CHAPTER 8

Disease Resistance in Plants: Examples of Historical and Current Breeding and Management Strategies*

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INTRODUCTION

Agriculture is, and will continue to be, the second most important human activity keeping mankind alive on this planet. From past, present and future perspectives, the goal of agriculture is to increase and optimize the production of food, fibre, stimulants and other plant and animal products. Major constraints, as well as soil and water, were and are plant pests, weeds and plant diseases. World food yield reduction through pathogens is estimated to be of the order of 20% (Russell, 1978). Until barely two centuries ago, ignorance of plant diseases was absolute and led to mysticism. Disease avoidance strategies knew only repent and quit sinning (Bible, Amos 4:9 'I have smitten you with blasting and mildew ... yet you have not returned to me said the Lord'). The Romans put the responsibility of the dreaded rust of cereals into the hands of two gods, Robigo and Robigus. Nevertheless, rational studies of plant disease did occur sporadically. Cleidemus (400 BC) described diseases on grapes, figs and olives. Theophrastus (300 BC) observed that the amount of disease was higher in low (probably more humid) spots than on high ground (Horsfall and Cowling, 1977). Albertus Magnus (1200–1280) conceived the idea that mistletoe was a parasitic plant and that wood decay fungi were exhalations of humid ingredients which condense and solidify in the cold air. However, the association of a fungus with a plant disease occurred only after the invention of the microscope (in 1667 R. Hooke observed teliospores of cereal rust), but erroneous interpretation still led to the mystical idea that diseases are spontaneously generated and not the expression of a parasitic interaction. Micheli (about 1726, Italy) showed that a saprophytic fungus was able to reproduce from spores, and Tillet, working on brining treatment (previously improved by Pluchet) of wheat, clearly showed that artificial inoculation of wheat seeds with the black dust of smut led to smutted crops. Control was possible by seed treatment with copper sulphate, as Pluchet had already shown with natural infections. Prévost, in

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Table 8.1 Evolution of knowledge of plant pathogens

Roman culture,	ca. 700BC	Cereals gods of rust Robigo and Robigus
Cleidemus,	400 BC	Diseases on grape, figs and olives
Theophrastus,	300 BC	The amount of disease was higher in low areas than on high ground
Bible, Amos 4:9		'I have smitten you with blasting and mildew ... yet you have not returned to me said the Lord'
Albertus Magnus,	1200–1280	Mistletoe was a parasitic plant and wood decay fungi were exhalations of humid ingredients which condense and solidify in the cold air
R. Hooke,	1667	A fungus was associated with a plant disease: teliospores of cereal rust
Micheli,	1726, Italy	Saprophytic fungus can reproduce from spores
Pluchet,	1746	Seed treatment with copper sulphate
Tillet,	1755	Artificial inoculation of wheat seeds with the black dust of smut led to crops with smut
Prévost,	*ca.* 1800	Plant diseases were caused by organisms reproducing themselves and parasite plants. Germination of spores and its inhibition by copper sulphate
DeBary,	*ca.* 1850	Established the living nature of plant parasites
Bailey,	1892	'Enemies often progress or develop as rapidly as do the host plant. I imagine that by the time we are able to breed scab-proof varieties our scab-fungus will have developed a capability to attack more uncongenial hosts'.
Biffin,	1907	Resistance to yellow rust of wheat controlled by a single recessive gene
Orton,	1909	Rational use of resistance and therefore the resistance breeding
Vavilov,	1920	Suggests the use of wild resistance sources

the early 1800s, again advanced the idea that plant diseases were caused by organisms reproducing themselves and parasitising plants. He even devised a method for observing germination of spores and their inhibition by copper sulphate. DeBary's work and the tragic consequences of the *Phytophthora* epidemic in the middle of the last century clearly established the living nature of plant parasites, finally opening the way for rational elaboration of strategies to avoid and control plant diseases (Table 8.1).

Prior to DeBary, recurrent losses probably went unremarked, and only exceptional epidemics leading to tragic famines were noted. However, empirically effective control strategies were developed from the Middle Ages, mostly based on crop rotation. The first truly rational control attempts involved chemicals. It is not surprising that the first great success in controlling epidemics is attributed to the use of sulfur dust to control powdery mildew of grapes in Europe in the autumn of 1854. Chemicals still are, and will be to a large extent in the future, the most convincing way to control pathogens. They fit in our science-orientated society: a cause is identified, an action can be taken and the success is visible. Quarantine, hygiene or sanitation (use of clean seed or propagation material) could only be taken into account once the infectious living nature of pathogens was established, and even then their real usefulness was recognized only slowly and applied generally only in this century. They have the disadvantage that their effects are much less evident and that they are mostly long-term strategies. Less susceptible plants, on the other hand, have been

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used unconsciously since the dawn of agriculture, as nature allows better survival of fitter plants which were selected automatically, while pathogens were a regular constraint. Later, deliberate selection of plants (cultivars) giving the best yield and/or best quality may often have also included deliberate selection for less susceptible plants. The rational use of resistance, and therefore resistance breeding, came into use only at the beginning of this century (Orton, 1909), and the search for resistance sources in natural ecosystems (wild resistance sources) began even later (Vavilov, 1920). A great stimulus to breeders derived from the recognition that a particular resistance to yellow rust of wheat was controlled by a single recessive gene (Biffin, 1907). To a large extent, breeding programs do (and today have to) consider the resistance aspect. To the scientists involved it has always been clear that the benefit to producers and the environment from the use of pathogen-resistant plants is extremely high, and is superior *per se* to other strategies. On the other hand, we cannot forget that the usefulness of a new cultivar will depend primarily on its yielding capacity and on the quality (appearance, content, etc.) of the product. A resistant cultivar is unlikely to be widely grown unless it has these qualities. Minor faults may often be compensated by the presence of resistance; this leads to economical considerations, and we can postulate that a resistant cultivar will be successful only if the benefits outweigh the disadvantages, a point which is often overlooked and leads back to the chemical control of pathogens. The advantage gained through resistance is often expressed only as a difference from a standard cultivar resulting from the lower cost of chemical control (product, application and equipment costs). As this is often low compared with the value of the crop, it is evident that resistant cultivars need to have almost all of the quality aspects of the susceptible control. Moreover, while other methods of control of a particular disease (e.g. crop rotation, quarantine or hygiene) are effective and are traditionally practised with no evident drawbacks, it is not useful to devote efforts to breeding for resistance against such diseases.

Definitions

Before venturing into breeding for resistance against pathogens, a few terms have to be defined and types of resistance clearly stated in order to have a common language. For this paper, an infectious disease is defined as a harmful alteration of the normal physiological state of a particular individual of a species by a pathogen of a particular species or *forma specialis*. A pathogen of a particular species can be (and is, in most cases) a non-pathogen on other plant species. All members of a non-host species are characterized by immunity or resistance against the non-pathogen. On the other hand, not all genotypes constituting the host species may be susceptible; some may be completely resistant, and their resistance is called host resistance and the pathogen is said to be avirulent on them (Andrivon, 1993). The interaction of the two organisms is then incompatible. Virulence therefore implies that the pathogen is able to infect and to reproduce on a particular host. The host is then said to be susceptible and the interaction compatible.

