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Quantitative trait loci for resistance against Yellow rust in two wheat-derived recombinant inbred line populations

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Abstract Yellow rust, which is a major disease in areas where cool temperatures prevail, can strongly influence grain yield. To control this disease, breeders have extensively used major specific resistance genes. Unfortunately this kind of resistance is rapidly lost due to pathogen adaptation. More-durable resistance against yellow rust can be achieved using quantitative resistance derived from cultivars with well-established durable resistance. The winter wheat Camp Remy has maintained a high level of resistance for over 20 years. In order to map quantitative trait loci (QTLs) for durable yellow rust resistance, we analysed a set of 98 F_8 recombinant inbred (RI) lines derived from the cross Camp Remy×Michigan Amber. We also mapped QTLs for adult resistance to yellow rust using the International Triticae Mapping Initiative RI population (114 lines derived from the cross Opata85×synthetic hexaploid). Two and five QTLs, respectively, were identified from these two populations. This work has highlighted the importance of the centromeric region of chromosome 2B and the telomeric regions of chromosomes 2AL and 7DS in durable yellow rust resistance. The same chromosomal regions are also implicated in resistance to other pathogens.

Keywords QTL mapping · Yellow rust · Durable resistance · Wheat

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Introduction

Diseases caused by the three rust fungi are a major limitation for wheat production worldwide. Yellow rust (*Puccinia striiformis* f.sp. *tritici*) is the most commonly occurring rust in temperate growing areas. Since this rust can affect the early stages of plant development, major infection can result in stunted and weakened plants, leading to yield losses as high as 50%, due to shrivelled grain and damaged tillers (Roelfs et al. 1992).

Developing resistant varieties is the most-efficient and environmentally sustainable means of reducing losses due to disease. Two kinds of rust resistance can be distinguished. The first, specific resistance (generally under mono- or oligo-genic control, and usually effective at the juvenile stage) is rapidly overcome in the field. The second, non-specific, adult or quantitative resistance is mostly polygenically determined, and is effective at the adult stage. This latter resistance is generally considered to be durable. The use of quantitative resistance in breeding programmes would be greatly facilitated by marker-assisted selection. With the advent of extensive genetic maps and the possibility of establishing the location of quantitative trait loci (QTLs) on these maps, the linkage of molecular markers to QTLs for quantitative disease resistance can be anticipated.

A number of RFLP-based wheat maps, generated from the International Triticae Mapping Initiative (ITMI) reference population Opata85 X W-7984, has been published (Nelson et al. 1995a, b, c; Van Deynze et al. 1995; Marino et al. 1996; Mingeot and Jacquemin 1999). More recently a microsatellite-based map has also been published (Röder et al. 1998). Genetic studies of certain varieties with a history of durable adult plant resistance have identified the importance of the 5BS-7BS translocation (Johnson and Law 1975; Law and Worland 1996, 1997), chromosome arms 7DS and 7BS (Pink and Law 1985), chromosome arms 5AS and 5DS (Pink et al. 1983) and chromosome 2D (Worland and Law 1986) in determining adult plant resistance to yellow rust.

Few studies have so far focused on QTL mapping for resistance against pathogens in wheat. These include analyses of resistance against leaf rust (Nelson et al. 1997; Messmer et al. 2000), powdery mildew (Keller et al. 1999), Karnal bunt (Nelson et al. 1998) and Fusarium head blight (Waldron et al. 1999). Major genes and QTLs for resistance to yellow rust have been identified in a number of recent studies (Chague et al. 1999; Börner et al. 2000; Peng et al. 2000; Singh et al. 2000).

Opata 85 is partially resistant to yellow rust and carries adult resistance (Broers 1997). Camp Remy, a winter wheat cultivar, has maintained a high level of resistance throughout its cultivation over more than 20 years, and so its resistance is considered to be durable. In the present study we have characterised and located QTLs for resistance to yellow rust in the ITMI reference population, as well as in a population derived from the cross between the two commercial varieties, Camp Remy×Michigan Amber.

