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# QTL Mapping for Adult-Plant Leaf Rust Resistance Genes in Chinese Wheat Cultivar Weimai 8

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**Abstract**

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The wheat cultivar Weimai 8 is a good source of adult-plant resistance to leaf rust in China. In order to understand the genetic background of the resistance genes, 179 $F_{2:3}$ families derived from the cross Weimai 8 and Zhengzhou 5389 (susceptible to leaf rust) were used to construct a linkage map. Results showed that there was a major QTL on chromosome 2AS, temporarily named *QLr.hbau-2AS,* responsible for the resistance. *QLr.hbau-2AS* from the resistant parent Weimai 8 was between the SSR markers *Xcfd36* and *Xbarc1138*, with an interval length of 2.58 cM. In the 2010–2011, 2011–2012 and 2012–2013 crop seasons, it explained 25.79, 71.55 and 60.72% of the phenotypic variation, respectively. The location of *QLr.hbau-2AS* was close to the leaf rust resistance gene *Lr37*. However, they may not be the same since *QLr.hbau-2AS* has a different virulence test response from *Lr37*. This study identified two closely linked flanking molecular markers *QLr.hbau-2AS* and these molecular markers will help marker-assisted selection in breeding resistant cultivars in the future.

**Keywords**: gene postulation; molecular marker; QTL loci; wheat leaf rust

Leaf rust caused by *Puccinia triticina* Eriks. is an airborne foliar fungal disease of wheat that can seriously disrupt plant photosynthesis and ultimately result in a significant loss of production. Leaf rust occurs in wheat growing areas all over the world. In China, the southwest and the northwest are the two most vulnerable regions where it causes serious yield losses (Huerta-Espion *et al.* 2011). Selection and deployment of resistant cultivars is the most economic and effective measure used for the wheat leaf rust control. Study of resistance genes in wheat cultivars is a prerequisite for disease resistance breeding and crop improvement. Furthermore, exploring disease resistance genes in a wheat cultivar is of great significance to understand genetic diversity.

There are two types of resistance to leaf rust in wheat; one is known as race-specific resistance which is conferred by a single gene. This resistance is very unstable, often lost due to a change in *P. triticina* pathotype. The other is known as non-race-specific resistance, usually expressed during adult-plant stage and is also called adult-plant resistance (APR) or slow rusting. This resistance shows characteristics of quantitative character unlike race-specific resistance and is also known as "field resistance" (Ribeiro *et al.* 2001). These genes controlling quantitative characters are called quantitative trait loci (QTL). Cultivars with slow rusting non-hypersensitive APR show slow development of rust symptoms and the ultimate disease level is low. It does not lead to sig-

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nificant losses, or losses that are much lower than in the susceptible controls. Slow rusting resistance reduces selection pressure greatly in *P. triticina* pathotypes and the pathogen population remains stable. Under such circumstances, both the host and the pathogen can coexist for a long time. Resistance in cultivars with slow rusting is more durable than the race-specific resistance. Epidemiologically, slow rusting resistance has a lower infection rate, long incubation period and small size of uredinia and lower numbers of spores/uredinia (Caldwell 1968). Genetic studies have shown that slow rusting resistance is a quantitative and durable character and is controlled by several genes (Johnson & Law 1973; Bjarko & Line 1988; Das *et al.* 1992).

