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# Inheritance and QTL mapping of leaf rust resistance in the European winter wheat cultivar 'Beaver'

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**Abstract** Genetic studies were conducted on an European winter wheat cultivar, Beaver, to determine the mode of inheritance of leaf rust resistance at seedling and adult plant growth stages using a recombinant doubled haploid population, Beaver/Soissons. Greenhouse studies indicated the involvement of genes *Lr13* and *Lr26* in governing leaf rust resistance at seedling growth stages, whereas, adult plant resistance (APR) in the field with pathotypes carrying virulence individually for *Lr13* and *Lr26* showed trigenic inheritance for the population. Marker regression analysis of adult plant field data indicated the involvement of six significant QTLs (chromosomes 1B, 3B, 3D, 4B, 4D and 5A) in year 2005, four QTLs (1B, 3B, 4B and 5A) in 2006, and six QTLs (1A, 1B, 3B, 4A, 4B and 5A) in 2007 for reducing leaf rust severity. QTLs on chromosomes 1B, 4B and 5A were considered the most important because of their detection across years, whereas QTLs on chromosomes 1A, 3B, 3D and 4A were either inconsistent or non-significant and unexplained. Based on an association of closely linked markers with phenotypic data, putative single gene stocks were identified for each consistent QTL and crossing was initiated to develop populations segregating for each to permit fine mapping of the identified regions.

**Keywords** Common wheat (*Triticum aestivum*) · Wheat leaf rust · Wheat brown rust · Resistance · Adult plant resistance (APR) · Genetic mapping

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## Introduction

Among the three fungal rust diseases of wheat, leaf rust caused by *Puccinia triticina* (*Pt*), occurs most regularly and is most widespread. More than 50 loci conferring resistance to *Pt* have been identified and catalogued in wheat (McIntosh et al. 2005), although many of these have been defeated by virulent pathotypes of the pathogen. A vast majority of the catalogued resistance genes are expressed at the seedling stage and are effective at all growth stages, and a few are expressed at post-seedling growth stages only. Adult plant resistances (APR) that have been genetically characterized to date are controlled either by hypersensitive and race-specific resistance genes like *Lr12*, *Lr22a*, *Lr22b*, *Lr35*, *Lr48* and *Lr49*, or non-hypersensitive and non race-specific resistance genes like *Lr34* and *Lr46*. Some genes from the former

category are believed to behave in a similar fashion to classical seedling resistances, lacking durability, as evidenced by the ineffectiveness of *Lr12* and *Lr22b* in some geographical areas (Park and McIntosh 1994). On the other hand, resistance that is non-hypersensitive and non-specific (often associated with slow rusting or partial resistance) is considered to be controlled by genetic factors of moderately high heritability and of additive and/or interactive action (William et al. 2006; Bjarko and Line 1988), and have a reputation for durability. It is noteworthy that both *Lr34* and *Lr46* have continued to condition leaf rust resistance despite the global deployment of cultivars possessing these genes over an extended period of time. In fact, Dyck (1991) hypothesized that *Lr34* became widespread due to its presence in genotypes like Chinese Spring that were commonly used as parents in breeding programs. Although *Lr34* and *Lr46* have proven durable thus far, it should not be assumed that *Pt* pathotypes with matching virulence will not appear in due course. Hence, over-reliance on these genes is risky and there is a need to characterize additional APR genes and deploy cultivars carrying them.

Many European winter wheats have been reported to possess high levels of APR and/or slow rusting to leaf rust (Singh et al. 2001; Pathan and Park 2006). However, little is known about the genetic basis of quantitative leaf rust resistance in European winter wheat germplasm (Messmer et al. 2000). Molecular markers provide a powerful tool to characterize quantitative traits such as slow rusting. Dissection of these traits into Mendelian factors or quantitative trait loci (QTL) helps to further identify specific regions on chromosomes that are associated with resistance. This is achieved by developing genetic linkage maps and carrying out precise phenotyping on defined genetic stocks, particularly recombinant doubled haploid (DH) populations (Nelson et al. 1997; Suenaga et al. 2003; Schnurbusch et al. 2004; William et al. 2006). Over the last 5 years, the John Innes Centre (JIC) has developed ten such DH mapping populations based on crosses between key European winter wheat germplasm (Snape et al. 2007). From a leaf rust resistance perspective, one of the most important populations from this set is based on a cross between the cultivar Beaver, which is known to carry leaf rust resistance that has been effective in Europe since its release, and is also effective in Australia (Singh et al. 2001), and the cultivar Soissons, known to be susceptible to leaf rust at adult plant growth stages in Australia (Singh et al. 2001).