Aggressiveness defines the amount of disease produced by a particular individual of the pathogen on the total range of hosts that it can infect; this non-specific attribute is quantitative. In addition, individuals may differ in their ability to infect a host for which they have the same specific virulence. In other words, they may differ quantitatively in their specific virulence. Quantitative differences in aggressiveness and virulence are difficult to distinguish experimentally. Fitness relates to the maintenance or increase in relative frequency of a particular pathogen genotype in the gene pool during evolution.

Parasitic fitness is therefore a basic attribute of a pathogenic strain within a population of pathogens (Andrivon, 1993).

Resistance of a host cultivar can be of two types: general, which allows less disease (development of the pathogen) than a susceptible cultivar by all genotypes of the pathogen, and is often referred to as horizontal resistance (elsewhere in the literature, general resistance is sometimes referred to as resistance of a particular host against several diseases); and specific, which allows no or less disease compared with a susceptible control cultivar by those pathogen genotypes that lack virulence specific to the resistance in question, and is often referred to as vertical resistance. The pathogen genotype that does have the appropriate virulence allowing it to induce normal disease is known as the pathotype. Different pathogen isolates with the same pathotype belong to the same physiologic race or, more commonly, race.

Inheritance of resistance mostly follows a Mendelian pattern, as does inheritance of virulence in the pathogen. Inheritance analysis can show two patterns: discontinuous variation or continuous variation. In the first case, the segregating progeny will segregate typically as susceptible and resistant individuals; this pattern is often found for differential resistance. General resistance, on the other hand, although often not exclusively, shows continuous variation in the segregating progeny, the individuals ranging from susceptible to resistant with a majority being intermediate.

From these data the underlying genetic base of the resistance can be of two types. The first type is monogenic (dominant or recessive, also referred to as the major gene), whose expression may be affected by other genes (modifiers). The majority of examples of differential resistance can be attributed to the effect of a single gene, as can the capacity of a particular pathotype to be virulent or avirulent. This led Flor (1942, 1956, 1971) to postulate the gene-for-gene theory: each pair of resistance/susceptibility alleles in the host has a matching pair of virulence/avirulence alleles in the pathogen. In other words, for each allele of resistance in the host, a particular allele of avirulence is present in the pathogen. Another theoretical model, the matching allele model, was recently proposed (Frank, 1994), but no experimental data are available to prove its fit to reality.

Resistance genetics focused its attention on sharply defined phenotypes, so differential resistance is also called qualitative resistance and results are expressed as compatible and incompatible interactions.

Considerably less information is available on quantitative interactions, even though they are arguably more common. The genetic basis underlying resistance may also be additive, where each gene contributes only partially to the level of resistance. Here, the expression of resistance is increased by the presence of more genes, also referred to as minor genes. As it is assumed that each minor gene has little effect and that many genes contribute (perhaps unequally) to this resistance (second type: polygenic resistance), it is also called quantitative resistance. However, resistance recognized as quantitative is often assumed to be polygenic, which may be incorrect in the absence of a genetic test since, as indicated above, a single gene may give quantitative resistance that segregates with a continuous distribution.

Resistance genes at different loci can control the same resistance mechanisms and can be overcome by the same race; these are duplicate genes and can be considered as one in breeding and effect. Conversely, resistance genes that are overcome by different races can be described as functionally different. Ephemeral resistance can be defined as resistance (vertical or horizontal) that selects rapidly for the pathogen genotype(s) that can over-

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come it. General or horizontal resistances are usually considered to be durable, since no races are known that can overcome them. On the other hand, Johnson (1981) defines durable disease resistance as resistance that lasts at least for the period during which the resistant cultivar is widely cultivated. Differential resistance can thus be durable because either virulence is extremely rare and/or it is linked to lower fitness (seldom the case over longer periods). Alternatively, planting strategies involving mixtures with functionally different resistances may inhibit virulent races from reaching epidemic levels (Wolfe, 1983, 1985, 1992, 1993; Gessler and Blaise, 1994).

To understand the complexity of a host–pathogen interaction and its analysis, the state of the genotypes should be known. In a diploid or dicaryontic organism it can be homozygous, meaning that the two alleles of a gene are identical, or heterozygous, where the two alleles are dissimilar. Often allelic relationships are not so simple, since the expression of a given allele may be affected by other non-allelic genes (e.g. epistatic effects). Many plant pathogens, such as the ascomycetes, are haploid with a single set of chromosomes carrying a single allele for each gene.

With new biomolecular methods allowing isolation and cloning of plant disease-resistance genes, it is necessary to state clearly what type of genes are envisaged. Plant resistance genes can not only be classified as described above, but also in relation to their function. Most genes of the potential ephemeral type are genes which allow the plant to recognize the pathogen (R-genes) and then activate a cascade of responses encoded by defence–response genes, such as the production of phytoalexins. These responses are often similar between plant species. Recognition genes often fit the gene-for-gene concept. Being potentially ephemeral, they are not desirable in breeding. Defence–response genes, on the other hand, cannot be used (or only in exceptional cases) as they code *per se* for non-specific phytotoxic products or cell letal functions (see Dixon et al., 1995; Innes, 1995; Young, 1995). Which type of genes (R-genes and/or defence–response genes) can and should be used in the construction of transgenic disease-resistant plants has to be considered very carefully.

THE CASE OF APPLE (*MALUS* × *DOMESTICA*)

Apple is not the most important fruit crop, but it is grown almost world-wide in temperate zones and requires massive fungicide inputs under intensive production systems. Increasing public pressure is demanding production systems with reduced inputs of pesticides. Integrated production (IP) systems are more and more popular in Europe. Although the fungicides used are decreasingly harmful to the environment and consumers, the input of pesticides is still questioned. In low-input systems high-quality apples are difficult or impossible to produce. Less susceptible cultivars or even resistant cultivars can be a step towards both production systems.

Genetics of the interaction scab–apple

Commercial apple cultivars have never been analyzed for segregation of genes for differential resistance against scab caused by *Venturia inaequalis*. Because of the variability of the scab population and the high frequency of virulence genes overcoming specific resistances of the commercial cultivars, breeders opted to use material resistant against all strains of *V. inaequalis* present at their location. This included other *Malus* species, often from other continents (Asia). For example, *M. baccata* Borkh. originates from the Himalayas, Siberia and eastern Asia. *M. floribunda* is known only as a cultivated species from Japan, probably originating from interspecies crosses from *M. kaida* × *M. baccata*, *M. ringo* × *M. spectatabilis* × *M. baccata* or others (Hegi, 1963). *M. micromalus* comes from China.