Currently, *Lr34* (Dyck 1977), *Lr46* (Singh *et al.* 1998), *Lr67* (McIntosh *et al.* 2011) and *Lr68* (Herrera-Foessel *et al.* 2012) are the only known genes of slow rusting resistance to leaf rust and their field performance is good. *Lr34* gene was identified in a Canadian cultivar PI58548 (Dyck 1977) and was located on chromosome 7D (Dyck 1987). Further research has shown that this locus also carries several genes including yellow rust resistance gene *Yr18* and powdery mildew resistance gene *Pm38* (Schnurbusch *et al.* 2004; Lillemo *et al.* 2008). This gene was cloned in 2009 (Krattinger *et al.* 2009). Singh *et al.* (1998) found *Lr46* on chromosome 1B. William *et al.* (2003) found and reported an AFLP marker *Pst*AAg*Mse*CTA-1 linked with *Lr46* and the precise position of *Lr46* was at the end of the chromosome 1B long arm. It was tightly linked with yellow rust slow rusting resistance gene *Yr29*. *Lr46* is widespread in Chinese wheat cultivars with different genetic backgrounds. The leaf rust slow-rusting resistance gene *Lr67* has a tight linkage with the yellow rust resistance gene *Yr46* (Herrera-Foessel *et al.* 2011). The recently discovered leaf rust slow-rusting resistance gene *Lr68* was located on the long arm of chromosome 7D. Compared to the number of qualitative traits of resistance genes, the number of leaf rust resistance QTLs is limited. Considering the important role in disease resistance breeding and broad application prospect, it is necessary to develop more effective QTLs and associated molecular markers.

The Chinese wheat cultivar Weimai 8 was developed by Weifang Agriculture Academy of Science (Shandong, China) by crossing 88-3149 (female parent) and Aus621108 (male parent). There are a number of valuable traits in Weimai 8 including strong tillering potential, cold resistance, compact plant type, high yield, resistance to powdery mildew and leaf rust (Chen & Zhang 2005). Weimai 8 is susceptible to most leaf rusts at the seedling stage, but at the adult plant stage it shows slow rusting resistance. This paper reports QTL analysis and molecular mapping of leaf rust APR genes in Weimai 8 and provides a theoretical basis and molecular markers for leaf rust resistance in wheat.

## MATERIAL AND METHODS

**Plant materials and *P. triticina* pathotypes**. The resistant (Weimai 8) and susceptible (Zhengzhou 5389) parents along with their 179 $F_{2:3}$ families were used for QTL mapping of leaf rust APR. There are seven Chinese *P. triticina* pathotypes used in the study. THTS, THTN, THTH and PHJN were used for seedling tests. THTT, THTS and THTQ were used for leaf rust APR tests. Leaf rust pathotypes were obtained from the Biological Control Center for Plant Diseases and Plant Pests of Hebei, Agricultural University of Hebei, China, which were named using the Prt-coding System (Long & Kolmer 1989).

**Seedling tests in the greenhouse**. Weimai 8 and TcLr37 were tested with 4 Chinese *P. triticina* pathotypes (Table 1). TcLr37 contains only one leaf rust resistance gene that is *Lr37*. Seedlings were grown in a growth chamber. When the first leaf was fully expanded, inoculations were performed by brushing urediniospores from the fully infected susceptible genotype Zhengzhou 5389 onto the new seedlings. Inoculated seedlings were placed in plastic-covered cages and incubated at 15°C and 100% relative humidity (RH) for 24 h in darkness. They were then transferred to a growth chamber programmed with 12 h light/12 h darkness at 18–22°C and 70% RH. Infection types (IT) were scored 10–14 days after inoculation according to the Stakman scale as modified by Roelfs *et al.* (1992). Plants with IT 0–2+ were considered to be resistant and those with IT 3–4 were susceptible.

**Adult plant field trials**. Both resistant and susceptible parents along with their 179 $F_{2:3}$ families

Table 1. Seedling infection types of Weimai8 and TcLr37 leaf rust resistance to 4 pathotypes of *Puccinia triticina*

| *Lr* gene or cultivar | Pathotype | | | |
|---|---|---|---|---|
| | THTS | THTN | THTH | PHJN |
| *Lr37* | 2+ | 3 | 4 | 4 |
| Weimai 8 | 4 | 3+ | 4 | 4 |

