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Molecular Breeding Approaches For Improving Rust Resistance In Wheat: Marker Assisted Gene Pyramiding

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Abstract

Among the diseases which affect wheat, rusts caused by fungal pathogens are prominent. The three important rusts of wheat, black or stem rust caused by *Pucciniagraminis*Pers.f.sp*tritici* Eriks. & Henn,brown or leaf rust caused by *Pucciniatriticina*Eriks. (Syn: *Pucciniarecondita*) and yellow or stripe rust incited by *Pucciniastriiformis*Westend. are known to cause significant losseswhen they appear in epidemic proportion.These are most destructive diseases worldwide and have the ability to destroy the entire wheat crop. They have the ability to form new races and can attack previously resistant cultivars, and have the capacity to move long distances with potential to develop rapidly under optimal environmental conditions resulting in serious yield losses. The presence of races capable of overcoming different resistance genes has been demonstrated for all three rust fungi.Growing resistant cultivars is the most economic, effective and environment-friendly approach to control the disease.Single major gene resistance in a cultivar may become ineffective soon after it is deployed because of continuous evolution of new pathotypesand slow hypersensitivity.Pyramiding two or more genes in one cultivar can enhance durability and the level of rust resistance.Molecular markers have made it possible to identify and pyramid valuable genes of agronomic importance in resistance breeding.

Key Words: Wheat; rust; marker assisted selection

Introduction

Wheat (Triticumaestivum L. 2n = 6x = 42, AABBDD genomes), the most important food crop of the world, is grown on about 225 m ha worldwide. It suffers from several diseases, which reduce its yield and quality. The major diseases of wheat are rusts, alternaria leaf blight, loose smut, Karnal bunt and powdery mildew. Stem, leaf and stripe rust diseases of wheat caused by *Pucciniagraminis* f. sp. *tritici (Pgt), P. triticina (Pt)* and *P.*

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striiformis f. sp. tritici (Pst) have caused significant yield losses during epidemics. Theyaffect the productivity and quality of wheat throughout the world and global food security (Brown *et al*. 2002, Fisher *et al.*2012,Kolmer*et al.* 2005). To prevent wheat rust epidemics, an international consortium known as the Borlaug Global Rust Initiative (http://www.globalrust.org/) was established. Stripe rust affects wheat crops in the early growth stagesleading to yield losses of more than 90 % on susceptiblecultivars when weather conditions are extremely favorableto the disease (Chen 2005; Sharma-Poudyal and Chen2011).Different yield lossesvarying from 5 to 70% could be caused by the diseasedepending on the leaf rust resistance genes (*Lr*genes)deployed and leaf rust isolates composition of the virulencein the region (Nagarajan and Joshi 1975; McIntosh *et al*. 1995).Similarly, stem rust also causes serious damage ranging from 50 - 70%. Yield losses depending on the severity of rust may reach upto 40% in susceptible cultivars (Knott 1989).Using resistant cultivarsis considered to be the most efficient and environmentfriendlyway to reduce the damage caused by rust. Although a number ofrust resistance genes have been identified in wheat(McIntosh *et al.* 2012), a major problem has been theirshort-lived effectiveness due to the fast emergence of virulentraces of the pathogen that are capable of overcomingthe resistance. Growing resistant cultivars is the most effective, economic and environmentally friendly way to control rust.

The presence of races capable of overcoming different resistance genes has been demonstrated for all three rust fungi. New races may arise through sexual recombination (not known for *P. striiformis*), mutation, or somatic hybridization followed by selection if a new race has a selective advantage. In India, inoculum for leaf rust and stem rust come from Nilgiri and Palani hills and for stripe rust from foot hills of Himalayas.Development of disease resistant varieties is one of the most economical methods of control of diseases like leaf rust. However, growing of rust resistant varieties having single gene for resistance results in rapid evolution of virulent biotypes of the pathogen, thereby making the resistance gene ineffective and the variety susceptible to rust. It is difficult to pyramid two or more disease resistance genes through conventional means, particularly where the resistance genes in question are effective against all the prevalent pathotypes. However, recent advances in molecular biology has made it possible to pyramid several genes in single line using marker assisted selection (MAS) and tagging of genes with molecular markers is prerequisite for MAS (Samsampour*et al.* 2009, Revathi*et al.* 2010, Bhawar*et al.* 2011, Dhillon*et al.* 2011). This review will briefly examine role of molecular markers in cereal rust diseases with particular emphasis on gene pyramiding approach to control rust in wheat. Other earlier notable

