Molecular mapping of the *Aegilops speltoides*-derived leaf rust resistance gene *Lr36* in common wheat (*Triticum aestivum*)

Zahra Pourkhorshid · Ali Dadkhodaie · Ali Niazi

Received: 30 November 2021 / Accepted: 22 January 2022 / Published online: 9 February 2022
© The Author(s), under exclusive licence to Springer Nature B.V. 2022

### Abstract

Leaf rust caused by *Puccinia triticina* Eriks. is the most prevalent wheat rust worldwide and occurs annually wherever wheat is grown. The most economical and environmentally friendly method to control this fungal disease is genetic resistance, which is achieved through deploying effective resistance genes. Tightly-linked molecular markers facilitate gene tagging and their deployment with other resistance genes, which in turn contribute to durable leaf rust resistance. The leaf rust resistance gene *Lr36* derived from *Aegilops speltoides* Taush and introgressed into hexaploid wheat by backcrossing, is located on chromosome 6BS. Despite detection of low frequencies of virulence for this gene, no tightly-linked marker is available in disease resistance breeding. Therefore, this research aimed at analysis of simple sequence repeats (SSR) markers linked with *Lr36* in 171 individuals of an F$_2$ population from a cross between the *Lr36*-carrying line (ER84018) and the susceptible cultivar; Boolani. Of 36 primer pairs on chromosome 6BS tested for polymorphism in parents and resistant and susceptible bulks, eight were polymorphic of which the markers *Xgwm88* and *Xcfd13* flanked *Lr36* by 3.8 and 5.2 cM, respectively. The identified markers were validated on 35 genotypes with different genetic backgrounds including few Australian wheat leaf rust differential sets, Iranian landraces and cultivars, and several cultivars and/or breeding lines from Switzerland and Afghanistan, which confirmed that they can be used in selection for *Lr36* in wheat breeding programs.

### Keywords
Brown rust · Bulked segregant analysis · Marker-assisted selection · Microsatellite markers · Resistance genes · Wheat

### Introduction

Wheat (*Triticum aestivum*) is a strategic crop all over the world. However, its production is often threatened by biotic and abiotic stresses (Arzani and Ashraf 2017). Among biotic stresses, fungal diseases including the three rusts; leaf, stripe, and stem rusts, result in significant annual yield losses and have detrimental effects on seed quality (McIntosh 1998). Leaf rust caused by *Puccinia triticina* Eriks. is the most prevalent wheat rust worldwide and occurs annually wherever wheat is grown (Kolmer 2013). The most economical and environmentally friendly method to control this disease is genetic resistance. However, rust pathotypes can mutate to virulence on wheat...
genotypes with previously effective resistance genes. Therefore, breeding for rust resistance can be a never-ending task that has to be supported by ongoing pathogenicity surveys to confirm continued resistance gene effectiveness.

Stacking multiple resistance genes is often difficult due to the unavailability of appropriate pathotypes and/or problems concerning their presence in combination with other resistance genes conferring lower infection types. Recent advances in molecular biology have facilitated the indirect selection of individual genes based on closely linked markers (Gupta et al. 1999), a procedure known as marker-assisted selection (MAS; Qureshi et al. 2018). Combinations of effective resistance genes not only increase the durability but, in many cases, also increase the degree of resistance. Simple sequence repeats (SSRs) or microsatellites are polymorphic tandem repeats of up to six base pairs (Tautz and Renz 1984) that can be identified and traced in genetic analysis or in selection. Though more comprehensive marker systems have been developed, SSR markers still provide valuable information for MAS in rust resistance programs because they are highly polymorphic, low cost to phenotype, and their analysis can be automated (Hayden et al. 2006). In addition, microsatellite consensus maps enable high-density maps of the wheat genome. In this context, more than 80% of primer sets are genome-specific and detect only a single locus in one of the three sub-genomes of bread wheat (A, B, or D). Moreover, publicly available databases provide opportunities to predict allele sizes in breeding populations and develop molecular and genomics strategies in gene mapping (Röder et al. 1998; Somers et al. 2004).