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were tested for leaf rust resistance in field nurseries at Baoding, Hebei Agricultural University during the 2010–2011, 2011–2012 and 2012–2013 cropping seasons. Field trials were conducted in randomized complete blocks with three replications. Each plot consisted of a single 1.5 m row with 30 cm row to row distance. About 100 seeds were sown in each row. Every tenth row was occupied by the highly susceptible line Zhengzhou 5389 to serve as a control and to facilitate rust development and spread of the spores within the experiment. Additional rows of Zhengzhou 5389 were planted crosswise and adjacent to the test rows. Equal amounts of *P. triticina* pathotypes THTT, THTS and THTQ were added with a few drops of Tween 20 (0.03%) and sprayed on spreader rows at the jointing growth stage (GS 32 according to Zadoks) at dusk using a sprinkling can. The average temperature in the middle of April, Baoding, was about 15°C during the day and 10°C during the night. Following the inoculation, plants were immediately covered with plastic films aiming to trap moisture within plant canopies and the films were removed in the next morning. The experimental field was kept moist by irrigation after inoculation.

**Leaf rust assessment**. Assessment of leaf rust was initiated when susceptible control Zhengzhou 5389 had more than 50% severity (the rust incidence area accounted for more than half of the whole leaf) and disease scoring was carried out once a week until the disease severity of Zhengzhou 5389 reached 100% (Peterson *et al.* 1948). Assessment of the final phenotypic disease severity (FDS) data was used for QTL analysis.

**DNA extraction and molecular marker screening**. Genomic DNA was extracted from the seedlings of the $F_{2:3}$ family using the CTAB protocol (Sharp *et al.* 1988). The DNA was quantified with a UV spectrophotometer, and diluted to a final concentration of 30 ng/µl. We used 3 replicates per year, and counted the average of the FDS replicates. Five families with the lowest FDS and five families with the highest FDS were selected. Both parental lines and these ten families were used for preliminary screening of molecular markers (Michelmore *et al.* 1991).

Using 1240 pairs of SSR markers and two pairs of scar markers distributed across 21 wheat chromosomes, we screened polymorphism among the two parents and ten families. The polymorphic molecular markers were further validated by 179 $F_{2:3}$ families.

Polymerase chain reactions (PCR) were performed in volumes of 10 µl with 1.0 U *Taq* of DNA polymerase (Zexing Biotechnology Co. Ltd, Beijing, China); 1× PCR buffer (25mM KCl, 5mM Tris-HCl, 0.75mM $MgCl_2$, pH 8.3); 100µM each of dNTP, 3 pmol of each primer and 30 ng of template DNA. The conditions of PCR were denaturation at 94°C for 5 min, followed by 35 cycles of 94°C for 1 min, 55–60°C (depending on the primer pair) for 1 min, 72°C for 1 min, and a final extension at 72°C for 10 min. The PCR product was mixed with 2 µl of formamide loading buffer (98% formamide, 10mM EDTA, 0.25% bromo-phenol blue, 0.25% xylene cynol, pH 8.0). The mixture was then loaded on 10% non-denaturing polyacrylamide gels or agarose gel for electrophoresis.

**Linkage map construction and QTL analysis**. The linkage map of detected QTLs was constructed by using polymorphic marker genotyped 179 $F_{2:3}$ families and the FDS of field trials in the Map Manager QTXb20. The software QTL Icimapping 3.2 was deployed to detect the QTL loci (Li *et al.* 2007) using composite interval mapping and the logarithm of odds (LOD) threshold of 2.5.

## RESULTS

**Seedling reactions**. In seedling tests with four *P. triticina* pathotypes (Table 1), Weimai 8 was susceptible to all the *P. triticina* pathotypes, which proved that the seedling resistance of Weimai 8 is not good. TcLr37 was resistant to THTS in this test. The seedling reactions to THTS of Weimai 8 and TcLr37 were different, so the leaf rust resistance gene in Weimai 8 may not be *Lr37*.

**Phenotypic evaluation**. The FDS of the susceptible control Zhengzhou 5389 was 100%; the FDS of Weimai 8 was 10%. Statistics of the three-year field testing results indicated that data was continuously distributed and supported quantitative inheritance (Figure 1). It can be inferred that Weimai 8 may carry some QTL for leaf rust resistance. The level of the disease in 2011–2012 was more serious than in 2010–2011 and 2012–2013, but overall incidence trends were consistent. The correlation coefficient of the 2010–2011 and 2011–2012 FDS of 179 $F_{2:3}$ families was 67.62%, 2010–2011 and 2012–2013 it was 66.04%, 2011–2012 and 2012–2013 it was 86.36%, indicating that there is a low genotype-by-year interaction in Weimai 8.

