CHAPTER 9

Epidemiology and Inoculum Thresholds of Seedborne Pathogens

I. EPIDEMIOLOGY

Epidemiology is the study of the development and spread of disease inoculum and of the factors affecting these processes, and deals with the effects of the biotic (host and pathogen) and abiotic environment. Van der Plank defined epidemics as the science of disease in populations involving the persistence and spread of inoculum and environmental factors affecting disease incidence on particular plant populations. Epidemiological studies are important in disease forecasting and disease control. Van der Plank applied mathematical models to epidemiological studies of plant diseases. Plant disease epidemic refers to the development and rapid spread of a disease on a particular kind of crop cultivated over a large area. Epidemics develop when a susceptible cultivar is planted over a large area in the presence of a virulent pathogen coupled with a favorable environment. The development and decline of epidemics is a balance between inoculum potential and disease potential:

\[
\text{Disease severity} = \text{Inoculum potential} \times \frac{\text{Disease potential}}{\text{capacity}} \times \text{proneness} \times \text{susceptibility}
\]

Inoculum potential is the number of infective propagules (inoculum density) and their pathogenic capacity. Disease potential is host susceptibility, which may be influenced by an unfavorable environment, nutritional imbalances, and/or a susceptible growth stage. Host susceptibility is controlled genetically, and development of an epidemic depends on the number of infective propagules, their pathogenic capacity, host susceptibility, and the effect of the environment on pathogen virulence and host proneness to disease. These same factors affect the epidemiology of seedborne pathogens.
The rate of seed infection to seed transmission or plant infection and subsequent establishment in the field and further spread of inoculum depends on the host, pathogen(s), environment, transmitting agents, and their interactions. Yield losses may be high, even with a low-percentage seed infection by certain pathogens. For example, as few as two Brassica seeds per 10,000 infected with X. campestris pv. campestris,4 less than 1% X. campestris pv. vesicatoria-infected tomato seeds,5 or 0.02% P. syringae pv. phaseolicola-infected seeds in bean6 can produce disease epidemics caused by these pathogens under suitable environmental conditions. The incidence of X. campestris pv. vignicola may be 62% from an initial inoculum of 1% infected cowpea seeds.7 If lettuce seed lots carry over 0.5% lettuce mosaic virus, significant yield losses may result if aphids are prevalent.8

Environmental conditions greatly influence the epidemiology of seedborne pathogens. During the summer in India (relative humidity 20 to 80%, and 25 to 34°C) the incidence of cowpea blight (X. campestris pv. vignicola) reaches 62% from an initial inoculum of 1% infected seeds, but the incidence of greengram leaf spot (X. campestris pv. phaseoli) is only 0, 3, and 32% from 1, 10, and 100% initial seed infestation, respectively. However, during the rainy season (relative humidity 50 to 95%, 24 to 30°C), both diseases become severe.7 As few as two infected seeds per 10,000 can cause an epidemic of blackrot (X. campestris pv. campestris) in cabbage. However, the environmental conditions influence the spread of primary inoculum. In 1976, seed infestations of 0.12, 0.06, and 0.02% resulted in epidemics. In 1977, epidemics resulted from infestations of 0.05% but not 0.01%.4 A population of 10⁴ to 10⁵ X. campestris pv. phaseoli per bean seed was required for production of infected plants under field conditions.9

