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Molecular Breeding for Septoria tritici Blotch Resistance in Wheat

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Septoria tritici blotch (STB) caused by the fungus *Mycosphaerella graminicola,* is one of the most important foliar diseases of wheat (*T. aestivum* spp., *aestivum* L.). Various practices such as crop rotation, application of fungicides, and deployment of genetic resistance have been utilised to control this disease and subsequently reduce yield losses. During the last 20 years, significant progress has been made in understanding host–pathogen interaction, inheritance of STB resistance, localisation of loci controlling STB resistance and identification of molecular markers associated with STB resistance in common wheat. We review the progress made on various aspects of molecular breeding for STB resistance especially on mapping and validation of qualitative and quantitative trait loci in common wheat.

Keywords: *Septoria tritici,* resistance, genetics, molecular markers, marker-assisted selection, wheat

Introduction

Common wheat (2n = 6x = 42, AABBDD genomes) is the major source of calories intake all over the world. Its production is threatened by various abiotic and biotic stresses especially by constantly changing populations of pathogen races. In order to stabilise the food production and to meet the food demand for growing population across globe, management of diseases is one of the key R&D priorities of various organisations. Septoria tritici blotch (STB), also known as speckled leaf blotch or septoria leaf blotch, caused by *Mycosphaerella graminicola* (Fuckel) J. Schröter. in Cohn (anamorph: *Septoria tritici* Roberge ex Desmaz), is one of the most ubiquitous and important necrotic leaf diseases of wheat worldwide, including Europe, North America, South America and Australia. This disease is characterised by irregular blotches that have thin yellow margins on the leaves, generally tan to brown. High humidity and moderate temperatures during vegetative growth promote disease development. Under epiphytotic conditions, STB can cause serious yield losses up to 60% in some wheat crops. Brennan and Murray (1998) estimated the potential loss caused by STB in Australia to be $152 m per year.

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Biology of the pathogen and epidemiology of the STB

M. graminicola belongs to phylum Ascomyceta and infects common wheat, durum (*Triticum durum* Desf) and spelt wheat (*Triticum aestivum* L. subsp. *spelta* (L.) Thell) (Gharbi et al. 2000; Goodwin 2007; Simon et al. 2010). It reproduces both asexually and sexually on host species and during a single crop growing season, it can complete several disease cycles. The fungus survives on crop residues mainly on leaf and stubble, and therefore survives from one season to the next. Sexual reproduction plays a significant role in genetic diversity of *M. graminicola* populations, resulting in high biological fitness (Chen and McDonald 1996). Molecular analysis of populations of *M. graminicola* has shown high gene flow within and between populations (McDonald and Martinez 1990a, 1990b; Schnieder et al. 2001).

During the last 20 years, tremendous progress has been made in understanding STB resistance due to the understanding of a variety of factors, including successful culture of the pathogen *in vitro*, long latent period of the disease, specific environmental conditions required for infection, variability in symptom expression; which often complicate the scoring of inoculated plants, and the knowledge of STB resistance gene locations on the wheat chromosomes (Goodwin 2007; Orton et al. 2011).

STB Infections occur during all stages of plant development; however, infection on the flag leaves can cause the most severe losses by reducing grain test weight (Paveley 1999). Under high humidity conditions, wind borne sexual spores (ascospores) germinate on leaf and penetrate through stomata (Kema et al. 1996). Fungal growth occurs in the leaf apoplast without invading plant cells or forming haustoria (Cohen and Eyal 1993). Once the fungus colonizes, it causes chlorosis and necrosis resulting in blotches on the leaf margins and alongside of leaf-veins. In addition to the necrotic tissue, this disease results in early leaf senescence and therefore reduces photosynthetic capacity (Palmer and Skinner 2002). Under optimum conditions, asexual pycnidiospores are formed. A combination of wind and rain provides the most favourable conditions for spread of both asexual and sexual spores for the STB disease development within and between paddocks and cause disease epidemics in wheat growing areas (Zhan et al. 1998).