**QTL mapping**. Overall, 13 molecular markers were found to be polymorphic by screening between the 10 resistant lines and 10 susceptible lines. They are *Xbarc124*, *Xwmc382*, *Xwmc407*, *Xbarc212*, *Xgwm512*, *Xgwm 614*, *Xbarc1114*, *Xgwm210*, *Xcfd36*, *Xgwm400*,

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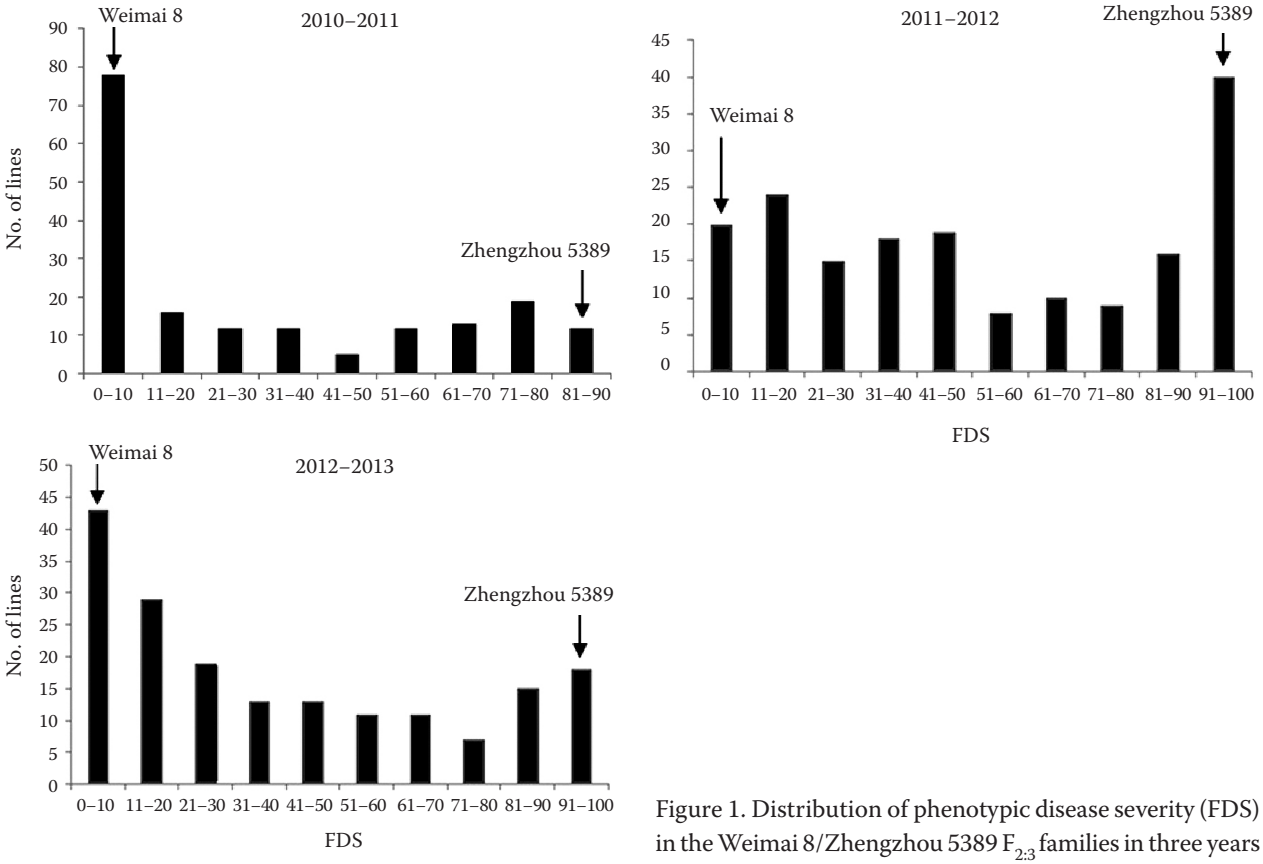