So far, more than 80 leaf rust resistance genes have been catalogued in wheat and its relatives (McIntosh et al. 2017; Qureshi et al. 2018), many of which have been mapped using molecular markers. These include but are not limited to Lr18, Lr23, Lr48, Lr53, Lr65, Lr70, Lr71, Lr73, and Lr80 (Aliakbari Sadeghabad et al. 2017; Chhetri et al. 2017; Dadkhodaie et al. 2011; Hiebert et al. 2014; Kumar et al. 2021; Mohler et al. 2012; Nsabiyera et al. 2016; Park et al. 2014; Singh et al. 2013).

The seedling stage leaf rust resistance gene, Lr36, which was transferred from Aegilops speltoides Taush (2n = 14) into hexaploid wheat by backcrossing, is located on the short arm of chromosome 6B (Dvořák and Knott 1990). This gene has been rarely deployed in wheat cultivars despite having no linkage to undesirable quality or agronomic characters (Dvorak and Knott 1990) and being effective in the USA, China, Poland, Kazakhstan, Pakistan, Egypt, and Iran (Aktar-Uz-Zaman et al. 2017; Ali et al. 2018; Czajowski and Czembor 2016; Koşhybae 2019; Li et al. 2016; Safavi and Afshari 2013; Zhang et al. 2019). Hence, it could be pyramided and deployed with other rust resistance genes. However, linked molecular markers that benefit its deployment in wheat breeding programs have not been reported (McIntosh et al. 2017). Therefore, this study describes mapping the gene Lr36 using SSR markers in a bi-parental population and validates the efficiency of closely-linked markers in different genetic backgrounds.

**Materials and methods**

Plant materials and leaf rust inoculation

The wheat line ER84018, carrying the leaf rust resistance gene Lr36 (Dvorak and Knott 1990), was crossed with ‘Boolani’, the Iranian susceptible cultivar, to produce F1 seeds. Subsequently, the resulting F2 population with 171 individuals and their F3 progenies were used for phenotypic and genetic analysis. Parental genotypes and all F2 plants were grown in 10 cm diameter pots in a temperature-controlled greenhouse (18 ± 2 °C).

The *P. triticina* pathotype FHTQQ (isolate no. 92-23; virulent for 2c, 3a, 3bg, 3ka, 10, 11, 14b, 16, 17, 26, 30, B), which produced infection types “0;1+N” and “33+” on ER84018 and Boolani (Table 1), respectively, was multiplied on a susceptible line. Fresh urediniospores were then mixed with talcum powder in a ratio of 1:4 and used to inoculate the test populations at the two-leaf stage. The inoculated plants were kept in a plastic-covered container at 100% humidity, 18 °C, and dark condition for 24 h prior to moving them to microclimate rooms at 18–24 °C. Infection types (ITs) were recorded according to the 1–4 scale described by McIntosh et al. (1995) approximately 10–12 days post inoculation (dpi) when the susceptible cultivar Boolani showed an IT of “33+”. Plants with ITs less than “2” were considered resistant and those with IT “3” and above were classified as susceptible.
After rust scoring, all 171 F2 plants were transplanted to 20 cm diameter pots and individually harvested. The segregation ratio from F2 population was confirmed by testing F3 families with the same pathotype as described before. Ten to 12 seedlings from each F3 family and both parents were inoculated as described previously, and phenotypes were recorded as homozygous resistant, segregating heterozygous, and homozygous susceptible.

Marker analysis

Rust-free leaf tissue from each F2 plant and both parents was used to isolate DNA using the CTAB method (Yu et al. 2017). A Nanodrop ND-1000 (Wilmington, USA) and 1% (w/v) agarose gel were used to evaluate the quality and quantity of DNA. Working solutions for both genomic DNA and primers were prepared in 200 µl volumes at a concentration of 50 ng µl⁻¹. As described by Michelmore et al. (1991), resistant and susceptible DNA bulks were made from equal amounts of DNA from 20 most resistant and 20 most susceptible F2 individuals.