Management of the STB

Various practices such as crop rotation, stubble management, seed dressing and foliar application of fungicide and deployment of genetic resistance have been employed to control this disease and subsequently reduce yield losses. The application of fungicides may be a useful tool for STB management. A range of 14-demethylase inhibitors of sterol biosynthesis and systemic fungicides belonging to the strobilurins (Qo inhibitor) have been employed for the control of *M. graminicola* (Goodwin et al. 1999; Jordan and Hutcheon 1999). However, the profitability of fungicide application needs to be considered, on case to case basis. Often, fungicide application is uneconomical for rain-fed and lower yielding environments. Moreover, pathogens tend to develop resistance to chemicals, particularly when they are constantly challenged. For example, in Europe, *M. graminicola* races have

developed resistance to strobilurin fungicides (Anonymous 2003; Chartrain et al. 2004b). Considering the capability of *M. graminicola* to overcome genetic resistance due to the high frequency of sexual reproduction in the population (Hunter et al. 1999), and increasing risk of developing resistance to fungicides and to the emerging need to develop sustainable wheat productivity, the identification of resistance gene sources representing current and historic commercial cultivars and landraces, and deployment of those sources is essential.

Evaluation of germplasm for STB resistance

Measuring levels of disease resistance for the purpose of breeding varieties, germplasm development and/or molecular genetic studies utilise similar techniques for evaluating reactions to the pathogen. Artificial inoculations are usually carried out to ensure uniform epidemics of disease which improves the estimation of resistance heritability. Various criteria such as percent necrosis bearing pycnidia and pycnidial density are used to assess disease severity, rely principally on scales or estimates of the percent of diseased leaf tissue at either seedling (intact and detached leaf) or at adult plant stages. Resistance to STB can be evaluated under glasshouse, laboratory (detached leaf assay) and field conditions. Glasshouse screening using an inoculum of laboratory – produced pycnidiospores removes some of the uncertainty inherent in field testing with its dependence upon wet seasons or spray irrigation (May and Lagudah 1992) and further reduce genotype × environmental interactions to a certain extent. The cultural conditions used to produce inoculum for artificial infections are well established. Spore suspensions of *M. graminicola* are generally raised from cultures grown on solid or liquid media (Ballantyne and Thomson 1995). For smaller experiments, the solid media is used and spores washed-off plates then diluted to the desired concentration. Spore inoculation concentrations range in the literature from $4 \times 10^6 – 1 \times 10^8$ spores per ml.

For evaluating seedling resistance, most studies report glasshouse inoculations. Growth for plants is generally maintained with conditions typically of 18°C night temperature to 22°C day temperature. Light is important to achieve good disease expression, repeated days of reduced light intensity can reduce symptoms. For uniform infection, seedlings are sprayed with a spore suspension at up to the third leaf stage and kept at 100% humidity for 48–72 h.

Sewell and Caldwell (1960) used detached leaf (excised wheat seedlings leaves) assay for the first time for testing resistance to *Septoria tritici*. Since then, this method has been used to identify sources of resistance to STB and subsequently to identify and validate molecular markers associated with this trait (Arraiano et al. 2001a, 2001b; Chartrain et al. 2004b, 2005b).

Assessment of adult plants under field conditions is important for the selection of resistant germplasm. Single pycnidia isolates or isolate mixtures are used for genetic analysis in most of studies. However, mixtures of isolates or natural infection are frequently used for germplasm evaluation in screening nurseries. Planting times are important to ensure natural conditions can be taken advantage of to promote infection. Inoculum is provided to

trials by either infected stubble or sprayed with fungal spore suspensions as in the glasshouse seedling assays (Paveley 1999; Brading et al. 2002; Chartrain et al. 2004a, 2004b).

Genetic variation and inheritance of STB resistance

Identification of resistance sources is the first step towards the development of varieties for target environments. Useful genetic variation for STB resistance exists within common wheat germplasm and has also been successfully exploited from a wide range of donor sources of resistance such as landraces, wild progenitors of wheat, synthetic wheats and durums. However, only a few genotypes have been examined in detail to investigate mode of inheritance and to locate loci controlling STB resistance (May and Lagudah 1992; Simon and Cordo 1997; Brown et al. 2001; Arraiano and Brown 2006; Ogbonnaya et al. 2008; Anderson et al. 2010; Zwart et al. 2010; Schilly et al. 2011).