Resistances in other *Malus* species were studied intensively and this indicated the existence of a large pool of resistances (Williams and Kuc, 1969). The available data allow various interpretations: (a) Vf-resistance from *M. floribunda* 821 is due to a single gene (Williams and Kuc, 1969, p. 226); (b) the original level of resistance in *M. floribunda* 821 was due to a group of closely linked quantitative genes; (c) resistance could be due to a qualitative gene giving a class 3 reaction type, closely linked with one or more quantitative genes always inherited to the modified back-cross progeny and due to a gene giving a class 1 reaction, not detected in the back-cross progeny (Williams and Kuc, 1969, p. 227). Crosby et al. (1992), reviewing the success of breeding apples for resistance against scab, list 48 cultivars resistant to scab, of which 37 carry the Vf-resistance. As Rousselle et al. (1974) and Gessler (1989) did earlier, they hypothesized that the phenotype of the Vf-resistance is enhanced or augmented by additional genes. The fraction of a segregating population carrying the Vf gene from a cross between a Vf carrier and a susceptible parent shows continuous gradation of the resistance reaction from no symptoms to restricted sporulation and even to full susceptibility.

To my knowledge, 19 independent resistance loci have been described to date (Bagga and Boone, 1968a). So far, only six resistances at different loci from six *Malus* species are named (Vf, Va, Vb, Vbj, Vr and Vm). As Dayton and Williams (1967) noted, it is still not clear whether all genes are truly different or whether some are identical genes that were transferred to non-homologous chromosomes (or loci) by aberration during the evolutionary development of *Malus*. On the other hand, there may be as many mechanisms of resistances as there are resistance genes present (Bagga and Boone, 1968a) and, correspondingly, the same number of different virulence genes. This can be established only after the corresponding virulences have been found and the resistance carriers have been tested accordingly.

The cultivar 'Nova Easygro' should carry the Vr-resistance since it was bred from a Russian seedling. This resistance is described as different and independent from all other resistances mentioned above (Dayton et al., 1953; Dayton and Williams, 1967). However, it was possible to show by the use of molecular DNA markers for the Vf locus that 'Nova Easygro' also carries the Vf gene (N.F. Weeden, personal communication, 1995; Gianfranceschi et al., 1996). The Vm resistance is different from the other single resistances as it is overcome by Race 5 (Williams and Brown, 1968).

Bagga and Boone (1968b) studied 41 scab-apples for crab resistance and found 25 independent loci rendering resistance. Some of them must be functionally different from each other because Williams and Kuc (1969, p. 228) found that they remained resistant to an inoculum consisting of a combination of isolates. Again we cannot deduce that the seven crab-apple selections which were resistant to the isolate used by Bagga but susceptible to the undefined inoculum used by Williams all had identical, or up to seven different, resistance genes. Similarly, we cannot deduce that the remainder had the same functional resistance but at different loci. Other examples where interpretation is impossible can be found in the literature. Recently (Gessler et al., 1993; Sierotzki et al., 1994a,b) it was shown that three popular cultivars considered to be susceptible have differential and

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Table 8.2 Major steps leading to scab resistance in apple

Aderhold,	1899	Particular apple cultivars seem to be more resistant than others. The observations were not consistent. Resistance/susceptibility of a single cultivar changes with the years. Accommodation of the scab fungus to the host
Aderhold,	1902	Recommendation to avoid spread of scab isolates and to test the behaviour of various cultivars at various sites over several years
Crandall,	*ca.* 1910	Collection of *Malus* species (see Crandall, 1926)
Wallace,	1913	Use of resistant apple cultivars offers little promise as a means of scab control, since varieties known to be fairly resistant changed to susceptible over the years
Crandall,	1914	Cross *Malus floribunda* 821 (Vf-resistance) × Rome Beauty (see Crandall, 1926 and Kellerhals, 1989)
Crandall,	1926	Full-sib cross between two F1 selections and selection of No. 26829-2-2
Hough and PRI-program,	1945	Developing scab resistant cultivars using Crandall's No. 26829-2-2 (see Hough et al., 1953 and Kellerhals, 1989)
Dayton et al.,	1970	Scab-resistant cultivar Prima (Vf)
Crowe,	1971	Scab-resistant cultivar Easygro (Vr from Russian seedling No. 12740-7A) (Crowe, 1975)
Williams et al.	1972	Scab-resistant cultivar Priscilla (Vf)
Williams et al.	1975	Scab-resistant cultivar Sir Prize (Vf)
Lespinasse et al.	1977	Scab-resistant cultivar Florina (Vf) (Lespinasse et al., 1985)
Norelli and Aldwinckle,	1993	Transgenic apple rootstock M26 with increased resistance to fire blight (see Norelli et al., 1994)

ephemeral resistances, each being active against populations of the pathogen collected from the other two cultivars. We may now assume that speculation that a gene-for-gene relationship exists (Boone, 1971) is also correct and valid for these hidden resistance genes in commercial cultivars. The total number of functionally different resistances present in wild *Malus* species and in cultivated commercial cultivars, almost all recognized as ephemeral, may be many times greater than expected.

Based on these assumptions, a set of old cultivars has been inoculated under controlled conditions with conidia from monoconidial isolates made from single lesions collected randomly and sparsely in various orchards. The results (Koch et al., 1996; Table 8.3) show that all cultivars carry resistances against particular isolates. Strongly sporulating lesions were observed when the inoculum originated from the same host cultivar. The isolates of an orchard with many cultivars carried more virulences than the isolates of an orchard with only a few cultivars.

The data described above indicate that many functionally different ephemeral resistance genes are present in old cultivars and other *Malus* species. A corresponding number of pathotypes is probably present in the world population of the scab pathogen, with the limitation, often found in other host–pathogen systems, that a number of the resistance genes have the same function, so that the number of different virulences may be lower.

Strategies to improve the durability of ephemeral resistance

By incorporating any resistance (or part of the immunity) into *Malus* × *domestica* we exert selection on the scab pathogen which leads to the emergence of a specific race (or

Table 8.3 Frequency (%) of *Venturia inaequalis* isolates capable of causing abundantly sporulating lesions or flecks with slight sporulation (in parentheses) on a set of eight cultivars. Monosporic isolates were obtained from single lesions randomly collected in an orchard planted with 26, 3 or 1 cultivars (adapted from Koch et al., 1996)

Origin of isolates	Ananas Reinette	Boskoop	Champagner Reinette	Glockenapfel	Golden Delicious	Gravensteiner	Jonathan	Klarapfel
Orchard with 26 cultivars								
Ananas Reinette (5 isolates)	100	20	80	(40)	(60)	20+(40)	(20)	20
Boskoop (4 isolates)	(75)	75+(25)	50+(50)	(75)	0	0	25	(50)
Champagner Reinette (10 isolates)	10+(30)	20+(30)	90+(10)	(10)	10+(10)	(10)	(20)	0
Glockenpfel (8 isolates)	25+(37)	12+(37)	25+(12)	87+(12)	12+(25)	20+(40)	(25)	(37)
Orchard with 3 cultivars								
Golden Delicious (15 isolates)	0	0	6.7	6.7	93+(6.7)	6.7	0	0
Jonathan (7 isolates)	28	14	0	0	57	0	29+(57)	0
Gravensteiner (4 isolates)	25	0	0	0	25	75	0	0
Orchard with 1 cultivar								
Golden Delicious	0	0	0	0	82+(9)	0	0	0

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races), as Parisi et al. (1993) and Roberts and Crute (1994) showed. Substituting susceptible cultivars by cultivars with Vf or any other single resistance may not be a long-term strategy as long as we maintain the concept of monoculture; we can predict that the resistance will be completely overcome and the cultivars will then be dependent only on their background resistance.

