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Durability of resistance against fungal, bacterial and viral pathogens; present situation

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Summary

In evolutionary sense no resistance lasts forever. The durability of a resistance can be seen as a quantitative trait; resistances may range from not durable at all (ephemeral, or transient) to highly durable. Ephemeral resistance occurs against fungi and bacteria with a narrow host range, **specialists.** It is characterised by a hypersensitive reaction (HR), major gene inheritance and many resistance genes, which often occur in multiple allelic series and/or complex loci. These resistance genes (alleles) interact in a gene-for-gene way with avirulence genes (alleles) in the pathogen to give an incompatible reaction. The pathogen neutralises the effect of the resistance gene by a loss mutation in the corresponding avirulence allele. The incompatible reaction is not elicited any more and the pathogenicity is restored. The pathogens can afford the loss of many avirulences without loss of fitness. Durable resistance against specialised fungi and bacteria is often quantitative and based upon the additive effects of some to several genes, the resulting resistance being of another nature than the hypersensitive reaction. This quantitative resistance is present to nearly all pathogens at low to fair levels in most commercial cultivars. Durable resistance of a monogenic nature occurs too and is usually of a non-HR type. Resistance against fungi and bacteria with a wide host range, **generalists**, is usually quantitative and durable. Resistances against viruses are often fairly durable, even if these are based on monogenic, race-specific, HR resistances. The level of specialisation does not seem to be associated with the durability of resistance.

Introduction

All plant species, including our crops, employ defence mechanisms to avoid or to resist pathogens and pests. Many of them are effective against whole groups of parasites, **broad resistance** (Parlevliet, 1981a). Phyto-alexins, produced by nearly 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 specialised on plant species with that broad resistance. The phaseolin in beans for instance is degraded and/or tolerated by *Colletotrichum lindemuthianum*, while *Uromyces appendiculatus* prevents the induction of phaseolin production.

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

Resistance in many crops to various pathogens is often ephemeral, a problem largely confined to resistances against specialised pathogens (Parlevliet, 1993). Because many of the important diseases in our crops are caused by such specialised pathogens much resistance breeding is directed towards this group of pathogens.

Table 1. Number of years that the resistance in five wheat cultivars to yellow rust and in five barley cultivars to powdery mildew remained effective in The Netherlands (Anonymous, 1955–1994)

Wheat	Years	Barley	Years
Tadorna	1	Ramona	3
Flevina	5	Aramir	5
Norda	8	Impala	5
Felix	15	Belfor	8
Arminda	18	Minerva	20

Durable resistance, a quantitative concept

In nature, there is a constant arms race between the attacking parasite and the defending host, which can result in remarkable coevolution (Ehrlich and Raven, 1964; Parlevliet, 1986; Thompson, 1994). In evolutionary sense all resistance is ultimately transitory. Absolute durability does not exist.

In agriculture the durability of a resistance varies too. From zero years – the resistance is neutralised already in the last stages of the breeding program (Thomas & Blount, 1976) – to over 130 years as for instance with the *Phylloxera* aphid resistance of grape rootstocks (Pouget, 1990).

Johnson (1981) defined **durable resistance** as a resistance that remains effective while being extensively used in agriculture for a long period in an environment conducive to the disease. This definition is difficult to use as it depends on quantitative parameters, like extensive use, long period, and conducive environment, while loss of effective resistance can also be gradual. However, as a better definition is not available we will stick to this definition.

Durability of resistance, a quantitative characteristic

As mentioned above the effectiveness of a resistance can last from very short (typical non-durable resistance) to very long (durable resistance). It can be seen as a quantitative characteristic, its expression depending on genotype and environment, whereby the pathogen is part of that environment.

Its quantitative nature is shown in Table 1. It is clear that the monogenic resistances of the cvs Tadorna, Ramona, Flevina, Aramir, Norda, Impala and Belfor were non-durable, while the monogenic resistance of cv Minerva lasted much longer. The monogenic resistance in cabbage to cabbage yellows, caused by *Fusarium oxysporum* f.sp. *conglutinans,* lasted even longer. It has been effective since the 1920s in almost all areas where it has been used. There is no evidence that the polygenic, partial resistance to barley leaf rust, *Puccinia hordei*, in the cvs Minerva and Vada (Parlevliet, 1978) was more effective in 1955, when they were released, than it is now.