May and Lagudah (1992) evaluated a large collection (376 accessions) of *T. tauchii* (syn. *Aegilops tauschii*) and synthetic hexaploid wheats, produced by hybridisation *A. tauschii* with tetraploid wheats for resistance to *M. graminicola* and found 90% of *A. tauschii* accessions resistant to STB. Genetic variation for STB resistance has been exploited in breeding programs to develop new varieties resistant to STB. As a result of targeted breeding, several varieties resistant to STB have been released for commercial cultivation in Australia and elsewhere. In Australia, two sources of resistance, WW15 and Frontana have been extensively utilised to develop varieties resistant to STB. Besides resistance to several foliar diseases (Kolmer et al. 2008), Frontana has also been extensively utilised as a donor source for introgression of alleles for aluminium resistance worldwide (Raman et al. 2005, 2008, 2010; Stodart et al. 2007), a 'must' trait for cultivation crops on acid soils. Besides, Farro Lunga, Inia-F66 and Canrock have also been used as donor sources to develop varieties for STB resistance in Australia.

Genetic inheritance studies suggest that resistance to *M. graminicola* in wheat is complex, due to both qualitative and quantitative genes (Rillo and Caldwell 1966; Rosielle 1972; Wilson 1979, 1985; Shaner and Finney 1982; Somasco et al. 1996; Arraiano et al. 2001b, 2007; Brading et al. 2002; Adhikari et al. 2003, 2004a, 2004b, 2004c; McCartney et al. 2003; Chartrain et al. 2005c; Arraiano and Brown 2006). The first gene, named *Stb1*, was identified in the common wheat cultivar Bulgaria 88 by Rillo and Caldwell (1966). Genes *Stb2* and *Stb3* were identified in Australia by Wilson (1985), and were derived originally from cv. Veranopolis and Israel 493, respectively. So far, 15 STB resistance genes have been identified (Goodwin 2007; Orton et al. 2011). In addition, several quantitative trait loci (QTLs) associated with STB resistance has been reported (http://www.shigen.nig.ac.jp/wheat/komugi/genes/symbolClassList.jsp, Eriksen et al. 2002, 2003).

Genetic studies which have aimed to identify QTL or genes associated with resistance have utilised populations either derived from single seed descent, intercross or from doubled haploidy (Arraiano et al. 2001b; Eriksen et al. 2003; Adhikari et al. 2004a, 2004b, 2004c; Simon et al. 2004; Chartrain et al. 2005a, 2005c; Raman et al. 2009b; Zwart et al. 2010). However, doubled haploid (DH) lines are not suitable to infer modes of inheritance. It is important to combine genetic analysis and inheritance studies because complex

modes of inheritance such as partially or semi-dominance and recessiveness have been described in several studies. For example, varieties such as Aurora, Besostaya 1 and Kavkaz (Danon and Eyal 1990), and Soalmouni and Coulter (McCartney et al. 2002), have partially dominant resistance while Colotana, Trakia (Danon and Eyal 1990), Seabreeze and Gala (Rosielle and Brown 1979; Wilson 1979) have recessive resistance. Three studies which have used a diallel approach to examine the inheritance of partial resistance have had similar findings (Simon and Cordo 1997; Zhang et al. 2001; Ramezanpour et al. 2010), i.e. that general combining ability is more important than specific combining ability and that large dominance effects were observed. The implications from these studies which, were mainly investigating genotypes with high levels of resistance, was phenotypic selection would be best delayed until later generations. Understanding the mode of inheritance of a qualitative and quantitative genes bridges the gap between QTL analysis and practical breeding and is essential for breeding programs to develop selection strategies.

Quantitative resistance vs. qualitative resistance

Two types of STB resistance, qualitative (controlled by race-specific major genes) and quantitative also called as partial or horizontal resistance (controlled by non-race-specific polygenes) to pathogens have been described in the literature. Race-specific resistance to *M. graminicola* seems to follow the classical gene to gene hypothesis (Flor 1942; Brading et al. 2002; McCartney et al. 2002) and is controlled by the interaction between resistance *(R)* gene in the host plant and corresponding avirulence *(Avr)* gene in the pathogen. Quantitative resistance does not follow gene – for gene hypothesis and seems to be more effective against different pathogen populations and is less likely to breakdown quickly.

