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ORIGINAL PAPER

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# Genetic analysis of durable resistance to yellow rust in bread wheat

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**Abstract** Yellow rust, caused by *Puccinia striiformis*, is one of the most damaging diseases affecting bread wheat in temperate regions. Although resistance to yellow rust is frequently overcome by new virulent races, a durable form of resistance in the French bread wheat Camp Rémy (CR) has remained effective since its introduction in 1980. We used 217 $F_7$ recombinant inbred lines (RILs) derived from the cross between CR and the susceptible cultivar Récital to identify and map quantitative trait loci (QTLs) involved in durable yellow rust resistance. Six significant QTLs that were stable over a 4-year period were detected. Two QTLs, denoted *QYr.inra-2DS* and *QYr.inra-5BL.2*, were located on the short arm of chromosome 2D and the long arm of chromosome 5B, respectively. Each explained on average 25–35% of the observed phenotypic variation and were probably inherited from Cappelle Desprez, a parent of CR that confers durable adult plant resistance to yellow rust. *QYr.inra-2DS* probably corresponds to the *Yr16* gene. The most consistent QTL, designated *QYr.inra-2BL*, was located on the centromeric region of chromosome 2B and explained 61% of the phenotypic variation in 2003. This QTL was responsible for seedling-stage resistance and may correspond to a cluster of genes, including *Yr7*. The remaining QTLs were mapped to the short arm of chromosome 2B ($R^2 = 22$–$70\%$) and to the long arm of chromosomes 2A ($R^2 = 0.20$–$0.40$) and 5B ($R^2 = 0.18$–$0.26$). This specific combination of seedling and adult plant resistance genes found in CR and CD may constitute the key to their durable resistance against yellow rust.

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## Introduction

Yellow rust, caused by the fungal pathogen *Puccinia striiformis* West., is one of the most damaging diseases affecting bread wheat in temperate regions. The deployment of disease-resistant varieties is the most effective approach to reduce fungicide use and minimize crop losses. Two types of resistance to yellow rust have been described: isolate- or race-specific resistance and quantitative resistance. The former is under monogenic control and can easily be overcome by new, virulent races of pathogen. To date, several race-specific resistance genes have been described for yellow rust resistance: 37 of these are designated *Yr1*–*Yr32* and include allele variations (for a review, see McIntosh et al. 1999, 2001), and an additional 24 have been allocated temporary names, such as *YrH52* (Peng et al. 1999) or *YrnsB1* (Börner et al. 2000). Quantitative resistance, usually under oligogenic or polygenic control, is generally more effective in the field and appears to be more durable. To date, only a few studies have been carried out to characterize quantitative resistance to yellow rust.

The bread wheat cultivar Camp Rémy (CR), which has been grown for more than 20 years in France, is known to possess seedling and adult plant resistance against yellow rust that is effective at all stages of plant growth and which remains effective against all known races of the pathogen. One of its parents, Cappelle Desprez, also resistant to yellow rust, was widely

cultivated without fungicide treatment in several western European countries during the 1960s and 1970s, comprising more than 75% of the acreage seeded in wheat crops. This cultivar is therefore considered to have a durable, moderate form of resistance that is effective at the adult plant stage (Law et al. 1978). Genetic and cytogenetic studies of Cappelle Desprez have implicated chromosome 2D (Worland and Law 1986), and the 5BS-7BS translocation (Law and Worland 1997) in adult plant resistance.

Boukhatem et al. (2002) detected two quantitative trait loci (QTLs) for yellow rust resistance in a population derived from the CR × Michigan Amber cross when this population was evaluated for resistance at the adult plant stage in the field. Partial scanning of the genome enabled the identification of two QTLs located on chromosome 2B and 2A that accounted for 46% and 15% of the total phenotypic variance, respectively. These authors reported that CR might also possess a specific seedling-stage resistance gene, *Yr7*, on chromosome 2B. De Vallavieille-Pope et al. (1990) demonstrated that CR may possess at least two or three race-specific resistance genes, including *Yr7*. Thus, other parts of the genome could also be implicated in the resistance derived from CR.

We report here the results of a genetic study carried out on a population derived from the CR × Récital cross in which both seedling and adult plant resistance against yellow rust was evaluated. Based on preliminary results, we focused on chromosomes 2A, 2B, 2D, and 5B to identify resistance factors in CR and its parents (Cappelle Desprez) in order to explain their common durability against yellow rust.