The environment can affect the durability considerably too (Parlevliet, 1993). The farming system can have a significant effect; the larger the proportion of an area covered by a crop the easier it is for a pathogen to develop new races. Sanitary and other measures taken to decrease the amount of inoculum too can reduce the possibilities for the pathogen to evolve new races. Agriculture itself represents an environment quite different from nature. In agriculture homogeneity tends to be the rule, in nature heterogeneity. In nature the reproduction of the host is often seriously affected by the pathogen (Burdon, 1987), in agriculture it is not as man takes care of that reproduction. Especially for a pathogen with a narrow host range there is no penalty for specialisation on its host in agriculture, but there can be in nature where a too high level of pathogenicity may endanger its host and so itself. The so called multiline effect on durability may therefore be considerably smaller in agriculture than in nature. An increased degree of specialisation in agriculture has been reported (Wahl et al., 1978). They observed that powdery mildew isolates from small grains or wild grasses in Israel have wider host ranges than isolates from small grains elsewhere. Isolates from wheat, barley and oats in the USA are fully specialized on their own cereal. They did not infect 27 grass species from various genera. Isolates from the three *ff. spp.* in Israel possess a much wider host range, attacking wild grasses from various genera.

So, durability of resistance is typically a quantitative characteristic and it is in this sense that durability is used here.

Durability of resistance, effect of the pathogen

Fungi and bacteria

Specialists. There is a group of pathogens in which races develop very easily and against which many race-specific resistance genes occur. Table 2 gives a representative sample of these pathogens. The resistance genes in their hosts, so notoriously non-durable,

Table 2. Crop-pathogen systems with many race-specific, non-durable resistance genes and many races known. B = biotrophic; HB = hemi-biotrophic; S = specialist, narrow host range (after Parlevliet, 1993)

Pathogen	Host	R-genes	
Fungi			
Puccinia hordei	barley	over 15	B,S
P. coronata	oats	over 30	B,S
P. sorghi	maize	over 25	B,S
P. triticina	wheat	over 40	B,S
Melampsora lini	flax	over 30	B,S
Blumeria graminis f.sp. *hordei*	barley	over 30	B,S
Bremia lactucae	lettuce	over 16	B,S
Cladosporium fulvum	tomato	over 11	B,S
Phytophthora infestans	potato	over 11	HB,S
Rhynchosporium secalis	barley	over 10	HB,S
Colletotrichum lindemuthianum	bean	over 10	HB,S
Magnaporthe grisea	rice	over 16	HB,S
Bacteria			
Xanthomonas oryzae pv. *oryzae*	rice	over 18	HB,S,
Pseudomonas syringae pv. *phaseolicola*	bean	5	HB,S,

are of the hypersensitive type. In this group many fungi and several bacteria, but no viruses are found. They have a biotrophic or hemibiotrophic nature.

Generalists. Resistance to generalists, such as *Sclerotinia sclerotiorum,* is as far as known highly durable and usually of a quantitative type (Bruehl, 1983, Parlevliet, 1989). No race-specificity has been reported within this group of pathogens.

Other pathogens. Between these two groups, representing the extremes, there are many pathogens with host ranges varying from fairly narrow to fairly wide of which either few or no races are known and against which resistance is in general considerably more durable than to the pathogens of the first group. Of those where some races have been identified (Table 3) the resistance is often highly durable. There are some fungal pathogens with a fairly narrow host range, where no races have been observed and where the monogenic resistance has been effective from the start (*Cladosporium cucumerinum* and *Corynespora melonis* in cucumber, *Periconia circinata* in sorghum, *Pseudocercosporella herpotrichoides* in wheat, *Cochliobolus victoriae* in oats).

Table 3. Host-pathogen systems with few races and a few race-specific resistance genes known: V = vascular wilt fungi; S = specialist. MS = moderately specialized; G = generalist; B = biotrophic; HB = hemibiotrophic (after Parlevliet, 1993)

Pathogen	Host	
Fungi		
Fusarium oxysporum f.sp. *lycopersici*	tomato	V,S,
〃 〃 f.sp. *pisi*	pea	V,S,
〃 〃 f.sp. *conglutinans*	cabbage	V,S,
Cochiliobolus carbonum	maize	N, S,
Ascochyta pisi	pea	HB, S,
Viruses		
Tobacco mosaic virus	tomato	B, MS
Virus X and virus Y	potato	B, MS
Peanut mottle virus	peanut	B, MS
Bean common mosaic virus	bean	B, MS
Bean yellow mosaic virus	bean	B, G
Soybean mosaic virus soy	bean	B, MS
Pea seedborne mosaic virus	pea	B, MS
Barley yellow mosaic virus	barley	B, MS

Viruses

Viruses are a quite different group of pathogens, with host ranges from quite narrow (Andean potato latent virus) to very wide (Tobacco mosaic virus, TMV, and

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

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

* Extremely susceptible control cultivar.

