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Combination of mechanisms of resistance to rust fungi as a strategy to increase durability

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SUMMARY – There has been some degree of success in achieving durable resistance even though we still do not fully understand what actually causes a resistance to be durable, nor how to distinguish durable resistance from non-durable resistance, nor even what criteria to use to decide whether a resistance is durable. We present some examples where resistance to cereal rusts that proved to be durable is of complex inheritance, and also others where durability is due to particular single genes that cause relatively small effects on the pathogen development, and yet seem to be race-non-specific and not based on hypersensitivity. Their effect can be greatly enhanced if they occur in combination with other, even when these may be race-specific, resistance genes. Combinations of different resistance mechanisms seem to be of great value to increase durability of resistance. It is generally agreed that the use of cultivars with single-gene race-specific resistance should be avoided, but such genes could still be of great help in enhancing and/or protecting durability. An alternative to the development of individual cultivars with long-lasting resistance might be the use of genetic diversity for resistance and diversity in its strategic deployment, and may include combining of race-specific with non-specific resistance. A key point is that the presence of genes conditioning complete resistance should not mask the small effects of the other genes during selection. Availability of markers is an advantage in combining genes for resistance.

Key words: Histology, *Puccinia graminis*, *P. recondita*, *P. striiformis*, resistance, rust, wheat.

RESUME – “Combinaison de mécanismes de résistance aux champignons de la rouille comme stratégie pour augmenter la durabilité”. Nous avons connu un certain degré de réussite dans l’obtention d’une résistance durable bien que nous ne comprenions pas encore pleinement ce qui fait réellement qu’une résistance soit durable, ni comment distinguer une résistance durable d’une résistance non durable, et pas même quels critères utiliser pour décider si une résistance est durable. Nous présentons quelques exemples où la résistance aux rouilles des céréales qui s’est montrée durable est d’hérédité complexe, et également d’autres où la durabilité est due en particulier à des gènes uniques qui causent des effets relativement faibles sur le développement du pathogène, et cependant semblent être non spécifiques à la race et non basés sur l’hypersensibilité. Leur effet peut être fortement augmenté s’ils sont en combinaison avec d’autres, même s’il s’agit de gènes de résistance non spécifiques à la race. Des combinaisons de différents mécanismes de résistance semblent être de grande valeur pour augmenter la durabilité de la résistance. Il est généralement admis que l’utilisation de cultivars ayant une résistance à gène unique et spécifique à la race devrait être évitée, mais ces gènes pourraient cependant être d’une grande utilité pour augmenter et/ou protéger la durabilité. Une alternative au développement de cultivars individuels avec une résistance de longue durée, pourrait être l’utilisation de la diversité génétique pour la résistance et de la diversité dans son déploiement stratégique, et peut inclure la combinaison de résistance spécifique à la race avec la résistance non spécifique à la race. Un élément clé est que la présence de gènes conditionnant la résistance complète ne devrait pas masquer les faibles effets d’autres gènes pendant la sélection. La disponibilité de marqueurs est un avantage pour combiner des gènes pour la résistance.

Mots-clés : Histologie, *Puccinia graminis*, *P. recondita*, *P. striiformis*, résistance, rouille, blé.

Rusts have threatened wheat production since the early times of agriculture. Greek and Roman classical authors were familiar with the cereal rusts and had a notion about ecological conditions favouring rust attack. Rust epidemics were so important that they were symbolised in religious ceremonies. Ovid (43 BC - 17 AD) described the tradition of igniting torches tied to the tails of foxes which were set loose in the Circus of Rome. This practice became established as an annual ritual to avert rust epidemics. A more sacred festival, the Robigalia, was celebrated annually on April 25 from about 700 BC in honour of the rust god Robigo. The pagan ceremony of the Robigalia was later incorporated into Christian tradition as the annual rite of St. Marks Day, or Rogation (Littlefield, 1981).