## Materials and methods

### Plant materials

A bread wheat population of 217 $F_7$ recombinant inbred lines (RILs), derived from the cross between CR and Récital, was created using single-seed descent. Récital is a susceptible French variety derived from the cross Mexique 267/5/(81.12/Besostaya1//HeineVII/3/Nord/4/Tadorna)/9369. CR (resistant) is derived from the cross (Cappelle Desprez/Thatcher//Cappelle Desprez/Garnet)/(Alsace/Nord Desprez//Széckacz//Providence). Cappelle Desprez and Nulli Cappelle 5BS-7BS were also included. Nulli Cappelle 5BS-7BS is a nullisomic line derived from Cappelle Desprez which only possesses 40 chromosomes because of the absence of the 5BS-7BS pair.

### Seedling resistance assessment

The RILs and the parents were sown in a growth chamber at 20°C and under a 16/8-h (light/dark) photoperiod until the second leaf appeared. The plants were then inoculated by spraying the leaves with a spore suspension (in mineral oil; Soltrol) of *Puccinia striiformis* pathotype 237 E141. This pathotype is endowed with virulence factors V1, V2, V3a + V4a, V6, V9, SD and SU (Robert et al. 2000). The inoculated plants were placed first in a growth chamber at 12°C and 100% relative humidity, in the dark, for 24 h to ensure spore germination and penetration and then returned to a growth chamber and grown at 20°C for 15 days under a 16/8-h (light/dark) photoperiod. At 12 days and 15 days after inoculation, symptoms of yellow rust resistance were recorded using the scale described by McNeal et al. (1971). The set of yellow rust standard differentials was included to confirm the virulence spectrum of the *P. striiformis* pathotypes for both the seedling and field assessment.

### Field trials and phenotypic assessment

The $F_6$–$F_8$ generations of the RILs, the $F_1$ plants and the parents were evaluated in the field at the INRA experimental farm in Le Rheu (Rennes-35, France) during the period 2000–2002 with the 237 E141 pathotype of *P. striiformis* and in 2003 with the 237 E141 V17 pathotype, which contains the additional virulence V17 factor (Bayles et al. 2000). Twelve seeds from each line were planted in a row at the end of October and arranged in a completely randomized bloc design with two replications. Récital was planted every third row as a susceptible spreader. In addition, Récital seedlings, cultivated in a growth chamber to the two-leaf stage, were artificially inoculated with *P. striiformis*. The plants were reared at 6–10°C in a growth chamber under a 10/14-h (light/dark) until the leaves were totally covered by yellow rust pustules and then transplanted into the experimental plot. To ensure a high level of disease pressure, we planted two or three inoculated plants in 30% of the Récital rows during January, February, and early March of each year.

Yellow rust scores were recorded on three separate occasions: at the time of rust appearance ($t_1$), corresponding to the growth stage DC32 (Zadoks et al. 1974), and after each cycle of pathogen multiplication on the susceptible Récital parent ($t_2$ and $t_3$), with $t_2$ corresponding to the growth stage DC39 and $t_3$ to the grain filling stage DC79 of Récital. The scoring method considered the percentage of leaf area covered by pustules on the three most heavily infested leaves, using a scale from 0 to 12 (Table 1). For each line, the area under the disease progress curve (AUDPC) was calculated using the formula: $\text{AUDPC} = (N_1 + N_2)(t_2 - t_1)/2 + (N_2 + N_3)(t_3 - t_2)/2$, where $N_I$ is the rust intensity recorded at $t_I$. At the end of the experiment, samples of *P. striiformis* spores were collected from the leaves to confirm the identity of the 237 E141 and 237 E141V17 pathotypes using a seedling-stage assessment.

Plant height was also recorded after flowering for each line of the CR × Récital population in 2001, 2002 and 2003.

**Table 1** Scale used for yellow rust scoring at the adult plant stage

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Percentage area infested on three leaves | 0 | 0.3 | 0.7 | 2 | 8 | 12 | 16 | 24 | 33 | 50 | 66 | 82 | 100 |
| Score | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |

**Table 2** Primer sequences, annealing temperature ($T_m$) and accession number of the EST markers

| Marker name | Accession | Putative function | Primer U | Primer L | $T_m$ (°C) |
|---|---|---|---|---|---|
| GC17 | BF428563 | Putative plant disease resistance polyprotein | GAG GTT TAT GCC ATA TCT GC | TCT TGG CCT GCT GAC ATA C | 58°C |
| GC18 | BE442858 | Peroxidase | ATT TCG TTC TGA TTA ATT CC | CCC AAA TAG TTG TGA TTA | 48°C |
| GC28 | BE442849 | Peroxidase | GGC CTG TTC AAG TCG GAC C | TAC AGT GTT CTG GCA GTG ACA TGG | 60°C |
| GC31 | BE591211 | Serine/threonine kinase receptor | CAT ATA GCT TTG GCG TTC TAT TGT | CTC ATC ATA TCG TTG CCT AAA GT | 55°C |

### Map construction

One hundred and ninety-four $F_7$ RILs were used for map construction. The phenotypic marker (named Rsp) corresponding to total juvenile resistance, 87 microsatellite markers and four expressed sequence tag (EST) markers were employed to establish a genetic linkage map of chromosomes 2A, 2B, 2D, and 5B. ESTs with putative defence-related functions were assigned to these chromosomes on the Chinese Spring deletion map (http://wheat.pw.usda.gov/wEST/binmaps/). In order to map the ESTs, specific primers were designed using the OLIGO6 software (Table 2). All markers were also evaluated on the variety Cappelle Desprez and the Nulli Cappelle 5BS-7BS line.