Tomato spotted wilt virus, TSWV, (Singh et al., 1995). The level of specificity does not seem to have any relation with the durability of virus resistance. Many viruses have developed some races (Table 3) among them viruses with a very wide host range such as TMV and TSWV. Others did not, such as potato leaf roll virus, groundnut bud necrosis virus and maize streak virus. Resistance to viruses, even when races developed, is often fairly durable even if the resistance is monogenic and of the hypersensitive type (Ross, 1983). Meiners (1981) concluded that all known resistances to pea viruses are race-specific but these resistances can be used very well as they appear to last long.

Apparently there are large differences in the flexibility of pathogen adaptation to introduced resistances. In addition it is difficult to deduce the expectation with respect to durability from the taxonomic position alone.

Durability of resistance, effect of the host

Fungi and bacteria

Resistance to specialists. Resistance to these pathogens is often very shortly effective (Table 4). This typical non-durable resistance (Table 2) is characterised 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. The flax-flax rust (*Melampsora lini*) pathosystem demonstrates this very well. Over 30 R-genes have been identified in 7 loci or tiny regions: K, L, M, N, P, D and Q. Regions N and P are linked, as well as regions N and K. The N region consists of at least two closely linked loci. The M region carries 4 closely linked loci. The L locus (14 alleles) does not fully behave as an allelic series, but neither behaves as closely linked loci (Islam & Shepherd, 1991).

These major R-genes operate on a gene-for-gene basis with avirulence genes in the pathogen. At infection the product of the avirulence gene is recognised by the product of the corresponding R-gene, inducing a complex series of events, including rapid and localised host cell death, that leads to resistance, the **hypersensitive reaction** (Moerschbacher and Reisener, 1997). Any mutation in the avirulence gene leading to non-recognition by the corresponding R-gene (loss mutation) restores the pathogenicity, unless the Avr-gene has an additional function (see below). The mutation can vary from a mutation at nucleotide-level (Joosten et al., 1994) to complete deletion of the avirulence gene (Joosten & de Wit, 1999). This loss of resistance, due to restored pathogenicity, is often described as 'virulence'. Specialised fungi and a few bacteria can accumulate such 'virulences' easily without measurable loss of fitness (Parlevliet, 1981b, Parlevliet, 1996). Stabilising selection in the sense of van der Plank (1968) does not seem to exist in this group of pathogens. The fact that a loss mutation can lead to restored pathogenicity is most likely the reason why the hypersensitive resistance to this group of pathogens is so easily overcome.

In bacteria there is now evidence that many Avr-genes have dual functions, a role in pathogenicity and a role in avirulence. The 'virulence' (pathogenicity) effects of these genes usually affect the population of the bacteria in the infected tissue and are discernible as causing changes in size, number or appearance of the lesions (White et al., 2000). A loss mutations in such a gene, leading to non-recognition by the corresponding R-gene, may or may not affect the 'virulence' function of the gene and so the fitness of the pathogen.

There are some major R-genes, such as the *Sr2* gene in wheat to wheat stem rust, the *Lr34* in wheat to wheat leaf rust, and the *ml-o* gene in barley to powdery mildew, that do not induce a hypersensitive reaction, but operate through a different mechanism. These genes are still effective after prolonged periods of exposure (Parlevliet, 1997).

Most of the durable resistance to this group of pathogens is of a quantitative nature based on the additive effects of some to several genes with smaller effects. This oligogenic or polygenic resistance is present at low to fair levels in most cultivars of nearly all crops to all important pathogens (Parlevliet, 1993). This quantitative resistance appears after introduced major R-genes become ineffective as Table 4 shows and is sometimes indicated as residual resistance. The level of this quantitative or residual resistance is often not inconsiderably and highly suitable for accumulating towards higher levels. The inheritance of quantitative resistance has been investigated in only a limited number of host-pathogen systems. The number of genes seems to range from 2 to 3 in maize to *P. sorghi* (Kim & Brewbaker 1977) and wheat to *P. triticina* (Broers & Jacobs, 1989), to several in wheat to *P. graminis* f. sp. *tritici* (Knott 1988), barley to *P. hordei* (Parlevliet, 1978), rice to *Xanthomonas oryzae* pv *oryzae* (Koch & Parlevliet, 1991) and maize to *Cochliobolus heterostrophus* and *Setosphaeria turcica* (Leonard, 1993). Black (1970) reported polygenic inheritance of field resistance in potato to *Phythophthora infestans* and so did Habgood (1974) in barley to *Rhynchosporium secalis*.