Materials and methods

Plant material

Reference population

The ITMI reference population consists of 114 RI lines developed from a cross between the hard red spring wheat cultivar "Opata 85" and a synthetic hexaploid wheat, W-7984, as described by Nelson et al. (1995a). The synthetic was produced from the cross between durum wheat (*Triticum turgidum* L.) cultivar "Altar 84" and *Triticum tauschii* CIMMYT accession W-219.

Gbx7 population

Ninety eight RI lines were derived from the cross Camp Remy (resistant parent)×Michigan Amber (susceptible parent), by advancing random individual F_2 plants to the F_8 generation by single-seed descent.

Disease screening

The RI lines were planted as hillplots in Gembloux (Belgium) in 1999 and 2000. Each population was sown in one replication in 1999 and two in 2000. The susceptible spreader cultivar Michigan Amber was planted in rows surrounding the RI line plots. In addition, in 2000, the RI lines of the Gbx7 population were planted in plots consisting of six 1.5 m rows planted 25 cm apart with 60 cm between plots. To initiate the disease epidemic, spreader plants were inoculated with spores collected during the previous year from naturally occurring yellow rust at Gembloux. The disease incidence (rust intensity: percentage of leaf area infected, based on the modified Cobb's scale, Peterson et al. 1948) was recorded twice at 10-day intervals beginning at growth stage 10.1 (Feekes scale, Large 1954). The rust intensity of each RI line is an average of the symptoms affecting the upper three leaves from five different plants. The rust intensity data were used to calculate the Area Under the Disease Progress Curve (AUDPC) for each RI line using the formula:

$$Y = [(Xi + Xi + 1)/2] (ti + 1 – ti),$$

where Y=AUDPC, Xi=the rust intensity recorded on the first date, Xi+1=the rust intensity recorded on the second date, and (ti+1–ti)= the number of days between the first date and the second date.

The infection types (IT) were scored using a 0 to 9 scale (McNeal et al. 1971) at growth stage 11.1 (Feekes scale, Large 1954).

Pathogenicity tests

Seedlings were inoculated at the 2-leaf stage with lyophilised *P. striiformis* uredospores dispersed in talc. The spores had been collected from naturally occurring yellow rust at Gembloux (Belgium). After 24-h incubation at 14°C in the dark at 100% humidity, the seedlings were held in a controlled environment cabinet, maintained at 14°C during an 8-h dark period, and at 20°C during a 16-h light period. IT scoring, based on the McNeal scale, was performed 15 days post-inoculation. Seven or eight seedlings were used per test.

RFLP analysis

Genomic DNA was extracted following Sharp et al. (1988). We used cDNA and gDNA probes provided by various laboratories: BCD, CDO and WG probes are described in Heun et al. (1991), ABC probes in Kleinhofs et al. (1993), PSR probes in Chao et al. (1989), and GBX probes in Mingeot and Jacquemin (1999). Southern blotting and hybridization were performed as described in Mingeot and Jacquemin (1999).

Microsatellite analysis

PCRs were performed as described (Röder et al. 1998), using publicly available GWM primer pairs. Fragment analysis was carried out on an automated laser fluorescence (ALF) sequencer. Denaturing gels (0.5 mm thick) were made up with 6% polyacrylamide using Readysol DNA/page (Pharmacia). The gels were run in 0.6×TBE buffer at settings of 900 V, 50 mA, and 35 W with 2-mW laser power and a sampling interval of 1 s. The gels were re-used three to four times. Standard size markers were included in each lane. Fragment sizes were calculated using the computer program AlleleLinks (Pharmacia Biotech) by interpolation with the size standards.