Segregation data for each marker were analysed using the MAPMAKER/EXP software, ver. 3.0 (Lander et al. 1987; Lincoln et al. 1992). Linkage groups were determined using a LOD (logarithm of the odds) score of 7.0 and a maximum distance between two markers of 40 cM. For each linkage group, the best marker loci order was determined using three-point and multi-point analyses and the ORDER or TRY commands. Genetic distances were calculated using the Haldane mapping function (Haldane 1919). Linkage groups were assigned to chromosomes by comparison with the International Triticeae Mapping Initiative (ITMI) map (Röder et al. 1998).

### Trait analysis

The statistical analysis of traits was performed using the SAS statistics package (SAS Institute, Raleigh, N.C.). Genotype, replication and year effects were tested using analysis of variance (ANOVA) and the PROC GLM procedure. For each year, the homogeneity of phenotypic variances between replications and genotypes was verified using Bartlett's test, and the normality of residual distributions was checked using the PROC univariate procedure and Shapiro's w-statistic. Broad-sense heritabilities for yellow rust resistance were calculated using the formula: $h^2 = \sigma^2_G/[\sigma^2_G + (\sigma^2_e/ r)]$, where $\sigma^2_G$ is the genetic variance, $\sigma^2_e$ is the residual variance and $r$ is the number of replications. For all tests, a probability level $P < 0.05$ was employed.

### QTL analysis

A one-way ANOVA with a probability level of $P < 0.001$ was employed to identify markers with significant effects on resistance. The QTL detection was performed using interval mapping (IM) (Lander and Botstein 1989) and composite interval mapping (CIM) (Zeng 1993, 1994) with the QTL CARTOGRAPHER software ver. 2.0 (Basten et al. 1997). In order to detect significant QTLs, a critical LOD threshold of 4.5 was established for AUDPC by conducting a permutation test with 1,000 permutations. Six cofactors were taken into account and a window size of 10 cM around the test interval was chosen for CIM analysis. For each QTL, the position corresponding to the maximum LOD, the additive effect and the part of the phenotypic variation it explained were estimated. The percentage of phenotypic variation explained by the whole model (total $R^2$) and by a QTL × QTL interaction was determined using multiple interval mapping (MIM).

## Results

### Seedling plant resistance

The resistant parent CR is considered to have an infection type equal to 2 on a scale from 0 to 9 (McNeal et al. 1971). We used the same value in considering plants to be resistant. Susceptible plants, such as Récital, exhib-

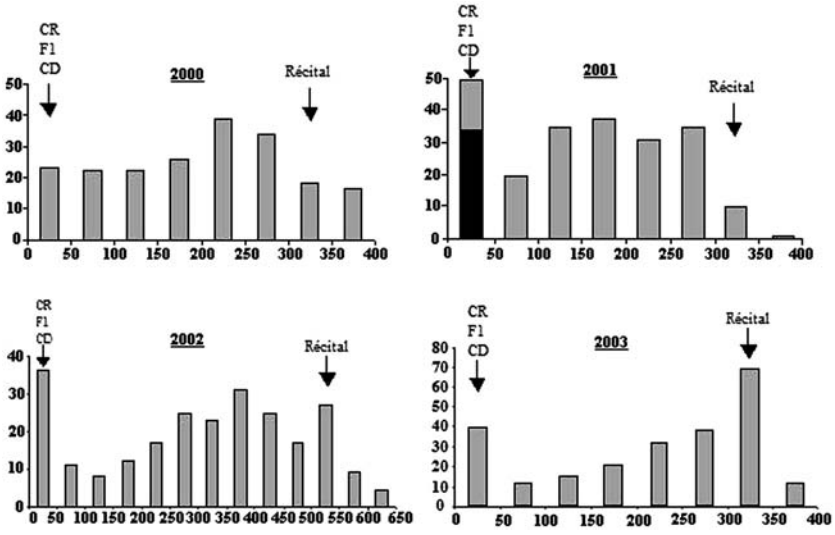


**Fig. 1** Phenotypic distribution of RILs derived from the CR × Récital cross for yellow rust intensity in 2000, 2001 and 2002 using the *Puccinia striiformis* 237 E141 pathotype and for 2003 using the 237 E141V17 pathotype. AUDPC values for CR, Récital, Cappelle Desprez (*CD*) and the $F_1$ generation are also included. The phenotypic distribution of RILs resistant at the seedling stage is represented in *black* for 2001