Resistance to specialised fungi and bacteria, whether major genic or polygenic is always **pathogen-specific**, i.e. directed to one pathogen species only (Parlevliet, 1981a). The polygenic resistance of barley to *P. hordei* is only effective to that pathogen species and does not operate to other *Puccinia* species, such as *P. striiformis* (Parlevliet, 1981a).

Resistance to generalists. Resistance to generalists is usually of a quantitative nature and highly durable. Resistance to one generalist may also give resistance to related other generalists.

Resistance to *Sclerotinia sclerotiorum* in the crops investigated is of the quantitative type and no race-specific effects have been observed (Boland, 1994). In sunflower the quantitative resistance (Table 5) also seems to operate to *S. minor* (Masirevic & Gulya, 1992).

Table 5. Seven sunflower lines tested for resistance to *Sclerotinia sclerotiorum* in two years. Resistance expressed as% diseased capitula (achenes without seeds), (Mündel et al., 1985)

Line	% capitula diseased	
	1982	1983
Lesaf 34C00	6	0.3
〃 〃 34A-YoYo	14	2
〃 〃 34B	25	4
Lesaf 15	45	4
Lesaf 16	49	22
RH3	56	29
Gila	62	21

Kernel ear rot in maize is caused by several generalists, *Fusarium moniliforme* and some other related *Fusarium* species and *Diplodia zeae*. Resistance to kernel ear rot is quantitative and polygenically inherited (Mesterhazy, 1989), and seems to operate to several of these pathogens (Gendloff et al., 1986; Mesterhazy, 1989).

Resistance to other pathogens. Monogenic, high-level resistance as well as quantitative resistance based on additive gene action occur frequently. Resistance to the former tends to be durable even when race-specific. Resistance to the latter is highly durable.

Viruses

Monogenic, complete resistance has been found in many crops to many viruses. The durability of these genes varies from very low, the *Tm1* gene in tomato to TMV, to very high, the *Tm2* gene in tomato and the *N*-gene in tobacco to TMV. The *Tm1* gene is in fact an exception. The majority of monogenic resistances are fairly durable to very durable. Interesting is the fact that resistances of the hypersensitive type to viruses are, in contrast to those against specialised fungi and bacteria, quite durable. The *Tm1* gene in tomato to TMV is of a non-hypersensitive nature, while the *Tm2* and $Tm2^2$ genes are of the hypersensitive type and much more durable. Fraser (1990) concluded that gene-for-gene relationships are assumed to exist and seem fairly durable (TMV-tomato; PVX and PVY-potato; BCMV-common bean; TMV-Capsicum).

Viruses often, but not always, develop races to these monogenic resistances, especially to the resist-

Table 6. Incidence (%) of groundnut bud necrosis virus of six groundnut genotypes in seven environments (four locations; Rajendranagar (RN), Narkoda (NAR), ICRISAT (ICR), Raichur (RAI) and three years) (Buiel & Parlevliet, 1995)

Genotype	Environment							
	RN 91	RN 92	NAR 93	ICR 91	ICR 92	RAI 91	RAI 92	Mean
JL 24	95	95	81	55	49	29	19	60.4
TMV 2	86	85	71	24	30	25	4	46.4
85/202-1	71	58	59	19	36	9	6	36.9
ICGV 89283	54	34	36	3	6	1	1	19.3
ICGV 86029	23	16	18	5	4	2	1	9.9
2169-5(9)	14	15	20	5	2	1	0	8.1

ances of the hypersensitive type, but these races do not seem to spread easily possibly due to impaired fitness (TMV-tomato R-genes *Tm2* and *Tm2*^{2}; several pea viruses-resistance genes to viruses). This is not surprising as the viral genome is very small and codes for only a restricted number of proteins. The viral-encoded proteins that are recognized by host R-proteins, are also required by the virus for replication, processing and/or envelopment (Nimchuk et al., 2001).