Thirty-six SSR primer pairs specific to chromosome 6BS (GrainGenes database, http://wheat.pw.usda.gov) were selected for evaluation of polymorphism on both resistant and susceptible parents and the corresponding bulks. Thereafter, each polymorphic marker (Table 2) was amplified on the entire F2 population to determine the number of recombinants between the marker and leaf rust response locus. Polymerase chain reactions (PCR) were conducted in final volumes of 20 µl, comprising 1 µl DNA (50 ng µl⁻¹) as a template, 1 µl of 50 ng µl⁻¹ each pair of forward and reverse primers (Metabion, Germany), 5 µl Taq DNA Polymerase 2× Master Mix Red (5 U µl⁻¹, Ampliqon, Denmark), and 12 µl ddH2O. The PCR conditions were incubation at 94 °C for 5 min, followed by 35 cycles of 94 °C for one minute,

Table 1 Leaf rust responses of Lr36-carrying genotype ‘ER84018’ and Lr36-lacking cultivar ‘Boolani’ to Puccinia triticina pathotype FHTQQ (isolate no. 92-23) at the seedling stage

| Parent  | Genotype  | Leaf rust reaction | Infection typea | Response |
|---------|-----------|--------------------|-----------------|----------|
| 1       | ER84018   | 0;1+N              | 0               | Resistant |
| 2       | Boolani   | 33+                | 3               | Susceptible |

a Infection types were scored based on a modified scale by McIntosh et al. (1995) where “0” = no visible uredinia; “;” = hypersensitive flecks; “1” = small uredinia with necrosis; “2” = small to moderate size uredinia with green islands surrounded by necrosis or chlorosis; “3” = moderate size uredinia with or without chlorosis; C = chlorosis; N = necrosis; ‘-’ and ‘+’ denote smaller or larger uredinia.

Table 2 Sequences and annealing temperatures for polymorphic primer sets used to map Lr36 in a wheat population from the cross between the leaf rust resistant line ‘ER84018’ and the susceptible cultivar ‘Boolani’

| Locusa | Sequence (5’–3’) | Annealing temperature (°C) |
|---------|------------------|---------------------------|
| barc198 | CGCTGAAAAAGAGTGCCGCATTATGA CGCTGCTTTTTCTCAGTTGACGGCA | 59 |
| wmc105  | AATGGTCATGAGGCTAGTCGA AAGGCACCTAACAGAGAGGG | 61.5 |
| gwm193  | TTTTGGCGACCCCTCAGCCAA AATGTTAGTGATGATTGGG | 58 |
| gwm88   | CACTACAATAGCGCTCAGC TCCATTGCTCCTCATAA | 59 |
| cfd13   | CCATAAACCAAGCGTCGATT TTTTTGCGATTGATGATGCTG | 56.5 |
| gwm518  | ATTCACAAAGGCGTGACAA CAGGGTGTGCTCATCAGTCA | 58 |
| barc101 | GCTTCTCCTACGATCAGCAAAAG GCGGCTGATCACTACATAGGCAAT | 63.5 |
| wmc486  | CGGAGTGGGATGCGATTTTT ATGCATGCGATACGCGGTA | 58.5 |

a The loci and their primer sequences were obtained from GrainGenes at http://wheat.pw.usda.gov/cgi-bin/GG3/browse.cgi
56–64 °C for one minute depending on the primer pair, 72 °C for one minute, and the final extension at 72 °C of 10 min. The amplified products were separated in 2% agarose gels containing 1× TAE buffer (54 g Tris–borate, 27.5 boric acid, 200 ml EDTA) and stained with DNA Safe Stain (SinaClon BioScience, Iran). The bands were visualized and photographed in a Gel documentation system (Gene Flash, Syn-gene BioImaging, Vaughan, Canada) under UV light. The band size of each amplicon was estimated using a DNA marker of 100 bp (DNA Ladder Plus, MBI Fermentas).

Statistical and linkage analyses

Alleles for the SSR markers were scored A, B, and H for the resistant, susceptible and heterozygous, respectively, and ‘−’ was used for missing values (PCR failure). The phenotypic data of F3 families were used to establish the genotypes of each F2 individual. The χ² test was used to evaluate the goodness of fit of observed and predicted ratios. Linkage analysis was conducted using JoinMap v0.5 (Van Ooijen 2019) with a threshold LOD ≥ 3.0 and the Kosambi mapping function (1943) based on the recombination frequencies.