Some centuries later, astute observations by French farmers that cereal rust was more severe near barberry bushes led in 1660 to the Barberry Laws requiring the destruction of barberry plants near grain

fields. This was long before the fungus causing stem rust was described (1797) and its life cycle understood (1927). In a similar way, long before Mendel laid the foundation of modern genetics, clever farmers and early plant breeders were empirically selecting resistant plants. The scientific basis of disease resistance breeding research was not laid until 1905 by Biffen, in PBI, UK, who demonstrated that resistance to yellow rust was inherited in a Mendelian way. Soon, extensive breeding programmes were initiated in many countries, and numerous rust resistant cultivars have been produced. To date, more than 45 single genes for leaf rust resistance, about 20 for yellow rust resistance and about 55 for stem rust resistance have been identified, many of them introduced from wild species. Many of these single genes for resistance to rust are already present in the A or B genomes (see Roelfs *et al.*, 1992) and thus, are relatively easily accessible for durum wheat breeding. New still unnamed genes are continuously being transferred to durum wheat from wild relatives (Bai *et al.*, 1998).

Despite the monumental strides made in breeding for rust resistance, for many decades (Stakman, 1946) breeders and pathologists have been aware that much of their work to introduce resistance in high yielding cultivars has been inadvertently ephemeral. For decades, selection for resistance was based on highly specific, clearly recognised complete resistance, which is usually controlled by a single gene, and this form of resistance has commonly proved ephemeral due to the evolution of virulent fungal isolates that negated the breeders' efforts and lead to spectacular "boom and bust" cycles.

Many terms and concepts have been proposed to describe and characterise other types of resistance. These include terms such as slow rusting, field, intermediate, quantitative, incomplete, general, partial, horizontal, adult plant, race-non-specific resistance, etc. Each of these terms has a specific meaning and specific implications, but they all describe a quantitative effect on the epidemic, and many of them often are taken to imply complex inheritance. The interest in these types of resistance is fuelled by the hope that they may be more durably effective against the target pathogen. However, according to Johnson (1984) resistance can only be identified as being durable when a cultivar is widely grown for a considerable period of time. For this reason durability can only be identified in retrospect. The fact is that we still do not understand what actually causes a resistance to be durable, nor how to distinguish durable resistance from non-durable resistance, nor even what criteria to use to decide whether a resistance is durable. The question is how to identify durable resistance and how to select for it. The problem is not an easy one, and is difficult to extrapolate from one pathosystem to another.

The great need for durable types of resistance has prompted scientists to design and propose several approaches to improve the durability of intrinsically "non-durable" types of resistance, or to identify and introduce resistance types that are intrinsically durable, if such resistance exists. The validity of such proposals have often been questioned, but research has made some progress in this particular field of science.

Among the strategies that have been proposed to increase the durability of "non-durable" resistance genes is the diversification of applied genes for resistance by the introduction of multilines or cultivar mixtures and through regional gene deployment. Also the introduction of more than one effective gene for complete resistance (multiple gene barriers or resistance gene pyramiding) is considered a potentially effective approach.

Several strategies have been suggested to exploit putative "alternative types of resistance" while avoiding the use of ephemeral single gene-controlled resistance. Robinson (1980) proposed accumulating minor genes for resistance to obtain durable resistance. Progeny from crosses between agronomically adapted local susceptible cultivars may show transgressive segregation for higher levels of resistance. The cultivars to be used for such a cross should show a fully susceptible infection type to avoid accumulation of race-specific hypersensitive resistance factors. Parlevliet (1978) described partial resistance (PR) as a type of resistance that retards epidemic development in the field, although plants show a compatible (non-hypersensitive) infection type. He also proposed a recurrent selection against extreme susceptibility to accumulate partial resistance in each recurrent selection cycle (Parlevliet and Van Ommeren, 1988). He also recommended selection against complete resistance to avoid major race-specific genes. Such complete major gene resistance would hamper the evaluation of the level of partial resistance in the germplasm, and hence, reduce the efficiency of the selection for partial resistance. However, the system of accumulating resistance by transgressive segregation from relatively susceptible lines although promising, has not resulted in successful cultivars and has not been a mainstream breeding system of any of the larger breeding programmes.