The STB resistance genes have often been identified using QTL mapping techniques (Goodwin 2007). This has been necessary due to the quantitative nature of phenotypes displayed by this host–pathogen interaction. The name of a 'QTL' as a qualitative gene in the literature has occurred mainly when a large percent of genotypic variation is explained by the QTL and/or a specific interaction with one or more isolates is observed. In classical genetics, QTL refers to quantitative genes that have low heritability, small genetic and accumulative effects. Less frequently tests for allelism have been carried out (Somasco et al. 1996; Chartrain et al. 2005a, 2005c, 2009; Arraiano and Brown 2006) in order to test whether major QTL and known *Stb* genes are same. For example, Raman et al. (2009b) mapped STB resistance conditioned by single major gene designated as *XStbWW2449, XStbWW1842* and *XStbWW2451,* in the DH populations derived from Chara/WW2449, Whistler/WW1842 and Krichauff/WW2451, respectively, located on the short arm of chromosome 1B. Therefore, it has been difficult to designate such major genes as known qualitative gene (most likely *Stb11*) with high degree of certainty. Nevertheless, such studies have provided insights on locations of STB resistance loci and associated molecular markers for marker-assisted selection.

Previous research has shown that different monogenic sources for STB resistance vary in providing effective resistance over period of time (Dublin and Rajararam 1996; Eyal

1999). However, the durability of qualitative STB resistance genes and therefore their long-term value to a breeding program has often been questioned. There are documented cases of resistance breakdown or gradual erosion of resistance (Krenz et al. 2008). For example, *Stb1* maintained its effectiveness for more than 25 years, whereas cv. 'gene' became highly susceptible within 5 years after its release to changes in virulence of *M. graminicola* (Cowger et al. 2000). It is often suggested that breeders wishing to introduce qualitative genes into breeding populations using molecular marker techniques should combine them with background levels of quantitative resistance. Successful pyramiding of qualitative and quantitative resistance to several diseases against appropriate pathogen populations has been achieved in other crop plants (Brun et al. 2010). Ultimately as greater understanding of how different resistance genes interact with pathogen avirulence genes will lead to the possibility of novel pyramids which have differing modes of action against the pathogen. The sequencing of *M. graminicola* genome provides the foundation to develop new strategies to uncover how resistance genes affect the pathogen (Goodwin et al. 2011).

Molecular mapping of STB resistance loci

Various molecular marker systems based upon restriction fragment length polymorphism (RFLP), randomly amplified polymorphic DNA (RAPD), amplified fragment length polymorphism (AFLP®), simple sequence repeats (SSR), sequence related amplified polymorphisms (SRAP), expressed sequence tags (EST) and Diversity Array Technology (DArT^{TM}) have been developed in wheat and subsequently employed to locate loci associated with qualitative and quantitative trait loci including for STB resistance (Röder et al. 1998; Gupta et al. 2002; Adhikari et al. 2004c; Akbari et al. 2006). However, identification of tightly linked markers has been challenging due to the low level of polymorphism between parental lines of genetic mapping populations. Recently, single nucleotide polymorphism (SNP) platform in wheat has also been developed, and it is anticipated that such markers will be routinely used for molecular analysis and marker assisted selection in the wheat breeding programs. For example, Trebbi et al. (2011) applied complexity reduction of polymorphic sequences (CRoPS®) technology and discovered a total of 2,659 of SNP markers in tetraploid durum wheat. Next generation sequencing technologies including assays for genotyping by sequencing (Elshire et al. 2011) and relevant bioinformatics tools are being developed rapidly, and will become an integral part of genetic linkage mapping, germplasm characterisation, comparative genomics, allele mining and developing trait-marker associations with various mapping strategies in the recent future. The International Wheat Genome Sequencing Consortium is currently sequencing the whole genome to enhance knowledge of the structure and function of the wheat genome, and will develop resources and technologies that will enable high density gene mapping. This will allow to develop robust and tightly linked markers for marker assisted selection and to facilitate map based cloning of resistance genes.