Marker validation

The efficiency of the linked markers to select Lr36-carrying genotypes was evaluated in 35 wheat genotypes with different genetic backgrounds. The validating genotypes included two groups; the first group comprised of Australian cultivars with known resistance genes used as differential testers in leaf rust studies. The second group included Iranian cultivars and landraces, Swiss cultivars and breeding lines, and Afghan genotypes. The genotypes in this group were classified as carrying/lacking Lr36 based on their pedigrees or rust response in the greenhouse. The DNA extraction protocol and PCR conditions were the same as mentioned earlier.

Results

Leaf rust phenotyping

The F2 population of 171 progenies segregated into 137 individuals with IT “0;1”, “1+2 N” and 34 plants with IT “3+” fitting a 3:1 ratio (P df=1 = 0.122). As eight F2 plants failed to set seed, the remaining 163 F3 families were used for progeny testing, of which 50, 80, and 33 were homozygous resistant (Lr36Lr36), heterozygous (Lr36lr36), and homozygous susceptible (lr36lr36) (Fig. 1), and conformed to the expected 1:2:1 ratio for a single locus (P df=2 = 0.165; Table 3).

Genotyping and linkage map

Of the 36 pairs of SSR primer sets located on chromosome 6B, eight showed polymorphisms between

![Fig. 1](image-url)
the parents and bulks and hence, were used to genotype the whole F2 population (Fig. 2). Based on PCR amplifications, the primer \textit{gwm88} produced a band of 160 bp in ER84018 while it amplified a 140 bp band in Boolani. The corresponding bands in the resistant and susceptible parents for \textit{cfd13} were 480 bp and 220 bp, respectively. The primer pair \textit{wmc486} amplified a fragment of 200 bp in the susceptible cultivar; Boolani, the susceptible progenies and the heterozygotes while no band was amplified in the resistant parent and the homozygous resistant offsprings. The remaining markers produced amplicons as presented in Table 4. The \( \chi^2 \) tests for segregation of these loci, indicated that all primers except \textit{wmc486} comply with the expected Mendelian ratio of 1:2:1 for co-dominant inheritance, whereas the \textit{wmc486} primer fitted a 3:1 ratio for dominance (Table 4) and at LOD \( \geq 3 \), showed a significant distortion, and was consequently categorized in a separate linkage group.

Overall, seven primer sets were mapped to one linkage group (with an LOD of 3 and greater) covering an interval of approximately 37.7 cM on chromosome 6BS. The closest markers were \textit{Xgwm88} and \textit{Xcfd13} flanking \textit{Lr36} at a distance of 3.8 and 5.2 cM, respectively. The markers \textit{Xgwm518}, \textit{Xgwm193}, and \textit{Xwmc105} were located 9.1, 10, and 15.2 cM from this gene, respectively. The marker \textit{Xbarc101} was mapped 17.5 cM proximally to \textit{Lr36} while the marker \textit{Xbarc198} was placed 20.2 cM distal to it (Fig. 3).

Marker validation

To validate their usefulness, the closely linked markers; \textit{Xgwm88} and \textit{Xcfd13} were used to evaluate 35 wheat genotypes from Australia, Iran, Switzerland

---

**Table 3** Frequencies of different phenotypes in F2 and F3 populations from the cross between the leaf rust resistant line carrying \textit{Lr36}; ‘ER84018’ and the susceptible cultivar ‘Boolani’ when infected with \textit{Puccinia triticina} pathotype FHTQQ (isolate no. 92-23)

| Generation\(^a\) | Observed\(^b\) | Expected ratio | Chi-square | \( P \) value |
|-----------------|--------------|----------------|------------|--------------|
|                 | R | Seg | S |                 |               |       |
| F2 plants       | 137 | -  | 34 | 3:1 | 2.38\(^c\) | 0.122 |
| F3 families     | 50  | 80  | 33 | 1:2:1 | 3.60\(^d\) | 0.165 |