reviews of cereal rust fungi are also available (Chester *et al.* 1946, Roelf*et al.* 1985, Roelf*et al.* 1992, Kolmer 2013, Tomar*et al.* 2014).

Three different rusts of wheat

The rusts are caused by fungal pathogens belonging to genus *Puccinia*, family Puccinaceae, order Uredinalis and class Basidiomycetes. Rust fungi are obligate parasites and survive only on living plants. Brown and yellow rusts are particularly important in the major wheat growing areas of north-western parts of the country. Black rust appears quite late in these areas and normally may not cause substantial damage except in wheat fields sown very late. A comparison of the characteristics of the three wheat rusts is presented in Table I.Wheat stem rust, caused by Pucciniagraminis f. sp. tritici (Pgt), is a devastating disease that can cause severe yield losses. A previously uncharacterized Pgt race, designated *Ug*99 (Uganda in 1999) has overcome most of the widely used resistance genes and is threatening major wheat production areas. Recently Saintenac*et al.* (2013) demonstrated that the *Sr*35 gene from *Triticummonococcum*confers near immunity to *Ug99* and related races. This gene is absent in the A-genome diploid donor and in polyploid wheat but is effective when transferred from *T. monococcum*to polyploid wheat. Chen *et al.* 2013 have conducted numerous studies to understand molecular mechanism of different types of stripe rust resistance using a transcriptomics approach. After various comparisons they have identified five genes to be involved in race specific all stage resistance controlled by *Yr*1, *Yr*5, *Yr*7, *Yr*9 and *Yr*15.Leaf rust resistance genes are located on 20 of the 21 chromosomes of hexaploid wheat (McIntosh *et al.*1995; McCallum *et al.*2012). Dakouri*et al.* 2013, characterize the seedling and adult plant leaf rust resistance genes present in a world collection (275 wheat accessions representing 42 countries), to evaluate their effectiveness under field conditions and to correlate the results with gene-specific molecular markers. Their work supportsthe claim that *Lr34* is the single most significant and durable gene in breeding programs for leaf rust resistance.

Table 1. Comparison of three different wheat rusts.

Characteristics	Leaf rust	Stem rust	Stripe rust
Pustule location	Leaf, mainly on the upper surface	Stem, leaf sheath, leaves and ear heads but the stem is often most severely affected	Leaf, upper surface; occasionally on head and seeds
Pustule color	Bright orange	Reddish-brown in color and elongated in shape	Orange-yellow
Pustule arrangement	Small, round-oval uredial pustules	Elongated, Single and random	Pustules develop along the leaf veins as long streaks
Pustule shape and size	Small clusters or irregularly scattered	Oval shaped or elongated; small to large	Round, blister-like; small
Optimum temperature for disease development	15-25ºC	18-30ºC	14-18^{0}C
Alternate hosts	Some cultivars of barley and some species of goat grass (*Aegilops spp.*)	Minor infection on certain cultivars of barley and rye	Barley, triticale, rye and over 18 species of grasses
Yield loss	In case of severe attack of the disease, plants mature early, produce light and shriveled grains and develop poor root system	Ripening and shrinking of the grains incurring the losses as much as 90% of grain yield	Reducing the number of kernels per spike, test weight and kernel quality
Latent period	7-10 days	7-10 days	10-14 days