The majority of molecular mapping studies have described qualitative resistance with specific interactions to single pycnidium isolates (Table 1). However, only a few resis-

tance genes such as the *Stb6* (Arraiano et al. 2001b; Brading et al. 2002; Eriksen et al. 2003; Chartrain et al. 2005a, 2005c; Zwart et al. 2010), has been studied comprehensively. Many of these mapping studies utilised relatively small populations. The development of high density map utilising larger populations comprising several hundred to thousands lines will allow us to map STB gene more precisely. Both linkage and QTL analysis has been extensively used to map STB resistance loci in common wheat (Table 1). Raman et al. (2009b) utilised bulked segregant analysis technique (Michelmore et al. 1998) to tag loci for STB resistance in the Australian DH population from Krichauff/WW2451. Resistant and susceptible 'bulks' were formed by pooling DNA from each of the resistant and susceptible lines and allele frequencies were compared. This strategy enabled to map a major gene for STB resistance. This approach is particularly useful when only a limited number of traits are to be mapped and resources (money and time) required for extensive genotyping are limited.

Table 1. Chromosomal locations and molecular markers that are linked with resistance to *M. graminicola* in wheat

Gene Symbol	Donor	Location of locus	Marker interval	Reference
Stb1	Bulgaria	5BL	*Xbarc74/AGC/MCTA-1*	Adhikari et al. (2004c)
Stb2	Veranopolis	3BS	*Xgwm533.1/Xgwm493*	Adhikari et al. (2004a)
Stb3	Iseael 493	7AS	*Xwmc83*	Adhikari et al. (2004a) Goodwin et al. (2011)
Stb4	Tadorna	7DS	*E-ACTG/M-CAAA5/Xgwm111*	Adhikari et al. (2004a)
Stb5	Synthetic 6X	7DS	*Xgwm44*	Arraiano et al. (2001b)
Stb6	Flame Hereward Kavkaz-K4500	3AS	*Xgwm369*	Brading et al. (2002), Chartrain et al. (2005a)
Stb7	Estanzuela Federal (ST6)	4AL	*Xwmc313*	McCartney et al. (2003), Chartrain et al. (2005a)
Stb8	Synthetic W7984	7BL	*Xgwm146/Xgwm577*	Adhikari et al. (2003)
Stb10	Kavkaz-K4500 L.6.A.4 1	1D	*Xgwm848*	Chartrain et al. (2005a)
Stb11	TE9111 TE9111	1BS	*Xbarc008*	Chartrain et al. (2005c)
Stb12	Kavkaz-K4500 L.6.A.4	4AL	*Xgwm219*	Chartrain et al. (2005a)
Stb15	Arina	6AS	*Xpsr904/Xpsr563a*	Arraiano et al. (2007)

Whole genome analysis has also been utilised to map loci associated with STB resistance at the seedling stage and at the adult stage in the DH populations derived from Chara (STB-susceptible)/WW2449 (STB-resistant), and Whistler (STB-susceptible)/WW1842 (STB-resistant) crosses that were also segregating for other agronomic traits such as wheat quality attributes, aluminium resistance and late maturity alpha amylase activity (Raman et al. 2008, 2009a, 2009b). Four QTLs for resistance to *M. graminicola*; *QStb.riso-2B, QStb.riso-3A.2 QStb.riso-6B* and *QStb.riso-7B* were identified in a DH population from Savannah (susceptible to STB)/Senat (resistant to STB) (Eriksen et al. 2002, 2003).

