Prime Scholars Library

Advances in Agronomy, Plant Breeding and Horticulture

Review

Vol. 8 (1), pp.10 - 17, January, 2020

Available online at
https://primescholarslibrary.org/

Strong rust resistant gene in wheat

Aluko M. S. and Aluko Tiwari

Directorate of Wheat Research, Karnal 132 001, India.

Abstract

Several rust resistance genes have been identified and used in breeding for resistance but new variants of the pathogen (referred to as races) overcome the resistance over a period of time. Most plant breeders and pathologists now advocate durable resistance to rusts based on multiple genes as best source of resistance as breeding for this type of resistance tends to produce long-lasting solutions. Wheat breeders are now increasingly focusing on the identification and incorporation of race non-specific resistance genes that may provide only partial resistance but when used in combination with other genes can condition highly effective resistance. Molecular markers are becoming available for many genes and their use in marker-assisted selection will certainly have a remarkable impact in practical breeding.

Keyword: Durable resistance, molecular markers, wheat, pathogen, *Lr34* gene.

INTRODUCTION

Wheat has accompanied humans since 3,000 to 4,000 BC. It has evolved in part by nature and in part by human manipulation from its primitive form (einkorn wheat) into the present main cultivated species; bread wheat (*Triticum aestivum* L.) Bread wheat is one of the most important staple crops worldwide, with a total production of over 600 millions tones annually. It is also the main staple food of India and occupies a central position in agricultural policies. The demand for wheat, based on production and stock changes, is expected to increase from the current level of approximately 625 million tons to around 813 million tons in 2030 and more than 900 million tons in 2050 (FAO, 2006).

Apart from the abiotic factors including scarcity of water and many agronomic implications, there are enormous biotic factors, which lower the yield, wherever the wheat is grown in the country. Wheat is attacked by a number of diseases such as rusts, smuts, bunts, leaf blight, powdery mildew and head scab which cause great losses to the quality and quantity of the produce. Among the biotic stresses, rusts are the most significant diseases of wheat in India and worldwide. Inspite of enormous progress made in rusts control in many countries (Saari and Prescott, 1985), the occurrence of rust diseases in cultivated wheat has significantly influenced the development of human civilization (Roelfs et al., 1992). The rust diseases of wheat have historically been one of the major biotic production constraints both in Asia and the rest of the world. Rusts remain the most important diseases of wheat worldwide because of their wide distribution, their capacity to form new races that can attack previously resistant cultivars, their ability to move long distances and their potential to develop rapidly under optimal environmental conditions that result in serious yield losses. Leaf rust, stem rust, and stripe rust comprise the three rust diseases of wheat.

The most efficient and environmentally friendly method to reduce yield losses due to the leaf rust pathogen is to use resistant cultivars (Knott, 1989). Chemical control is not justified under low yielding and low priced circumstances such as those found in many developing countries. Rust resistance genes offer a cost-effective strategy to reduce losses in wheat from attack by rust pathogens (Spielmeyer et al., 2005). 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.

Abbreviations: HR, Hypersensitive reaction; **QTL,** quantitative trait loci; **DNA,** deoxyribonucleic acid; **BACs,** bacterial artificial chromosomes; **wESTs,** wheat expressed sequence tags; **MAS,** marker-assisted selection; **GS,** genomic selection.

For decades, selection for resistance was based on highly specific, clearly recognized 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. The breakdown of genetic resistance occurs due to the evolution of the local pathogen population because of selection for mutants, recombinants or immigrants that were better adapted to the resistant cultivar. All pathogens are variable with respect to host resistance but the virulent is in itself a variable quality. Self-pollinating cereal crops being grown over large area provide a platform for the capture and multiplication of pathotypes that are highly virulent on particular varieties, even though such pathotypes may remain rare and in hidden state during the development of the variety.

