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GENETIC AND BREEDING ASPECTS OF DURABLE RESISTANCE OF CROPS TO PATHOGENS

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ABSTRACT

Rapid adaptation of pathogens to introduced resistances is largely restricted to biotrophic or hemi-biotrophic pathogens with a narrow host range and an air-borne or splash-borne dispersal, and to resistances of the major genic types that operate on a gene-for-gene basis with avirulence genes in the pathogen, resulting in a hypersensitive reaction, a post-haustorial resistance. Durable resistance against these pathogens rests basically on a different resistance mechanism (pre-haustorial) and not on the number of genes. Selection for durable resistance against such pathogens should consist of removing the most susceptible entries (selection against susceptibility) and of removing the (near-) completely resistant entries (selection against non-durable, major genes). Among the remaining entries selection for agronomic performance can be carried out. Another approach could be the exclusive use in the crossing programme of cultivars with known durable resistance and of cultivars with a high level of residual resistance after the major gene resistance has been overcome.

Key Words: **Broad resistance, generalist, hypersensitivity, pathogen-specific resistance, pre-haustorial resistance, post-haustorial resistance, quantitative resistance, race-specific resistance, specialist, specificity**

RÉSUMÉ

L'adaptation rapide des pathogènes aux résistances introduites est largement limitée aux pathogènes bitrophiques et hemi-biotrophiques avec un évantail restreint d' hôtes et un systeme de dispersion par voie aérienne ou par éclaboussement, et aux résistances des types génétiques majeurs fonctionnant sur une base de gènes pour gènes avec les gènes avirulents dans le pathogène ce qui résulte en une réaction, hypersensible et une résistance post-haustorielle. La résistance durable contre ces pathogènes dépend surtout d'un mécanisme different de résistance (pré-haustoriel) et non pas du nombre de gènes. La sélection pour une résistance durable contre de tels pathogènes devrait consister au retrait de la plus part des implantations prédisposées (sélection contre la prédisposition) et le retrait des implantations (presque) complètement résistantes (sélection contre les gènes majeurs, non durables). Parmis le reste des implantations une sélection pour la performance agronomique peut être entreprise. Une autre approche consisterait en l'utilisation exclusive dans le programme de croisement de cultivars avec une résistance durable connue et de cultivars avec un haut niveau de résistance résiduelle une fois que la résistance génétique majeure a été maîtrisée.

Mots Clés: **Résistance prononcée, généraliste, hypersensibilité, résistance spécifique aux pathogènes, résistance pre-haustorielle, résistance quantitative, résistance spécifique aux races, spécialiste, spécificité**

INTRODUCTION

All plant species, and so our crops, employ defence mechanisms to ward off or to resist pathogens and pests. Many of them are effective against whole groups of parasites, broad resistance (Parlevliet, 1981). Phyto alexins produced by practically all plant species form such a broad resistance, whereby each plant species produces its own phyto alexins; phaseolin by beans, pisatin by peas, etc. Pathogens that have overcome such a broad resistance in the course of evolution have specialized on the plant species with that specific broad resistance. The phaseolin in beans for instance is circumvented by the bean rust, *Uromyces phaseoli*, and degraded and/or tolerated by *Colletotrichum lindemuthianum*.

The major pathogens of the important crops often belong to these specialized pathogens, and are characterized by a narrow host range. *Puccinia hordei* and *Phytophthora phaseoli*, pathogenic on barley and on lima beans, respectively, are examples of such specialists. *Phytophthora cinnamoni*, causing root rot in over 1000 woody plant species of widely different plant families, and *Sclerotinia sclerotiorum*, affecting hundreds of plant species in over 60 families, are typical generalists.

The fact that the resistance in cultivars often appears ephemeral is largely confined to resistances to such specialized pathogens. It is in such pathosystems that the durability of resistance has become a problem.

Johnson (1981) defined durable resistance as resistance that remains effective while being extensively used in agriculture for a long period in an environment conducive to the disease. This is not a qualitative definition as "long period" and "extensive use" are both in principle quantitative concepts. And indeed the durability of resistance is a typical quantitative trait, which can vary from zero year, the resistance is neutralized already in the lasts stages of the breeding programme (Thomas and Blount, 1976), to over 130 years as is the case with the woolly aphid resistance in some apple cultivars (Niks *et al.*, 1993) and the Phylloxera aphid resistance of grape rootstocks (Pouget, 1990). Table 1 illustrates this with resistance within pathosystems.

The durability of resistance is not determined only by the genetics and mechanism of the resistance. Parlevliet (1993) showed that the farming system can have some effect as well. It is also possible to obtain a more lasting effect from non-durable resistance genes by using them in certain strategic systems such as multilines, cultivar mixtures, gene deployment and multiple gene use. The exploitation of such strategies has been extremely limited up to the present, due to severe limitations of these strategies (Parlevliet, 1993).

Table 1. Number of years that the resistance to yellow rust in wheat cultivars and to powdery mildew in barley cultivars remained effective in The Netherlands (Anonymous, 1955-1983)

Wheat cultivars	Year	Barley Cultivars	Years
Tadorna	1	Ramona	3
Flevina	5	Belfor	8
Felix	15	Minerva	20

Among the specialized pathogens, especially the biotrophic and hemi-biotrophic, fungal pathogens with an air and splash borne type of dispersal (Table 2) are notorious for their ability to overcome introduced resistances (group 1). Resistance to the other pathogens, the generalists (group 2), and a broad group of pathogens, group 3, not belonging to either the first or the second group, is in general durable. Resistance to the generalists is usually of a quantitative nature, resistance to the pathogens of group 3 can be simply or complexly inherited (Parlevliet, 1989, 1993).