In this study we report on (i) genetic analysis of seedling and adult plant leaf rust resistance in cultivar Beaver and (ii) identification of QTLs and localization of markers near the genomic regions responsible for leaf rust resistance in Beaver under Australian field conditions in which artificial epidemics of leaf rust were generated with specific pathotypes of *Pt*.

## Materials and methods

### Population development

The recombinant DH population comprised 65 lines and was developed at the JIC using the standard wheat × maize technique from the $F_1$ between Beaver and Soissons (Snape et al. 2007). Beaver (Pedigree: Hedgehog/Norman/Moulin), an English feed wheat developed by PBI Cambridge and released commercially in 1989, and Soissons (Pedigree: Jena/HN35), a French bread-making quality wheat developed by Desprez and released in 1987, were previously shown to be resistant and susceptible to leaf rust at adult plant growth stages, respectively, in Australian studies (Singh et al. 2001).

### Seedling resistance assessment

The DH population, parents and a set of Australian leaf rust differential genotypes were screened in the greenhouse with *Pt* pathotypes 26-0 (PBI Accession No. 640153, avirulent on *Lr13* and *Lr26*); 53-1,(6),(7),10,11 (810043, virulent on *Lr13* and avirulent on *Lr26*); 104-2,3,6,9 (840412, avirulent on *Lr13* and virulent on *Lr26*) and 10-1,3,9,10,11,12 (040120, virulent on *Lr13* and *Lr26*). Plants of each genotype were established as clumps of 10–12 seedlings (four per 9-cm diameter pot). Pathotype nomenclature and classification, and the method of sowing, are described in McIntosh et al. (1995).

Inoculations were performed on seedlings at the 1–1.5 leaf stage. Urediniospores suspended in a light mineral oil (Shellsol®, 10 mg spores per 10 ml oil per 200 pots) were atomised over the seedlings using a hydrocarbon propellant pressure pack. The inoculated seedlings were incubated for 18–24 h at ambient

temperatures in a dark room, in which mist was generated by an ultrasonic humidifier. The seedlings were then moved to a temperature and irrigation-controlled greenhouse compartment at 20–22°C, and disease response assessments were made after 12–14 days. Infection types were recorded on the basis of the infection type (IT) scale given by Stakman et al. (1962), with slight modifications as proposed by Luig (1983).

### Field phenotypic assessment

Fifteen seeds from each line of the DH population and the parents were sown as 0.5 m rows with a clump of a rust spreader adjacent to each row. An artificial leaf rust epidemic was created by misting urediniospores of relevant pathotypes suspended in mineral oil (Shellsol®) over spreader rows using an ultra low volume applicator (Microfit®, Micron Sprayer Ltd.). Inoculations were performed on clear afternoons when there was a high likelihood of overnight dew. The pathotypes used in the field in 2005 (53-1,(6),(7),10,11 and 104-1,2,3,(6),(7),9,11) carried virulence individually for *Lr13* and *Lr26,* but not in combination. The same pathotypes were used in the field in 2006, along with a third pathotype that combined virulence for *Lr13* and *Lr26* (10-1,3,9,10,11,12).

Three observations at fortnightly intervals recorded disease severity (percentage leaf area affected) and host response using a modified Cobb scale (Peterson et al. 1948). The disease severity and host response data were combined into a single value, the coefficient of infection (CI), by multiplying severity by a constant for host response, where MR, MR-MS, MS and S = 0.25, 0.50, 0.75 and 1.0, respectively. For example, a disease score 50 MR was represented by the CI = 12.50 (50 × 0.25).