GENE FOR GENE INTERACTION

Flor (1946), studied inheritance of pathogenicity in the pathogen and inheritance of resistance in the host using flax (*Linum usitatissimum*)-flax rust (*Melampsora lini*) as a model system and concluded, that for every gene that conditions resistance in the plant, there is a corresponding and complementary gene that conditions avirulence in the pathogen (Flor, 1971), and only the corresponding avirulence gene can initiate the hypersensitive reaction (HR) leading to incompatibility. Resistance and avirulence inherit in most cases in a dominant manner while, susceptibility and virulency in a recessive manner. This system has the implication that resistance will not remain effective if the pathogen acquires the corresponding virulence by losing the avirulence alleles that elicits resistance either by deletion or by genetic change. Gene-for-gene system occurs most clearly in pathosystems where a biotrophic, highly specialized pathogen is involved such as cereals with various rusts, smuts and bunts, and with the powdery mildew. There are nevertheless numerous examples of interactions in which no such relationship has been found. Examples include Flag smut disease of wheat (McIntosh et al., 1983), eyespot disease of wheat (Scott and Hollins, 1977), potato aphid on tomato coffee berry disease (Vander, 1981) and many diseases of sugarcane (Robinson, 1976). Ellingboe (1982) stated that about 95% of all analyzed disease resistance operates on gene-for-gene pattern with the pathogen.

TYPES OF RESISTANCES IN PLANTS

Quantitative resistance is defined as resistance that varies in continuous way between the various phenotypes of the host population, from almost imperceptible (only a slight reduction in the growth of the pathogen) to quite strong (with little growth of the pathogen). Quantitative resistance occurs at various levels to nearly all-important pathogens in most cultivars of the crops (Xavier et al., 2001). Its expression depends upon the genotype and environment, whereby the pathogen is part of that environment. 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. Incomplete or partial resistance is assumed to be under polygenic control and such resistance will be race-non-specific. This does not establish that all durable resistance is controlled by many genes, but, even where it is, this need be no deterrent to its use by plant breeders who frequently select for characters, such as yield, that are also controlled by many genes. Polygenes need not be widely dispersed through a genome but many be gathered in groups (Arnold and Brown, 1968; Thoday, 1961) thus facilitating their manipulation by breeders. However, in several host pathogen systems which demonstrated durability, polygenic resistance race-specific effects were demonstrated (Parlevliet, 1997). Parlevliet and Zadoks (1977) realized that in gene-for gene relationships, the race-specific effects are of same size as the gene effects. Major resistance genes are associated with clear, identifiable races, while the polygenes result in only small race-specific effects, insufficient for unambiguously identifying races.

Horizontal, uniform, race-non specific or stable resistance can be discerned according to Vanderplank (1968) from vertical, differential, race specific or unstable resistance by a test in which a number of host pathogens (cultivars) are tested against a number of pathogen genotypes traces of isolates. Vertical (qualitative, major) resistance is specific to pathogen isolates based on single or very few genes. Partial resistance (horizontal, quantitative or minor gene) is characterized by slow development and reproduction of the pathogen. Partial resistance leads to delayed onset of infection (longer latent period), a reduced final extent of attacked leaf area. Race-specific is used to describe resistance that interacts differentially with pathogen races; it is applied both to complete resistance and the components of incomplete resistance that so interact. However, non specificity is only recognized by the absence of specificity and because all tests are of limited size, the presence of race-non-specificity can never be proved. At most, resistance that has not shown any specificity after prolonged testing could be classified as apparently non specific (Scott et al., 1980).

The concept of horizontal (Vanderplank, 1963; Robinson, 1973), uniform or non race specific resistance which by definition is permanent because any variety possessing it should be effective to the same degree against all the races of a pathogen regardless of their differences in specific virulence on other varieties. This is

contrasted with vertical (Vanderplank, 1963; Robinson, 1973), differential (Robinson, 1969) or race specific resistance towards which the biotypes of a pathogen show obvious differences in specific virulence and which often proves temporary (Robinson and Chiarappa, 1975). The operation of all resistance and virulence genes in a natural population is therefore seen as one integrated system. All resistance genes in the host population, whether they are major or minor genes are considered to interact in a gene-for- gene way with virulence genes either major or minor, in the pathogen population. Populations with a polygenic resistance based on gene-for-gene action have an increased level of resistance.