This review discusses genetic durable resistance and its use as this seems the most effective approach to obtaining durable resistance.

INHERITANCE OF RESISTANCE: GENERAL ASPECTS

Already in 1905 Biffen reported the monogenic inheritance of resistance in wheat against yellow rust. An ever increasing flow of reports made it clear that in many cases resistance is inherited in

a simple way. Dominance is very common. Monogenic recessive resistance occurs less frequently. Resistance against pathogens of group 1 (Table 2) is often characterized by the presence of many major resistance genes, usually of a dominant nature, while linkage between the resistance loci, multiple allelic series and complex loci occur frequently. This is demonstrated very well by the flax-flax rust (*Melampsora lini*) pathosystem. There are some 30 resistance genes known, which are located on only five loci or rather five tiny regions, designated K, L, M, N and P. L appears to be truly one locus with 13 different resistance alleles. The M and N loci are in fact tiny regions, consisting of several very closely linked loci, each with one or more resistance alleles. The seven resistance alleles on region M occur on at least four very closely linked loci (Mayo and Shepherd, 1980). These different resistance genes on the same locus can be distinguished only by their different race-specificities. There seems to be a coevolution between these resistance genes and the genes in the pathogen.

Resistance is also often based on several genes with relatively small additive effects, minor genes or polygenes (Parlevliet, 1981). Both types of inheritance, the major gene and the minor gene resistance, often occur together.

TABLE 2. Crop-fungal pathogen systems with many race-specific, non-durable resistance genes and many races known. B=biotrophic; HB=hemi-biotrophic; S=specialist, narrow host range; A=air borne; AS=air and splash borne; SS=splash and seed borne; W=water borne; (after Parlevliet, 1993)

Pathogen	Host	R-genes	Dispersal
Fungi			
Puccinia hordei	barley	over 11	B,S, A
P. coronata	oats	over 30	B,S, A
P. sorghi	maize	over 25	B,S, A
P. recondita f.sp. *tritici*	wheat	over30	B,S, A
Melampsora lini	flax	over 30	B,S, A
Erysiphe graminis f.sp.*hordei*	barley	over 30	B,S, A
Bremia lactucae	lettuce	over 13	B,S,AS
Fulvia fulva	tomato	over 11	B,S, A
Phytophthora infestans	potato	over 11	HB,S,AS
Rhynchosporium secalis	barley	over 10	HB,S,AS
Colletotrichum lindemuthianum	bean	over 10	HB,S,SS
Magnaporthe grisea	rice	over 15	HB,S, A
Bacteria			
Xanthomonas campestris pv *oryzae*	rice	over 12	HB,S, W

TABLE 3. Reactions of 16 combinations between two host loci, R and S coding for resistance, and two pathogen loci, A and B coding for avirulence. The host is assumed to be a diploid, the pathogen a haploid, while a + represents compatibility, a - incompatibility (resistance x avirulence)

Host genotype for resistance (R,S) and susceptibility (r,s)	Pathogen genotype for avirulence			
	A,B	-,B*	A,-*	-,-*
rr ss	+	+	+ **	+
RR ss	-	+	-	+
rr SS	-	-	+	+
RR SS	-	-	-	+

*: - means absence of the avirulence function
**: Differential interaction

THE GENE-FOR-GENE INTERACTION

The resistance genes in the pathosystems shown in Table 2 often operate in a gene-for-gene interaction with avirulence genes in the pathogen as shown in Table 3. In this table, the so-called virulence alleles are represented with a- to indicate the absence of avirulence (virulence is an empty concept, see below). The recognition in the gene-for-gene interaction is between the product of the resistance allele, R or S, and the product of the corresponding avirulence allele, A or B respectively. After this recognition, a series of reactions start leading to incompatibility, the hypersensitive reaction of the host (a - in Table 3). If either the host product (R or S) or the pathogen product (absence of A or of B) is not formed, there is no incompatibility; the normal pathogenicity is expressed. So in all combinations of Table 3, a+ means normal expression of pathogenicity. When a cultivar with an effective resistance gene is released, say RR in Table 3, a new race of the pathogen, say -B in Table 3, appears and the resistance is neutralized ("broken") by the race with the new virulence. This latter is actually wrong. There is no new virulence. The normal pathogenicity reappears because the avirulence allele A has become ineffective due to a mutation, which causes either a change in the avirulence product not recognized by the resistance allele anymore or the non-production of that product. These are so called loss mutations, easy to perform by the pathogen; the reason why this type of resistance is so easily neutralized (non-durable resistance).

DESCRIPTION OF RESISTANCE

Scientists have tried to describe host resistance in various ways such as: 1) *Expression of the resistance* (complete, partial, field, residual, quantitative, seedling and adult plant resistance); 2) *Inheritance of resistance* (major gene, minor gene and polygenic resistance); 3) *Specificity of resistance* (race-specific, vertical, race-non-specific, horizontal and pathogen-specific resistance); 4) *Mechanism of resistance* (hypersensitive, non-hypersensitive, post-haustorial and pre-haustorial resistance).

It is useful to discuss the above mentioned terms with the assumed durability of the resistances they try to indicate.