### Genetic linkage map and QTL analysis

A genetic linkage map was created from 312 polymorphic loci comprised of 121 SSR, 49 AFLP, and 135 DArT markers, and the major genes *Glu-A1*, *Glu-B1*, *Glu-D1*, *Rht-B1*, *Rht-D1*, *B1* (presence/absence of awns) and *Ha* (grain hardness). The map covered 1,526 cM, corresponding to an average density of 12.6 cM per marker. Initial QTL analysis was performed using the programme QTL-Café, available from http://web.biosciences.bham.ac.uk/labs/kearsey. Single marker ANOVA, marker regression analysis (Kearsey and Hyne 1994), and interval mapping (Lander and Botstein 1989) were applied on leaf rust disease CI data to identify putative QTLs and markers linked to genomic regions associated with leaf rust resistance.

## Results

### Parental response

Beaver showed low ITs with pathotypes 26-0 (avirulent on both *Lr13* and *Lr26*) and 53-1,(6),(7),10,11 (virulent on *Lr13* and avirulent on *Lr26*), and behaved similarly to the *Lr26* control Mildress (Table 1). With pathotype 104-1,2,3,(6),(7),9,11 (avirulent on *Lr13* and virulent on *Lr26*), Beaver displayed a mesothetic infection type very typical of *Lr13* and similar to the *Lr13* control Egret, and with pathotype 10-1,3,9,10,11,12 (virulent on *Lr13* and *Lr26*), it displayed a high IT. Based on the infection type pattern and a comparison with controls, Beaver was postulated to carry *Lr13* and *Lr26*. Soissons showed high ITs with all three pathotypes, indicating that it lacked effective seedling resistance to these pathotypes. In the field in 2005, Beaver was resistant while Soissons was susceptible (predominant field pathotypes virulent on *Lr13* and *Lr26* separately but not in combination), and similar responses were seen in 2006 and 2007 (predominant field pathotypes virulent on *Lr13* and *Lr26* in combination).

### Genetic analysis and mapping of seedling inheritance

The lines within the entire population varied in their seedling responses to three *Pt* pathotypes (Table 2). Of 65 lines tested, 50 produced a resistant phenotype (ITs ranging from; to X+3+) and the remaining 15 were susceptible when tested with pathotype 26-0, avirulent on both *Lr13* and *Lr26*. This conformed to a satisfactory fit for segregation at two loci, likely *Lr13* and *Lr26*. All 12 lines that showed a mesothetic IT with 26-0 were susceptible when tested with the *Lr13*-virulent pathotype 53-1,(6),(7),10,11, whereas the remaining resistant and susceptible lines behaved in a fashion similar to that recorded with pathotype 26-0. The segregation pattern observed with pathotype 53-1,(6),(7),10,11 conformed to segregation at a

**Table 1** Leaf rust response of parental genotypes and controls tested in the greenhouse and field with four pathotypes of *Puccinia triticina*

| Parent | Infection type | | | | Postulated gene(s) | Field disease score | | |
|---|---|---|---|---|---|---|---|---|
| | A | B | C | D | | 2005[a] | 2006[b] | 2007[b] |
| Beaver | ;1− | ; | X + 3 | 3+ | *Lr13*, *Lr26* | 0 | TR | TR |
| Soissons | 3+ | 3+ | 3+ | 3+ | None | 80S | 80S | 70S |
| Egret | X+3 | 3+ | X++3 | 3+ | *Lr13* | 80S | 80S | 80S |
| Mildress | ;1= | ; | 3+ | 3+ | *Lr26* | 70S | 80S | 80S |

*A* 26-0; *B* 53-1,(6),(7),10,11; *C* 104-1,2,3,(6),(7),9,11; *D* 10-1,3,9,10,11,12

Prevalent pathotypes [a]53-1,(6),(7),10,11 and 104-1,2,3,(6),(7),9,11; [b]53-1,(6),(7),10,11 and 104-1,2,3,(6),(7),9,11 and 10-1,3,9,10,11,12

**Table 2** Leaf rust response of progeny of the Beaver/Soissons doubled haploid population in seedling greenhouse tests and adult plant field tests

| Resistance (screening) | Pathotype/year | No. of families | | $\chi^2$ fit (R:S) | $P >$ | No. of genes |
|---|---|---|---|---|---|---|
| | | Res (R) | Sus (S) | | | |
| Seedling (greenhouse) | A | 50 | 15 | 3:1 | 0.7 | 2 |
| | B | 38 | 27 | 1:1 | 0.1 | 1 |
| | C | 32 | 33 | 1:1 | 0.9 | 1 |
| | D | 0 | 65 | – | – | – |
| APR (field) | 2005[a] | 59 | 6 | 15:1 | 0.3 | 4 |
| | 2006[b] | 55 | 10 | 7:1 | 0.5 | 3 |
| | 2007[b] | 55 | 10 | 7:1 | 0.5 | 3 |

