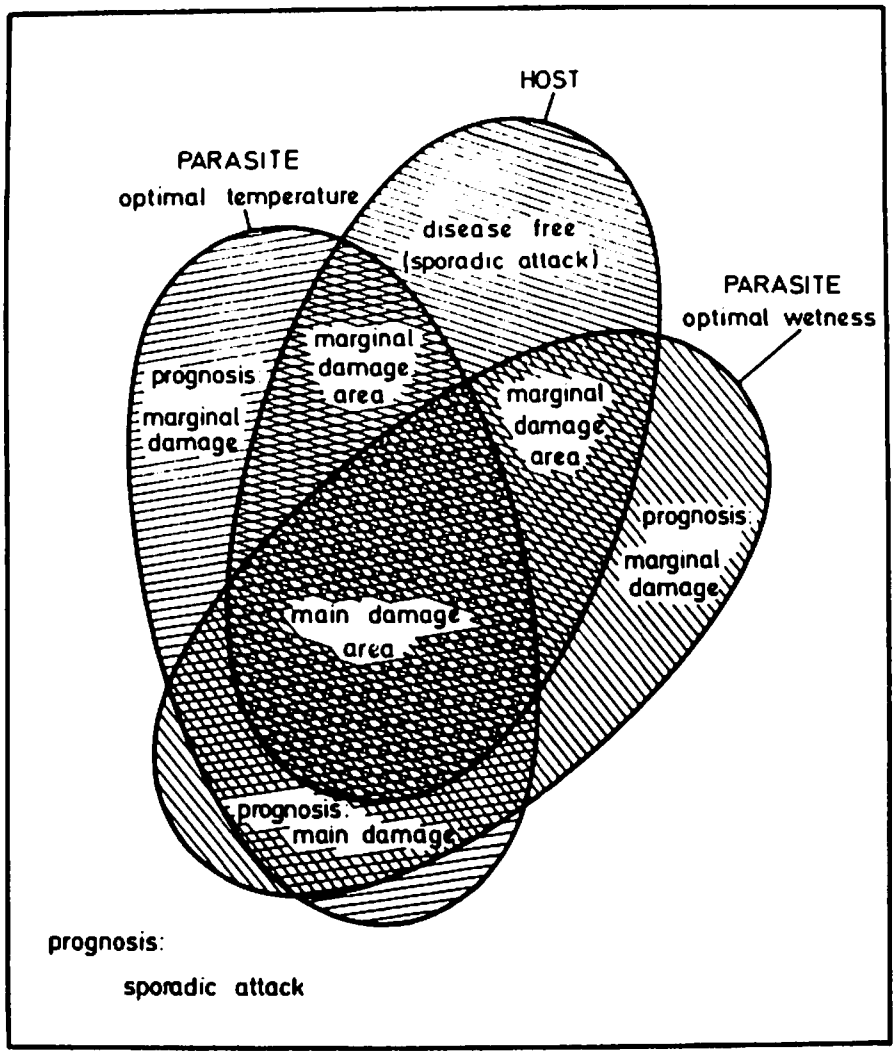

Fig. 15. Model to explain actual and potential occurrence of a plant pathogen, depending on distribution of the host and on the two predominant climatic factors temperature and wetness (from Weltzien 1978).

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