Expression. Complete, seedling and adult plant resistances usually refer to simply inherited resistances, easy to select for, but mostly non-durable in the pathosystems of group 1 (Table 2). Adult plant resistance is expressed only in the more mature stages of the plant and is sometimes considered as durable, a wrong notion, as race-specific non-durable resistance genes can be expressed in any stage of the plant or in the adult plant stage only.

Partial, field and residual resistance are terms identifying quantitative resistance. Quantitative resistance shows a continuous variation in disease severity among host genotypes from very low (high resistance) to extremely high (no resistance, extreme susceptibility). Residual resistance is the resistance that becomes visible (and was hidden) after a non-durable resistance is overcome by a new race of the pathogen (Table 4). Residual

TABLE 4. Yellow rust, *Puccinia striiformis*, disease severity in percentage leaf area affected of six Kenyan wheat cultivars before and after their release as commercial cultivar (Danial *et al.*, 1994)

Year of release	Cultivar	Before release	After release			
			1987	1989	1991	Mean
1981	Paa	0	70	60	30	53
1982	Kenya Popo	0	10	30	15	18
1982	Kenya Kulungu	0	5	30	30	22
1984	Kenya Kima	0	10	30	10	17
1984	Kenya Tumbili	0	40	60	20	40
1989	Pasa	0	-	-	30	30
-	Morocco*	90	90	90	90	90

* Extremely susceptible control cultivar

resistance is nearly always present behind the major-resistance genes introduced by the breeders (Parlevliet, 1993), and the level of this quantitative resistance can be quite good as the cultivars K. Popo, K. Kulungu and K. Kima in Table 4 show. Their disease severity is much lower than that of the very susceptible control.

Quantitative resistance is often considerably more durable than complete or near-complete resistance. But there are exceptions. In a few cases typical quantitative resistance "broke down" equally fast as many complete resistances did. In such cases a monogenic inheritance controlled that resistance. The field resistance against rice blast, *Magnaporthe grisea*, in the rice cultivars St-1 and Chugoku illustrates this. The resistance became ineffective within a few years and appeared to be based on a single dominant gene *Pi-f* (Toriyama, 1975). The *R10* gene in potato against late blight, *Phytophthora infestans*, is also somewhat quantitative in its expression, but it appeared as race-specific and non-durable as the other major R-genes of potato (Turkesteen, 1993).

Genetics. The R-genes in Table 2 are examples of major genes, each gene being responsible for complete or near-complete resistance. The partial resistance of maize to *Puccinia sorghi* (Kim and Brewbaker, 1977) and of wheat to *Puccinia recondita* (Broers and Jacobs, 1989) are based on a few (2 or 3) additive genes. The field resistance of potato to *Phytophthora infestans* (Black, 1970), the quantitative resistance of maize to *Cochliobolus heterostrophus* and *Setosphaeria turcica* (Leonard, 1993) and of barley to *Rhynchosporium secalis* (Habgood, 1974, 1976), the partial resistance of barley to *Puccinia hordei* (Parlevliet and Kuiper, 1985), and the partial resistance of rice to *Magnaporthe grisea* (Roumen, 1994) are all of the polygenic type.

Typical non-durable resistance is restricted to the major, monogenic type of resistance to specialized pathogens of group 1. Monogenic resistance to pathogens of group 3 is generally much more durable. Examples are monogenic resistances against *Cochliobolus carbonum* and *Periconia circinata* in maize, against *Cladosporium cucumerinum* and *Corynespora melonis* in cucumber, against *Pseudocercosporella herpotrichoides* in wheat and against *Helminthosporium victoriae* in oats. The polygenic examples, mentioned above have not shown any erosion of their quantitative resistance despite the fact that, in the six polygenic examples small race-specific effects were reported (see 4th section). In the case of barley-*P. hordei*, there are strong indications that the polygenes in the host operate on a gene-for-gene basis with polygenes in the pathogen (Parlevliet, 1978b).

When major resistance genes become ineffective because a new race of the pathogen appears, it sometimes occurs that the effect of the resistance gene is not completely lost. Some resistance is still visible, a residual, or shadow effect (Riley, 1973; Pederson and Leath, 1988), not to be confused with the residual resistance of Table 3 which is due to other genes also present besides the neutralized major gene. Such resistance would form part of the quantitative resistance. Koch and Parlevliet (1991), describing a clear residual effect of the defeated *Xa-4* resistance gene in rice against *Xanthomomas campestris* pv *oryzae*, doubted whether residual effects of defeated resistance genes are a common phenomenon.

Specificity. Race-specific, pathotype-specific, specific and vertical resistance are synonymous with each other, which is also the case for non-race-specific, horizontal or general resistance. These terms describe the specificity of the resistance towards genotypes within the pathogen species, *forma specialis* (in fungi) or pathovar (in bacteria). The former type of resistance operates against specific genotypes, races of the pathogen only, while resistance of the latter type is assumed to operate against all genotypes of the pathogen. Race-specific resistance is often considered to be non-durable, while non-race-specific resistance is generally thought to be durable (Van der Plank, 1968). Both hypotheses do not hold all the time. When host genotypes with quantitative resistance are tested against different pathogen genotypes small race-specific effects can be found (Table 5, where the cultivars IR50 and IR37704 show a small differential interaction with the isolates W6-1 and JMB8401-1). In all six examples of polygenic resistance mentioned (4th section) small race-specific effects have been reported and it is probable that polygenic resistance to specialized

pathogens usually goes together with small race-specific effects. These small race-specific effects are often unnoticed. Only when the experimental error is kept small do they become visible. Because of this, pathosystems with such small race-specific effects resemble true non-race-specific pathosystems (Parlevliet and Zadoks,1977; Parlevliet, 1985). There are no indications that polygenic, quantitative types of resistances with small race-specific effects are not durable. The partial, polygenic resistance in barley to barley leaf rust shows clearly small race-specific effects (Parlevliet, 1978b), but is very durable (Habgood and Clifford, 1981; Parlevliet, 1993).