Populations of pathogens are genetically variable and therefore respond to selection pressure. In natural systems, one apple tree represents a single genetic entity. Mankind changed this situation drastically by creating large and dense, genetically uniform host areas (monoculture). Seemingly, the Greeks propagated apple clones by grafting, and the Romans cultivated particular cultivars in orchards and had disease problems with them. Until the beginning of this century, the planting system consisted of single trees in meadows and non-arable land, most of the trees originating from seedlings (Morgan and Richards, 1993). Today's intensive orchard forms planted with only a limited number of cultivars were adopted only during this century, mostly thanks to the systematic selection of rootstocks with particular growth characteristics (Hatton, 1939). Parallel to the disappearance of the sparse planting system of trees of unequal genome, the disease situation worsened. With the introduction of dense orchards, chemical disease control was introduced. Even today we can observe that scab does not cause a total loss on single standing trees of 'older' cultivars (cultivars not popular anymore). Under similar conditions, however, scab can cause total or near total loss in an unsprayed, intensive single-cultivar orchard. Even if the destabilizing effect of increasing aggregation of plants with the same genome is poorly substantiated (Zadoks, 1993), we can postulate that this change drastically shifted the relationship between the host (now a single entity) and the parasite. Previously, variability was required to enable the pathogen to infect more than a single entity (by sexual ascospores), followed by asexual reproduction from a successful infection. Now, however, uniformity gives an advantage to the pathogen. Scab can damage trees to such an extent that trees are defoliated early and some individuals are no longer able to survive competition from others, leading to a thinning of the close stand. However, humans have intervened by reducing pathogen damage through protection by pesticides. As the efficacy of the pesticides and of pesticide scheduling improved, host density was maintained or increased. At this stage, the pathogen population also responds to the fungicides, and resistant strains or sub-populations are selected. We are now dependent to such an extent on the use of pesticides to maintain an equilibrium between pathogen and host that we have forgotten how this situation arose; the current unnatural situation is now considered natural.

Can we return to the earlier system of dispersed trees of various cultivars (as described above for apple)? I am not advocating this because it is unrealistic. Can we therefore continue on our current road? Again this is unrealistic, or at least short-sighted. Few classes of fungicides have been developed, and the probability of finding new products is diminishing. Moreover, even with the best strategy of use, fungicide resistance is likely to emerge. Indeed, by definition, no chemical strategy is sustainable because of either resistance or toxicity to humans or other organisms. In the co-evolution of a host and a pathogen in a particular environment, the main stabilizing factors are, to different extents, distances between hosts, inter-cropping and the genetic variability of the host population. As the first two factors cannot easily be altered, the latter remains.

A possible strategy is cultivar mixing (Wolfe, 1983; Blaise and Gessler, 1994), where each cultivar needs to have a different resistance. A simple mixture of only three cultivars greatly limits the production of primary lesions, but we have now to prove or demonstrate the validity of the theory for this host–pathogen system. However, we do not know the resistance-gene composition of all cultivars, and old cultivars do not correspond to our needs. Therefore modern cultivars need to be analyzed accordingly or new cultivars need to be bred.

Breeders have two options. The first is to breed a set of cultivars each having a functionally different resistance. These unrelated cultivars, each with one or more resistances, may differ so much that in an appropriate planting system that adaptation of the fungus is difficult if not impossible. In other words, occurrence of a super-race (a single isolate able to overcome all mixtures of resistance components) may be much less likely in appropriate cultivar mixtures than in one cultivar with the same total number of resistances, even without considering the possibility of stabilizing selection *sensu* Vanderplank (1982; see Schaffner et al., 1992, for similar data on the response of populations of the barley mildew pathogen to large-scale use of cultivar mixtures).

The recognition of functionally different resistances is possible if the resistances are ephemeral, but only through the use of differential races. Such a selection scheme would be very cumbersome. An alternative would be to transfer the genome segment carrying the resistance from one cultivar into the target cultivar. The target cultivar could be a popular commercial cultivar. By incorporating functionally different resistances, the concept of near-isogenic lines (NIL) could be adopted. Although this cannot yet be done, it may well be possible in the foreseeable future, but it is not clear whether this will be acceptable to consumers. Moreover, by incorporating the resistances into a single genetic background, we may simplify the problem for the pathogen of overcoming the resistances (Wolfe, 1993) and thus select a fit super-race even in a resistance-mixture concept.

The second option is to breed cultivars that contain the greatest possible number of resistance genes (pyramiding). The presence or absence of each gene could be recognized by serial testing of the progenies of correctly selected parents with inoculum of races carrying the appropriate virulences. Alternatively, the races could be mixed in the inoculum to reach the same conclusion in fewer tests, except that genes giving incomplete resistance may be missed. This system could again be used for resistances known to be ephemeral, i.e. if we have the corresponding virulent races which would allow identification of a second (or third?) resistance. However, selection for super-races may be stronger than in the NIL concept, particularly for a parasite such as *V. inaequalis* which reproduces sexually each year. There has been much discussion about the advantages and disadvantages of both strategies (Wolfe, 1993), mostly by experts on annual plants. The choice of strategy (multi-cultivar mixture, NIL or pyramiding resistance genes) may not be relevant to the apple system because the constraints on success may be of a completely different nature.

A more elegant approach than selecting for resistance in the glasshouse or field is to identify or mark the genome segment carrying the resistance information and to select progeny by the presence of such markers. The success of marker-assisted breeding is based on the assumption that resistances at different loci are also functionally different. Since the resistance genes are often not easily identified, it may be better to concentrate on marker-assisted breeding, particularly because of its high feasibility and cost–benefit ratio. An alternative to classical breeding is the transformation of a desired cultivar through incorporation of genes leading to resistance. Two types of genes can be considered. (a) The traditional resistance genes which allow the host to recognize the pathogen

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(R-genes). Such genes, as already mentioned, are (potentially) ephemeral, and the strategies stated above would be needed to render these resistances durable. (b) Defense response genes (reviewed by Dixon et al., 1995), which may well lead to stable resistance, as, for example, would the constitutive expression of one or more phytoalexins. An example is the introduction of the gene encoding the lytic protein attacin E which is active against a broad spectrum of gram-negative bacteria. Aldwinckle and Norelli recently obtained a transgenic apple rootstock expressing this gene and being significantly more resistant to fire blight (*Erwinia amylovora*) than the parent root stock M26 (Norelli et al., 1994). For apples, a strategy based on defense–response genes must be considered very carefully as regards the side effects on the plant itself and toxicity to consumers.

COFFEE

Coffee is, in global trading, the highest revenue cash crop, with US$11–12 billion year^{-1}. Several countries are highly dependent on this crop. Coffee originates from tropical Africa, an important center of origin being Ethiopia. The genus *Coffea* includes about 90 species. Commercially important are *C. arabica* and *C. canephora*. *C. arabica* has excellent quality. *C. canephora*, on the other hand, is more resistant to diseases (*C. canephora subspecies robusta*). *C. arabica* is tetraploid and self-fertile while *C. canephora* and most other species are diploid and cross-pollinating. The basic number of chromosomes is 11 (Van der Graaff, 1986; Lutzeyer et al., 1993). The most important diseases are coffee-rust (caused by *Hemileia vastatrix*) and coffee-berry disease (caused by *Colletotrichum coffeanum*).