In the International Triticeae Mapping Initiative (ITMI) population, three QTLs, *QStb.ipk-1DS, QStb.ipk-2DS* and *QStb.ipk-6DS* conferring resistance at seedling-stage, and two QTL, *QStb.ipk-3DL* and *QStb.ipk-7BL* for resistance at adult-stage were identified (Simon et al. 2004). Chartrain et al. (2004a) reported a QTL, *QStb.psr-6B.1* for partial resistance to STB in Riband on chromosome 6B. A weak QTL, *QStb.psr-7D.1*, providing partial resistance to Portuguese isolate IPO92006, was detected in the *Xcdo475b-7B – Xswm5-7B* marker interval in chromosome 7DS (Arraiano et al. 2007). In spelt wheat, Simon et al. (2010) mapped the adult resistance locus *QStb.ipk-7D2* was found on the short arm of chromosome 7D in a similar position to the locus *Lr34/Yr18* known to be effective against multiple pathogens. These findings suggest that different loci control STB resistance in different genetic backgrounds under different environments.

Eriksen et al. (2003) detected a QTL *QHt.riso-3A.2* for height at approximately the same position as the QTL *QStb.riso-3A.2* for resistance to STB. Previous studies have shown significant negative correlation between flowering time/maturity and STB infection (Tavella 1978; Arama et al. 1999). However, no association between plant height, heading data and resistance to STB was reported in wheat (Simón et al. 2005). This suggests that STB resistance genotypes with early maturity and short height can be selected in the breeding programs.

Although, QTL mapping studies provide comprehensive information on the nature of inheritance, and location, magnitude and allelic effects of QTLs, much of the information tends to be 'population' specific. In biparental populations, generally two alleles at each locus are sampled and therefore trait-marker association may not be highly relevant to diverse genetic backgrounds. Association mapping can be utilised for investigating linkage disequilibrium close to loci of interest in a diverse germplasm (Flint-Garcia et al. 2003; Hirschhorn and Daly 2005; Breseghello and Sorrells 2006a; Buckler et al. 2009; Raman and Gustafson 2010) and therefore offers an alternative to linkage and QTL mapping and validating loci. Currently, this approach is increasingly being followed to identify loci of agronomic important traits including for disease resistance genes. Although, genetic structure and close pedigrees in a given breeding program tends to have important consequences in determining trait-marker associations, several algorithms to control such factors have been developed and implemented in such studies. This mapping approach has been employed to identify 'significant' trait-marker associations in wheat (Breseghello and Sorrells 2006a, 2006b; Tommasini et al. 2007; Raman et al. 2010; Ghavami et al. 2011). However, this and other approaches such as mapping as you go (Podlich et al. 2004) have not been investigated in determining marker-STB resistance associations.

Validation and marker assisted selection

During last two decades, several major and minor loci for STB resistance and molecular markers tracking such loci have been identified. Often some QTL have minor effects and may be environment specific. Little or no effort has been made to evaluate the stability of these QTL and or of qualitative genes over diverse wheat growing environments to test their usefulness in achieving long-term durable control of the disease. While some wheat

breeding and germplasm enhancement programs are making progress in selecting desirable alleles of major STB resistance genes, many breeding programs are unwilling to invest to introgress multiple QTL having small effects especially those which have not been validated in a wide range of germplasm. Only a few studies reported validation and verification of STB resistance-molecular marker linkages in the literature. For example, Raman et al. (2009b) validated the linkage between STB resistance and SSR markers; WMC230, BARC119b and KSUM45 that were mapped within 5cM from *XStbWW* and predicted the STB resistance with over 94% accuracy in the 79 advanced breeding lines. Such results provide some confidence to the wheat breeding programs for their usefulness in monitoring STB resistance in different background. Recently, Goodwin et al. (2008) validated the linkage between markers and *Stb3* gene that was mapped on the short arm of chromosome 6D previously (Adhikari et al. 2004a). Genetic and physical mapping showed that *Stb3* in fact maps on the short arm of chromosome 7A. More comprehensive analysis should be done to predict their usefulness to characterise different resistance sources and identify allelic variants of STB resistance genes. Cloning of genes controlling resistance will allow gaining such understanding. To increase marker adoption for improvement of STB resistance in wheat breeding programs, more sources of resistance need to be examined in detail (Orton et al. 2011). However, the disperse nature of identified resistance genes and QTL conditioning STB resistance throughout the wheat genome presents a challenge to breeders in choosing optimal crosses.