OVERCOMING THE RESISTANCE

Resistance introduced in a cultivar remains effective till the pathogen does not become virulent on the cultivar. Favorable changes in the environment undoubtedly play a role in reducing the effectiveness of resistance. Increased pathogenicity in pathogens is probably the commonest cause for failure of resistance. The rate of evolution of pathogen is dependent on many factors, not least the size of the pathogen population and its inherent variability. The resistance in a widely grown cultivar becomes ineffective after passage of time, as the pathogen that was earlier not able to defeat the immune system of this cultivar has evolved to greater pathogenicity on getting favorable conditions or by an increase in susceptible host plants other than the cultivar under test. Such environmental factors could also include reduction in resistance in the host cultivar due to changes in the conditions of cultivation, for example, higher or lower fertility or moisture. 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. Durable resistance is a retrospective, empirical description of resistance that has remained effective during prolonged and widespread use in an environment conducive to the disease (Johnson, 1984), having no priori genetic or physiological characteristics. Although the concept of durable resistance and resistance gene deployment has been around for several decades, durable resistance has remained an elusive goal for most crop improvement programs. Resistance in a cultivar is described as durable if the cultivar has been grown over a large-scale experiment and such tests are repeated at many locations for many years. It refers to long lasting resistance.

CONFIRMING DURABLE RESISTANCE

A cultivar to possess durable resistance must undergo some tests to prove its durability. The cultivar under test is grown in an environment that supports the disease development for a longer period on a larger scale, if the cultivar is able to survive without loosing its resistance then it is believed to have durable resistance. Strength wise this is the strongest method against all methods to check the durability of a cultivar. Two other types of tests are also mainly used. The first method is multilocational testing. The cultivar must show durability of resistance at diverse locations irrespective of the conditions. One major limitation of this method is that, the total area occupied by such tests for this testing cultivar used to be small compared with the area occupied by a cultivar in commercial use. The total size of the selective screen for new pathogen races thus remains relatively small in such tests, even when they are repeated many times, and the test for durable resistance is weak (Johnson, 1978). Second method is testing of resistance with many races of a pathogen from an existing collection. This is a weak test for durability, particularly of a newly introduced source of resistance. (Johnson, 1984)

MECHANISM OF DURABLE RESISTANCE

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 and Rajaram, 1992; Line and Chen, 1995).

DEGREE AND GENETICS OF DURABLE RESISTANCE

Resistance in a cultivar should be of an appropriate degree or amount to inhibit the development of epidemics. Level of resistance and the durability are not related to each other. Resistance even being durable can be incomplete in nature and vice versa also. So, merely by looking at the degree of resistance recognition of durable resistance cannot be recognized or defined. Many varieties, which display durable resistance to rust disease, are not completely resistant such kind of resistance is said to be partial resistance or slow resistance. Examples of partial resistance to some

biotypes of *P. striiformis* and higher susceptible to others are to be found in data from yellow rust nurseries (Stubbs et al., 1974; Zadoks, 1961). It is likely that the incorporation of partially resistant varieties from diverse sources, without an adequate test of durability, will often result in partial resistance, which is incomplete, from genetically diverse population of plants. Caldwell (1968) described this type of resistance to the wheat rust fungi as general or slow rusting resistance because it was manifested as slow development of disease on a cultivar compared to a specific check cultivar despite a compatible host-pathogen interaction.

It can be assumed that the concept of horizontal resistance as race-non-specific resistance is mainly of interest to plant breeders because it holds the promise of providing durable resistance.

However, not all-partial resistance of wheat to yellow rust is durable. Examples of partial resistance to some biotypes of *P. striiformis* and higher susceptible to others are to be found in data from yellow rust nurseries (Stubbs et al., 1974; Zadoks, 1961). It is evident therefore that the selection, of varieties from collections grown in nurseries or from other sources merely because they are incomepletely or partially resistant to yellow rust will not ensure the durability of that resistance. At present however, the breeders will rarely have any information about the genetical control of partial resistance in any variety, particularly from foreign sources (Johnson, 1978). It is likely that the incorporation of partially resistant varieties from diverse sources, and without an adequate test of durability, will often result in partial resistance, which is incomplete.