Monogenic resistance of the race-specific type to typical generalists has been reported several times, in contrast to fungal generalists. The R-genes to TMV mentioned above and the monogenic resistances in tomato to TSWV (Finlay, 1953; Paterson et al., 1989) are examples of it.

Quantitative resistance to viruses seems, as with fungi and bacteria, durable. This type of resistance is often characterised by a reduced incidence. All plants can be infected, but the chance of infection is reduced as exemplified by the resistance of groundnut to groundnut bud necrosis virus, a tospovirus related to tomato spotted wilt virus (Table 6). The differences in resistance are large and are expressed in a wide range of environments (Table 6) and the resistance seems polygenic. This type of quantitative resistance is widespread and is assumed to be polygenic (Ross, 1983; Parlevliet, 1993). Quantitative resistance against the potato viruses PVA, PVM, PVS, PVX, PVY and PLRV has been observed to occur at various levels and in many cultivars (Ross, 1983).

Table 7. 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[3]
IR50	38	48[1]	33	–
IR37704	38	37	21[2]	–
IR66	20	24	18	21b
IR36	12	12	14	13c
IR64	7	9	6	7d

[1] Significantly higher ($p < 0.01$) than the value of 36 expected when genetic interaction is absent.
[2] Significantly lower ($p < 0.01$) than the value of 37 expected when genetic interaction is absent.
[3] 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.

Durability of resistance, effect of race-specificity

Race-specificity is often considered as a clear indication of lack of durability and durable resistance is in that view expected to be non-race-specific. The facts, however, do not support this view.

The resistance genes of the hypersensitive type are clearly of a race-specific type. However, these R-genes against viruses are considerably more durable than those against the specialised fungi and bacteria.

The quantitative resistance to fungal generalists seems of a non-race specific nature, but the quantitative resistance to at least several fungal specialists

Table 8. Seven host-pathogen systems with a polygenic, quantitative resistance where small race-specific effects have been reported

Host	Pathogen	
Barley	*Rhynchosporium secalis*	(Habgood, 1976)
Barley	*Puccinia hordei*	(Parlevliet, 1977)
Wheat	*Puccinia triticina*	(Kuhn et al., 1978)
Potato	*Phythophthora infestans*	(Bjor & Mulelid, 1991)
Maize	*Cochliobolus heterostrophus*	(Leonard, 1993)
Maize	*Setosphaeria turcica*	(Leonard, 1993)
Rice	*Magnaporthe grisea*	(Roumen, 1994)

(Table 8) is not of the non-race-specific type, although all quantitative resistances based on some to several genes are durable. Table 7 shows such small race-specific effects in the rice-rice blast pathosystem and Table 8 gives a number of pathosystems with such small race-specific effects.

The experiments of Nelson et al. (1965) with northern leaf blight, *Setosphaeria turcica,* and of Kolmer and Leonard (1986) with southern leaf blight, *Cochliobolus heterostrophus,* corroborate the race-specific nature of polygenic, quantitative resistance. In both pathogens a selection experiment was done by crossing isolates obtained from the largest lesions with one another after which the progenies were inoculated on inbred lines with good levels of quantitative resistance. Again isolates obtained from the largest lesions were intercrossed and the progenies inoculated onto the same inbred lines. This selection was done for 3 cycles. In both experiments the lesion size increased significantly indicating increased aggressiveness. The mean diameter of the *Cochliobolus heterostrophus* lesions on the resistant inbred line 316 increased from 6.25 mm to 7.40 mm (18%). The selected pathogen was also tested on three inbred lines different from line 316. The increase in lesion size was significantly smaller, varying between 7 and 10%, indicating the cultivar-specific effect of the selection. Despite the fact, that both pathogens are able to accumulate genes for increased aggressiveness under experimental conditions, it does not seem to happen in the field. As the quantitative resistance has not shown any sign of erosion; it is considered to be highly durable (Leonard, 1993).

Even broad resistance, the resistance operating to wide groups of pathogens and considered as highly durable can be race-specific as will be discussed below.

Race-specificity therefore is a poor indication for the durability of a resistance.

Durability of resistance, no resistance is sacrosanct

In the introduction it was already mentioned, that in evolutionary sense no resistance would last forever. Even in the relatively short period of modern agriculture this appears to be true.

The adaptation of the specialised fungi and bacteria to the introduced R-genes of the hypersensitive type (Table 1) is often so fast, that it is questionable whether these R-genes have a protective function in nature. Several R-genes in potato to *Phytophthora infestans* and in lima beans to *P. phaseoli* (Thomas & Blount, 1976) were already neutralised in the breeding phase.