Impact of rusts and its control

Most of the grain yield losses incurred due to leaf and stripe rusts are attributed to infection of the flag leaf, which is thought to be responsible for greater than 70% of grain filling. The yield losses are significant, if the flag leaf is heavily infected prior to grain fill.It appears from historical records that rust epidemics have occurred from time to time in India (Nagarajan and Joshi 1975), the earliest on record dates back to 1786. In 1946-47, the rust epidemics caused a loss of nearly 2 million tonnes of wheat. Leaf and stripe rust appeared in epidemic form in North Western Region of the country which resulted into loss of 0.8 to 1.5 million tonnes of wheat in 1971-1973 (Joshi 1975). Sonalika epidemic of leaf rust caused approximately one million tonnes loss in 1980 (Joshi *et al*. 1984). The losses are lesser in the areas where environment is marginally warmand not suited for disease. The disease becomes severe

only when conditions are unusually favorable such as susceptible cultivars, presence of adequate inoculum load and/ or altered cultural practices. In such situations losses upto 100% may occur (Roelfs*et al*. 1992).

Controlling rust is a complicated job because of constant change in strains (races) of the pathogens. Pathogenic activity of rust can either be curtailed bythe cultivation of rust resistant variety or by use of chemicals (fungicides). Chemical control is more effective when rust diseases are identified on susceptible varieties early in the growing seasons. However chemical control of rust pathogens is inefficient, expensive and cannot be adopted by small and marginal farmers. Hence the development of genetic resistance to rusts in host is advocated which is economical, effective and environment-friendly approach to prevent the losses caused by rust epidemics (Kolmer 1996). Wheat production largely depends on the resistance carried by the diverse and well characterized genes. Qualitative resistance, which confers major gene-specific resistance against some pathogen races, is easiest to incorporate into breeding programe and is usually considered a gene-for-gene type of resistance. Several rust resistance genes have been transferred in the genetic background of popular Indian cultivars using conventional backcross breeding (Sivasamy*et al.* 2009). To date, nearly 57 stem rust resistance genes, 71 leaf rust resistance genes and about 53 stripe rust resistance genes have been identified in wheat germplasms and related species that confer resistance to rusts in wheat (McIntosh *et al.* 2012).

Rust resistance genes of alien origin

Although many rust resistance genes have been identified in present day Indian wheat cultivars but most of them are ineffective to prevailing virulent races of rusts. Several genes of alien origin still exhibit resistance to leaf and stem rusts,a few of them have been reported to have yield penalty. However, not all genes are disadvantageous rather few of them enhance yield potential, e.g., 1BL/1RS (*Lr*26/*Sr*31/*Yr*9/*Pm*8) translocation from *Secalecereale*. The nexus between alien gene and yield can be broken (Penrose *et al*. 1998) if selection strategy is carefully adopted in segregating populations. Several alien genes have been transferred into bread wheat from its wild relatives like *Agropyron*, *Aegilops* and some *Triticum*spp. (McIntosh *et al*. 1995, Sivasamy*et al.* 2009). Some of the wild species have one genome in common with wheat genome, which facilitate the search for new resistance genes and help in mapping. The genes from secondary and tertiary gene pool require specific techniques for transferring them into common wheat.

The progenitors of wheat carry different genomes (*Triticum*sp.,viz.,*Aegilopsspeltoides*carring S genome, similar to that of B,*Triticumboeoticum* having A genome and *Aegilopssquarrosa*possesingD genome) are still a good sources of resistance to diseases. A frequent way to

transfer the resistance genes into wheat is to use wheat lines with translocation of a chromosome fragment carrying the gene from wild species. This was done in the case of genes, *Lr19*, *Lr24* and *Lr29* derived from *Agropyronelongatum*(Procunier*et al.* 1995, Schachermayr*et al*. 1995, Prins*et al.* 1996).A significant number of leaf rust resistance genes originate from *Triticumaestivum*, but some resistance genes were originally introgressed into common wheat from wild species (Table 2).Most of the genes conditioning resistance to stripe rust have originated from wheat itself and very few gene(s) have been transferred from wild species (Tomar and Menon 2006).