BREEDING FOR DURABLE RESISTANCE

Durable resistance is incomplete/ partial so its expression tends to be less stable than that of the very high levels of race-specific resistance which are often not durable as intermediate levels of resistance to rust diseases are more readily influenced by environmental factors in comparison to high levels of resistance (Johnson, 1978). Two problems associated with instability of these intermediate levels of durable resistance is that firstly severe infections are undesirable and may also be thought to indicate the emergence of increased virulence in the pathogen population. Secondly as resistance is strongly affected by the environment and its heritability is reduced by the environmental affect so chances of proper selection of correct, genetically resistant plants reduced. Both these problems would be reduced where varieties with higher levels of durable resistance are used as parents (Johnson, 1978). As durable resistance usually (not always) is governed by several genes rather than by one major gene so breeding to attain durable resistance is more difficult because several genes need to be transferred at one time, thus requiring large populations for selection, as well as multiplying the usual problem with linkage drag (undesirable genes that are tightly linked to the desired ones).

Breeding for durable resistance is too much challenging and difficult as the source genotype itself may be poorly adapted and a sufficient number of minor genes may not be present in a single source genotype. For checking the presence of minor genes in the source genotypes, reliable molecular markers are not available. One approach suggested in the literatures is to use recurrent selection schemes to accumulate several minor genes in a single genetic background. The breeders select the lines with the lower levels of disease severity and by doing that continuously over the seasons, the level of durable resistance will increase (Parlevliet and Van Ommeren, 1988). There is however one complication that if there is no durable major gene around it has to be taken into account. Selection for resistance alone will not generate important popular cultivars, unless it should carry some other traits like good quality and high yield also. The germplasm made of combination of minor genes could be used in transferring these genes to adapted local cultivars.

DURABLE RUST RESISTANCE GENES

Genetic diversity and durability are the two most important features of the resistance for the global wheat improvement. Genetic analysis to understand the genetic basis of such resistance could aid the directed transfer of resistance as well as the search for additional genes that could contribute to new durable resistance gene combinations. Deployment of such resistance will provide a long-term genetic solution to rust control in Asia and other countries. Till date three rust resistance genes *Lr34/Yr18/Pm38*, *Lr46/Yr29/Pm39* and *Sr2/Yr30* are considered as durable rust resistance genes. Also *Yr36* is now considered to carry durable resistance but it need to be deployed and has different gene structure. These genes do not provide the host plant with complete immunity against a set of leaf rust (*Puccinia triticina*) races; instead they can delay the infection process or reduce the development of symptoms caused by a wider range of leaf rust races on adult plants. Durable rust resistance genes available for breeding programme are discussed below.

Durable resistance to leaf rust is thought to be more difficult to obtain than to stem rust (Rubiales and Niks, 1995). But resistance against leaf rust has been identified that appears more durable than usual. 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

(Pasquini, pers. comm.). 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, 1992). 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 racespecific resistance genes it possesses others that contribute to quantitative adult plant resistance that does not appear to be race-specific (Johnson, 1984).

The ability of *Lr34* to interact with other minor/major genes provides effective or durable leaf rust resistance to cultivars. The resistance phenotype displayed by this gene includes longer latent period, fewer uredina and smaller uredina size (Drijepondt and Pretorius, 1989; Rubiales and Niks, 1995). Because *Lr34* and *Yr18* work mostly in adult plants and in combination with other rust resistance genes it is difficult for breeders to determine if they are present when a wheat plant displays resistance, or if resistance is caused by other resistance genes. Duo of *Lr34* and *Yr18* has long been recognized as carrying the most durable forms of resistance. Other rust resistance genes may be more effective against specific strains of rust, but their effectiveness is eventually overcome by new strains. Genes *Lr34* and *Yr18* confer slow rusting resistance to leaf and stripe rust, respectively, and are known to be pleiotropic or completely linked to each other (McIntoch, 1992; Singh, 1992). Although genes *Lr34* and *Yr18* may not provide adequate resistance under high disease pressure when present alone (Ma and Singh, 1996; Singh and Gupta, 1992; Singh and Huerta-Espino, 1997), they could contribute to achieving acceptable levels of resistance in combination with other slow rusting genes (Singh et al., 2001; Singh and Rajaram, 1992). Because *Lr34* and *Yr18* work mostly in adult plants and in combination with other rust resistance genes it is difficult for scientists to determine if they are present when a wheat plant displays resistance, or if resistance is caused by other resistance genes. Since slow rusting resistance is quantitatively, inherited, quantitative trait loci (QTL) analysis has been applied to map genes conferring it. Deoxyribonucleic acid (DNA) markers are more accurate than determining if a gene is present or not based on the response of the plant to infection.