A highly significant correlation was found between barley seed infection with Pyrenophora graminise and plant infection, tiller infection, and yield reduction. The ratio of infected seeds to infected seedlings equaled 1:0.4, of infected tillers to yield loss equaled 1:0.9; and of infected seeds to yield loss equaled 1:0.3. The threshold of seed infection at which production was not significantly lower than the control was 14%. Therefore, seed treatment was advisable under northern and central Italy conditions, when the percentage of seed infection in commercial seed lots was above this level. However, a tolerance near zero was recommended in prebasic and basic seeds.10 There was a direct correlation (r = 0.76) between the level of seed infection of A. brassicae and seedling infection in Brassica.11 The ratio of seedborne infection to seed rot and seedling mortality was 1:088 in canola and mustard.12 As the incidence of seed infection by Stagonospora nodorum at planting of wheat increased from 1 to 40%, the intensity of subsequent disease increased but the relationship of seed infection to disease was nonlinear. Seed infection of 10% supplied enough inoculum to cause a severe epidemic (Figure 9-1).13 A significantly high correlation (r = 0.86) between seed infection to plant infection was observed in loose smut of wheat, but the ratio of seed infection to plant infection differed between cultivars.14 The ratio of A. raphani infection of radish to infected plants approached 1:1.15 A high correlation (r = 0.76, P = 0.01) was shown between safflower seed contamination by teliospores of Puccinia calcitrane var. centaureae and incidence of disease.16

In France, 5 infected bean seeds per 1000 resulted in an epidemic of halo blight of bean, whereas 1 per 20,000 did not.17 Incidence of X. c. pv. campesiris in crucifers was high in field plots that contained 0.03% infected plants, but less disease developed when the initial level was 0.01%.18 Seedborne C. m. subsp. michiganensis did not affect tomato seed germination or seedling emergence, but primary cankers developed where the incidence had a ratio of 10:8 and 10:9 during 1987 and 1988, respectively. The seedborne pathogen also reduced yield.19 C. m. subsp. nebraskensis was transmitted to maize seedlings at 0.1 to 0.4% from seeds inoculated by vacuum infiltration.20 The possibility of transmission of maize white line mosaic virus in maize was less than 0.01%.21 Concomitans cascad-cadag viroid was seed transmitted at a rate of 1 in 300 coconuts.22

The threshold level for pathogenicity of any seedborne inoculum is not constant since it is affected by inoculum level, inoculum location, seed- and soilborne
microflora, soil temperature, moisture, pH, environmental factors, insect vectors, wind velocity, etc. Epidemics vary with different pathogens. For example, *Mycosphaerella pinodes* and *Phoma pinodella* caused greater reductions in yield than *Ascochyta pisi*. *A. pisi* required a higher incidence of seed infection than *M. pinodes* and *P. pinodella* to lower yields. The ratio of seed infection to plant infection varies depending upon cultivar; the rate of seed infection to tomato plant infection of *F. oxysporum* f. sp. *lycopersici* was 1:0.75, 1:0.70, and 1:0.36 for cvs. Monalbo, Eclaireur, and Exhibition, respectively.24

A. Classification of Seedborne Diseases Based on Epidemiology

Seedborne diseases can be classified into either monocyclic or polycyclic, based on their epidemiology.25

1. Monocyclic Diseases

These are diseases in which the seedborne pathogen is closely associated with the host after infection until symptom production, and reinfection of the host does not occur. However, inoculum transfer may result in seed infection. Epidemics due to such infections occur only when seeds are heavily contaminated or infected. Typical examples are barley and wheat loose smut and wheat bunt fungi, in which plants show symptoms only at ear formation. In cv. Sonora-64, a susceptible wheat genotype, a direct correlation was found between embryo and seedlings having more than 50% of the tissue invaded with hyphae and field expression of the disease.26

A system was developed in India for predicting loose smut of wheat utilizing the seed infection and transmission ratio. Seed infection of wheat loose smut was determined by an embryo count and seed transmission ratio based on tiller infection in the field. The data were fitted into the equation

\[ Y = KE \]

for calculating the predicted value of loose smut incidence, where \( Y \) = predicted incidence of loose smut, \( E \) = percent loose smut infection in the seed lot, and \( K \) = the constant for seed transmission of the loose smut pathogen in the cultivar used based on the previous 3 years.27

In the United Kingdom, the leaf stripe of barley pathogen does not re-infect leaves and was considered as a monocyclic disease. The initial seedborne inoculum is the total inoculum available each year.28 The diseases *Gloeotinia grani-gena* (blind seed disease) and *Claviceps purpurea* (ergot), which invaded only inflorescence, also were regarded as monocyclic diseases by Hewett,29 but conidia produced by both fungi on infected plants resulted in secondary spread. These diseases should be considered polycyclic diseases.