Map construction

For the ITMI population, the published genetic map (Nelson et al. 1995a, b, c; Van Deynze et al. 1995; Marino et al. 1996) now consists of more than 1,000 genetic markers. The map has been enhanced by the addition of 279 microsatellite-derived (Röder et al. 1998) and 160 RFLP-derived (Mingeot and Jacquemin 1999) loci. Marker segregation data are available from the graingenes database (http://wheat.pw.usda.gov.). For the Gbx7 population, microsatellite and RFLP segregation data were analysed using MAPMAKER version 3.0 (Lander et al. 1987). Linkage groups were determined with a lodscore of 3 as a threshold. Two-point, three-point and multi-point analysis were used in order to determine the best order of marker loci within the linkage groups. Loci whose location was ambiguous were placed in the interval in which they were best fitted using the "try" command. The Kosambi function (Kosambi 1944) was used to calculate centimorgan distances. The chromosomal assignment of linkage groups and the approximate position of centromeres were deduced from published wheat maps (Nelson et al. 1995a, b, c; Marino et al. 1996; Röder et al. 1998; Mingeot and Jacquemin 1999).

QTL analysis

Quantitative resistance to yellow rust as measured by AUDPC and IT was analysed with marker data by interval regression mapping using MAPMAKER-QTL (Lander and Botstein 1989). A LOD threshold of 3 is required to detect QTLs with a significance level of 0.05 (Lander and Boststein 1989).

Results

Characterisation of the yellow rust population

Camp Remy, the resistant parent of the Gbx7 population, is known to carry the race-specific resistance genes *Yr3a/Yr4a/Yr7*, along with at least one more resistance, which could be *Yr6*, *Yr8* or *Yr9* (De Vallavieille-Pope et al. 1990). The spreader cultivar (Michigan Amber) was inoculated with uredospores from naturally occurring yellow rust. In order to ensure that these specific resistance genes did not interfere with the quantitative non-specific resistance, we characterised the virulence spectrum of the inoculum. To achieve this, a set of 21 European and world differential varieties were inoculated at the seedling stage. The results of this pathogenicity test (Table 1) indicated that all the specific resistance genes present, or probably present in Camp Remy (except for *Yr8*), are matched. Compair, which was resistant in this test, carries *Yr19* in addition to *Yr8* (Chen et al. 1995). Thus in the pathogen population used in this work there is no pathotype to match the combination *Yr8/Yr19*, but we cannot exclude the existence of a pathotype matching *Yr8* alone.

Table 1 IT scoring of wheat differential cultivars infected at the seedling stage with a naturally occurring population of yellow rust at Gembloux (Belgium)

Cultivars	Resistance genes: *Yr*	IT (McNeal scale)
Nord Desprez	3a, 4a	9
Anza	A	8
Clement	9, 2	8
Heines VII	2, +	8
Hybrid 46	3b, 4b	8
Kalyansona	2	8
Strubes Dickkopf	SD	8
Suwon x Omar	Su	8
Vpm1	17	8
Austerlitz	6, +	7
Chinese 166	1	7
Federation x Kavkav	9	7
Heines Kolben	6, 2	7
Reichersberg 42	7, +	7
Vilmorin 23	3a, 4a	7
Heines Peko	6, 2, +	6
Lee	7, +	6
Spaldings Prolific	Spa	4
Carstens V	CV	1
Compair	8, 19	0
Moro	10, +	0

Detection and localisation of QTLs

ITMI population

For QTL localisation, we first used evenly spaced loci (according to Nelson et al. 1995a, b, c; Van Deynze et al. 1995; Marino et al. 1996). For this population, only IT data were employed to search for QTLs. A LOD threshold of 3 is required for declaring QTL linkage to a locus (significant linkage), but reporting suggestive linkage (LOD score greater than 2) is worthwhile for further confirmation (Lander and Kruglyak 1995).