\(^a\)The total number of F3 differs from that of F2 because some of them did not set seed

\(^b\)R and S represent resistant and susceptible phenotypes, respectively in F2. R, Seg and S indicate resistant (\textit{Lr36Lr36}), segregant (\textit{Lr36lr36}) and susceptible (\textit{lr36lr36}) in F2, respectively. The resistant individuals showed resistance ITs “0;1” and “1+2 N", the susceptible showed the ITs “33++" and the segregating families represented a combination of the above- mentioned ITs to the \textit{Puccinia triticina} pathotype FHTQQ

\(^c\)Non-significant at \( p=0.01 \) (df = 1)

\(^d\)Non-significant at \( p=0.01 \) (df = 2)

---

**Fig. 2** Polymorphic markers on 2% agarose gel. A The \textit{Xgwm88}, \textit{Xcfd13}. M shows 100 bp DNA ladder. \textit{P1}; leaf rust resistant parent (ER84018), \textit{P2}; susceptible parent (Boolani), \textit{R}; resistant line, \textit{S}; susceptible line and \textit{H}; segregating in F2 population from a cross between the two parents when tested with pathotype FHTQQ (isolate no. 92-23)
and Afghanistan. The markers Xgwm88 and Xcfd13 amplified 140 and 220 bp amplicons, respectively, in all the genotypes that lacked Lr36 (Supplementary Table 1).

**Discussion**

Wheat relatives are valuable resources for resistance breeding to biotic stresses, especially rust diseases. Though the Aegilops speltoides-derived leaf rust resistance gene, Lr36, has been mapped on the short arm of 6B (Dvorak and Knott 1990), it has not been deployed in wheat cultivars likely due to the unavailability of a diagnostic marker for its selection. Therefore, phenotyping and genotyping were performed on an F2 population and its F3 progenies to map it using SSR markers.

In the current study, P. triticina pathotype FHTQQ (isolate no. 92-23) showing a very low IT on Lr36-carrying genotype and a high IT on Boolani, was used to phenotype the F2 and F3 populations derived from the cross between these two genotypes. Most F2 individuals were resistant and showed a segregation ratio of 3:1 indicating that the resistance in ER84018 was controlled by a single dominant gene which was confirmed by a segregation ratio of 1:2:1 in the F3 generation.

To map Lr36, SSR primers specific to chromosome 6BS were evaluated for polymorphism between the two bulks of resistant and susceptible, of which eight revealed clear polymorphisms. As generally expected with SSR markers, all tested markers displayed co-dominant inheritance except Xwmc486 that fitted to a 3:1 genotypic ratio. This marker showed a null allele in the resistant plants (amplified only in Boolani, the susceptible lines and the heterozygotes) and therefore, could not differentiate heterozygotes from the susceptible homozygotes. Altering of the annealing site has likely led to the loss of the amplicon, resulting in null alleles (Naik Vinod et al. 2015).

The most closely-linked markers to Lr36 were the flanking markers; Xgwm88 and Xcfd13 at 3.8 and 5.2 cM followed by Xgwm518 at 9.1 cM. The order of markers was slightly different from that of the SSR consensus map developed by Somers et al. (2004). This is likely due to the translocation of Lr36 from Aegilops speltoides to wheat, which has led to different recombination frequencies depending on the distance from the breakage points. Additionally, the type and sample

| Marker    | Product size (bp) | Ratio** | χ²   | P-value |
|-----------|-------------------|---------|------|---------|
|           | Resistant^a |
| Xbarc198  | 150               | 32:87:34| 2.93 | 0.231   |
| Xwmc105   | 220/350           | 36:80:39| 0.28 | 0.869   |
| Xgwm193   | 190               | 42:77:30| 2.10 | 0.349   |
| Xgwm88    | 160               | 45:86:27| 5.34 | 0.069   |
| Xcfd13    | 480               | 36:89:38| 1.43 | 0.489   |
| Xgwm518   | 160/210           | 33:99:29| 8.70 | 0.012   |
| Xbarc101  | 170               | 48:86:27| 6.23 | 0.044   |
| Xwmc486   | 200               | 42:121  | 0.05 | 0.823   |
|           | Susceptible^b |