Parlevliet and Zadoks (1977) compared two models of polygenic pathosystems with one another. In the one system the host polygenes operated in a gene-for-gene interaction with the pathogen polygenes, causing small race-specific effects. In the other system the host polygenes did not operate in a gene-for-gene way with the pathogen polygenes, representing true non-race-specific resistance. The models also showed that the polygene-for-polygene system was more durable (less liable to adaptation in the pathogen population) than the system where the polygenes were not operating in a gene-for-gene system, assuming all other variables such as resistance mechanisms being the same. This study also brought forward the fact that the assumption of Van der Plank (1968), that non-race-specific resistance is durable because of its non-race-specific character, is unjustified, although it has been accepted by the majority of researchers without any sign of challenge.

Pathogen-specific resistance represents specificity at the species level and not at a within-species level like the race-specificity, or at an above-species level like the broad resistance. Resistance against specialized pathogens is usually, if not always, pathogen-specific and directed against one species or even one *forma specialis* or pathovar of the pathogen (Parlevliet, 1981). The resistance, even the quantitative one, in wheat, barley, oat, rye and maize to one *Puccinia* species for instance is not effective to any of the other *Puccinia* species that are pathogenic on the same crop.

Mechanism. Mechanisms of broad resistance (see 1.), such as the phytoalexins but also preformed structural or chemical barriers, are doubtless durable. The pathogen has great difficulties to overcome such hindrances. An interesting case is the resistance of oat to the take-all fungus, *Gaeumannomyces graminis* var. *graminis*, a pathogen that attacks many grasses, including the cereals, but not oat. Oat is resistant because of avenacin (a preformed chemical barrier toxic to various fungi), which prevents the growth of this and other soil borne pathogens (Turner, 1953). In Wales, and in a few other parts of the world, forms of this pathogen have been found that overcome the resistance of oat, because it produces avenacinase, an enzyme hydrolyzing avenacin into products that are less toxic to the

TABLE 5. Relative number of sporulating lesions in leaves of the main tiller of six rice cultivars inoculated by three isolates of Magnaporthe grisea. Means of three independent but similar experiments. The number of lesions of the extremely susceptible CO39 is set at 100%. (Roumen, 1992)

Genotype	Isolate			Mean
	Po6-6	W6-1	JMB8401-1	
CO39	100	100	100	100a***
IR50	38	48*	33	-
IR37704	38	37	21**	-
IR66	20	24	18	21b
IR36	12	12	14	13c
IR64	7	9	6	7d

* : Significantly higher ($P < 0.01$) than the value of 36 expected when genetic interaction is absent.
** : Significantly lower ($P < 0.01$) than the value of 37 expected when genetic interaction is absent.
***: Values followed by different letters are significantly different (Bonferroni's test for inequalities; alpha = 0.5). No mean is given where a significant interaction is present.

pathogen (Turner, 1961). Despite the fact that such take-all pathogen forms have been found decades ago, oat is still resistant to this pathogen seen on a world wide basis. The forms pathogenic on oat do not seem to spread; they occur as isolated cases. The resistance therefore must be judged as durable.

With the durability of pathogen-specific resistance, the situation is more complex. The resistance so easily overcome is predominantly found among the specialized biotrophic and hemi-biotrophic fungi with an airborne and or splashborne type of dispersal (see 1st section.). This ephemeral resistance is the resistance based on the hypersensitive reaction, where the cells invaded by the pathogen, as well as neighbouring cells, collapse resulting in a tiny necrotic lesion. The growth of the pathogen is stopped or seriously hindered, allowing sometimes for a slight production of spores. This hypersensitivity is basically induced after haustoria are formed in the invaded cells, it is a post-haustorial resistance (Niks, 1986). The genes causing this hypersensitivity are usually race-specific major genes operating on a gene-for-gene basis with avirulence genes in the pathogen (see 3rd section).

As was discussed (see 3rd section) the normal pathogenicity reappears when such a major resistance gene is neutralized. This at the same time explains why residual resistance is expressed as soon as the major resistance gene (of the hypersensitive type) is defeated. The level of pathogenicity allowed by the host's quantitative resistance is now expressed. This quantitative resistance has been found to occur at high frequencies in all pathosystems where a serious inventory was carried out (Parlevliet, 1993), and seems in general far more durable than the resistance of the hypersensitive type.

The partial resistance of barley to barley leaf rust, *Puccinia hordei* is a typical example of this quantitative resistance (Table 6). Partially resistant cultivars have less uredia (reduced infection frequency), that appear later (longer latency period) and produce fewer spores (reduced sporulation rate) (Clifford, 1972; Neervoort and Parlevliet, 1978). The combined effects of these resistance components cause large differences in disease severity in the field (Parlevliet and van Ommeren, 1975). The partial resistance does not evoke a hypersensitive type of response (Clifford, 1972), the uredia are basically of a susceptible type (Parlevliet and van Ommeren, 1975), and the resistance is expressed in all stages of plant growth (Clifford, 1972), but is best expressed at heading (Parlevliet, 1975). The partial resistance in the field is very strongly correlated with the latency period measured in the young flag leaves (r ranging from 0.92 to 0.97, Parlevliet and van Ommeren, 1975; Parlevliet *et al.*, 1985). Latency period in the young flag leaves is therefore a good estimator of partial resistance in barley to *P. hordei*, primarily because the components are pleiotropically controlled by the same genes (Parlevliet, 1986). The latency period in the young flag leaves, and so the partial resistance, is polygenically inherited (Parlevliet, 1978a; Parlevliet and Kuiper, 1985). This polygenic resistance showed small race-specific effects. The size of these race-specific effects is similar to the size of the effect of the individual polygenes (Parlevliet, 1978b), but the resistance is very durable (Habgood and Clifford, 1981).