Johnson (1984) advocated the use of cultivars in which the resistance has proven durable in the past as sources of resistance for future cultivars. Indeed, astute breeders have identified cultivars in which the

resistance genes appear to remain effective despite extensive use in international breeding and agricultural programmes. Usually the mechanistic basis of the apparently durable resistance is unknown, but there are indications that durability usually depends on a combination of genes affecting several mechanisms of resistance (Martens and Dyck, 1988; Roelfs, 1988; McIntosh, 1992; van Ginkel *et al.*, 1992; Line and Chen, 1995).

For example, the stem rust resistance derived from the variety Hope (*Sr2* complex) in combination with other genes seems to have provided the foundation for durable resistance to stem rust in CIMMYT germplasm (Roelfs, 1988; Van Ginkel and Rajaram, 1992). *Sr2* is a gene that causes a reduced receptivity in the adult plant stage. This gene does not provide immunity and under high inoculum densities is often characterized by susceptible type lesions. *Sr2* alone gives "slow rusting", but its effect on the epidemic is enhanced when combined with other genes (Sunderwirth and Roelfs, 1980). Also the gene *Sr36* derived from *T. timopheevii* that confers a slow rusting resistance due to low receptivity appears to offer useful protection in combination with genes for specific resistance (Rowell, 1982) and has been described as providing durable resistance since the 1960's (McIntosh, 1992).

Although there is some understanding of resistance to rusts in bread wheats, little is known about resistance in durum wheats. However, the above mentioned *Sr2* was identified early in durum wheat and then transferred to the bread wheat cvs Marquillo, Hope and H-44 that are in the pedigree of numerous modern bread wheat cultivars. Additional sources of durable resistance to stem rust in durum wheat have been reported, like the durum wheat cv. Glossy Huguenot, effective in Australia over the past 90 years (Hare, 1997). Interestingly, its resistance is also quantitative and based on a reduced number of pustules in adult plants and a delayed onset of disease.

Durable resistance to leaf rust is thought to be more difficult to obtain than to stem rust. But resistance against leaf rust has been identified that appears more durable than usual. Especially resistance in the bread wheat cultivars Americano 44D, and Frontana, derived from resistant landraces, appears to be durably effective. The resistance in Frontana has been reported to be based on four additive genes, one of which is *Lr34*. Such durable resistance to leaf rust is presumably the result of a few particular gene combinations. It appears that *Lr12* and *Lr13*, both genes for hypersensitive resistance that is expressed only in the adult plant stage, in combination with *Lr34*, are the basis of most of this resistance (Roelfs, 1988; Van Ginkel and Rajaram, 1992). Also the resistance of Pavon 76 is believed to be durable. Pavon has displayed slow rusting since its release in 1976, and this has been ascribed to two genes, one of which has been identified and named *Lr46*. In addition, Pavon 76 possesses *Lr1*, *Lr10* and *Lr13* (Singh *et al.*, 1998). The effect of *Lr46* is similar to that of *Lr34* (Rubiales *et al.*, 1999). Both *Lr34* and *Lr46* confer quantitative resistance that increases latency period and decreases infection frequency and uredium size, and both are more clearly expressed in adult plants than in seedlings. The resistance of *Lr34* and *Lr46* is based on reduced rates of haustorium formation in the early stages of infection and is not associated with a hypersensitivity reaction of the plant cells (Rubiales and Niks, 1995; Rubiales *et al.*, 1999). In all these aspects they are therefore similar to the polygenic partial resistance of wheat to *P. recondita* (Niks, 1983; Jacobs, 1990).

Durable resistance to leaf rust has been reported in the Italian durum wheat cv. Creso (Pasquini and Casulli, 1993). Its resistance has remained effective since 1975 but there is no good information on the mechanistic or genetic control of the resistance although it seems that a combination of race-specific and partial resistance genes might be involved (M. Pasquini, pers. comm.).