We detected five QTLs with a LOD score greater than 2 (Table 2). The localisation of these QTLs is presented in Fig. 1. The first QTL (QYR3) on chromosome 2B is associated wih a highly significant LOD score (7.39), the second (QYR4) on chromosome 7D with a significant LOD score (3.36), and the three others QYR5, QYR6 and QYR7 on, respectively, chromosomes 5A, 3D and 6D, are suggestive only (LOD scores of, respectively, 2.80, 2.76 and 2.43). For QYR3, QYR4, QYR5 and QYR6, the allele for increasing resistance originates from the resistant parent Opata85. For QYR7, the allele for resistance was from the susceptible parent W-7984.

Gbx7 population

To evaluate disease resistance we used both AUDPC (based on disease intensity) and IT. Both traits generate quantitative non-normal distributions. Angular transformation failed to normalise either distribution, but a logarithmic transformation did normalise the AUDPC distribution. Therefore un-transformed IT data and log-transformed AUDPC data were used for analysis. AUDPC and IT were highly correlated (correlation coefficient 0.82 in 1999; 0.88 and 0.89 in 2000 for the first and second replication, respectively). Replicates within a year (correlation coefficient 0.91 and 0.94 for IT and AUDPC respectively) and between years (correlation coefficient 0.87 and 0.86 for IT; 0.73 and 0.76 for AUDPC) were

Table 2 QTLs for yellow rust resistance identified using IT data for the ITMI RI lines population. Length is given in cM using the Kosambi function. Position indicates the position of the highest LOD score for the respective QTL. LOD: is the highest LOD score for the respective QTL. %VE: is the percentage of explained total variance for the respective QTL. Effect: is the estimated additive effect of substituting one allele of Opata85 by one allele of W-7984

QTLs	Chromosome	Interval	Length	Position	LOD	% VE	Effect
QYR3	2B	Cdo405–bcd152	7.4	4	7.39	30.7	+3.12
QYR4	7D	Wg834–bcd1438	6.9	4	3.36	13.9	+2.09
QYR5	5 A	Fbb209–abg391	18.7	10	2.80	15.0	+2.16
QYR6	3D	Cdo407–ksuA6	5.7	4	2.76	11.7	+1.91
QYR7	6D	Bcd1510–ksuD27	21.8	12	2.43	13.1	–2.02