It has long been known that the location of *Lr34* and *Yr18* is on wheat's 'D genome'. *Lr34* gene has remained durable, and no evolution of increased virulence toward *Lr34* has been observed for more than 50 years. Despite the importance of adult plant resistance genes (Panter and Jones, 2002), no such gene has been cloned. Previous studies have localized the codominant gene *Lr34* on the short arm of chromosome 7D between the two markers gwm1220 and SWM10 (Spielmeyer et al., 2008; Bossolini et al., 2006). Simon et al. (2009) showed that the *LR34* protein resembles adenosine triphosphate binding cassette transporters of the pleiotropic drug resistance subfamily raising the possibility of similar defense mechanisms in non–host resistance and durable resistance to an adapted pathogen. Simon et al. (2009) and his team isolated *Lr34* gene using a resistant wheat line, knocking out genes until they found the one that offered protection. Lagudah et al. (2006) utilized the knowledge accrued from colinearity of rice chromosome 6S and the *Lr34/Yr18* region of wheat chromosome 7DS to identify orthologous wheat expressed sequence tags (wESTs) as well as diploid D genome bacterial artificial chromosomes (BACs) in an attempt to further characterize and develop potentially useful molecular markers for the *Lr34/Yr18* gene region. Five allele-specific markers (cssfr1 and cssfr5) were developed based on a 3 bp deletion in exon 11 of the *Lr34*-gene, and one marker (cssfr6) was derived from a single nucleotide polymorphism in exon 12. Validation of reference genotypes, well characterized for the presence or absence of the *Lr34/Yr18/Pm38* resistance locus, demonstrated perfect diagnostic values for the newly developed markers (Lagudah et al., 2009).

Marker assisted targeted transfer of durable resistance gene *Lr34* into widely grown Indian genotypes and the subsequent deployment of their derivatives will be an attractive strategy for achieving long-term objective of rust control. Leaf rust resistance gene *Lr46* a slow rusting gene, is located in chromosome 1BL and is linked (or pleiotropic) to gene *Yr29* that confers moderate levels of adult-plant resistance to stripe rust (William et al., 2003). Gene *Lr46/ Yr29* also affect all components of slow rusting and are associated with durable resistance (Singh, 1992; Singh et al., 1998; Suenaga et al., 2003). The effect of *Lr46* resembles that of *Lr34* and other wheats reported with partial resistance. At macroscopic level, *Lr46* produced a longer latency period than observed on the susceptible recurrent parent Microscopically, *Lr46* increased the percentage of early aborted infection units not associated with host cell necrosis and decreased the colony size. The effect of *Lr46* is comparable to that of *Lr34* in adult plant stage, but in seedling stage its effect is weaker than that of *Lr34*. Considering the increasing worldwide use of *Lr46* and other adult-plant genes for durable rust resistance, it is essential to obtain a greater understanding of their mechanisms of resistance. The Powdery Mildew resistance associated with the *Lr34/Yr18* and *Lr46/Yr29* loci has recently been named *Pm38* and *Pm39*, respectively (Lillemo et al., 2008). The most successful wheat stem rust APR gene, *Sr2*, has provided partial resistance to all stem rust races since its deployment in

the 1920s (McFadden, 1930, McIntosh et al., 1995). *Sr2* provides a degree of resistance to race Ug99. When present alone, the *Sr2* gene confers slow rusting that is not adequate under heavy disease pressure, but does provide adequate resistance in combination with other minor genes. Unfortunately, not much is known about the other genes in the *Sr2* complex and their interactions. Knott (1988) has shown that adequate levels of multigenic resistance to stem rust can be achieved by accumulating approximately five minor genes.

Resistance conferred by *Sr2* has been particularly durable, and is characterized by slow rust development and low terminal rust responses on field-grown, adult plants (McIntosh et al., 1995). Results show that durable stem rust resistance gene *Sr2* is closely linked to minor gene *Yr30* conferring yellow rust resistance (Singh et al., 2000). It is recessively inherited, making it difficult to detect in segregating populations, especially in the presence of other rust-resistance genes (Brown 1993; McIntosh et al., 1995). The use of durable resistance to rusts has been a mainspring of sustainable wheat production worldwide. Molecular markers can be used to tag rust resistance genes and further to be used in improvement the efficiency of selection in plant breeding by marker-assisted selection (MAS). MAS is a powerful alternative to facilitate new gene deployment and gene pyramiding for quick release of rust resistant cultivars. The selection of genotypes with combinations of non-racespecific resistance genes defining durable resistance over year as well as race specific genes at seedling stage is a task of prime importance for molecular marker assisted selection. A promising alternative is genomic selection (GS), which utilizes genome-wide marker coverage to predict genotypic values for quantitative traits (Rutkoski et al., 2011). In turn, GS can reduce the selection cycle length of a breeding program for traits like APR that could take several seasons to generate reliable phenotypes