Germination, penetration, vescicle formation and the formation of the primary infection hyphae are not affected by the partial resistance (Niks, 1981, 1982). The partial resistance becomes effective as soon as haustoria mother cells are formed; a pre-haustorial resistance (Niks, 1986). Haustoria formation is partially prevented by the host cells to be invaded from the haustorium mother cells through the formation of cell wall appositions (papillae) on the inside of the cell walls opposite the sites of attachment of the haustorium mother cells (Niks, 1986).

This pre-haustorial resistance mechanism occurs in other pathosystems as well, and is not restricted to polygenic resistances. Jacobs (1989) observed it in the partial resistance of wheat against *Puccinia recondita* f. sp. *tritici* (Broers and Jacobs, 1989). In barley the resistance conferred by the *ml-o* major resistance gene against powdery mildew, *Erysiphe graminis* f. sp. *hordei*, is based on pre-haustorial cell wall appositions (Jorgensen, 1993). Cultivars carrying this resistance have been grown since the late seventies and covered an acreage of over 700,000 ha in Europe in 1990 (Jorgensen, 1993) without signs of erosion of this resistance.

In wheat, the leaf rust resistance gene *L34* is of a non-hypersensitive type and resembles the partial resistance in cultivars such as Akabozu and BH1146. The resistance of this gene is based on a pre-haustorial resistance (Rubiales and Niks, 1995).

ADAPTATION TO POLYGENIC AND PRE-HAUSTORIAL RESISTANCE

In the preceding paragraphs it was shown that the durable resistance to specialized fungi of group 1 is primarily determined by the mechanism of the resistance and not by the number of genes involved. In a few cases, scientists have studied the ability of the pathogen to adapt to such durable resistance. In two experiments a recurrent selection for increased aggressiveness of maize pathogens was carried out by isolating the spores of the largest lesions and intercrossing these (Leonard, 1993). In maize the quantitative resistance to Northern leaf blight (*Setosphaeria turcica*) and to Southern leaf blight (*Cochliobolus heterostrophus*) is typically polygenic and is expressed by smaller and fewer lesions. The increased aggressiveness was measured as an increase in the lesions. In the second pathosystem the lesions had increased 17% after 3 cycles of recurrent selection (Leonard, 1993). This increased aggressiveness showed cultivar-specific effects, the mirror-image of the race-specific effects reported in polygenic resistance and predicted by Parlevliet and Zadoks' polygene-for-polygene model (Parlevliet and Zadoks, 1977). It appears possible to increase the aggressiveness by rearranging the pathogen genes, but it does not seem to happen in the field.

In a third experiment a recurrent selection for increased aggressiveness against the barley *ml-o* resistance gene in a barley powdery mildew isolate was carried out. It appeared possible to increase the aggressiveness of the isolate (probably through spontaneous mutations), but the resistance was only reduced to a relatively small extent; in the field such more aggressive mutants have not been found (Jorgensen, 1993).

These examples proved that even against durable resistance the pathogen is in principle able to increase its aggressiveness. In farmers' fields, nature for the pathogen, it does not seem to happen, probably because of fitness problems going together with such increases in aggressiveness. In the case of the third example, there are indications of a loss of fitness in the more aggressive isolate (Jorgensen, 1993).

Since it seems theoretically possible to adapt at least partially to such durable resistance, one may expect this also to happen in reality at least very incidentally and this is indeed the case. The field resistance of potato to late blight is considered to be polygenically inherited and durable (Black, 1970; Turkesteen, 1993). The field resistance in the foliage and in the tubers is, to a fair extent, independent of each other (Anonymous, 1953-1994; Turkesteen, 1993). Pimpernel, a Dutch cultivar released in 1953, has a fairly high field resistance to this pathogen both in the foliage and in the tubers. This resistance was still fully effective in The Netherlands in 1988 when it was removed from the recommended cultivar list. From other countries, where it was grown and still is grown, no reports of a reduced resistance are known, except one. In Norway it was observed in the late 1980's that Pimpernel's tuber resistance had become less. This appeared to be due to an isolate that was more aggressive on the tubers of this cultivar (Bjor and Mulelid, 1991).

SELECTION FOR DURABLE RESISTANCE

Durable resistance against the groups 2 and 3 (see 1st section) seems straightforward; any resistance can be used. However, the quantitative resistance is often not easy to identify. The breeder has to discern the more resistant entries from the less resistant ones. This calls for a homogeneous exposure to the pathogen, a situation usually not existing and often difficult to realize.

Soil-borne pathogens tend to be heterogeneously distributed. A homogeneous exposure to the pathogen is often not easy and the accuracy of the disease assessment is then far from satisfactory. Soil-borne pathogens that spread upward to the leaves and ears offer other problems in the screening of resistance. Resistance to such pathogens (*Septoria nodorum*, *Septoria tritici*, *Fusarium culmorum*) is often severely confounded with earliness and tallness.