As a further complication, there are indications that leaf rust populations collected on durum wheats could be classified in two separate species (Anikster *et al.*, 1997). The two groups differ in the aecial host, and are morphologically distinct and genetically isolated from each other. Also an earlier report suggested that leaf rust populations specific for durum wheats in some areas are distinct from those of bread wheat (Huerta-Espino and Roelfs, 1989). Further studies are needed to determine the effectiveness of resistance genes to one or both of the putative species.

The concept of partial resistance was established in the pathosystem barley/*P. hordei* and it was suggested that similar types of resistance would also occur in other rust and mildew pathosystems. Also in wheat/*P. recondita* (Jacobs, 1990), wheat/*P. graminis* (Broers and López-Atilano, 1995) and wheat/*E. graminis* (Pearce *et al.*, 1996) PR was found to occur. However PR to yellow rust appears to be rare in modern wheat cultivars (Danial, 1992), although it was found to be common in old winter wheats (van Dijk *et al.*, 1988).

It is important to note that slow epidemic progress in the field is not necessarily due to partial resistance *sensu* Parlevliet (1978), nor that such a resistance is necessarily durable. Slow epidemic progress can also be due to an incompletely acting race-specific hypersensitive resistance, that may (or may not) be expressed only in the adult plant stage. A third phenomenon that may cause “slow rusting” is the presence of one or more genes for complete resistance in the host where mixed pathogen populations contain virulent and avirulent genotypes (Parlevliet, 1983). Thus, monocyclic greenhouse experiments with a virulent isolate are needed to investigate whether the slow rusting observed in the field is partial resistance *sensu* Parlevliet (1978).

Durable resistance to yellow rust has been described (Johnson, 1984; van Dijk *et al.*, 1988; Line and Chen, 1995; Zhang, 1995), but in contrast with leaf rust, there is no clear phenotypic distinction in reaction type of cultivars that express durable resistance and those in which resistance was not durable. High levels of resistance in durably resistant cultivars were associated with low infection type, just as in the resistance of slow-rusting cultivars that later became susceptible (Johnson, 1992b). Durable resistance that does not show race-specificity to yellow rust has been described in the adult plant stage. For instance, the cultivar Cappelle Desprez has durable yellow rust resistance and in addition to some race-specific resistance genes it possesses others that contribute to quantitative adult plant resistance that does not appear to be race-specific (Johnson, 1984). Milus and Line (1986) reported temperature-sensitive minor-genes for resistance that they considered race-non-specific and suggested could be considered durable. Van Dijk *et al.* (1988) considered that partial resistance combined with temperature sensitive resistance were the major components of the durable resistance found in old winter wheat cultivars in the Netherlands. In contrast to the temperature sensitive resistance described by the American workers, those reported by van Dijk *et al.* (1988) appeared to be race-specific.

Histological studies on interactions between plants and rusts can help both to discern the various resistance mechanisms and to combine them in a genotype in the hope to increase durability. The commonly used hypersensitivity resistance, typically conferred by single genes with race-specific effectiveness, is due to a posthaustorial defence mechanism. The plant cell collapses after the rust fungus has started to form a haustorium in that cell. Mechanisms of resistance acting before the formation of haustoria also exist, and their application should contribute to the diversity of defence of the cereal crops to the rust fungi. Rust infection can be hampered at very early stages of fungal development, from spore deposition to stomata recognition, resulting in a reduced penetration of the fungus into the tissue. However, in most known cases in cereals, infection units usually develop normal haustorial mother cells. Nevertheless, haustorium formation can still be prevented by papilla deposition within host cells attacked by haustorial mother cells. This type of resistance is very common in nonhost interactions (Rubiales and Niks, 1992a). It also is the mechanism responsible for the partial resistance of some barley cultivars to barley leaf rust (Niks, 1983) and of some wheat cultivars to wheat leaf rust (Jacobs, 1990). In addition, pre-haustorial resistance to wheat leaf rust has also been reported to occur in some diploid wheat accessions (Niks and Dekens, 1991), and in wheat cultivars that carry particular resistance genes such as *Lr34* and *Lr46* (Rubiales and Niks, 1995; Rubiales *et al.*, 1999).