Breeding

As with apple, breeding a high-quality perennial crop is time-consuming and success is slow (see Muller, 1986; Van der Graaff, 1986). The market for resistant plants or seeds is small and resistance has to be durable. Coffee breeding was therefore of no interest to private breeders. Coffee is bred in public institutions in producer countries. Great regional differences can be observed among these institutions. The CENICAFE (Centro National de Investigationes de Café de la Federacion Nacional de Cafeteros de Colombia) can be regarded as the leading institution for research and breeding, together with the CIFC (Centro de Investigação das Ferrugens do Cafeeiro) in Portugal (Oeiras). These institutions generally carry a big deficit; international collaboration is often hindered by economic and political problems. Sinking world prices for coffee do not favour investment in research.

Coffee rust

Coffee rust struck in 1868 in Ceylon (Sri Lanka), completely devastating the large British-owned plantations. Since then, coffee rust has spread to all coffee-planting areas, fortunately (for producers and coffee drinkers) with less impact, thanks to less favourable climates and/or the use of fungicides.

Host–pathogen interaction

About 30 races of the pathogen have been described so far at CIFC based on the interactions of dominant host resistance genes and pathogen virulence genes. The resistance genes can give complete protection, but most have been overcome; a few single genes and some combinations are still effective against the complete population of *H. vastatrix*. An important source of resistance are the Timor hybrids (probably spontaneous hybrids between *Coffea arabica* and *C. canephora*), which possess the genes Sh 6–9. These hybrids are often used in breeding.

Horizontal resistance can be found in *C. arabica* and in *C. canephora*. In *C. canephora* the pathogen response ranges from almost susceptible to immunity. Breeding strategies include the use of transgressive segregation in interspecies crosses of *C. arabica* with the introduction of resistance from *C. canephora* and the use of these resistances in *C. canephora* itself by back-cross breeding and the pyramiding of the vertical resistance gene in new selections. Male-sterile lines can be used to produce hybrids. As may be expected, introduction of general resistance is problematic because the resistance may be completely or partially lost during back-crossing.

Impact

Resistant cultivars of *C. arabica* from breeding programs are only slowly finding their way into cultivation. In 1986, the rust control strategy was generally based on the replacement of *C. arabica* by resistant *C. robusta* (Van der Graaf, 1986), a strategy which leads to lower quality since *C. robusta* is inferior to *C. arabica.*

However, in Colombia more than 200 000 ha are currently planted with the cultivar mixture 'Colombia' which is uniform for high quality, dwarf stature and high yield, but heterogeneous for a range of rust-resistance genes defined through collaboration with CIFC in Portugal (Lutzeyer et al., 1993). The lines derive from crosses between the tall, rust-resistant population of 'Hibrido de Timor' and high-quality dwarf varieties of *C. arabica* (e.g. 'Caturra'). Appropriate lines are selected in F5 and composed in mixtures on the basis of agronomic trials in Colombia and resistance screening in Portugal. Mixtures of different compositions are released in different years depending on the current structure of the pathogen population (Moreno-Ruiz and Castillo-Zapata, 1990). This highly dynamic use of the available resistance genes in a mixture strategy gave durable resistance for some 20 years. The use of pathogen-resistant cultivars allows important savings in pesticide costs, amounting in Colombia alone to US$28 million (calculated after the official plant protection recommendations).

Coffee berry disease

The coffee berry disease (CBD) originated in Africa; it can be devastating in higher zones of Kenya. The situation regarding resistance against CBD is very unclear. Until now no race has been detected which is able to overcome any resistance, and the resistances are assumed to be general. The mode of inheritance is unclear, but selection of resistant cultivars seems to be promising. Recently in Kenya a new cultivar ('Ruiru 11') has been introduced with good resistance against CBD and partial resistance against rust, and this cultivar is being propagated. It is also a hybrid originating from the Timor hybrids and *C.*

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The use of 'Ruiru 11' would avoid all the fungicide treatments which are now necessary to control CBD and rust, and which are a large proportion of the variable costs (28%) in Kenya, amounting to US\$560 ha^{-1} (Omondi, 1994; Opile and Agwanda, 1993). Theoretically, Kenya could save about US\$10–19 million in direct fungicide costs on its 35 000 ha of coffee. In Ethiopia, CBD-resistant cultivars cover the highest surface percentage of any coffee-producing country, although this only amounted to 5% in 1986.

Even though encouraging rust- and CBD-resistant cultivars are available, the area covered by disease-resistant cultivars world-wide is still extremely small.

FRENCH BEANS (*PHASEOLUS VULGARIS*)

The importance of beans is less evident from commercial statistics, since beans are produced as high-quality food for own or local consumption. They are an important source of protein and also vitamins. As a basic food the crop plays an essential role in many Third World countries. Indeed, in some regions of Africa it is the most important food source. It is consumed as green pods or as seed, and different cultivars have been selected for each purpose. The genetic variability is immense (Cepts and Debouck, 1991), with more than 40 000 registered entries at the gene resource bank of CIAT (Centro International de Agricoltura Tropical; Hidalgo, 1991). The most important diseases include anthracnose caused by *Colletotrichum lindemuthianum*, which is important in colder humid climates and can be transmitted by seed, and rust caused by *Uromyces appendiculatus* (Beebe and Corrales, 1991; Singh, 1991).

Breeding

A leader in breeding and research on beans is CIAT, which has the global mandate for the crop. Breeding at the local level is also important in the USA, Italy, France and The Netherlands, etc.

The breeding strategy is negative selection: in the absence of major genes, minor genes should be accumulated in breeding populations. Beans are intrinsically easy to breed since generation times are short and the plant is a typical outbreeder (cross-pollinating; $n = 11$). On the other hand, the crop has to be highly adapted to regional requirements, therefore breeding for resistance is often of secondary importance.

Anthracnose

C. lindemuthianum was used as an example to demonstrate the presence of different races in a fungal pathogen population. Initially there was thought to be little variability in the fungus, but many different races have now been identified. However, the possibility of obtaining durable resistance has been described as good because of the slow rate of spread of the fungus and the rarity of sexual recombination (see Beebe and Corrales, 1991; Singh, 1991). Thirteen vertical resistances have been described, which cover a large range of the world population of *C. lindemuthianum* (ARE, MEXIQUE1, MEXIQUE2, TO, TU, etc.). The corresponding genes are described as complementary dominant, simple dominant or double dominant, and even some recessive. More recently, resistance sources have been found which give resistance against all known races of *C. lindemuthianum*. Even if these resistances are controlled by single genes they cannot be regarded as vertical. Attempts to incorporate partial general resistance are problematic because such resistance is often not expressed in seedlings in the glasshouse. Yield has been static over the last 40 years, but the cultivars used have changed, especially in intensive agriculture, due to mechanization and market requirements. Disease-resistant cultivars are now available for almost any requirements (Silbernagel and Hannan, 1988; Allavena and Ranalli, 1989; Beebe and Corrales, 1991; Singh, 1991).