^aThe resistant individuals displayed a range of ITs i.e. “0;1” and “1+2 N” to Puccinia triticina pathotype FHTQQ

^bThe susceptible individuals showed high ITs of “33+” to the same pathotype

^cThe ratios represent the number of individuals in which bands corresponding to homozygous resistant, heterozygous and homozygous susceptible were amplified, respectively

^dDifferences in total number of observed genotypes are due to the non-amplification of the corresponding band(s) is some individuals

^eNo band was amplified

^nsNon-significant

*Significant at p = 0.05 (df = 2)
size of the tested population might have contributed to the altered genetic distances (Liu et al. 2013).

The gene \( Lr36 \) was conclusively mapped on the short arm of 6B similar to the two tightly linked leaf and yellow rust resistance genes \( Lr53 \) and \( Yr35 \) as reported by Dadkhodaie et al. (2011). According to their findings, these genes segregate independently from the gene \( Lr36 \), and the marker \( Xbarc198 \) was distal to \( Lr53 \) at an approximate distance of 28 cM while our results located this marker at a distance of 20.2 cM from \( Lr36 \). Therefore, it could be inferred that the gene \( Lr36 \) is located distal to \( Lr53 \).

Both markers were tested on a panel of 35 wheat genotypes from different backgrounds. The Australian...
cultivars are known to carry known resistance genes. The Iranian cultivars are mostly derived from CIM-MYT germplasms, which lack Lr36 in their pedigrees. Previous phenotypic evaluation of these genotypes along with landraces had shown they do not carry this gene (A. Dadkhodaie, unpublished data). Similarly, phenotyping and pedigree information evidently showed the absence of this gene in Swiss and Afghan genotypes. Therefore, the results of genotyping with these markers were in agreement with those of previous studies confirming their reliability in screening for Lr36.

Overall, though sequence-based genotyping and single nucleotide polymorphism (SNP) markers have dominated in genomic selection, SSR markers could facilitate finding neighboring SNPs in gene regions and fine mapping. In the present study, we developed PCR-based molecular markers (SSR) for the leaf rust resistance gene Lr36 for the first time. Both linked markers; Xgwm88 and Xcfd13 gave clearly scorable bands and categorized resistant, susceptible, and heterozygotes. Furthermore, their application in tracking Lr36 in diverse wheat lines and genotypes produced unambiguous and precise outcomes and confirmed the marker–trait association. Therefore, both markers could be utilized for marker-assisted selection in breeding programs. Since this gene is still effective in many countries across the world, these markers not only enable researchers to combine it with other seedling and adult plant rust resistance genes but also lay the foundations for its map-based cloning and fine-mapping.

Authors’ contribution ZP conducted the research and drafted the manuscript; AD designed the experiment, supervised the study, and revised the manuscript and AN helped to improve the manuscript.

Funding Partial financial support was received from Shiraz University, Higher Education Office, to conduct this research.

Availability of data and materials The data that support the findings of this study are available on request from the corresponding author.

Code availability Not applicable.

Declarations

Conflict of interest The authors declare that there is no conflict of interest.

Ethics approval Not applicable.

Consent to participate Not applicable.

Consent for publication Not applicable.

References

Aktar-Uz-Zaman M, Tuhina-Khatun M, Hanafi MM, Sahebi M (2017) Genetic analysis of rust resistance genes in global wheat cultivars: an overview. Biotechnol Biotechnol Equip 31(3):431–445. https://doi.org/10.1080/13102818.2017.1304180

Ali B, Munir I, Iqbal A, Ahmad MA, Maqsood I, Hafeez M (2018) Molecular characterization of wheat advanced lines for leaf rust resistant genes using SSR markers. Microb Pathog 123:348–352. https://doi.org/10.1016/j.micpat.2018.07.029