Quantitative resistance to viruses carries a similar problem. Homogeneous exposure to the virus is difficult to obtain, especially if an animal vector (aphids, nematodes) is involved. Screening methods that identify small differences in resistance are not readily available. Resistance to barley yellow dwarf virus in wheat and oats is quantitative in nature and screening is far from easy. In barley it is easier as two major genes have been identified. In potatoes the quantitative resistance to the leaf roll, the X, the A and the Y viruses is expressed as a reduced frequency of plants becoming infected (showing symptoms) when exposed. To obtain a fairly accurate assessment of the differences in frequency of diseased plants, it is essential that, of each entry, a sufficient number of plants (20 to 30) is tested per season per entry. This precludes screening for this resistance in the early stages of the selection programme when the number of plants per entry (clone) is still small.

Good screening methods that can identify small differences in quantitative resistance are absolutely essential in order to accumulate this resistance and such screening methods are not available for many pathogens in these two groups.

In case of pathogens of group 1 (see 1st section) the breeder should observe certain rules. One important rule is that each pathosystem has its own particular aspects. It is easy to make a general statement that one should select for quantitative resistance, but by doing this one might overlook some serious problems and some useful possibilities.

Problems: Not all quantitative resistance is genetically of the durable type. There are race-specific, non-durable major genes with an incomplete expression, such as the *Pi-f* resistance gene in rice to rice blast (Toriyama, 1975), and the *R10* resistance gene in potato against late blight (Turkesteen, 1993).

In pathosystems, such as shown in Table 2, many cultivars carry one or more major genes that are partially overcome because the pathogen population consists of a mixture of races corresponding to the various resistance genes in the cultivars grown. In the breeders' selection plots, such partially ineffective resistance genes show up as quantitative resistance (Parlevliet, 1983), confounding the quantitative resistance really present. In the presence of fully effective non-durable major genes, quantitative resistance is not expressed.

Possibilities: If one would select only for quantitative resistance, a resistance such as the *ml-o* resistance in barley to powdery mildew would be discarded. Also linkage with other more easily recognizable traits should be used when possible (indirect selection). The stem rust resistance gene *Sr-2* in wheat controls a high level of partial resistance. It occurs in many cultivars worldwide where stem rust was a problem. Even after 60 years of exposure it is still effective and it is genetically linked with false black chaff due to head and stem melanism (Van Ginkel and Rajaram, 1993). Selection for this false black chaff will select the *Sr-2* resistance gene.

If, however, one chooses to select for quantitative resistance the selection is in general not difficult, provided no effective or partially effective major genes are present (see *problems* above). This is so because many of the pathogens in this group are airborne (Table 2) and it is fairly easy to obtain a uniform exposure to airborne inoculum, while the assessment of the amount of tissue affected too is more reliable with biotrophic fungi. However,

TABLE 6. Number of barley leaf rust pustules per tiller and relative grain yield of barley populations before and after three cycles of recurrent selection for increased partial resistance (decreased pustule number) against barley leaf rust

Population/commercial cultivars	No.of pustules	Relative yield
Mean of unselected populations	5200	100
Selected population	275	126
Best line from selected population	75	132
Cv Mamie, extremely susceptible	11000	76
Cv Sultan, susceptible	7200	97
Cv Vada, partially resistant	500	123

there are only a few important host-pathogen systems where the host is free or mostly free of effective non-durable major genes. Groundnut - *P. arachidis*, barley - *P. hordei* and maize - *P. sorghi* are examples of this type. Selection for increased levels of partial resistance is relatively easy in such cases. In barley, it was shown that three cycles of recurrent selection with a mild selection for partial resistance against barley leaf rust, in the F_2 and F_3 in each cycle, was very effective to accumulate partial resistance (Parlevliet and Van Ommeren, 1988a). In each selection stage the 30% most affected F_2 plants or F_3 lines were removed. Among the remaining plants or lines selection for improved agronomic performance was carried out. Table 6 summarizes the results. This mild selection, in fact a selection against susceptibility, increased the level of partial resistance from fairly susceptible, slightly lower than that of cv Zephyr, to significantly higher than that of the fairly resistant cv. Vada. At the same time the agronomic performance as measured by grain yield improved greatly (Parlevliet and van Ommeren, 1988b)

That selection against susceptibility can be very effective in the real world is supported by the soybean - *Pseudomonas glycinea* pathosystem in the USA (Wilcox, 1983). This bacterial blight occurs throughout the soybean growing areas. Major gene resistance and the existence of eight races have been reported. Soybean breeders in the USA have never put much emphasis on breeding for a high level of resistance. They have eliminated the highly susceptible lines continuously during the breeding procedures. The current cultivars are therefore relatively free from bacterial blight during most of the growing season, whereas unselected accessions from abroad quite often become heavily infected in the same breeders' nurseries.

However, in most of the economically important pathosystems, major non-durable resistance genes are present, often even very frequently. This makes selection for quantitative resistance far from easy. How should one select for quantitative resistance in such host-pathogen systems? Before a selection strategy is laid out one has to take a few facts into consideration: (i) The major genes present in the population to select from may vary from totally ineffective to completely effective to the races present in the area. Part of the major genes are effective to some races and ineffective to other races. These latter genes may easily give the impression that quantitative resistance is present when the pathogen population consists of a mixture of races. The entries may show different levels of disease severity where the lesions have a susceptible infection type. But the differences in disease severity are solely due to these major genes. So the conscious use of race mixtures should be avoided if one wishes to select for quantitative resistance. Instead one should use one race with as wide a virulence range as possible (Parlevliet, 1983). (ii) If the breeder finds entries with a disease severity of zero or close to that (trace) he should realize that this high level of resistance is unlikely to come from accumulated quantitative resistance. Most likely it is the expression of an effective major gene and therefore most likely not durable. The advice is to remove such lines.