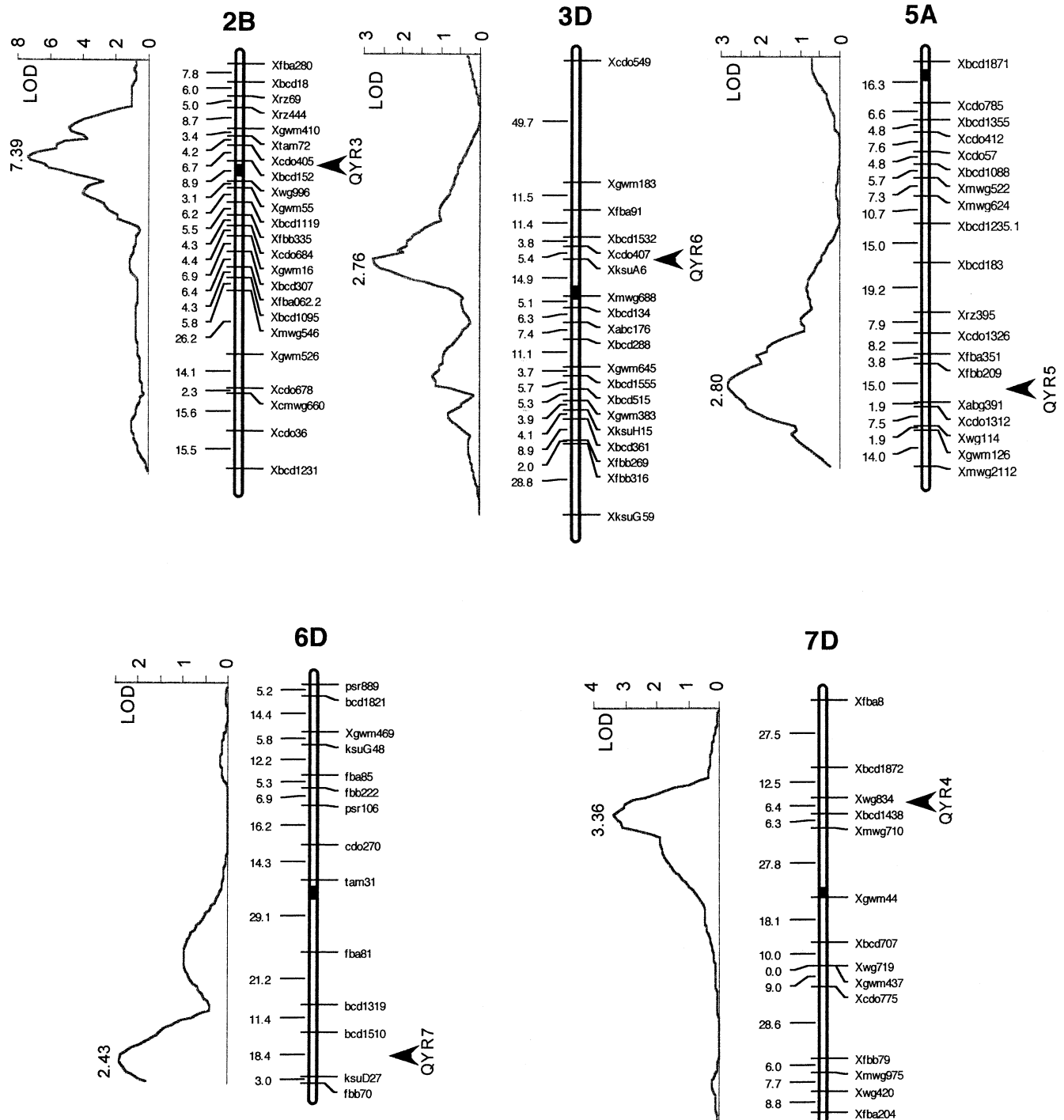

Fig. 1 QTL localization on the ITMI population-derived map. Only chromosomes where a QTL for yellow rust resistance is detected are shown. Chromosome short arm are toward the top of the map. *Arrows* indicate QTL positions; immediate markers from each *arrow side* are flanking markers. LOD score curves for interval mapping produced by Mapmaker are placed on the left of each chromosome. Maximum LOD scores are shown near peaks

also highly correlated. In the ANOVA analysis there was no significant effect of replication, so for each of the two data sets (IT and AUDPC) we have used the means obtained from the whole data set (all replicates and all years) to search for QTLs.

The Gbx7 map is still being developed. So far, segregation data for 147 loci have been obtained. These loci are distributed among 33 linkage groups. Thirty four loci (23%) are unlinked and, for these, we used ANOVA analysis to search for QTLs. For the 33 linkage groups,

Table 3 QTLs for yellow rust resistance identified using IT data or the AUDPC logarithm for the Gbx7 RI lines population. Length is given in cM using the Kosambi function. Position indicates the position of the highest LOD score for the respective QTL. LOD: is the highest LOD score for the respective QTL. %VE: is the percentage of explained total variance for the respective QTL. Effect: is the estimated additive effect of substituting one allele of Camp Remy by one allele of Michigan Amber

QTLs	Chromosome	Interval	Length	Position	LOD	%VE	Effect
*** IT ***							
QYR1	2B	Gwm47–Gwm501	3.5	2	12.01	46.0	+3.90
QYR2	2 A	Gwm356–Gwm382	23.9	2	2.99	15.4	+2.37
AUDPC logarithm							
QYR1	2B	Gwm47–Gwm501	3.5	2	11.84	45.8	+3.24
QYR2	2 A	Gwm356–Gwm382	23.9	6	1.70	10.7	+1.53