Aliakbari Sadeghabad A, Dadkhodaie A, Heidari B, Razi H, Mostowfizadeh-Ghalamfarsa R (2017) Microsatellite markers for the Triticum timopheevi-derived leaf rust resistance gene Lr18 on wheat 5BL chromosome. Breed Sci 67(2):129–134. https://doi.org/10.1270/jsbbs.16148

Arzani A, Ashraf M (2017) Cultivated ancient wheats (Triticum spp.): a potential source of health-beneficial food products. CRFSFS 16:477–488. https://doi.org/10.1111/1541-4337.12262

Chhetri M, Bariana H, Wong D, Sohail Y, Hayden M, Bansal U (2017) Development of robust molecular markers for marker-assisted selection of leaf rust resistance gene Lr23 in common and durum wheat breeding programs. Mol Breed 37(3):1–8. https://doi.org/10.1007/s11032-017-0628-6

Czajkowski G, Czembor PCz (2016) Virulence of Puccinia triticina on triticale in Poland. In: 20th EUCARPIA General Congress, Plant Breeding, the Art of Bringing Science to Life, ETH Zurich. Switzerland. Agroscope, ISS, 210 pp

Dadkhodaie NA, Karaoglug H, Wellings CR, Park RF (2011) Mapping genes Lr53 and Yr35 on the short arm of chromosome 6B of common wheat with microsatellite markers and studies of their association with Lr36. Theor Appl Genet 122(3):479–487. https://doi.org/10.1007/s00122-010-1462-y

Dvofák J, Knott DR (1990) Location of a Triticum speltaoides chromosome segment conferring resistance to leaf rust in Triticum aestivum. Genome 33(6):892–897. https://doi.org/10.1139/g90-134

Gupta PK, Varshney RK, Sharma PC, Ramesh B (1999) Review molecular markers and their applications in wheat breeding. Plant Breed 118(5):369–390. https://doi.org/10.1046/j.1439-0523.1999.00401.x

Hayden MJ, Stephenson P, Logojan AM, Khatakar D, Rogers C, Eldsen J, Sharp PJ (2006) Development and genetic mapping of sequence-tagged microsatellites (STMs) in bread wheat (Triticum aestivum L.). Theor Appl Genet 113(7):1271–1281. https://doi.org/10.1007/s00122-006-0381-4

Hiebert CW, McCallum BD, Thomas JB (2014) Lr70, a new gene for leaf rust resistance mapped in common wheat.

Hiebert CW, McCallum BD, Thomas JB (2014) Lr70, a new gene for leaf rust resistance mapped in common wheat.
accession KU3198. Theor Appl Genet 127(9):2005–2009. https://doi.org/10.1007/s00122-014-2356-1

Kolmer J (2013) Leaf rust of wheat: pathogen biology, variation and host resistance. Forests 4(1):70–84. https://doi.org/10.3390/f4010070

Kosambi D (1943) The estimation of map distances from recombination values. Ann Eugen 12(1):172–175. https://doi.org/10.1111/j.1469-1809.1943.tb02321.x

Koshybaev M (2019) Reaction of isogenes Thatchher wheat lines on the North-Kazakhstan population of Puccinia triticina and resistance of wheat cultivars. J Mycol Plant Pathol 53(3):162–169. https://doi.org/10.1134/S0026364819030073

Kumar S, Bhaward SJ, Gangwar OP, Sharma A, Qureshi N, Kumar VN, Khan H, Prasad P et al (2021) Lr80: A new and widely effective source of leaf rust resistance of wheat for enhancing diversity of resistance among modern cultivars. Theor Appl Genet 134(3):849–858. https://doi.org/10.1007/s00122-020-03735-5

Li G, Xu B, Bai G, Carver BF, Hunger R, Bonman JM, Dong H (2016) Genome-wide association mapping reveals novel QTL for seedling leaf rust resistance in a worldwide collection of winter wheat. TPG. https://doi.org/10.3835/plantgenome2016.06.0051

Liu Z, Bowden RL, Bai G (2013) Molecular markers for leaf rust resistance gene Lr42 in wheat. Crop Sci 53:1566–1570. https://doi.org/10.2135/cropsci2010.09.0532