This leads to the general advice, when major resistance genes against pathogens of group 1 are present, to remove the most susceptible entries (selection against susceptibility) and to remove the (near-) completely resistant entries (selection against non-durable major genes). Among the remaining entries selection for agronomic performance can be carried out. Another approach could be the exclusive use in the crossing programme of cultivars with known durable resistance and of cultivars with a high level of residual resistance after the major gene resistance has been overcome.

REFERENCES

Anonymous, 1953-1994. Beschrijvende rassenlijst voor landbouwgewassen no's 28 to 69 (Descriptive cultivar lists of arable crops no's 28 to 69). CPRO, Wageningen, The Netherlands.

Bjor, T. and Mulelid, K. 1991. Differential resistance to tuber late blight in potato cultivars without R-genes. *Potato Research* 34: 3-8.

Black, W. 1970. The nature and inheritance of field resistance to late blight (*Phytopththora infestans*) in potatoes. *American Potato Journal* 47: 279-288.

Broers, L.H.M. and Jacobs, T. 1989. The inheritance of host plant effect on latency period of wheat leaf rust in spring wheat. II. Number of segregating factors and evidence for transgressive segregation in F_3 and F_5 generations. *Euphytica* 44: 207-214.

Clifford, B.C. 1972. The histology of race non-specific resistance to *Puccinia hordei*, Otth. in barley. In: *Proceedings of the Third. European Mediterranean Cereal Rusts Conference I*, pp. 75-79. Research Institute of Crop Production, Prague.

Danial, D.L., Stubbs, R.W. and Parlevliet, J.E. 1994. Evolution of virulence patterns in yellow rust races and its implications for breeding for resistance in wheat in Kenya. *Euphytica*: (In press).

Habgood, R.M. 1974. The inheritance to *Rhynchosporium secalis* in some European spring barley cultivars. *Annals of Applied Biology* 77: 191-200.

Habgood, R.M. 1976. Differential aggressiveness of *Rhynchosporium secalis* isolates towards specified barley genotypes. *Transaction of the British Mycological Society* 66: 201-204.

Habgood, R.M. and Clifford, B.C. 1981. Breeding barley for disease resistance: The essence of compromise. In: *Strategies for the Control of Cereal Disease.* Jenkyn F.J. and Plumb R.T. (Eds.), pp. 15-25. Blackwell Scientific Publications, Oxford.

Jacobs, Th. 1989. Haustorium formation and cell wall appositions in susceptible and partially resistant wheat and barley seedlings infected with leaf rust. *Journal of Phytopathology* 127: 250-261.

Johnson, R. 1981. Durable resistance: Definition of, genetic control, and attainment in plant breeding. *Phytopathology* 71: 567-568.

Jorgensen, J.H. 1993. Durability of resistance in the pathosystem: barley-powdery mildew. In: *Durability of Disease Resistance.* Jacobs Th. and Parlevliet J.E.(Eds.), pp. 159-176. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Kim, S.K. and Brewbaker, J.L. 1977. Inheritance of general resistance in maize to *Puccinia sorghi* Schw. *Crop Science* 17: 456-461.

Koch, M. and Parlevliet, J.E. 1991. Residual effects of the *Xa-4* resistance gene in three rice cultivars when exposed to a virulent isolate of *Xanthomonas campestris* pv *oryzae*. *Eyphytica* 55: 187-193.

Leonard, K.J. 1993. Durable resistance in the pathosystems: maize-Northern and Southern leaf blights. In: *Durability of Disease Resistance.* Jacobs Th. and Parlevliet J.E.(Eds.), pp. 99-114. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Mayo, G.M.E. and Shepherd, K.W. 1980. Studies of genes controlling specific host-parasite interactions in flax and its rust. I. Fine structure analysis of the M group in the host. *Heredity* 44: 211-227.

Neervoort, W.J. and Parlevliet, J.E. 1978. Partial resistance of barley to leaf rust, *Puccinia hordei*. V. Analysis of the components of partial resistance in eight barley cultivars. *Euphytica* 27: 33-39.

Niks, R.E. 1981. Appressorium formation of *Puccinia hordei* in partially resistant barley and two non-host species. *Netherlands Journal of Plant Pathology* 87: 201-207.

Niks, R.E. 1982. Early abortion of colonies of leaf rust, *Puccinia hordei*, in partially resistant seedlings. *Canadian Journal of Botany* 60: 714-723.

Niks, R.E. 1986. Failure of haustorial development as a factor in slow growth and development of *Puccinia hordei* in partially resistant barley seedlings. *Physiological and Molecular Plant Pathology* 28: 309-322.

Niks, R.E., Ellis, P.R. and Parlevliet, J.E. 1993. Resistance to parasites. In: *Plant Breeding, Principles and Prospects.* Hayward, M.D. Bosemark N.O. and Romagosa I. (Eds.), pp. 422-447. Chapman & Hall, London.

Parlevliet, J.E. 1975. Partial resistance of barley to leaf rust, *Puccinia hordei*. I. Effect of cultivar and development stage on latent period. *Euphytica* 24: 21-27.