Bean rust

Various dominant monogenic vertical resistances against rust have been described and a gene-for-gene relation has been postulated (Christ and Groth, 1982a,b). The bean rust population is genetically highly variable because sexual recombination frequently occurs. It is therefore likely that vertical resistance has little future in resistance breeding. Through the use of an international rust nursery, the variability of the pathogen in relation to virulence is being analyzed to find resistance sources which are effective against the whole pathogen population. Habtu and Zadoks (1995) analyzed components of partial resistance (latent period, infection efficiency, sporulating capacity, infectious period and pustule size). The contribution of the various components to the resistance varies from cultivar to cultivar. Some of these components could be used in selection for partial resistance. Accumulation of genes contributing to such a resistance could be feasible with a method of screening for each component. This type of resistance could be truly horizontal.

Impact

The impact of resistance breeding can be regarded as high in intensive agriculture. Without resistance, production would be seriously hampered. However, there is still much room for improvement in sustainable agriculture. Trials in Rwanda showed that through the use of fungicides the average local yield of 873 kg ha^{-1} could be increased by 343 kg ha^{-1}. Disease represented the second most important constraint after the lack of fertilizer (increment through fertilization 571 kg ha^{-1}; Voss and Graf, 1991). Yield increment is vital for countries with dense populations such as Rwanda. Resistant cultivars are particularly important since money for fungicides is not available. Several cultivars were developed in the 1950s and 1960s with ARE resistance, but they were overcome in the early 1970s by a specific race (Messiaen, 1981). This virulence is now widely distributed in South America. Locally important cultivars are available with other resistance genes.

Progress in breeding still continues. In 1975/76 almost no generally resistant material was available, but by 1983/84, 33 out of 100 breeding populations were resistant in all localities where tests were made (Van Schoonhoven and Voyset, 1991). The old strategy of small farmers to mix cultivars (Panse, 1988) has probably helped to extend the durability of vertical resistances. Breeding needs to adopt a holistic point-of-view and cannot concentrate on a single cultivar and pathogen.

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WHEAT (*TRITICUM AESTIVUM*)

Globally, wheat is one of the most important food crops, and is planted on 220 million ha; almost 600 million tonnes were produced in 1990 (ca. 120 kg per capita; FAO, 1990). Productivity varies from 0.4 t ha^{-1} (Somalia) and 1.2 t ha^{-1} for Africa to 4.8 t ha^{-1} in Europe, with The Netherlands reaching the highest yield with 8 t ha^{-1}. The climatic and pedologic conditions contribute most to this variability, but other major determinants are inputs such as fertilizer and pesticides. Western Europe planted about 7.6% of the world surface area of wheat in 1987 and produced 15.7% of the world production; this required 78.2% of all fungicides used world-wide on wheat (corresponding to US$430 million in 1987; Verreet, 1991).

Resistance breeding therefore has different goals and different impacts in various countries. In Western Europe it can lead to a more ecological production system, and in Third World countries to more stable and higher yields.

Breeding

Wheat-breeding programs are present in almost all developed countries as well as the major Third World countries. In addition, CIMMYT (Centro Internacional de Mejoramiento de Maiz y Trigo, Mexico) is financed internationally, and has the largest collection of genetic material and an exceptionally productive breeding program. Cultivars selected at CIMMYT now cover almost a quarter of the world wheat-growing area ($>$ 50.7 million ha; Singh and Rajaram, 1992). Breeding concept (Simmonds and Rajaram, 1988) as well as general knowledge (Saunders, 1991) are among the most advanced for all crops.

Wheat includes several species, of which bread wheat (*Triticum aestivum*, hexaploid; $n = 7$, self-pollinating) is dominant. Local varieties, durum wheat (*Triticum durum*), *Triticum spelta* and wild relatives (grasses) can be used as sources of disease resistance (Dosba et al., 1982). Many resistance genes are catalogued and already introgressed in breeding lines. The most important diseases are rusts (especially leaf-rust caused by *Puccinia recondita*), mildew (*Erysiphe graminis*), and leaf and glume blotch (*Septoria* spp.) (Verreet, 1991), but other diseases of more local importance are also considered in resistance breeding (yellow rust, eyespot, stem rust, etc.).

Powdery mildew

Fifteen major genes (Pm1 ... Pm15) giving resistance against one or more races of the powdery mildew fungus have been identified so far (McIntosh, 1988; Fried et al., 1993), all of which have proved to be ephemeral. The cultivar 'Walter', used in Switzerland, is an excellent example: with its increasing popularity the frequency of the corresponding virulence in the mildew population also increased to levels rendering the resistance ineffective (Winzeler et al., 1990). Similar boom and bust cycles are well documented for other nations and other cereals and diseases. Minor genes conferring quantitative resistance, described as slow-mildewing, are widely used in breeding and have given sufficient protection at lower levels of production. However, they may not compete with the use of fungicides at high production levels. Selection in the breeding material has to avoid the trap of vertical resistance (Bartos et al., 1990), and breeders try to expose their material to inoculum of all known virulences (Winzeler et al., 1990).

Leaf rust

Resistance breeding against leaf-rust could follow the same strategy as that for powdery mildew, but the situation is more complex. Genetic variability in the leaf-rust population is high, partly in response to the 34 known major genes for resistance (Lr1, Lr2, ...). Several single genes and combinations are known to have been overcome by the pathogen. In North America more than 100 distinct races have been identified using differential cultivars. The cultivar 'Mironovskaja 808' carrying Lr3 became susceptible just 2 years after its introduction in 1968 (Bartos et al., 1990). In former Czechoslovakia the gene Lr26 became ineffective in the same year that varieties with the gene ('Kavkaz', 'Aurora') were introduced. A long list of such events is described (Russell, 1978). Zadoks (1972) pointed to the probable presence of minor genes inducing a slow-rusting effect. Breeding for slow rusting has to overcome obstacles such as epistatic effects of major genes in the material. Moreover, some are expressed only in adult plants and others are temperature-dependent for their expression (Fried et al., 1993).

For particular resistance-gene combinations no corresponding pathotype has yet been found. For example, the cultivar 'Frontana' is still resistant (it has Lr13, Lr34 and two further unidentified resistance genes; Singh and Rajaram, 1992). Similarly, no pathotype overcoming the genes Lr9 and Lr24 has yet been identified. Breeding for resistance against leaf-rust is still based mainly on major genes and adopts the strategy of pyramiding the best combination of genes. However, even with conventional pedigree selection procedures a good resistance level can be selected from parents with an intermediate resistance level (Pieters et al., 1991).

Glume blotch

Glume blotch, caused by *Septoria nodorum*, is often mentioned in conjunction with *S. tritici* (leaf blotch) since they both infect the leaves, but damage due to glume blotch may be more serious since it can move from the leaves to the glumes. Resistance against this disease may be indirect, based on the distance between leaves, and between the flag-leaf and the spike. Tall cultivars with long internodes are less affected; this resistance is a form of disease escape based on morphology. Under artificial infection conditions, the cultivar may be susceptible. Direct resistance against glume blotch in the leaves and glumes is controlled by different genes (Fried and Meister, 1987). Both are polygenic and additive (Ecker et al., 1989; Wilkinson et al., 1990). Selection is cumbersome, since the development of the disease has to be observed over long periods because it is highly weather-dependent (Brönnimann, 1968). It is almost impossible to determine the resistance level in single plants early in the selection program. The current Swiss breeding program is based on careful determination of the resistance level of possible parents and selection of the progeny late in the program (F6 generation). Development of genetic markers for quantitative trait loci (QTL) could advance *Septoria* breeding greatly.