McIntosh RA (1998) Breeding wheat for resistance to biotic stresses. Euphytica 100(1–3):19–34. https://doi.org/10.1007/978-94-011-4896-2_11

McIntosh RA, Wellings CR, Park RF (1995) Wheat rusts: an atlas of resistance genes. CSIRO Publications, Melbourne, p 200

McIntosh RA, Dubcovsky J, Rogers J, Morris C, Xia XC (2017) Catalogue of gene symbols for wheat. Komugi wheat Genetic Resources Database. http://shigen.nig.ac.jp/wheat/komugi/genes/macgene/supplement2020.pdf

Michelmore RW, Paran I, Kesseli RV (1991) Identification of markers linked to disease-resistance genes by bulked segregant analysis: a rapid method to detect markers in specific genomic regions by using segregating populations. PNAS 88(21):9828–9832. https://doi.org/10.1073/pnas.88.21.9828

Mohler V, Singh D, Singrør C, Park RF (2012) Characterization and mapping of Lr65 in spelt wheat “Altgold Rotkorn.” Plant Breed 131(2):252–257. https://doi.org/10.1111/j.1439-0523.2011.01934

Naik Vinod BK, Sharma JB, Sivasamy M, Prabhu KV, Tomar RS, Tomar SMS (2015) Molecular mapping and validation of the microsatellite markers linked to the Secale cereal-derived leaf rust resistance gene Lr45 in wheat. Mol Breed 35:61. https://doi.org/10.1007/s11032-015-0234-4

Nsabiyera V, Qureshi N, Bariana HS, Wong D, Forrest KL, Hayden MJ, Bansal UK (2016) Molecular markers for adult plant leaf rust resistance gene Lr48 in wheat. Mol Breed 36(6):1–9. https://doi.org/10.1007/s11032-016-0488-5

Park RF, Mohler V, Nazari K, Singh D (2014) Characterization and mapping of gene Lr73 conferring seedling resistance to Puccinia triticina in common wheat. Theor Appl Genet 127(9):2041–2049. https://doi.org/10.1007/s00122-014-2359-y

Qureshi N, Bariana H, Kumar VN, Muruga S, Forrest KL, Hayden MJ, Bansal U (2018) A new leaf rust resistance gene Lr79 mapped in chromosome 3BL from the durum wheat landrace Aus26582. Theor Appl Genet 131(5):1091–1098. https://doi.org/10.1007/s00122-018-3060-3

Röder MS, Korzun V, Wendeheke K, Plaschke J, Ne Tixier MH, Leroy P, Ganal MW (1998) A microsatellite map of wheat. Genetics 149:2007–2023

Safavi SA, Afshari F (2013) Virulence factors of Puccinia triticina on wheat and effectiveness of Ls genes for leaf rust resistance in Ardabil. Arch Phytopathol 46(10):1246–1254. https://doi.org/10.1080/03235408.2013.764055

Singh D, Mohler V, Park RF (2013) Discovery, characterization and mapping of wheat leaf rust resistance gene Lr71. Euphytica 190(1):131–136. https://doi.org/10.1007/s10681-012-0786-x

Somers DJ, Isaac P, Edwards K (2004) A high-density microsatellite consensus map for bread wheat (Triticum aestivum L.). Theor Appl Genet 109(6):1105–1114. https://doi.org/10.1007/s00122-004-1740-7

Tautz D, Renz M (1984) Simple sequences are ubiquitous repetitive components of eukaryotic genomes. Nucleic Acids Res 12(10):4127–4138. https://doi.org/10.1093/nar/12.10.4127

Van Ooijen JW (2019) JoinMap® 5, Software for the calculation of genetic linkage maps in experimental populations of diploid species. Kyazma B.V., Wageningen, pp 1–61

Yu G, Hatta A, Periyannan S, Lagudah E, Wulff BBH (2017) Isolation of wheat genomic DNA for gene mapping and cloning. In: Periyannan S (ed) Wheat rust diseases methods and resistance of wheat cultivars. J Integr Agric 16:1093–1104. https://doi.org/10.1007/s11032-016-0488-5

Publisher’s Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.