It is easier to control monocyclic diseases because the use of clean seed can be achieved using seed certification or seed treatment with systemic or nonsystemic fungicides.

2. Polycyclic Diseases

These are diseases in which seedborne pathogens after infection produce inoculum on the host capable of being carried to other host plants, which may result in disease (Figure 9-2). The seedborne inoculum gives rise to infected seedlings scattered throughout the field. The inoculum multiplies repeatedly and spreads throughout the growing period of the host when conditions are favorable.2 The inoculum may be carried by rain splash, wind, vectors, and other means. Epidemics of polycyclic diseases are influenced by the environment. Under unfavorable conditions even a low initial seedborne inoculum can result in an epidemic. Many seedborne pathogens belong to this group. Some examples are given.

Fava bean seeds infected with *A. fabae* produced seedlings with leaf lesions, inoculum from which spread for up to 10 m in an average season and usually infected the new seed crop. Seed lots with 1% infected seeds were suitable for crop production, but little or no *A. fabae* was tolerated in seeds intended for multiplication. Infection in British-grown commercial seeds was reduced by seed selection. Standards adopted in the seed scheme eliminated *A. fabae*. Theoretically, 1% seed infection gave one diseased seedling per 40 m² (18 cm between rows).29 *A. fabae* in *Vicia faba* increased from initial seed infection levels of 8.4 to 20% at harvest.30
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In Wisconsin and the United Kingdom, respectively. Under British conditions it was predicted that 0.025% seed infection would give rise to 0.0025% primary infection and that this would produce 4% infection in the mature crop with a $r$-value of 0.15. The model has been used to determine tolerance levels for seed infection and to compare the effectiveness of foliar sprays and seed treatments. A reduction in primary inoculum derived from infected seeds either by exclusion through seed testing or seed treatment gave effective disease control. Seed treatment reduced initial inoculum ($x_0$) and foliar sprays affected the rate of disease increase ($r$). Effective control of halo blight was achieved by seed treatment with the antibiotics kasugamycin or streptomycin, which reduced seed infection by 98%, or with the foliar sprays with cupper oxichloride or streptomycin sulfate.

It was found that 5 crucifer seeds per 10,000 infected with *X. c. pv. campestris* resulted in a high incidence of black rot, but a single diseased seedling did not. Laboratory seed assays capable of detecting 1 infected seed per 10,000 could predict field severity of black rot. Thus it was suggested that a tolerance of 1 infected seed per 10,000 be accepted for direct seeding of cabbage for head production, but a 0 tolerance be accepted for seeded production. Infected cabbage seeds with *X. c. pv. campestris* up to 1% resulted in a high disease incidence in the direction of the prevailing wind. The severity of black rot infection in brussels sprouts in central New York State was related inversely to the distance from infected transplants.

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Factors inherent in the host

Phytoalexins are toxins formed in host cells in response to certain stimuli, including products secreted from infection hyphae, germinating conidia, and application of substances such as CuCl₂ or HgCl₂ in suitable concentrations. Often microorganisms that stimulate formation of phytoalexins are those which do not actively parasite tissues. Production of phytoalexins is essentially a response of living cells; the substances are formed as a direct result of infection and appear as a local response to infection. Tissues of the healthy host are considered usually not to contain phytoalexins. The importance of phytoalexins in disease resistance remains to be established. Briefly, in the definition of Müller (1956), phytoalexins describe antibiotic principles present in hypersensitive host tissues.

A phytoalexin in pea, pisatin, has been studied in detail (Cruickshank and Perrin, 1961, 1963a, 1963b, and others). Pisatin may be produced in pods of pea by inoculation with conidia of Sclerotinia fructicola which is non-pathogenic to pea. The substance is fungistatic to a variety of fungi not normally parasitic on pea, as well as on six parasites on this host, but on only in higher concentrations. Pisatin is not produced by mechanical damage but as a response to drops of CuCl₂ or HgCl₂ placed on the pods, whereas under these conditions CaCl₂ and NaCl are ineffective. Cruickshank (1963a, 1963b) considers pisatin to be a critical factor in resistance of pea to fungal parasitic in this host.