Adaptation to quantitative resistance based on some to many genes is apparently very difficult for pathogens. There are no reliable reports about erosion of such resistances, not even to specialised fungi and bacteria. However, there is one exception, the field resistance to late blight, *P. infestans,* in potato cultivar Pimpernel, a cultivar without known R-genes and carrying a good level of quantitative resistance in both foliage and tubers. The field resistance of the foliage to this pathogen is polygenically controlled (Black, 1970) and is highly durable (Turkensteen, 1993). The field resistance of the foliage and the quantitative resistance of the tubers are not associated (Anonymous 1953–1994; Turkensteen, 1993) but tuber resistance is as durable as foliage resistance (Anonymous, 1953–1994). However, Bjor & Mulelid (1991) reported some erosion of the tuber resistance of cv Pimpernel. Nevertheless, the resistance appeared durable since its fairly high resistance remained effective over its commercial life time in The Netherlands (Anonymous, 1953–1988). Moreover, its resistance remains to be effective in other countries where it is still grown. The isolates found in Norway, showing an increased aggressiveness on tubers of cv Pimpernel, had no such increased aggressiveness on other cultivars, indicative of the race-specific character of this quantitative resistance.

Even broad resistance is not sacrosanct. Saponins are plant glycosides that occur in a great many plant species, and have been implicated as pre-formed determinants of resistance to fungal attack. A number of fungi that succeed in breaching these antimicrobial plant defences produce saponin-detoxifying enzymes

Table 9. Frequency (Freq.) of resistance types in 4 groups of pathogens and the mean durability (Dur.) of that resistance type

Type of resistance	Fungi/bacteria				Viruses	
	Specialists		Generalists		Specialists and generalists	
	Freq.	Dur.	Freq.	Dur.	Freq.	Dur.
Monogenic-HR[1]	++++[2]	–[3]	–		++	+ to +++
Monogenic-non-HR	+	+++	–		++	– to ++++
Polygenic	++++	++++	+++	++++	++++	++++

[1] Hypersensitive type of resistance.
[2] Frequency; from extremely low or absent (–), to low (+), to moderately frequent (++), to frequent (+++), to nearly always present (++++).
[3] Durability; from typical non-durable (–), to moderately durable (+), to fairly durable (++), to durable (+++), to highly durable (++++).

(Osbourn, 1996). This was demonstrated for the Take-all disease, caused by *Gaeumannomyces graminis.* It is a soil borne pathogen infecting the root systems of grasses. Wheat, barley and rye can be infected by it, but oat is resistant as its roots produce avenacin, protecting it from various pathogens including take-all (Turner, 1953; Schaefer, 1994). In Wales, an isolate was found able to attack oat, because it produced avenacinase, an enzyme that hydrolyses avenacin into products less toxic to the pathogen (Turner, 1961). Isolates able to attack oat have been found elsewhere too, but they do not spread. Oat therefore remains resistant to take-all.

Conclusions

Fungi and bacteria

Specialists. Monogenic resistance of the hypersensitive type is typically non-durable. All other monogenic resistances are considerably more durable.

The quantitative resistance based on the additive effects of some to many genes is highly durable. It occurs in most cultivars to most important pathogens (Table 9).

Generalists. The resistance is largely of a quantitative type and highly durable.

Other pathogens. Most monogenic resistance, even when race-specific, is quite durable. The quantitative resistance is very durable. It occurs in most cultivars to most pathogens.

Viruses

Many monogenic resistances, also those of the hypersensitive and race-specific type, are quite durable, while only a few R-genes can be classified as ephemeral. Such notoriously non-durable R-genes can be genes against typical generalists. Quantitative resistance, usually expressed as a reduced incidence, is very durable. It occurs in most cultivars in most crops to most viruses (Table 9).

Race-specificity is not a good indicator of the durability of resistance.

It is important to realise, that adaptation to the hypersensitive R-genes by fungal and bacterial specialists is quite different from the adaptations to polygenic quantitative resistance or to broad resistance. In the former a loss mutation in the avirulence gene results in restoring the pathogenicity. In the latter, exemplified by the cereals/take-all pathosystem, the mutation is due to a gain mutation; the pathogen must add a new characteristic or change an existing characteristic in a specific way. Adaptation by a loss mutation is expected to be much easier than adaptation through a gain mutation. This is the main reason for the large difference in durability.

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