Table 2. Leaf and stripe rust resistance genes and their origin

Gene	Location	Origin
Lr1	5DL	*T. aestivum*
Lr9	6BL	*Ae. umbellulata*
Lr13	2BS	*T. aestivum*
Lr14	7BL	*Ae. ventrocosa*
Lr16	2BS	*Ae. ventrocosa*
Lr19	7DL	*Agropyronelongatum*
Lr20	7AL	*T. aestivum*
Lr 21	1DL	*Ae. tauschii*
Lr22a	2DS	*Triticum tauschii*
Lr 23	2BS	*T. aestivum*
Lr24	3DL	*Th. elongatum*
Lr25	4BS	*Secale cereale*
Lr26	1BL	*Secale cereale*
Lr27	3BS	*T. aestivum*
Lr28	4AL	*Ae. speltoides*
Lr29	7DS	*Th. elongatum*
Lr31	4BS	*T. aestivum*
Lr34	7DS	*Thatcher*
Lr35	2B	*Ae. speltoides*
Lr37	2AS	*Ae. ventricosa*
Lr38	6DL	*Th. intermedium*
Lr39	2DS	*Ae. tauschii*
Lr47	7AS	*Ae. speltoides*
Lr50	2BL	*T. timopheevii*
Lr51	1BL	*Ae. speltoides*
Lr52	5BS	*Thatcher*
Lr56	6A	*Ae. sharonensis*
Lr58	2BL	*Ae. triuncialis*
Lr62	6AL	*Aegilopsneglecta*
Lr68	7BL	*Thatcher*

Gene	Location	Origin
Yr5	2BL	*T. spelta album*
Yr7	2BL	*T. aestivum*
Yr9	1BL-1RS	*Secale cereale*
Yr10	1BS	*Triticumspelta*
Yr15	1BS	*T. dicoccoides*
Yr16	2D	*T. aestivum*
Yr17	2AS	*Ae. Ventricosa*
Yr18	7DS	*T. aestivum*
Yr24	1BS	*T. dicoccoides*
Yr26	1BS	*Haynaldiavillosa*
Yr27	2BS	*T. aestivum*
Yr28	4DS	*Ae. Tauschii*
Yr29	1BL	*T. aestivum*
Yr30	3BS	*T. aestivum*
Yr31	2BS	*T.aestivum*
Yr32	2AL	*T. aestivum*
Yr40	5DS	*Ae. Geniculata*
Yr42	6AL	*Ae. Neglecta*
Yr43, Yr44	2BL	*T. aestivum*
Yr53	2BL	*T. aestivum*

Early history of rust resistance breeding

First crosses of wheat based on Mendel's genetic principles, aiming to transfer disease resistance, were carried out by Biffen (1905). He found monogenic inheritance of yellow rust resistance in wheat. Such inheritance has been later confirmed in other crops and pathogens. The discovery of physiologic races in cereal rusts (Stakman 1914) enabled exact genetic analyses of resistance. Studies on sources of resistance, particularly of wild relatives of crops, were performed by Vavilov (1919, 1935) who also described geographic centers of origin of cultivated plants and resistance sources. Besides studies of inheritance of resistance, the genetics of virulence was also studied, which was often found monogenic (Flor 1942). From his results on genetics of resistance in flax and virulence in flax rust,Flor (1956) developed the gene for gene hypothesis. Person (1959) demonstrated practical applications of this hypothesis, e.g. for the postulation of resistance genes using pathogen races with known virulence(s). Polygenic resistance became more popular when Vanderplank (1963) published his analysis and conclusions on vertical and horizontal resistance. Durability of disease resistance as defined by Johnson (1981) attracted attention particularly in the last decade when several conferences were devoted to this aspect of resistance. Recent developmentsin molecular biology haveopened many new prospects for resistance breeding.