A closely related phytoalexin is phaseollin which is induced by inoculation on French bean, Phaseolus vulgaris with conidia of S. fructicola, non-pathogenic to this host (Müller, 1958; Cruickshank and Perrin, 1963b). Bailey (1973) substantiated that phaseollin is induced when and where bean cells undergo necrosis and show browning as they do in response to incompatible hyphae or, as in his experiments, a virus, tobacco necrosis virus. There seems to be a connection between a rather "slow" process of cell death and phytoalexin production. Similar inhibitory substances have been induced in rice by Pyricularia oryzae, blast of rice, resistant cultivars producing more of the substance than susceptible cultivars (Uehara, 1958) and by Xanthomonas oryzae, bacterial blight of rice (Uehara, 1960). Other phytoalexins seem to be induced by species of Pseudomonas and Xanthomonas in Phaseolus bean (Klement and Lovernkovich, 1961, 1962, 1964). Post-invasion fungal inhibition produced in oats, resistant or susceptible to Drechslera victoriae (Nishimura and Scheffer, 1965; Samaddar and Scheffer, 1970), seems not to be due to phytoalexins because the inhibitions affect pathogenic and non-pathogenic forms equally.

Hypersensitivity to infection is of extreme importance; particularly in resistance to obligate parasites. Cells invade by the parasite, sometimes even in advance of invasion, are killed and therefore become unsuitable substrate for the parasite which commonly lives in living cells. The parasite is confined to a local necrosis and is eventually killed or sealed off. This reaction is equivalent to immunity.

Hypersensitivity has been studied extensively in Phytophthora infestans by Müller and his co-workers, in obligate rust and mildew fungi, and in viruses (local lesion reactions). Also non-obligate parasites may meet hypersensitive response in resistant plants, e.g. in resistant cultivars of Phaseolus bean to Colletotrichum lindemuthianum, anthracnose, in cotton to Xanthomonas malvacearum, bacterial wilt (Thiers and Blank, 1951).

Király, Barna and Ersek (1972) question the phytoalexin theory of resistance. Working with Phytophthora infestans, Puccinia graminis and Uromyces phaseoli they showed that both hypersensitivity reaction and production of phytoalexin, occurring simultaneously, are the effects of the pathogens' death, not the cause. An unknown resistance mechanism includes the release of endotoxins and causes host necroses and phytoalexin production. Van Der Plank (1975) regards phytoalexins as preformed antibiotics protecting the plant against secondary infections.

18.4 GENETIC IMPULICATIONS OF SUSCEPTIBILITY AND RESISTANCE

Inheritance of disease resistance in plants follows Mendel's laws. Bifen (1905, 1912) was the first to pronounce this. He demonstrated that the F₂ progeny of a cultivar, highly resistant to yellow stripe rust of wheat, Puccinia striiformis, crossed with a susceptible cultivar, segregated in the ratio of three susceptible to one resistant.

Inheritance of resistance to a definite disease may be monogenic, oligogenic or polygenic. The number of genes for resistance varies greatly with host and with pathogen, as does also the mode of inheritance.

Single dominant gene resistance has been established in tomato to race 1 of Fusarium oxysporum f.sp. lycopersici. Fusarium wilt (Bohn and Tucker, 1940), in pea to pea wilt, Fusarium oxysporum f.sp. pisi race 1 (Wade, 1929) and near wilt, F. oxysporum f.sp. pisi race 2 (Hare, Walker and Delwiche, 1949), in sorghum to milo disease, Periconia circinata, a widely distributed seed-borne soil inhabitant that produces an exotoxin causing disease in certain cultivars (Bowman et al., 1937; Leukel, 1948; Scheffer and Pringle, 1961), and in barley to loose smut, Ustilago nuda and black smut or semi-loose smut, U. nigrá (Cherewick and Buchannan, 1969). Zauemyer and Harter (1943a) showed that resistance in bean to bean southern mosaic virus is controlled by a single dominant gene. Resistance in some cultivars of onion to pink root, Pyrenochaeta terrestris, is controlled by a single recessive gene (Jones and Perry, 1956;
Factors inherent in the host