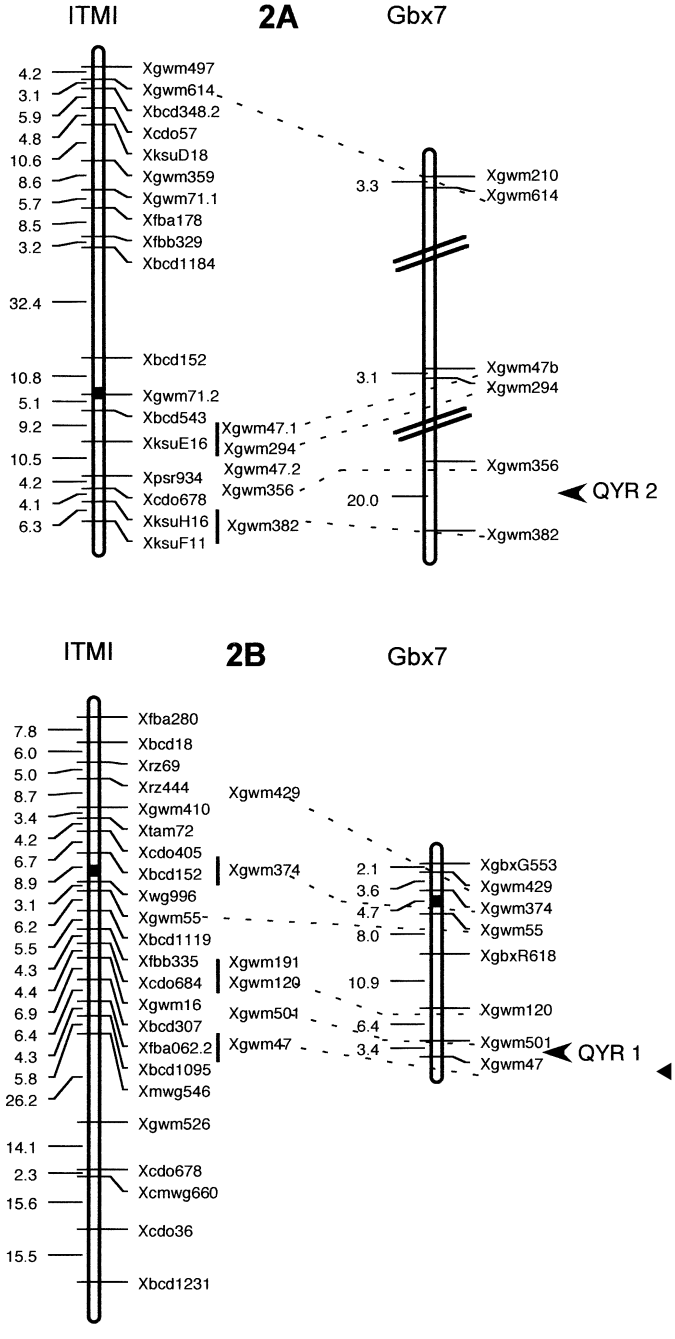

Fig. 2 QTL localization on the Gbx7 population derived-map. Alignment of the linkage map based on Gbx7 population (on the right) and the linkage map based on ITMI population (on the left) is shown; gwm-designated microsatellite markers are indicated (Röder et al. 1998). Concerning the ITMI linkage map, microsatellite loci mapped with a LOD>2.5 are integrated into the framework; the other microsatellites were placed in the most-probable interval (Röder et al. 1998). Only chromosomes where a QTL for yellow rust resistance is detected are shown. Chromosome short arms are toward the top of the map. *Arrows* indicate QTL positions; immediate markers from each arrow side are flanking markers

interval regression mapping using Mapmaker-QTL has been applied.