*A* 26-0; *B* 53-1,(6).(7),10,11; *C* 104-1,2,3,(6),(7),9,11; *D* 10-1,3,9,10,11,12

Prevalent pathotypes [a]53-1,(6),(7),10,11 and 104-1,2,3,(6),(7),9,11; [b]53-1,(6),(7),10,11 and 104-1,2,3,(6),(7),9,11 and 10-1,3,9,10,11,12

single locus, likely *Lr26*. When assessed with the *Lr26*-virulent pathotype 104-1,2,3,(6),(7),9,11, the lines segregated in the ratio of 38 mesothetic resistant: 33 susceptible, a satisfactory fit for segregation at a single locus, likely *Lr13*. All lines showed susceptible responses when tested with pathotype 10-1,3,9,10,11,12, virulent on *Lr13* and *Lr26*.

The seedling resistance locus *Lr26*, detected with pathotype 53-1,(6),(7),10,11, was mapped to 1RS. Because Beaver carries the 1BL/1RS translocation, detailed mapping to this arm is not possible. The second seedling resistance gene (*Lr13*) mapped to 2BS with the closest marker being wPt8492 located at 43 cM, in comparison to the consensus map of Somers et al. (2004).

## Phenotyping and genetic analysis of APR

The leaf rust reaction of the 65 lines in the field varied with the seedling genotypes involved and the year of assessment, although similar disease score patterns were observed during years 2006 and 2007. There was continuous phenotypic variation from 0 to 40 MS (CI 0 to 30) within the lines classified as resistant. The Chi square distribution of the entire population supported a four gene segregation ratio (15:1) during year 2005. However, the pathotypes used in the field during 2005, while individually virulent for *Lr13* and *Lr26*, did not combine virulence for these genes. Hence, for a reliable estimation of APR, lines with the genotype *Lr13* + *Lr26* were excluded from adult plant screening analysis, resulting in the remaining lines giving a satisfactory fit for a 7:1 ratio ($P > 0.7$) expected for segregation at three genetically independent loci. The pathotypes used in 2006 and 2007, however, carried virulence on all seedling genotypes and the rust response (R:S) distribution for the whole population also showed a good fit for segregation at three independent loci ($P > 0.5$ for both years). The combined analysis of years 2005 (excluding lines

**Table 3** Chromosome (chrm) and QTL location (lctn), and potential linkage to loci with adult plant resistance to leaf rust in a doubled haploid population (Beaver/Soissons) in field tests over 3 years

| Year | Chrm. | QTL Lctn (cM) | Simulated QTL position | Nearest marker | Significance |
|---|---|---|---|---|---|
| 2005 | *1B* | 1BL/1RS[a] | | | |
| | 3BS | 32 | 26.4 ± 7.7 | s15/m34.3 | 0.007a |
| | 3D | 8 | 8.0 ± 5.9 | gdm72 | 0.016* |
| | *4B* | 8 | 9.2 ± 6.5 | wPt-5334 | 0.003* |
| | 4D | 4 | 5.2 ± 4.8 | s13/M23.2 | 0.007* |
| | *5AS* | 12 | 9.1 ± 4.0 | wPt-1931 | 0.011* |
| 2006 | 1A | 10 | 14.0 ± 11.6 | Psp3027 | 0.053b |
| | *1B* | 1BL/1RS[a] | | | |
| | 3BL | 84 | 71.6 ± 23.8 | wPt-5946 | 0.062c |
| | 4A | 54 | 48.4 ± 13.1 | wPt-6447 | 0.008* |
| | *4B* | 6 | 8.3 ± 7.8 | wPt-1708 | 0.028* |
| | *5AS* | 10 | 7.8 ± 4.5 | wPt-1931 | 0.036* |
| 2007 | 1A | 10 | 13.7 ± 10.8 | psp3027 | 0.041* |
| | *1B* | 1BL/1RS[a] | | | |
| | 3BL | 84 | 73.5 ± 22.4 | wPt-5946 | 0.073 |
| | 4A | 52 | 47.1 ± 11.9 | wPt-6447 | 0.051 |
| | *4B* | 6 | 8.4 ± 7.7 | wPt-1708 | 0.031* |
| | *5AS* | 8 | 7.0 ± 4.5 | wPt-8756 | 0.049* |