Nichols et al., 1965), and resistance in pea to pea (seed-borne) mosaic virus seems also to be conditioned by a single recessive gene (Hagedorn and Gritton, 1973). The outer-scale resistance of onion to smudge, Colletotrichum dematiu, is based on three gene pairs that determine the formation of the phenolic colour compounds which inactivate the pathogen (Rieman, 1931; Clarke, Jones and Little, 1944). According to the gene combination, colour of the outer-scale and disease reaction may range from red or yellow and high resistance through pink or cream and intermediate resistance to white and susceptibility.

Inheritance of resistance may not follow the same pattern in different cultivars. In two cultivars of barley resistance to net blotch, Drechslera teres, has been found conditioned by two dominant genes (Shands, 1951), in another cultivar by one completely dominant gene, possibly supplemented by one or more modifying genes (Schaller, 1955); three dominant genes were found in barley by Mode and Schaller (1958), and in soybeans between three susceptible and seven resistant and semi-resistant cultivars Omar, Selim and Khalifa (1970) established three dominant genes for susceptibility and three dominant genes for resistance, out of which were two for semi-resistance.

Resistance to specific pathogenic races of a pathogen can usually be referred to specific genes. Resistance against bean anthracnose, Colletotrichum lindemuthianum, race alpha, beta and gamma, is controlled by independent gene pairs, resistance being dominant (Andrus and Wade, 1942). Also resistance to flax rust, Melampsora lini, is dominant, based on many distinct independent genes. Flor (1946, 1947, 1954, 1955, 1959) has substantiated a theory of gene-to-gene relationship between resistance in the host and pathogenicity in the fungus, in which pathogenicity is recessive to non-pathogenicity. Each differential cultivar of a list of standard differentials, established by Flor, has a unique gene for resistance to flax rust. Twenty-six of such genes have been identified (Flor, 1955; Flor and Comstock, 1972).

In wheat resistance to bunt, Tilletia caries and T. foetida, is based on a number of major genes and some minor genes. Each of the dominant genes controls resistance against a certain group of races, e.g. M, gene seven races of T. caries and four races of T. foetida, while gene H controls resistance to eight races of T. caries and three races of T. foetida, some of which, however, are controlled by M. Omar was the first cultivar which combined these two genes for resistance (Holton et al., 1956). Factors for resistance to bunt may be strong or weak, they may be independent or linked, and effective against a single race or against several. By crossing resistant with susceptible cultivars there may be transgressive segregation (Fischer and Holton, 1957, who discussed in detail inheritance of factors for resistance to bunt, oat smuts and loose smut of barley and wheat).

Epidemiological implications of susceptibility (18.5)

While usually specific genes for resistance are related to specific pathogenic races, inheritance of general resistance, or field resistance, or horizontal resistance (discussed in Section 18.5) to non-specific pathogenic races is generally polygenic, though oligogenic inheritance also occurs. This type of resistance may be due to a range of general morphological and physiological characters working together in reducing possibilities for establishment of infection such as thickness of cuticle and epidermis, epicuticular wax coating, size and number of stomata, hairiness of leaves or fruits, closed or open flowers, chemical defences such as in onion against smudge, early maturation determining escape from late season diseases, etc.

Polygenic resistance is more subject to influence by environmental factors such as modifications of host nutrition than is monogenic resistance, which usually is completely stable but which may fail by the appearance of a new pathogenic race.