REFERENCES

Arnolds MH, Brown SLJ (1968). Variation in the host parasite relationship of a crop disease. J. Agric. Sci., 71: 19-36.

Bossolini E, Krattinger SG, Keller B (2006). Development of simple sequence repeat markers speciific for the *Lr34 r*esistance region of wheat using sequence information from rice and Aegilops tauschii, Theor. Appl. Genet., 113: 1049-1062.

Brown GN (1993). A seedling marker for gene *Sr2* in wheat. In: Imrie BC, Hacker JB (eds) Proceedings of the tenth Australian plant breeding conference, vol. 2. Conference Organizing Committee, Gold Coast, pp. 139-140

Caldwell RM (1968). Breeding for general and/or specific plant disease resistance. In: Proc. 3rd Int. Wheat Genetics Symp. (Ed. K.W . Finlay and K.W . Shephard). (Aust. Acad. Sci., Canberra, Australia) pp. 263-272.

Drijepondt SC, Pretorius ZA (1989). Greenhouse evaluation of adult-plant resistance conferred by the gene *Lr34* to leaf rust of wheat. Plant Dis., 73: 669-671.

Ellingboe A (1982). Genetical aspects of active defence. In active defence Mechanism in Plants, ed. R.K.S. Wood, New York : Plenum pp. 179-92.

FAO (2006). http:// faostat.fao.org

Flor HH (1946). Genetics of pathogenicity in Melampsora lini. J. Agric. Res., 73: 335-357.

Flor HH (1971). Current status of the gene-for-gene concept, Ann. Rev. Phytopathol., 9: 275-296.

Johnson R (1978). Practical breeding for durable resistance to rust disease in self pollinating cereals. Euphytica, 27: 529-40.

Johnson R (1984). A critical analysis of durable resistance. Ann. Rev. Phytopathol., 22: 309-330.

Johnson R, Law CN (1975). Genetic control of durable resistance to yellow rust (*Puccinia strriformis*) in the wheat cultivar Hybride Bersee. Ann. Appl. Biol., 81: 385-391.

Johnson R (1992). Reflections of a plant pathologist on breeding for disease resistance, with emphasis on yellow rust and eyespot of wheat. Plant Pathol*.,* 41: 239-254.

Knott DR (1989). The Origin and Evolution of Wheat. In: The Wheat Rusts - Breeding for Resistance. Springer-Verlag, Berlin, Monographs Theor. Appl. Genet., pp. 1-6.

Knott DR (1988). Using polygenic resistance to breed for stem rust resistance in wheat. *In* N.W . Simmonds & S. Rajaram, eds. Breeding strategies for resistance to the rusts of wheat, Mexico, DF, CIMMYT p. 39-47.

Lagudah ES, Krattinger SG, Herrera-Foessel S, Singh RP, Huerta-Espinosa J, Spielmeyer W, Brown-Guedira G, Selter LL, Keller B (2009). Gene-specific markers for the wheat gene *Lr34/Yr/18/Pm38* which confer resistance to multiple fungal pathogens. Theor. Appl. Genet., 119: 889-898.

Lagudah ES, McFadden H, Singh RP, Huerta-Espino J, Bariana HS (2006). Molecular genetic characterization of the *Lr34 /Yr18* slow rusting resistance gene region in wheat. Theor. Appl. Genet., 114: 21-30.

Lillemo M, Asalf B, Singh RP (2008). The adult plant rust resistance loci *Lr34/Yr18* and *Lr46/Yr29* are important determinants of partial resistance to powdery mildew in bread wheat line Saar. Theor. Appl. Genet., 8: 1155-1166.

Line RF, Chen X (1995). Successes in breeding for and managing durable resistance to wheat rusts. Plant Dis., 79: 1254-1255.