Figure 1. Distribution of phenotypic disease severity (FDS) in the Weimai 8/Zhengzhou 5389 $F_{2:3}$ families in three years

*Xbarc1138*, *sc372*, and *sc385*. These molecular markers are all located on chromosome 2A. Using the Map Manager QTXb20 and QTL Icimapping 3.2 analysis, we found that with 13 molecular markers in one linkage group, the length was 20.5 cM (Figure 2). There was a wheat leaf rust resistance QTL locus, located on chromosome 2AS, temporarily named *QLr.hbau-2AS*. The QTL may be between the two flanking markers *Xbarc1138* (Figure 3) and *Xcfd36* (Figure 4) with an interval length of 2.5 cM. In 2010–2011, the site explained 25.79% of the phenotypic variation; in 2011–2012, the site explained 71.55% of the phenotypic variation; and in 2012–2013, the site explained 60.72% of the phenotypic variation (Table 2). Another QTL was detected in the 2010–2011 crop season in addition to *QLr.hbau-2AS* with a genetic distance of 2 cM. Based on the position alone, the two QTLs are probably the same. However, the estimates shown in Table 2 indicate otherwise. In all three environments, the QTL in the *Xbarc1138-Xcfd36* interval has a strong negative additive effect and a much smaller negative dominance effect. In contrast, the second QTL that was detected in 2010–2011 has a weak negative additive effect and a larger positive dominance effect. If in fact the plants were in the $F_{2:3}$ generation

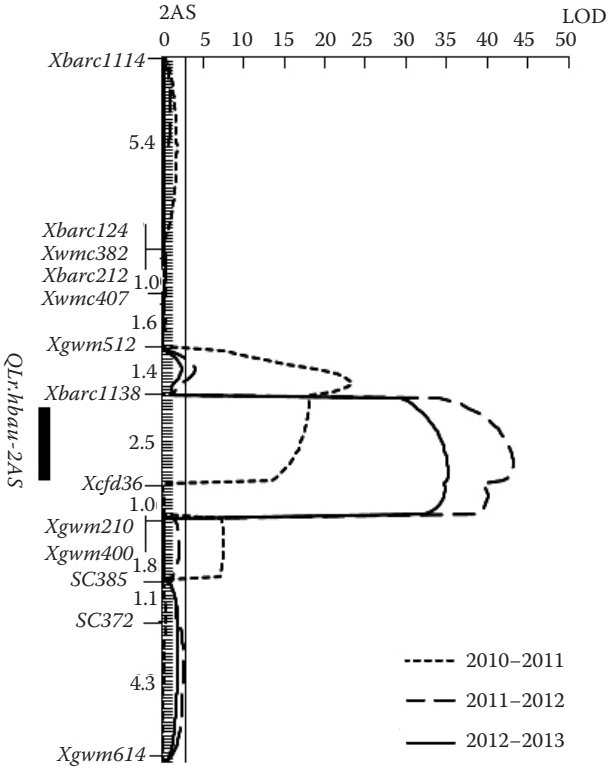


Figure 2. The positions of the quantitative trait loci (QTLs) conferring adult-plant resistance to leaf rust on 2AS

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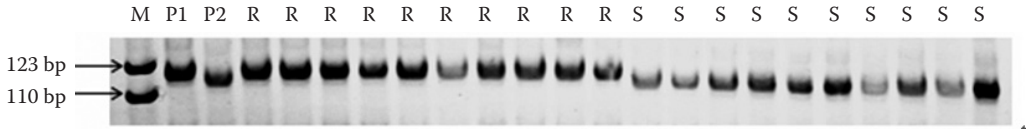