The importance of environment to the final effect of resistance is relevant to the testing techniques to be applied in breeding programmes. Standard conditions and optimum conditions for development of disease must be provided. In pea wilt monogenic resistance breaks down at high soil temperatures (Schroeder and Walker, 1942; Wells, Hare and Walker, 1949) and in acid soil (Hubbeling and Mathur, unpublished). Resistance to Fusarium wilt in cultivars of cotton, tomato and pea breaks down in the presence of parasitic nematodes (see Section 16.7) and resistance to dry rot, Phoma lingam, by cauliflower mosaic virus in cultivars of swede (Smith, 1960; see Section 16.14). In addition to field tests, breeding materials should be tested in plant houses where temperature, light and humidity can be controlled. In any breeding programme standard testing conditions must be provided which can be maintained uniform from season to season. An essential aspect is establishment of plant disease gardens where attempts are made to accumulate presently and potentially important pathogens under favourable conditions for abundant disease development. But here it must be emphasised that such 'nurseries of all evils' must be carefully established and controlled. Sometimes the accusation that the most important distributors of new plant pathogens are plant pathologists appears to be deplorably justified!

18.5 EPIDEMIOLOGICAL IMPLICATIONS OF SUSCEPTIBILITY AND RESISTANCE

Any resistance in crop plants carries implications for the population dynamics of disease development in crops. The character of resistance has direct bearing on the epidemics of the disease concerned.
Factors inherent in the host

In the previous sections mechanistic and genetic implications of resistance have been discussed. However, the mechanisms and genetics of resistance are not necessarily directly related to their epidemiological consequences.

There are two types of resistance in plants which may well be placed in the two epidemiological categories which Van Der Plank (1963) has called 'vertical' and 'horizontal'; other authors have used the terms 'specific' (race specific) resistance, and 'general' or 'generalised' (race non-specific) resistance (Stakman and Christensen, 1960). General resistance is almost synonymous with 'field resistance'. Useful discussions on the terminology of resistance have been given by Robinson (1969, 1971) and the Federation of British Plant Pathologists (1973).

Vertical (race specific) resistance operates against specific pathogenic races or pathotypes (i.e. 'vertical pathotypes') but not against others. It may be expressed as nearly complete resistance, immunity, as known in resistance of recent potato cultivars against certain races of potato blight, Phytophthora infestans. The response of host to pathogen is often a 'yes or no' response — the effect is qualitative.

The epidemiologic effect of this type of resistance is delay of the onset of the epidemic because of reduction of initial inoculum. The disease gets a poorer start.

Horizontal (race non-specific) resistance operates against all pathogenic races of a pathogen. It may be explained as that resistance which is left when a cultivar is attacked by all existing races. The response is a 'more or less' response — the effect is quantitative.

Characteristic of this kind of resistance is a reduction in the rate of increase which by definition includes the process of infection, incubation and reproduction. The development of the disease is slowed down.

The terms 'vertical' and 'horizontal' refer to the effect of the two types of resistance in a graphic presentation with a vertical and a horizontal axis (Van Der Plank, 1968; see Fig. 18.1). The advantages of these terms of mathematical origin are that they are free from ambiguity, that they are interrelated, and that their definitions have a mathematical precision. The terms have the virtue of recalling the diagrams vividly! In genetics the two terms are generally covered by the long-used standard terms 'qualitative' and 'quantitative' inheritance.

As mentioned, horizontal resistance is effective against all races and is independent of new races that may appear. But, as shown in Fig. 18.1, this kind of resistance may vary substantially from one cultivar to another, as well as from one host species or genus to another within a wide host range.

Obviously, horizontal resistance is by nature stable while vertical resistance is unstable. On the other hand, usually a higher degree of resistance is obtained by vertical than by horizontal resistance.

Epidemiological implications of susceptibility (18.5)

There is a certain risk of retrogression in breeding for resistance if in attempts towards vertical resistance the role of horizontal resistance is neglected. By improving vertical resistance one may obtain reduced horizontal resistance. If a cultivar with high vertical resistance but with a low horizontal resistance is crossed with a cultivar in order to improve the vertical resistance of this cultivar, the resulting hybrid may, in addition to better vertical resistance, receive the lower horizontal resistance of the parent cultivar. If the race population of the new cultivar is changed with a breakdown of the vertical resistance, then this cultivar is in a worse position than its predecessor. Loss of horizontal resistance due to negligence in breeding for vertical resistance has been termed the 'Vertifolia effect' because the potato cultivar Vertifolia (in which the vertical resistance is monogenic) provides a conspicuous example of this effect (Van Der Plank, 1968).