We detected two IT-QTLs that reach the LOD threshold of 3 (Table 3). The first (QYR1) is located on chromosome 2B and accounted for 46% of the total variance; the second (QYR2) is on chromosome 2 A and accounted for 15.4% of the total variance. In both cases, the QTL allele for increasing resistance was derived from the resistant parent Camp Remy. Using AUDPC we failed to reach the LOD threshold for QYR2 (LOD score=1.7), but QYR1 was detected with the same LOD score, and accounted for a similar proportion of the total variance as for IT (Table 3, Fig. 2).

Composite Interval Mapping (CIM) using QTL Cartographer (Basten et al. 1994, 1999) analysis gave comparable results to the Simple Interval Mapping (data not shown).

Discussion

In order to evaluate resistance levels we have used the two traits AUDPC and IT. These two kinds of resistance assessment gave comparable results, as in both cases we detected QTLs located in the same regions with approximately the same LOD score and the total proportion of variance explained. A similar high correlation between IT and AUDPC has also been observed in other studies (Chen and Line 1995; Ma and Singh 1996), and it has been postulated that the genes controlling IT have a strong effect on disease intensity (Chen and Line 1995).

Using two RI line populations, we have detected only a few QTLs involved in the quantitative resistance to

yellow rust. This is particularly clear in the case of the ITMI population, where only two QTLs with a significant LOD score were identified. Three others were identified, but only with suggestive LOD scores. For the other population we also identified only two QTLs, but in this case the genome scanning was only partial. This result is not unique to our work. Young (1996) has pointed out that it is common to find only three to five QTLs involved in quantitative resistance (frequently just one or two QTLs predominate), although there are examples of several (>10) QTLs involved in quantitative resistance.

QYR1 and QYR3 are located 37-cM apart on chromosome 2B. Given the errors involved in assigning QTLs by interval mapping, we cannot unambiguously declare them to be distinct loci. But if the confidence intervals (Lander and Botstein 1989) of QYR1 and QYR3 (respectively 6 and 8 cM) are considered, it seems likely that they are in fact distinct. QYR2 maps to a chromosome in homoeologous group 2 (2AL), which suggested the possibility that this QTL is homoeologous to the QTLs on 2B. However, upon transferring the QTL positions on the ITMI map, the genetic distance between the molecular markers nearest to QYR1 and QYR2 (respectively, *Xfba062.2* and *Xcdo678*) is 46 cM (Fig. 2, chromosome 2BL). Thus these two QTLs are probably not homoeologous.

Our results indicate that chromosome 2B carries important factors for resistance to yellow rust, since two major QTLs (QYR1 and QYR3) arising from two different resistance sources (Camp Remy and Opata85) are both localised there. QYR3 maps between the RFLP loci *Xcdo405* and *Xbcd152*. The former maps 4.2 cM from *Xtam72*, which has previously been identified as the most-closely linked RFLP locus to the gene *Lr23* which determines a race-specific resistance against leaf rust (Nelson et al. 1997). In addition, the *Per2–2B* (peroxidase) locus, which maps 10-cM distally to *Xtam72*, is linked to a QTL (also detected using the ITMI population) for leaf rust resistance (Faris et al. 1999).

QYR1 is located on the chromosome 2B segment between markers *gwm501* and *gwm47* (Fig. 2). From the reference map (Fig. 1), *gwm501* lies about 37 cM from *Xcdo405* and about 23 cM from the centromere on chromosome arm 2BL. A group of three race-specific resistance genes (for yellow rust *Yr7* and *Yr5* and for stem rust *Sr9*) has also been mapped on this chromosome arm, 2BL, about 15 cM from the centromere (Hart et al. 1993). De Vallavieille-Pope et al. (1990) reported that Camp Remy carries *Yr7*. Again we have a QTL (QYR1) coinciding with the location of a specific resistance gene (*Yr7*). This situation has been described in a number of different resistance systems (resistance to rice blast: Yu et al. 1991; resistance to potato late blight: Leonards-Schippers et al. 1994; resistance to leaf rust: Faris et al. 1999).