Parlevliet, J.E. 1978a. Further evidence of polygenic inheritance of partial resistance in barley to leaf rust, *Puccinia hordei*. *Euphytica* 27: 369-379.

Parlevliet, J.E. 1978b. Race-specific aspects of polygenic resistance of barley to leaf rust, *Puccinia hordei*. *Netherlands Journal of Plant Pathology* 84: 121-126.

Parlevliet, J.E. 1981. Race-non-specific disease

resistance. In: *Strategies for the Control of Cereal Disease.* Jenkyn F.J. and Plumb R.T. (Eds.), pp. 47-54. Blackwell Scientific Publications, Oxford.

Parlevliet, J.E. 1983. Can horizontal resistance be recognized in the presence of vertical resistance in plants exposed to a mixture of pathogen races? *Phytopathology* 73: 379.

Parlevliet, J.E. 1985. Resistance of the non-race-specific type. In: *The Cereal Rusts. II. Diseases, Distribution, Epidemiology and Control.* Roelfs, A.P. and Bushnell, W.R. (Eds.), pp. 501-525. Academic press, Orlando, Florida, USA.

Parlevliet, J.E. 1986. Pleiotropic association of infection frequency and latent period of two barley cultivars partially resistant to barley leaf rust. *Euphytica* 35: 267-272.

Parlevliet, J.E. 1989. Identification and evaluation of quantitative resistance. In: *Plant Disease Epidemiology, Vol. 2: Genetics, Resistance and Management.* Leonard K.J. and Fry W.E. (Eds.), pp. 215-248. McGraw-Hill Publ. Comp., New York.

Parlevliet, J.E. 1993. What is durable resistance, a general outline. In: *Durability of Disease Resistance.* Jacobs Th. and Parlevliet J.E.(Eds.), pp. 23-39. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Parlevliet, J.E. and Kuiper, H.J. 1985. Accumulating polygenes for partial resistance in barley to barley leaf rust, *Puccinia hordei.* I. Selection for increased latent periods. *Euphytica* 34: 7-13.

Parlevliet, J.E., Leijn, M. and Van Ommeren, A. 1985. Accumulating polygenes for partial resistance in barley to barley leaf rust, *Puccinia hordei.* II. Field evaluation. *Euphytica* 34: 15-20.

Parlevliet, J.E. and van Ommeren, A. 1975. Partial resistance of barley leaf rust, *Puccinia hordei.* II. Relationship between field trials, microplot tests and latent period. *Euphytica* 24: 293-303.

Parlevliet, J.E. and van Ommeren, A. 1988a. Accumulation of partial resistance in barley to barley leaf rust and powdery mildew through recurrent selection against susceptibility. *Euphytica* 37: 261-274.

Parlevliet, J.E. and van Ommeren, A. 1988b. Recurrent selection for grain yield in early generations of two barley populations. *Euphytica* 38: 175-184.

Parlevliet, J.E. and Zadoks, J.C. 1977. The integrated concept of disease resistance; a new view including horizontal and vertical resistance in plants. *Euphytica* 26: 5-21.

Pederson, W. and Leath, S. 1988. Pyramiding major genes for resistance to maintain residual effects. *Annual Review of Phytopathology* 26: 369-378.

Pouget, R. 1990. Histoire de la lutte contre le Phylloxera de la vigne en France (1868-1895). INRA, Paris.

Riley, R. 1973. Genetic changes in hosts and the significance of disease. *Annals of Applied Biology* 75: 128-132.

Roumen, E.C. 1992. Small differential interactions for partial resistance in rice cultivars to virulent isolates of the blast pathogen. *Euphytica* 64: 143-148.

Roumen, E.C. 1994. The inheritance of host plant resistance and its effect on the relative infection efficiency of *Magnaporthe grisea* in rice cultivars. *Theoretical and Applied Genetics* 89: 489-503.

Rubiales, D. and Niks, R.E. 1995. Histological characterization of *L34*, a major gene conferring non-hypersensitive resistance to wheat leaf rust. *Plant Disease.* (In press).

Thomas, C.A. and Blount, V.L. 1976. Race D of *Phytophthora phaseoli. Plant Disease Reproduction* 60: 308.

Toriyama, K. 1975. Recent progress of studies on horizontal resistance in rice breeding for blast resistance in Japan. In *Proceedings of Seminar on Horizontal Resistance to Blast Disease of Rice.* pp. 65-100. CIAT, Series CE9, Columbia.

Turkesteen, L.J. 1993. Durable resistance of potatoes against *Phytophthora infestans.* In: *Durability of Disease Resistance.* Jacobs Th. and Parlevliet J.E. (Eds.), pp. 115-124. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Turner, E.M.C. 1953. The nature of oats to the take-all fungus. *Journal of Experimental Botany* 4: 264-271.

Turner, E.M.C. 1961. An enzyme basis for pathogenic specificity in *Ophiobolus graminis*. *Journal of Experimental Botany* 12: 169-175.

Van der Plank, J.E. 1968. *Disease Resistance in Plants*. Academic Press, New York.

Van Ginkel, M., and Rajaram, S. 1993. Breeding for durable resistance to diseases in wheat: An international perspective. In: *Durability of Disease Resistance*. Jacobs Th. and Parlevliet J.E.(Eds.), pp. 259-272. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Wilcox, J.R., 1983. Breeding soybeans resistant to diseases. *Plant Breeding Review* 1: 183-235.