The strategy of resistance breeding

Johnson (1981) defined durable resistance as "resistance that remains effective in a cultivar that is widely grown for a long period of time in an environment favorable to the disease". Major gene resistance against biotrophic pathogens such as rust and mildew is generally highly unstable and non-durable.Usually the mechanism of 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; McIntosh 1992; Chen and Line 1995). Among the strategies that have been proposed to increase the durability of "non-durable" resistance is the diversification of 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 combination or resistance gene pyramiding) is considered to be a potential approach. McIntosh (1992b) recommended the concept of anticipatory breeding for resistance to control wheat rusts, which means, if the pathogen evolves, the breeders should already have well advanced resistant lines for replacement of varieties that became susceptible. Some of the important strategies proposed for resistance breeding are use of multiline varieties,Varietal mixtures/mosaics,

Varietal diversification through gene deploymentand Oligogenic combinations or Gene pyramiding.

Pyramiding involves the accumulation of several resistance genes against the same pathogen into a single line or cultivar. Each of the resistance gene is either effective (pathogen avirulent) or defeated (pathogen virulent) (Melchinger 1990). Knott, (1989) described gene pyramiding as an important resistance breeding strategy that was applied worldwide for the control of the rust diseases of wheat. However, resistance that is based on a single gene can easily be overcome by a change in virulence at a single locus in the pathogen (Johanson 1984). With a limited number of resistance gene in hexaploid wheatand with the difficulties and associated problems of transferring genes from related species through traditional breeding, combinations of genes (referred to as gene pyramiding) are believed to provide durable resistance to both virulent and avirulent races of a pathogen than single genes (Nelson 1978). In other words gene pyramiding is a breeding strategy whereby host resistance genes are combined together with the objective of prolonging their usefulness in crops(Kloppers and Pretorius1997).Multigenic combinations have been produced with the help of available genetic information, reaction pattern against individual pathotypes in seedling stage, genetic linkages and cytological markers (Tomar*et al*.2006, 2007). However, the selection of genotypes carrying two or more genes using traditional host-parasite interaction is very time consuming and often not possible due to lack of isolates with specific virulence and difficulty of identifying one resistance gene in the presence of another gene. Since the identity of the two resistance genes is difficult through conventional genetic analysis, the pyramiding of different resistance genes can be supported by the use of molecular markers.

DNA markers as new tools for resistance breeding

The use of DNA markers in plant and animal breeding has opened a new realm in agriculture called ‘molecular breeding’ (Rafalski and Tingey 1993). The main advantage of using molecular markers for the introgression of resistance genes into cultivars is a gain in time (Tanksley*et al*. 1989; Melchinger 1990). Resistance gene introgression is normally conducted by crossing a donor line with an agronomically superior cultivarfollowed by repeated testing, selfing and backcrossing until the donor genome is diluted out of the cultivar, retaining only the desired resistance gene. The use of DNA markers could speed up this process by three plant generations, by allowing selection of the resistant offspring that contains the lowest amount of the donor genome in every generation (Tanksley*et al.* 1989). DNA markers for many rust resistance genes have been identified to date. Availability of molecular markers can facilitate pyramiding of durable resistance genes into an elite cultivar background in less time and in a cost effective manner (Tanksley*et al*. 1989). A compilation of several markers for useful genes including

rust resistance genes for wheat is available at the MAS wheat web site (http://maswheat.ucdavis.edu). RFLP and PCR based markers such as RAPD, SSR and ISSR markers have been used to tag a number of resistance genes in wheat (Gupta *et al.* 1999).

The usefulness of a molecular marker in MAS depends on the following criteria. The technique used to generate the markers must be reliable, relatively simple to perform, and capable of processing a large number of samples per unit time. In addition, selection for the marker must signify selection for the gene if progeny testing, to identify individuals with the desired genes, is to be reduced. For plant breeders, the most useful application of MAS is to use DNA-based markers for basically three purposes:

1. Tracing favorable allele(s) either dominant or recessive, across the generations in order to accumulate favorable alleles,
2. Identifying the most suitable individuals among segregating progenies, based on the allelic composition of a part or of the entire genome and
3. Breaking the possible linkage of favorable alleles with undesirable loci (Francia*et al*., 2005).