Breeding for resistance must take both types into consideration, as is observed in the current breeding of, for instance, rice for resistance against blast, Pyricularia oryzae, although little is known about horizontal resistance to blast (Ou, 1972).

![Fig. 18.1. Diagram of the resistance to potato blight, Phytophthora infestans, of the foliage of two potato cultivars, Kennebec and Maritta. The resistance is shown shaded to 16 races of the pathogen. Against certain races, e.g. (0), (2), (3) and (4), both cultivars show vertical and complete resistance. Against other races, e.g. (1), (1,2), (1,3) and (1,4), resistance is horizontal. This is small in Kennebec and moderate in Maritta. (Van Der Plank, 1968)
Crop epidemics depend, of course, on the susceptibility of cultivars in the crop concerned. Fields crops commonly involve large populations of plants which are genetically similar; many are clones, and they are usually of the same age and arranged uniformly in fixed positions. If all plants are equally susceptible and exposed to infection, and the relevant factors of weather are known, prognosis of an epidemic can be accomplished. When resistant cultivars are grown, then modifications in the course of the epidemic are introduced.

The rate of increase in any plant disease depends on the susceptibility of the host and the environmental conditions such as temperature and humidity. In the initial phase, when conditions begin to be adequate for infection, the development is usually slow; in the final phase naturally it slows down with the steady decrease of available uninfected host tissue; in the intervening period the rate of increase is highest. Thus, the typical course of an epidemic describes an S-formed curve, as shown in Fig. 18.2 (Van Der Plank, 1968).

![Graph showing the effect of vertical and horizontal resistance on the development of an epidemic](image)

The epidemiological implications of susceptibility and the two types of resistance may be explained by this figure. Curve A represents a host with no vertical resistance and very little horizontal resistance. B a host with vertical resistance and with little horizontal resistance as in A, resulting in delayed infection depending on building up of inoculum of such pathogenic races against which the vertical resistance of the B-host does not work; C is a host with no vertical resistance, as in A, but with considerable horizontal resistance slowing down development of infection of any pathogenic race; finally, in D, vertical resistance is combined with considerable horizontal resistance, providing both a poor start and a general slowing down of the epidemic.

Two examples of hazards due to crop homogeneity favouring epidemics are Victoria blight susceptible oats which in 1945 covered 80 per cent of the oat acreage in the U.S.A. (98 per cent in Iowa) and suffered such heavy losses from Drechsleraavenue that resistant cultivars had to be introduced (Simons and Murphy, 1961); and Texas male sterile cultivars of maize grown on four million acres in the U.S.A. in 1970, when a race of D. maydis caused a destructive southern blight epidemic on these cultivars (Moore, 1970) which then had to be replaced by 'normal cytoplasm' cultivars. To counteract such consequences of lack of heterogeneity in self-pollinated crops plants, development of multi-line cultivars, consisting of a blend of compatible lines of different genotypes, is a possibility (Jensen, 1952; Borlaug, 1953, 1959).

Van Der Plank (1963) emphasises the importance of vertical resistance as a form of sanitation comparable with other means of sanitation, such as eradicant chemicals, and seed and soil disinfections. These procedures reduce inoculum, thus slowing down initiation of infection. Vertical resistance does not reduce the rate of increase. When races are not controlled by resistance, their reproduction is normal.

Horizontal resistance, on the other hand, reduces the rate of increase — the epidemic progresses more slowly than in susceptible cultivars. The effect of horizontal resistance is comparable with the effect of protective fungicides. A fungicide layer on a leaf surface largely prevents germination of spores and establishment of infection, thus providing reduction of infection rate.