Cloned disease resistance genes

So far, 20 resistance (R) genes have been cloned in plants (Michelmore and Meyers 1998; Feuillet et al. 2003; Huang et al. 2003; Yahiaoui et al. 2004; Srichumpa et al. 2005; Cloutier et al. 2007; Krattinger et al. 2009). Several allele variants and the haplotypes of cloned genes including of disease resistance genes in various crops including wheat, barley and rice have been reported (Latha et al. 2004; Malysheva et al. 2004; Srichumpa et al. 2005; Yahiaoui et al. 2006). Classical genetic and molecular analysis data suggest that most of these R genes are clustered and may contain sequences related to gene function and may not be related structurally (Ellis and Jones 1998; Leister et al. 1999). For example, Raman et al. (2009b) located *XstbWW* between the breakpoints 0.52 to 0.84 in the deletion lines 1BS-4 (0.52FL)/IBS-9 (084FL) where several other disease resistance genes to stripe rust, leaf rust and fusarium head blight: *YrH52* (Peng et al. 1999, 2000), *Yr15* (Sun et al. 1997; Peng et al. 2000), *Yr24* (Zakari et al. 2003), *QLr-sfr.1* (William et al. 1997; Schnurbusch et al. 2004), and FHB (Shen et al. 2003), *Yr9, Yr10, Yr26, Yr3a-c, Yr51*; Pm2, Pm3, Pm8, Pm10, Pm22, Pm28, Pm32, QPm.vt-1BL, Lr10, Lr24, Lr26, Lr33, Lr44, Lr46+Yr29*, Sr14, Sr18, Sr31,* and *snn1* for *Stagonospora nodorum* leaf blotch have also been localized on short arm of chromosome 1 (Tosa et al. 1987; Peusha et al. 1996, 2000; Liu et al. 2004; Dilbirligi et al. 2004; Erayman et al. 2004; McIntosh et al. 2004; Tyrka and Chelkowski 2004). Other STB resistance genes such as *Stb2* also map in the same genomic region where *Qfhs.ndsu-3BS* that confers resistance to *F. graminearum* (Buerstmayr et al. 2003), *Lr27* for leaf rust resistance (Faris et al. 1999), *Phaeosphaeria*

(an amorph *Stagonospora nodorum*) causing *Septoria nodorum* (Schnurbusch et al. 2003) and yellow or stripe rust resistance loci are located (Manilal et al. 2003). More recently, physical map of 3B chromosome has been constructed (Paux et al. 2008). At least 40 genes and QTLs have been identified on chromosome 3B (http://wheat.pw.usda.gov/GG2/maps.shtml). Physical maps of different chromosomes would allow fine mapping of the target genes. To date, no gene controlling STB resistance has been cloned. However, the wheat genome is currently being sequenced under international wheat sequencing consortium and will provide insights into gene organisation and their functions, diversity, evolution and the origin of different genes controlling STB resistance. Next generation sequencing approaches will also reveal new candidate genes, identify targets for expression analysis and comprehensively investigate the extent of linkage disequlibrium especially in the vicinity of STB resistance genes. Diagnostic or perfect markers for resistance genes which can be used on multiplexing platforms such as SNPs or DArT will assist enrichment strategies in breeding programs.

Conclusions

It is now clear that major resistance genes will be overcome by time, as has been seen in many crop plants. Therefore, there is constant need to identify new sources of both qualitative and quantitative resistance genes. It is probable that pyramiding of both qualitative and quantitative genes will provide more durable resistance to STB disease that remains effective for a long period of time in cultivars which are widely grown in an environment favourable for disease (Johnson 1984). In addition to genetic resistance, deployment of agronomic practices such as use of rotation and stubble management will further reduce pathogen inoculums for subsequent crops. Development and validation of tightly linked molecular markers that are amenable for high throughput marker screening and cost effective systems will enable to increase their adoption in the wheat breeding programs. Marker assisted selection in an early generation will allow to enrich the desired traits (alleles) in the population. In order to understand host–pathogen interaction, there is a need to develop a set of isolates for key resistance genes. While a resource like this may help to characterise varieties and parents within breeding programs, it is likely to be expensive and slow for rapid selection in segregating populations.

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