Stem rust

Stem rust caused by *Puccinia graminis* sp. *tritici* presents analogous problems to leaf rust. It is absent or only a minor problem in some countries due to eradication of the haploid host barberry. Resistance is also based on major vertical genes. Noteworthy is the explication of virulence frequency observations made in North America by Vanderplank (1982). In particular cases the frequency of individuals carrying a particular virulence gene combination is significantly lower than expected. Therefore, Vanderplank postulated a stabilizing effect on the pathogen population due to an epistatic effect which caused two virulences to dissociate. If this could be proved it may lead to a new strategy in breeding.

Impact

Wheat resistance breeding has often produced cultivars resistant against some diseases with clear success in practice, but in most cases the pathogens were able to overcome those resistances sooner or later; in other cases the popularity of resistant cultivars decreased, not because of susceptibility but because new and more productive cultivars were released. Zwatz (1992) reported the progress of disease resistance in cereals in Austria. Noteworthy is the change of the average response to diseases of all cultivars used in Austria between 1975 and 1990: for leaf rust it changed from an average of 6.9 to 5.6 (on a scale 1–9, with 1 indicating completely immune and 9 indicating highly susceptible), which is a clear success. For other diseases the change was less obvious, and in the case of glume blotch, there was a slight increase in susceptibility (from 6.84 to 6.91), possibly due to a gradual height reduction of the popular varieties. Similar results have been reported from other countries (Zimmermann and Strass, 1991). Using even partially resistant cultivars the use of fungicides is economically no longer justified, neither to increase nor to stabilize yield (risk insurance; Zwatz, 1990).

In Switzerland, the disease resistance range of the recommended cultivars also shows clear progress, although in the late 1980s the situation has been clouded by the dominance of a single cultivar, rather susceptible to leaf rust, and to the intensification of production. Therefore no practical effect can be demonstrated other than a clear increase in the use of fungicides. Fortunately, new and highly productive cultivars with good resistance qualities are slowly replacing the susceptible cultivars. The potential for savings is high, since US$12–15 million are spent on fungicides in Switzerland per year on the 100 000 ha planted with wheat (Winzeler et al., 1990). Multi-line cultivars (NIL) carrying various resistances were developed (Fried et al., 1992), but have not been used in practice for regulatory reasons.

TOMATO (*LYCOPERSICON ESCULENTUM*)

The modern Western European kitchen is almost unimaginable without the tomato. Tomato production is associated with the technical revolution in agronomy. The demands of consumers (from industrial use to local consumption) has led to intensive breeding of new cultivars. The tomato has an important role not only in the developed world but also in Third World countries; the total production in 1992 was 70.4 million t on 2.9 million ha. Yield ranges from a high of 433 t ha^{-1} in The Netherlands (Western

Europe 37 t ha^{-1}, USA 58 t ha^{-1}) to a low of 18 t ha^{-1} for Asia (FAO, 1992). Tomato is a self-fertilizing crop with a high degree of cross-pollination under natural conditions; it is easy to breed and to maintain as a pure stand. Tomato is highly susceptible to a great number of pathogens, which can seriously limit production.

Breeding

Disease resistance breeding has been a clear goal for a long time. Resistances to the major diseases are controlled by single, mostly dominant genes, e.g. resistance to *Fusarium* wilt (genes I-1 to I-3), *Verticillium* wilt (Ve), leaf mould (*Fulvia fulva* (= *Cladosporium fulvum*) genes Cf1–Cf5), *Septoria* etc. Resistance to bacterial wilt (*Pseudomonas solanacearum*) and bacterial spot (*Xanthomonas campestris* pv. *vesicatoria*) is polygenically inherited (Callow, 1993). Cultivars which are resistant in a particular area may be susceptible in another. For example, the cultivars 'Venus' and 'Saturn' are resistant in the USA, but susceptible in Mexico. Most single-gene resistances are known to be race-specific. The original source of resistance is usually an individual of a related wild species found in the Andes. As most cultivars used in commercial planting today have resistances against several pathogens, the main diseases can be controlled by the appropriate choice of cultivar. Even if most of the resistances are vertical and the pathogen population adapts accordingly, it is easy to change to cultivars carrying another resistance. Virulence changes in pathogen population are relatively slow (*Fusarium* and *Verticillium* wilt are soil-borne) and often clearly hampered by the closed environment (glasshouses) in high-production systems. Breeding in this case is predominantly one step ahead of the pathogen. New techniques (transgenic plants) may even increase the advantage of the breeders over the various pathogens. Nevertheless, not all problems due to diseases have been resolved as new problems can arise quickly.

Impact

In the late 1970s and early 1980s plastic tunnels increasingly replaced field planting of tomato in southern Switzerland. Tunnels are now being replaced by glasshouses. All popular cultivars in 1994 carried resistance against TMV virus, *Verticillium*, *Fusarium* (mostly F2), the nematode *Meloidogyne incognita* and corky root rot caused by *Pyrenochaeta lycopersici*. Some cultivars also had resistance against *Fusarium* crown and root rot (*F. oxysporum* sp. *radicis-lycopersici*) and *Cladosporium* leaf mould. *Cladosporium* is often a problem, and appropriate studies showed that locally some of the Cf-genes are ineffective. However, the presence of cultivars with many efficient resistances is not due to farmers deliberately selecting resistant cultivars to avoid disease problems. It is dictated by the quality requirements of the market. Farmers do not acknowledge resistance; for them it is an irrelevant surplus. The disease problems that still occur, such as grey mould (*Botrytis cinerea*), black mould (*Alternaria alternata* f. sp. *lycopersici*) and in particular cases leaf mould, are due to lack of resistance in the cultivars and are resolved by the use of fungicides. Problems due to wilts which occurred with particular cultivars in the early 1980s were resolved by using grafts on resistant roots.

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CONCLUSIONS

The arguments for increased efforts to breed resistant cultivars and their increased use can be separated into two groups: the first is more valid for the developed world, where production currently covers the needs of its population, and the second for a large group of countries—with the majority of the human population—which cannot produce enough to satisfy their needs.

The requirements of consumers and environmental concerns impose strong pressure to change intensive production systems towards systems with lower pesticide inputs. Resistance breeding is regarded as highly relevant, since widespread adoption of disease-resistant cultivars would solve many of the concerns expressed by environmental movements and consumers. However, a crucial question for the success of resistant cultivars is the faith of the producer in the marketing quality of the cultivars and in the superiority of the cultivar because of its resistance. If the producer is not convinced of the durability of the resistance, it will be difficult to substitute well-known susceptible cultivars by unfamiliar resistant cultivars.