Ma H, Singh RP (1996). Contribution of adult plant resistance gene *Yr18* in protecting wheat from yellow rust. Plant Dis., 80: 66-69.

Martens JW, Dyck PL (1988). Genetics of resistance to rust in cereals from a Canadian perspective. Can. J. Plant Pathol., 11: 78-85.

McFadden ES (1930). A successful transfer of emmer characteristics to vulgare wheat. J. Am. Soc. Agron., 22: 1020-1034.

McIntosh RA, Wellings CR, Park RF (1995). In: Wheats Rusts, An Atlas of Resistance Genes. Alexa C.G., (ed.). CSIRO Publishers. Australia. pp. 29-82.

McIntosh RA (1992). Close genetic linkage of genes conferring adult plant resistance to leaf rust and stripe rust in wheat Plant Pathol., 41: 523-27.

McIntosh RA, Luig NH, Milne DL, Cusick J (1983). Vulnerability of triticales to wheat stem rust. Can. J. Plant Pathol., 5: 61-69.

Panter SN, Jones DA (2002). Age-related resistance to plant pathogens. Adv. Bot. Res., 38: 251-280.

Parlevliet JE, Van Ommeren A (1988). Accumulation of partial resistance in barley to barley leaf rust and powdery mildew through recurrent selection against suscepptibilty. Euphytica, 37: 261-274.

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

Parlevliet JE (1997). Durable resistance. In: H.Hartleb, R.Heitefuss and H.H. Hoppe (Eds.), Resistance of crop Plants against Fungi, Gustav Fisher, Jena Germany, pp. 238-253.

Parlevliet JE, Zadoks JC (1977). The integrated concept of disease resistance; a new view including horizontal and vertical resistance in plants. Euphytica, 26: 5-21.

Pasquini M, Casulli F (1993). Resistenza “durevole” a Puccinia recondita f.sp. tritici ed Erysiphe graminis f.sp. tritici in frumenti duri italiani. Phytopathol. Medit., 32: 135-142.

Robinson RA (1973). Horizontal resistance. Rev. Plant. Pathol., 52: 483-501.

Robinson RA (1976). Plant Pathosystems. Berlin: Springer-Verlag. pp.

184.
Robinson RA (1969). Disease resistance terminology. Rev. Appl. Mycol., 48: 593-606.

Robinson RA, Chiarappa L (1975). The proposed FAO international programme on horizontal resistance to crop pests and diseases. FAO Pl.

Roelfs AP, Huerta-Espino J, Marshall D (1992). Barley stripe rust in Texas. Plant Dis., 76: 538.

Roelfs AP (1988). Resistance to leaf and stem rust in wheat. Pages 10-22 in: Breeding Strategies for Resistance to the Rusts of Wheat. NW Simmonds and S Rajaram, eds. CIMMYT, Mexico D.F Protection Bull., 23: 125-129.

Rubiales D, Niks RE (1995). Characterization of *Lr34* a major gene conferring non-hypersensitive resistance to wheat leaf rust. Plant Dis., 79: 1208-1212.

Rutkoski J, Heffner E, Sorrells M (2011). Genomic selection for durable stem rust resistance in wheat. Euphytica, 179: 161-173,13.

Saari EE, Prescott JM (1985). World distribution in relation to economic losses. *In*: *The Cereal Rusts,* vol. 1. A. P. Roelfs and WR Bushnell, eds. Academic Press, Orlando, USA, pp. 259-298.

Scott PR, Hollins TW (1977). Interactions between cultivars of wheat and isolates of *Cercosporella herpotrichoides*. Trans. Br. Mycol. Soc., 69: 397-403.

Scott PR, Johnson R, Wolfe, MS, Lower HJB, Bennett FGA (1980). Host specificity in cereal parasites in relation to their control. Appl. Biol., 5: 349-393

Simon GK, Evans SL, Wolfgang S, Ravi PS, Julio H-E, Helen M, Eligio B, Liselotte LS, Beat K (2009). A Putative ABC Transporter Confers Durable Resistance to Multiple Fungal Pathogens in Wheat. Science, 323: 1360-1363.

Singh RP, Gupta AK (1992). Expression of wheat leaf rust resistance gene Lr34 in seedlings and adult plants. Plant Dis., 76: 489-491.