Marker-based breeding may revolutionize the process of cultivar developmentby eliminating or reducing the need for field trials and making itpossible to select individuals or lines with crossovers very near to a gene ofinterest, potentially removing “linkage drag” that frequently comes from the donor parent.Identification of molecular markers for resistance genes can efficiently facilitatepyramiding major genes into a valuable background in less time and make it more costeffective. Recently there have been advances in the mapping and development of molecular markers of several rust resistance genes (Prins*et al.* 2001; Gupta *et al.* 2006; Sun *et al.* 2009; Cheng *et al* 2010,Foessel*et al.* 2012; Spielmeyer*et al.* 2013;Xu*et al.*2013;Heile*et al.* 2013).

MAS and gene pyramiding

Molecular markers are used for two purposes in resistance breeding: (a) to monitor the incorporation of designated resistance genes or QTLs into elite wheat genotypes, (b) to identify resistance genes in varieties and lines where the genetic background is unknown (Vida *et al*. 2009). Gene pyramiding makes use of molecular markers linked to rust resistance genes for both these purposes. In addition, MAS can provide specific advantages in resistance breeding allowing faster response to a break down in resistance, rapid introgression of multiple genes from diverse germplasms, pyramiding and selection or rare recombinants with tightly linked resistant genes (Michelmore2003). Pyramiding of rust resistance gene through traditional phenotypic-based technology is difficult when different resistance genes produced similar infection types.The application of molecular marker technology can tackle such complex problems. Marker assisted pyramiding approach has been earlier used in wheat and other crops (Samsampour*et al.* 2009;Revathi*et al.*

2010;Bhawar*et al.* 2010;Charpe*et al.* 2012;Suh*et al.* 2013, Tiwari*et al*. 2014).

Better understanding of virulence variations and mechanisms of rust fungus wheat interactions is necessary for the development and deployment of more effective and durable resistant cultivars (Webb *et al.* 2006). Nevertheless, recent technological advances have enabled the use of genomic approaches to study virulence variation, development and evolution in rust and other obligate biotrophic fungi (Duplessis*et al.* 2011;Spanu*et al.* 2010). Among the rust fungi that have been sequenced, the genome sequences of Pgt and Melampsoralarici-populina (Mlp) were generated by Sanger sequencing (Duplessis*et al.* 2011). In 2011, a rough draft sequence of a US isolate of *Pst* was generated with short reads from next-generation sequencing (NGS) (Cantu *et al.* 2011). Recently a significantly improved draft genome (~110 Mb) of a Chinese Pst isolate, CY32, using a 'fosmid-tofosmid' sequencing strategy has been generated (Zheng*et al.* 2013) that combines the use of a fosmid library and Illumina sequencing.

Conclusion and Future prospects

The rust diseases of cereals represent a continuous challenge to plant breeders and plant pathologists in developing crop cultivars with long lasting resistance. Due to the continual evolution and selection of rust races new sources of effective rust resistance genes must be added to breeding germplasms regularly to maintain rust resistance.The use of DNA markers in plantbreeding i.e., molecular breeding has accelerate the breeding process compared to conventional breeding, and to pyramid combinations of genes that could not be readily combined through other means. New varieties or lines with genes that control the desired traits can be produced by combining MAS with traditional breeding.

Continued advances in the genomics of crop plants and their related wild species mayallow greater expansion and exploitation of the gene pool available for improvement of rust resistance. Advances in whole genome sequencing of the wheat rust pathogens will lead to the development of highly diagnostic SNP markers that could be used in real time to detect new races and to trace their spread and worldwide migration. The sequencing of additional rust resistance genes and the future characterization of avirulence/virulence genes in the wheat rusts will shed further understanding on themolecular and biochemical pathways that lead to resistance response in cereal plants.

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