Even the habit of plants (erect versus horizontal leaf attitude) can affect spore deposition and rust epidemic development (Gasowski, 1990). Resistance due to inhibition or reduction of uredospore germination is rare, but has been reported for *P. striiformis* on different parts of the wheat leaves and leaf surfaces (Russell, 1976) and in some non-cereal crops where reduced germination has been ascribed to reduced wettability of the leaf surface (Cook, 1980). Nevertheless, even on nonhost plants, reports of reduced rust spore germination are rare and it seems the rule rather than exception that urediospores germinate well. On many nonhost species, however, germ tubes appear to have difficulty in locating and recognising stomata. This is the rule if the non-host species is unrelated or only distantly related to the host species. On lettuce, for example, the germ tubes of barley leaf rust have great difficulty in finding stomata, apparently due to the entirely different epidermal morphology (Niks, 1981). In cases where the non-host is more closely related to the host, the epidermal structure is usually very similar to that of the host species, and hence, the germ tubes are well able to find the stomata. Most rust species collected from grasses efficiently found and penetrated stomata of barley (Niks, 1986). However, on some plant species closely related to the host, the rust may not be able to locate stomata efficiently. This is the case for some accessions of wild barleys inoculated with several leaf rusts (Rubiales and Niks, 1992b; Rubiales *et al.*, 1996). This phenomenon may be attributed to excessive wax covering the stomata hiding morphological features that normally induce the differentiation of the appressorium (Rubiales and Niks, 1996). Another possible resistance mechanism may cause the cessation of the fungal growth during penetration through the stoma into the plant, or poor development of the substomatal vesicle. For resistances affecting these stages of fungal development there is, however, hardly or no evidence yet.

Thus, we suggest that combining different resistance mechanisms acting at different stages of the infection process could provide multiple barriers that are not easily overcome by simple race-type changes of the pathogen. Evaluation of the mechanisms involved in rust resistance that has proven to be durable should get higher priority. Utilisation of a wide array of resistance mechanisms in modern germplasm would contribute considerably to durability. There is still a range of resistance mechanisms not exploited in wheat breeding like those reducing spore deposition, germination, appressorium formation or development of substomatal vesicle.

The examples cited above suggest that resistance to cereal rusts that has proved to be durable is often due to particular genes that cause relatively small effects on the pathogen development, and seem to be race-non-specific and not based on hypersensitivity. Their effect can be greatly enhanced if they occur in combination with other, maybe race-specific, genes. Combination of different resistance mechanisms seems to be of great value to increase durability of resistance. It is generally agreed that the use of cultivars in which the resistance depends solely on a single gene for race-specific hypersensitivity should be avoided. Also, creation of new effective combinations of genes already known individually to have been overcome by the pathogen should be avoided (Johnson, 1992a,b). But race-specific genes could be of great help in enhancing and/or protecting durability. The key to the development of cultivars with long-lasting resistance might be diversity. This might include genetic diversity in the type of resistance and diversity in their strategic deployment, and may involve combining of race-specific with non-specific resistance. An important point is that the presence of genes for complete resistance should not mask the small effects of the other genes during selection. Availability of markers is an advantage to combine genes for resistance. The availability of morphological markers for *Lr34* (leaf tip necrosis, association with resistance genes to other rusts) and *Sr2* (pseudoblack chaff on glumes or upper stems) facilitated their widespread utilisation in breeding programmes. Molecular markers are becoming available for many genes (see Gupta *et al.*, 1999) and their use in marker-assisted selection will certainly have a remarkable impact in practical breeding.

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