Chromosomes in *italics* represent significant QTLs detected during all years

*a* minor non-significant effect detected at 30 cM in 2006, *b* minor non-significant effect detected at 6 cM in 2005, *c* minor non-significant effect detected at 100 cM in 2005

[a] Beaver carried 1BS/1RS translocation so it was not possible to locate the gene to a location within this arm as no recombination takes place between the 1BS and 1RS segments

* $P = 0.05$

with genotype *Lr13* + *Lr26*), 2006 and 2007 again implied trigenic inheritance of APR ($P > 0.6$).

## QTL mapping of APR

Marker regression analysis indicated the involvement of six significant QTLs (chromosomes 1B, 3B, 3D, 4B, 4D and 5A) in year 2005, four QTLs (1B, 3B, 4B and 5A) in 2006, and six QTLs (1A, 1B, 3B, 4A, 4B and 5A) in 2007 for reducing leaf rust severity. The QTLs and the closest marker detected for each during years 2006 and 2007 were identical (except the 5A marker), though the significance levels varied (Table 3). QTLs on 3B, 3D and 4D were only detected during 2005, whereas QTLs 4B and 5A were detected consistently during all three years (Fig. 1). The QTL on chromosome 1BS was also detected consistently for 3 years, however, Beaver carries the 1BS/1RS translocation so it was not possible to map the gene to a location within this arm as no recombination takes place between the 1BS and 1RS segments The different results across years obtained from the mapping are likely attributable to differences in the pathotypes used in the field in 2005 compared to 2006 and 2007. Data for years 2006 and 2007, however, were considered more meaningful because of the presence of virulence on all seedling genes and gene combinations. QTLs on chromosomes 1B, 4B and 5A were considered the most important because of their consistent recurrence. The reduction in disease severity associated with these three QTLs was calculated by comparing mean differences in disease severity of lines with and lines without marker alleles for both years (2006 and 2007). Depending upon the QTL and year involved, an 11–20% reduction in leaf rust severity was observed with respect to marker allele data (Table 4). The additive effects for all these three QTLs were highly significant.

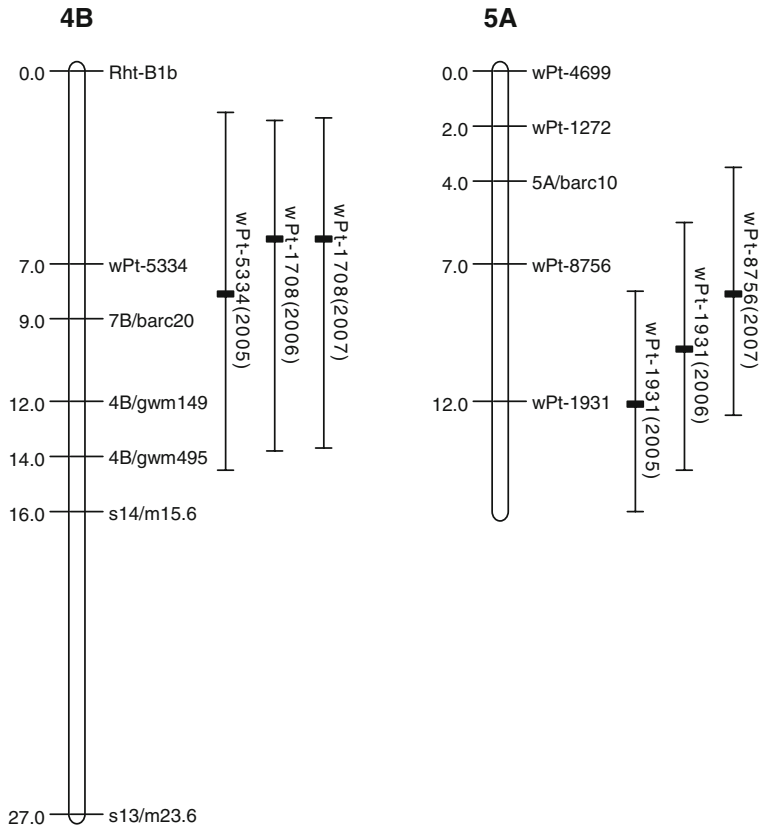


**Fig. 1** Partial linkage maps of chromosomes 4B and 5A and simulated location of QTLs associated with respective chromosomes during years 2005, 2006 and 2007 in the doubled haploid population Beaver/Soissons. The *solid bar* indicates the most likely position of QTLs identified