Moreover, farmers will switch to resistant cultivars only if there is a pathogen problem without an easy solution. If resistant cultivars are already in use, or the problem is not pressing, the choice of cultivar is dictated by other reasons. Only clear awareness of the risk of a particular pathogen becoming a problem could influence a possible 'wrong choice'.

The responsibility of scientists, administrators and politicians in countries with insufficient food production is to favour strategies to increase and maintain this production with the constraint that the increase should not be detrimental to the environment (sustainable agriculture). As, for economic reasons, inputs can be increased only to a limited extent, all possible measures should be taken to help stabilize and increase yields. Inputs to control diseases are very cost-effective under conditions of high financial yield. For example, a 10% loss in wheat under the Swiss production system is equivalent to a loss of 300–400 ECU ha^{-1}, but only to 200 kg ha^{-1} or about 22 ECU in low production systems due to price and yield differences. Under such conditions only resistant cultivars are cost-effective; they may also help to avoid sudden, unexpected epidemics due to particular weather patterns. In developing countries, it is even more important to strive for durability of disease resistance, or at least to minimize the impact of a loss of resistance. The choice of breeding strategies depends more on the crop, the availability of a resistance type and a source, and the pathogen than on use in intensive or sustainable agriculture (see also Singh, 1986; Callow, 1993). In all cases, the goal of breeding strategies should be, as far as possible, to produce cultivars with durable resistance.

The usefulness of resistance in controlling pathogen epidemics and possible approaches to increase durability have been discussed elsewhere (Wolfe and Gessler, 1992). One approach is to use quantitative resistance, inherited oligo- or polygenically. It has been observed that the durable resistance of some cultivars are often of this type, but the assumption that any cultivar with resistance inherited this way is durable is dangerously optimistic (Wolfe, 1993). Moreover, as the above examples show, this type of resistance is rarely available and often involves time-consuming breeding strategies and selection procedures.

The use of major resistance genes has often led to rapid breakdown of the resistance. Here the assumption that major genes are in all cases ephemeral is too pessimistic. Which type of resistance should be used has to be considered case by case.

Apple culture appears to be a classic case, since it carries the complexity which we should also expect in other systems. The Vf-resistance introgressed into apple was tested carefully over 40 years in scab-favourable situations all over the world; breakdown of the resistance was not expected (Crosby et al., 1992). However, it occurred (Parisi et al., 1993), and made breeders and producers doubt the relevance of resistant cultivars. As documented by more than 100 years of observation and research, the breakdown of resistance in apple is a natural and common phenomenon, being so frequent (specific resistances in commercial cultivars) that we do not notice it except in special cases (e.g. the Vf-resistance).

Commercial cultivars differ from each other in their level of resistance; some of those differences were never affected regardless of the area covered by a single cultivar. In these cases the resistances can truly be called general, but some cultivars that are moderately susceptible in one area are highly susceptible in others and are ranked accordingly (Gessler, 1994; compare Govi, 1996 to Götz and Silbereisen, 1989).

Parents for specific crosses should be cultivars with truly general resistance even in breeding steps where strong resistance is introgressed from an other source. Many problems currently seem insoluble (e.g. recognition of several functionally different resistances in a single individual), but new biotechnological aids are now being developed. Some of these, such as gene transfer, are being questioned and may even delay widespread application of biotechnology because of patent protection. Also they may have negative effects due to their own success, reducing variability in the crops instead of increasing it, and thus increasing dependence on inputs (e.g. herbicide-resistant cultivars) and the risk of breakdown (large areas with the same genotype). On the other hand, some biotechnological methods can greatly increase the efficiency of breeding. In tomatoes produced for processing, traditional breeding has rather reduced the genetic variability and increased reliance on inputs. Some argue that genetic engineering would reverse this trend (Hauptli et al., 1990).

However biotechnology can be of more immediate advantage by integration with classical breeding. Plant genome maps can associate the information on traits of interest with DNA markers. Once these traits can be identified, they can be selected, manipulated and complemented (see Allen, 1994).

The problem of recognizing different resistances in a single individual due to major genes can be overcome with modern genomic markers closely linked to the R-genes. Quantitative trait loci (QTL) markers are much more difficult to find, but not impossible. They can be useful for selecting individuals with as many QTLs as possible and with major gene resistance. The use of QTL markers could also avoid the *Vertifolia* effect, defined by Vanderplank (1963, 1968) as loss of general resistance in the process of breeding for vertical resistance. The erosion of general resistance is due to the unrecognized loss of additive minor genes during back-crossing.

Instead of pyramiding R-genes in a single cultivar, several cultivars adapted for planting in mixtures, each with a functionally different resistance, can be bred and selected. As Wolfe (1992) points out, different resistances should be kept in separate cultivars in order to maximize varietal diversity and as a consequence to maximize diversity within the pathogen population. Planting strategies oriented towards the use of cultivar mixtures can stabilize yield and increase durability compared with mono-culture (Wolfe, 1993).

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The use of such complementary strategies is imperative in a sustainable agriculture (low- or no-input production systems). Their use may also be advantageous in intensive agriculture (see Wolfe, 1993). With the appropriate cultivar combination, mixtures can be devised that impede the pathogen from reaching intolerable epidemic levels. Genetic uniformity in a crop (field) is much more risky than diversity. Natural, successful 'planting systems' are usually based on genetic variability; our concept for the next century should follow this lesson. Enhancing sustainability, increasing production and decreasing pesticide input are possible by coordinated action on the entire cropping system. Breeders who have worked traditionally on a particular set of problems in a crop must increasingly consider modification of cultivars to fit within the overall cropping pattern of a particular climate and society. Of prime importance is the maintenance of genetic variability, in order to have a reservoir of adaptability that acts as a buffer against harmful environments (FAO, 1993b), agricultural practices and other changes.

OUTLOOK

Modern agriculture will have to increase production of essential food crops substantially over the next 50 years. Production has more than doubled in the last 35 years, and this pace should be maintained. The main factors for these increases were newly cultivated land, and pesticide and chemical fertilizer use (FAO, 1993a). Pesticides will also be one of the main factors of increment in agricultural production in the near future. Strong arguments can be developed in favour of pesticides and chemical fertilization even with respect to sustainable agriculture (Deichner, 1995). However, considering consumer perceptions in the industrialized nations, the current information status of farmers in the Third World, and economic factors, plant resistance against pathogens should be favoured. Breeding can and will produce such plants. Under particular conditions transgenic resistant plants may also be developed. The problem arising will not be the availability of resistant cultivars, but their introduction in practice, since a new cultivar is often associated with a change in characteristics. Moreover, even if this can be accomplished, plant resistance can only be of importance if strategies granting durability of resistance can be implemented. The primary goal of plant breeding and agronomy should be the development of strategies—appropriate to crop and local situation—rendering resistance durable. Otherwise, plant pathogens will cause sporadic and erratic high losses, or regular losses when no fungicides are used. We recognize the high benefit/cost ratio of plant resistance against pathogens. This should be an incentive to develop durable resistance. However, as the beneficiaries (farmers) are not directly and visibly the same organizations and persons as those carrying the costs (mostly state-financed agencies), this argument generally fails. Scientists cannot count on support in proportion to benefit to the same extent as do their colleagues involved in pesticide research and application.

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