Chromosome 7D is also important for resistance to yellow rust as we detected a QTL on it (QYR4), mapping between *Xwg834* and *Xbcd1438*. Using the ITMI population, Nelson et al. (1997) localised on this same region of chromosome 7DS the adult plant non-specific resistance gene *Lr34* against leaf rust. The adult plant yellow rust resistance gene *Yr18* is closely linked to *Lr34* (McIntosh 1992; Singh 1992). Opata85 is known to carry *Yr18* (Singh 1992), thus QYR4 could be due to the effect of *Yr18*. *Lr34* is also closely linked to *Bdv1*, a gene encoding resistance to barley yellow dwarf virus (Singh 1993). Finally, *Lr34* enhances stem rust resistance due to its inactivation of a suppressor of resistance to stem rust (Kerber and Aung 1999). From all these observations, we conclude that this region of chromosome arm 7DS seems to be very important not only for yellow rust resistance but also for resistances to all the wheat rusts, and for resistance to barley dwarf virus as well. Whether all these resistance effects are due to close linkage between several different genes, or due to pleiotropy of one or maybe two genes remains to be demonstrated.

QYR2, which is only detected with IT data, is located on a chromosomal region (distal end of 2AL) known to be implicated in disease resistance. The powdery mildew race-specific resistance gene *Pm4* has been mapped to chromosome 2AL close to the loci *Xbcd1231* and *Xcdo678* (Ma et al. 1994). More recently, Waldron et al. (1999) localised a QTL for resistance to Fusarium head blight on chromosome 2AL close to *XksuH16*. In the present study, QYR2 has been mapped close to *gwm356* (Fig. 2). Röder et al. (1998) localised this locus in the same chromosomal region as *XksuH16* and *Xcdo678* (Fig. 2). Here again different resistance systems (powdery mildew resistance, Fusarium head blight resistance and yellow rust resistance) are controlled by genes located in the same chromosome region.

Li et al. (1999) mapped a set of Defence Response (DR) genes and demonstrated that the DR loci are not randomly distributed throughout the wheat genome, but rather are located in clusters and/or in the distal gene-rich regions of the chromosomes. They also observed that the homoeologous group-7 chromosomes possessed the most DR loci, followed by those in group 2. Faris et al. (1999) demonstrated that QTLs with large effects are located in regions of putative resistance (R) genes. These results also indicated that many minor resistance QTLs may arise from the action of DR genes. In this regard it is interesting to consider QYR5. *Mpc1*, a DR gene implicated in the flavonoid metabolic pathway maps to chromosome 5A between *Xfba351* and *Xcdo1312* (Fig. 1). This is exactly the location of QYR5 (between *Xfbb209* and *Xabg391*). Although identified only with a suggestive LOD score (2.80), this QTL therefore deserves more attention, as it appears to coincide with *Mpc1*.

Camp Remy is known to carry durable resistance to yellow rust. This cultivar was obtained from the cross Hardi×Atou, both of which are derived from the cultivar Cappelle-Desprez. Cappelle-Desprez is a well-known example of a cultivar with durable yellow rust resistance, since it has maintained an adequate level of resistance despite being widely used for many years. As described for other resistance systems [reviewed in Young (1996)],

durable resistance can be due to a combination of quantitative and qualitative resistance genes. This seems to be the case for Camp Remy, as it carries both race-specific resistance genes (De Vallavieille-Pope et al. 1990) and, as our results here indicate, it must also have at least two QTLs affecting non-specific resistance.

Conclusions

This research has highlighted the importance of the centromeric region of chromosome 2B and the telomeric region of chromosomes 2AL and 7DS in yellow rust resistance. These chromosomal regions are also implicated in other resistance systems. Durable resistance could result from a combination of quantitative and qualitative resistance genes and this seems to be the case in Camp Remy.

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