**Table 4** Significant QTLs for adult plant resistance to leaf rust involved in a doubled haploid population (Beaver/Soissons) and their contribution to disease severity and additive effects during years 2006 and 2007

| Chromosome location | Marker | Disease severity reduction (%) | Additive effect |
|---|---|---|---|
| 1BL | 1BL/1RS | 17.3 | 7.5 |
| 4BL | *wPt-1708* | 12.4 | 7.0 |
| 5AS | *wPt-1931/wPt-8756* | 11.2 | 6.9 |

## Discussion

Although a population size as small as 65 DH lines is not ideal for Mendelian and QTL analysis and a larger population would have been desirable, the results obtained were highly consistent across years. Genetic studies of the Beaver/Soissons DH population supported the involvement of two genes (*Lr13* and *Lr26*) in governing seedling leaf rust resistance in the cultivar Beaver. The mapping of seedling data showed the involvement of the 1BL/1RS and 2BS regions, supporting the presence of *Lr26* and *Lr13*, respectively. Gene *Lr13* was reported as 3.9 cM from the centromere on chromosome 2BS (McIntosh et al. 1995), but in this study, it could not be mapped accurately due to the low number of markers mapped closer to this locus, however, if the gene involved is not *Lr13*, it had the same or very similar pathogenic specificity. The gene *Lr16* is located at the proximal end of chromosome 2B (McCartney et al. 2005), and because all four pathotypes used in the seedling studies were not capable of distinguishing *Lr13* from *Lr16*, it is possible that the gene may be *Lr16* instead of *Lr13*. However, additional testing of Beaver using pathotype 76-3,5,9,10 +*Lr37* (virulent on *Lr13* and *Lr26* and avirulent on *Lr16*) demonstrated susceptibility, indicating the gene present in Beaver is not *Lr16*.

High levels of APR were detected in the population, the inheritance of which was controlled by either four genes (year 2005) or three genes (years 2006 and 2007). The differences in the results were likely due to differences in the virulence spectra of the pathotypes

used in the field in 2005 from those used in 2006 and 2007. In 2005, a combination of pathotypes was used that carried virulence individually for *Lr13* and *Lr26*, but not in combination. Until early 2005, combined virulence for *Lr13* and *Lr26* had not been detected in Australia, but in 2005, a new pathotype 10-1,3,9,10,11,12 carrying this virulence combination was detected (R.F. Park, unpublished). This pathotype was used in the field in 2006 and 2007 as the combination of virulence for *Lr13* and *Lr26* made it particularly suitable to assess APR in the Beaver/Soissons population. The data for years 2006 and 2007 is therefore more meaningful in interpreting the inheritance of APR to leaf rust in Beaver, and suggested a trigenic inheritance. This estimate of gene number does, however assume that the genes are not linked and that they have equal effects.