Figure 3. The specific PCR fragments amplified of parents and $F_{2:3}$ families with SSR marker *Xbarc1138*
M – PBR322 marker; P1 – the resistant parent Weimai 8; P2 – the susceptible parent Zhengzhou 5389; R – the resistant plants in $F_{2:3}$ families; S – the susceptible plants in $F_{2:3}$ families

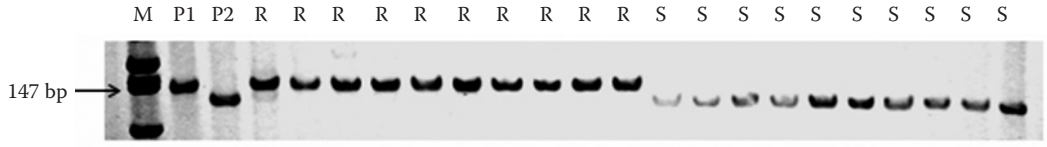


Figure 4. The specific PCR fragments amplified of parents and $F_{2:3}$ families with SSR marker *Xcfd36*
M – PBR322 marker; P1 – the resistant parent Weimai 8; P2 – the susceptible parent Zhengzhou 5389; R – the resistant plants in $F_{2:3}$ families; S – the susceptible plants in $F_{2:3}$ families

Table 2. QTL for phenotypic disease severity (FDS) based on the composite interval mapping analysis

| Years | Chromosome | Left marker | Right marker | LOD score | Additive | Dominate | Var. (%) |
|---|---|---|---|---|---|---|---|
| 2010–2011 | 2A | *barc1138* | *cfd36* | 18.1051 | –21.4503 | –2.5875 | 25.7901 |
| 2010–2011 | 2A | *wms400* | *SC385* | 7.4921 | –10.5552 | 26.8303 | 9.4510 |
| 2011–2012 | 2A | *barc1138* | *cfd36* | 43.1124 | –37.9774 | –6.4349 | 71.5499 |
| 2012–2013 | 2A | *barc1138* | *cfd36* | 35.1736 | –34.8843 | –7.6860 | 60.7152 |

LOD – logarithm of odds

in 2010–2011 but were in later generations in the other years, this would explain why this QTL was detected only in the first year because the families became more and more homozygous leading to the dominance effect becoming no longer detectable.

## DISCUSSION

QTL Icimapping 3.2 analysis showed that the LOD score was very high in three years FDS, so the probability of existing *QLr.hbau-2AS* was very high. The correlation coefficient of FDS in three-year field study was also very high, and *QLr.hbau-2AS* explained 25.7%, 72% and 61% of the phenotypic variance in three years respectively and proved that the leaf rust resistance of QTL *QLr.hbau-2AS* was less affected by the environment and is relatively stable. The relatively low percentage explained by the QTL in 2010–2011 was probably due to the fact that scores taken from $F_2$ plants were not highly accurate.

At present only one designated gene *Lr37* is located on chromosome 2AS (Bariana & McIntosh 1993). The resistance of *Lr37* is very good. It is known that *Lr37* was from chromosome 2N of *Aegilops ventricosa* which was transferred to the wheat genome, and the 2NS/2AS translocation cultivar VPM1 was bred (Maia 1967). Studies at Sydney University found that VPM1 harbours three rust resistance genes: the leaf rust resistance gene *Lr37*, stem rust resistance gene *Sr38* and yellow rust resistance gene *Yr17*. These three genes are closely linked to the wheat chromosome 2AS (Bariana & McIntosh 1993) and have been used all over the world (McIntosh *et al.* 1995). Helguera *et al.* (2003), using RFLP marker *cmwg682*, positioned *Lr37-Yr17-Sr38* linkage group at 10 cM of chromosome 2AS from the telomere or centromere 2AS. According to the bread wheat high-density SSR marker map by Somers *et al.* (2004), *QLr.hbau-2AS* was positioned at the end of the short arm of chromosome 2A, close to the location of *Lr37*. Although the pedigree of Weimai 8 was not clear, according to the race test results, *QLr.hbau-2AS* may not be the same as *Lr37*.

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