Singh RP, Huerta-Espino J (1997). Effect of leaf rust resistance gene *Lr34* on grain yield and agronomic traits of spring wheat. Crop Sci., 37: 390-395.

Singh RP, Rajaram S (1992). Genetics of adult plant resistance to leaf rust in 'Frontana' and three CIMMYT wheats. Genome, 35: 24-31

Singh RP (1992). Association between gene Lr34 for leaf rust resistance and leaf tip necrosis in wheat. Crop Sci., 32: 874-878.

Singh RP, Huerta-Espino J, William M (2001). Slow rusting genes based resistance to leaf and yellow rusts in wheat. In Eastwood R, Hollamby G, Rathjen T, and Gororo N, September 2001 (eds). Wheat Breeding Society of Australia: Proceedings of the Assembly 10, 16-21, Mildura, Australia. pp. 103-108.

Singh RP, Huerta-Espino J, Rajaram S (2000). Achieving near-immunity to leaf and stripe rusts in wheat by combining slow rusting resistance genes. Acta Phytopathol. Entomol. Hung., 35: 133-139.

Singh RP, Mujeeb-Kazi A, Huerta-Espino J (1998). *Lr46:* a gene conferring slow rusting resistance to leaf rust in wheat. Phylopathology, 88: 890-894.

Spielmeyer W, McIntosh RA, Kolmer J, Lagudah ES (2005). Powdery mildew resistance and *Lr34/Yr18* genes for durable resistance to leaf and stripe rust co-segregate at a locus on the short arm of chromosome 7D of wheat. Theor. Appl. Genet., 111: 731-735.

Spielmeyer W, Singh RP, McFadden H, Wellings CR, Huerta-Espino J, Kong X, Appels R, Lagudah ES (2008). Fine scale genetic and physical mapping using interstitial deletion mutants of *Lr34/Yr18*: Adisease resistance locus effective against multiple pathogens in wheat. Theor. Appl. Genet., 116: 481-490

Stakman EC (1946). Plant pathologist's merry-go-round. J. Hered*.,* 37: 259-265.

Stubbs RW, Fuchs E, Vecht H, Basset EJW (1974). The international survey of factors of virulence of *Puccinia striiformis* Westend. In 1969, 1970 and 1971. Sticht. Nederlands Graancentrum Tech. Bericht, p. 88.

Suenaga K, Singh RP, Huerta-Espino J, William HM (2003). Microsatellite markers for genes *Lr34/Yr18* and other quantitative trait loci for leaf rust and stripe rust resistance in bread wheat. Phytopathology, 93: 881-890.

Thoday JM (1961). Location of polygenes. Nature, Lond. 191: 368-370.

Van Dijk KV, Parlevliet JE, Kema GHJ, Zeven AC, Stubbs RW (1988). Characterization of the durable resistance to yellow rust in old winter wheat cultivars in the Netherlands. Euphytica*,* 38: 149-158.

Van Ginkel M, Rajaram S (1992). Breeding for durable resistance to diseases in wheat: An international perspective. In: Durability of Disease Resistance*,* Jacobs, Th. and Parlevliet, J.E. (eds).Kluwer Academic Publ., Dordretch, pp. 259-272.

Vander Graaff NA (1981). Selection of arabica coffee types resistant to coffee berry disease in Ethiopia. Meded. Land-bouwhogesch. Wageningen, 191: 1-110.

Vanderplank JE (1963). Plant Diseases: Epidemics and Control. New York: Academic, p. 349.

Vanderplank JE (1968). Disease Resistance in Plant Infection. New York: Academic, p. 206.

William HM, Singh RP, Huerta-Espino J, Ortiz-Islas S, Hoisington D (2003). Molecular marker mapping of leaf rust resistance gene *Lr46* and its association with stripe rust resistance gene *Yr29* in wheat. Phytopathology, 93: 153-159.

Xavier Ribeiro Ribeiro do vale FX, Parlevliet JE, Zambolim L (2001). Concepts in plant disease resistance. Fitopatologia Brasileira, 26: 577-589.

Zadoks JC (1961). Yellow rust of wheat: studies in epidemiology and physiological specialization. Tijdschr. Plant Ziekt, 67: 69-256.