QTL analysis of APR detected significant and consistent putative loci on chromosomes 1B, 4B and 5A in all three years, which on this basis were considered as the most important in conferring the observed APR to leaf rust. These three loci had significant additive effects and consistently contributed to lowering leaf rust response over years. The QTL on chromosome 1B was mapped to the short arm, which segregated for the 1BL/1RS translocation. Because Beaver carries the 1BS/1RS translocation, it was not possible to map the gene to a location within this arm as no recombination takes place between the 1BS and 1RS segments. Four genes conferring resistance to *Pt*, *Lr26* (Mettin et al. 1973), *Lr33* (Dyck 1987), *Lr44* (Dyck and Sykes 1994) and *Lr46* (Singh et al. 1998), have been located on chromosome 1B. In our study, *Lr26* was ineffective in the field and is therefore unlikely to be the contributor towards the 1B QTL. Gene *Lr33*, located on the long arm of chromosome 1B with $3.1 \pm 1.2$ crossover units from the centromere (Dyck 1987), is not effective in Australia and all Australian pathotypes are considered virulent on this gene at the seedling stage, but very minor levels of resistance can be detected in adult plants (McIntosh et al. 1995). Although, the chromosomal arm location of the 1B QTL detected in this study and that reported for *Lr33* differ, it is possible that the 1B QTL may still be an effect of *Lr33* and the difference in location in the two studies could be because of its recombination within the centromeric region. Gene *Lr44*, derived from an accession of *T. aestivum* spelta (Dyck and Sykes 1994), is also reported to be linked to *Lr33* with a recombination value of $4 \pm 1.0$ cM (Dyck and Sykes 1994) and can give lower levels of protection at seedling and adult plant growth stages under Australian field conditions (McIntosh et al. 1995). Hence, the presence of *Lr44* also cannot be discounted. The only catalogued APR gene with partial effects on chromosome 1B is *Lr46* (Singh et al. 1998), located on the distal end of the long arm, a location distant from the present results. To confirm whether the 1B located QTL is attributable to *Lr46*, a closely linked marker csLV46 developed by Lagudah et al. (unpublished) was applied to both parents (data not shown). Beaver showed the presence of the *Lr46* allele, consistent with the 1B QTL being contributed by *Lr46*. It is possible that the number of markers mapped in the target region is either low, resulting in an erroneous location or, less likely, that the 1B QTL involved is different from *Lr46*. In the field, Beaver also showed high levels of leaf tip necrosis (*Ltn*), a morphological trait associated with gene *Lr34/Yr18* (Dyck 1991; Singh 1992) on 7DS, indicating the possibility of the presence of *Lr34*, although no QTL was detected on chromosome 7D. When Beaver was tested with marker csLV34, closely linked to *Lr34* (Lagudah et al. 2006), it was negative, indicating that gene *Lr34* is most probably absent in Beaver. Nevertheless, the *Ltn* phenotype was also reported as closely linked with the *Lr46/Yr29* locus and controlled by second leaf tip necrosis gene, *Ltn2* different from *Ltn1*, linked with *Lr34/Yr18* (Rosewarne et al. 2006). Many other European wheats like Mec, Pegaso, Rialto, which show high levels of APR to leaf rust (Singh et al. 2001; Pathan and Park 2006) and *Ltn*, also amplified *Lr46* marker indicating the likely frequent occurrence of *Lr46* in European winter wheats (data not presented).

Depending upon the year of testing, the 4B QTL was located on the short arm with the closest marker being either *wPt1708* or *wPt5334* located at around 28–29 cM, in comparison to the consensus map of Somers et al. (2004). It is possible that this QTL is the same as a QTL detected in a Pavon 76/Avocet S RIL population, considered to be contributed by Avocet S and located 31 cM from the closest marker *Xgwm368* (William et al. 2006). Bansal et al. (2008) mapped the hypersensitive APR gene *Lr49* also on 4BL at approximately 12 cM from the 4B QTL identified by William et al. (2006) but it is presumed that the QTL detected in this study is less likely because of *Lr49* based on its mapped location.

The 5AS located QTL contributed by Beaver is unique as no known leaf rust resistance has been catalogued on chromosome 5A, and is therefore a candidate locus for uncharacterized and new APR to leaf rust. This QTL is temporarily designated as *QTLBvr5AS*, and mapped at 11 cM (marker *wPt1931*) and 16 cM (*wPt8756)* from the centromere relative to the consensus map of Somers et al. (2004). An association analysis of historical bread wheat germplasm from CIMMYT (Crossa et al. 2007) is the only report that has identified chromosome 5A as contributing to leaf rust resistance. The remaining QTLs on chromosomes 1A, 3B, 3D and 4A were either inconsistent or non-significant and remain unexplained.

On the basis of the results presented here, it can be suggested that APR to leaf rust in Beaver is complex, conditioned by several QTLs with small but different genotypic effects and with a major contribution of chromosomes 1B (1B/1R), 4B and 5A. Based on an association of closely linked markers with phenotypic data, putative single gene stocks were identified for each QTL and crossing was initiated to develop populations segregating for each to permit fine mapping of the identified regions. Future work will focus on developing closely linked markers for the resistance loci identified using these putative gene stocks, and the utilization of these markers to pyramid the genes with other resistance genes to diversify resistance to leaf rust.

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