**Table 3** ANOVA of genotype and replication effects and proportion of phenotypic variation for yellow rust intensity obtained for each year among RILs from the CR × Récital cross. The results shown were obtained using the AUDPC trait

| | *df* | MS | *F* | *P* | $h^2$ |
|---|---|---|---|---|---|
| 2000 | | | | | |
| Lines | 213 | 22,626 | 19.3 | 0.0001 | |
| Replication effect | 1 | 228 | 0.02 | 0.8916 | |
| Error | 199 | 1,175 | | | |
| | | | | | 0.95 |
| 2001 | | | | | |
| Lines | 216 | 20,413 | 16.36 | 0.0001 | |
| Replication effect | 1 | 31,086 | 2.89 | 0.0899 | |
| Error | 216 | 1,109 | | | |
| | | | | | 0.95 |
| 2002 | | | | | |
| Lines | 217 | 54,734 | 41.5 | 0.0001 | |
| Replication effect | 1 | 42,703 | 1.53 | 0.2175 | |
| Error | 215 | 1,120 | | | |
| | | | | | 0.98 |
| 2003 | | | | | |
| Lines | 213 | 21,511 | 20.74 | 0.0001 | |
| Replication effect | 1 | 818 | 0.07 | 0.7895 | |
| Error | 205 | 1,034 | | | |
| | | | | | 0.95 |

ited an infection type of 8–9. The analysis of five plants per RIL enabled us to designate lines as resistant or susceptible. In total, 34 RILs were found to be resistant at the seedling stage.

## Severity of yellow rust in the field

The frequency distributions of the yellow rust disease scores for mean RIL values showed continuous phenotypic variation in the AUDPC (Fig. 1). All plants belonging to resistant lines at the seedling stage were still resistant at the adult plant stages (Fig. 1). The distribution showed an increase in the number of susceptible lines in 2003 in comparison with the other 3 years. The residual distributions were normal during all years according to Shapiro's w-statistics (data not shown). Heritabilities within years were high, ranging from 0.95 in 2000, 2001 and 2002 to 0.98 in 2003. High levels of correlation were observed between replications in all years (0.82 in 2000, 0.81 in 2001, 0.92 in 2002 and 0.89 in 2003), and no significant replication effects were detected (Table 3). Consequently, the mean of the two replications was used for QTL detection. Significant genotype effects for each year (Table 3) and a significant year effect (data not

**Table 4** Summary of the QTLs for yellow rust resistance detected by CIM in the CR × Récital population for 2000, 2001, 2002, and 2003

| QTL | Linkage group | Position (cM) | 2000 | | | 2001 | | | 2002 | | | 2003 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | LOD score | $R^2$ | Additive effect | LOD score | $R^2$ | Additive effect | LOD score | $R^2$ | Additive effect | LOD score | $R^2$ | Additive effect |
| *QYr.inra-2AL* | 2A | 41.28 | 5.61 | 0.22 | −60.02 | 5.43 | 0.20 | −55.59 | 6.08 | 0.24 | −98.78 | 5.37 | 0.40 | −81.24 |
| *QYr.inra-2BS* | 2B | 58.8 | 7.03 | 0.22 | −56.49 | 10.72 | 0.56 | −80.79 | 5.96 | 0.42 | −117.49 | 17.92 | 0.70 | −98.14 |
| *QYr.inra-2BL* | 2B | 96.35 | 21.70 | 0.42 | −89.77 | 23.40 | 0.43 | −87.72 | 24.62 | 0.45 | −146.22 | 35.52 | 0.61 | −104.44 |
| *QYr.inra-2DS* | 2D | 47.16 | 5.70 | 0.24 | −56.55 | 5.81 | 0.25 | −56.20 | 7.99 | 0.42 | −116.15 | 10.9 | 0.69 | −95.68 |
| *QYr.inra-5BL.1* | 5B | 145.9 | 9.70 | 0.26 | −72.03 | 6.59 | 0.18 | −58.41 | 8.75 | 0.24 | −108.22 | 9.04 | 0.26 | −77.53 |
| *QYr.inra-5BL.2* | 5B | 173.45 | 11.17 | 0.32 | −74.38 | 10.46 | 0.31 | −70.62 | 9.66 | 0.29 | −112.32 | 10.79 | 0.34 | −69.17 |
| Total $R^2$ for the multiple QTL model | | | | 0.57 | | | 0.81 | | | 0.57 | | | 0.81 | |

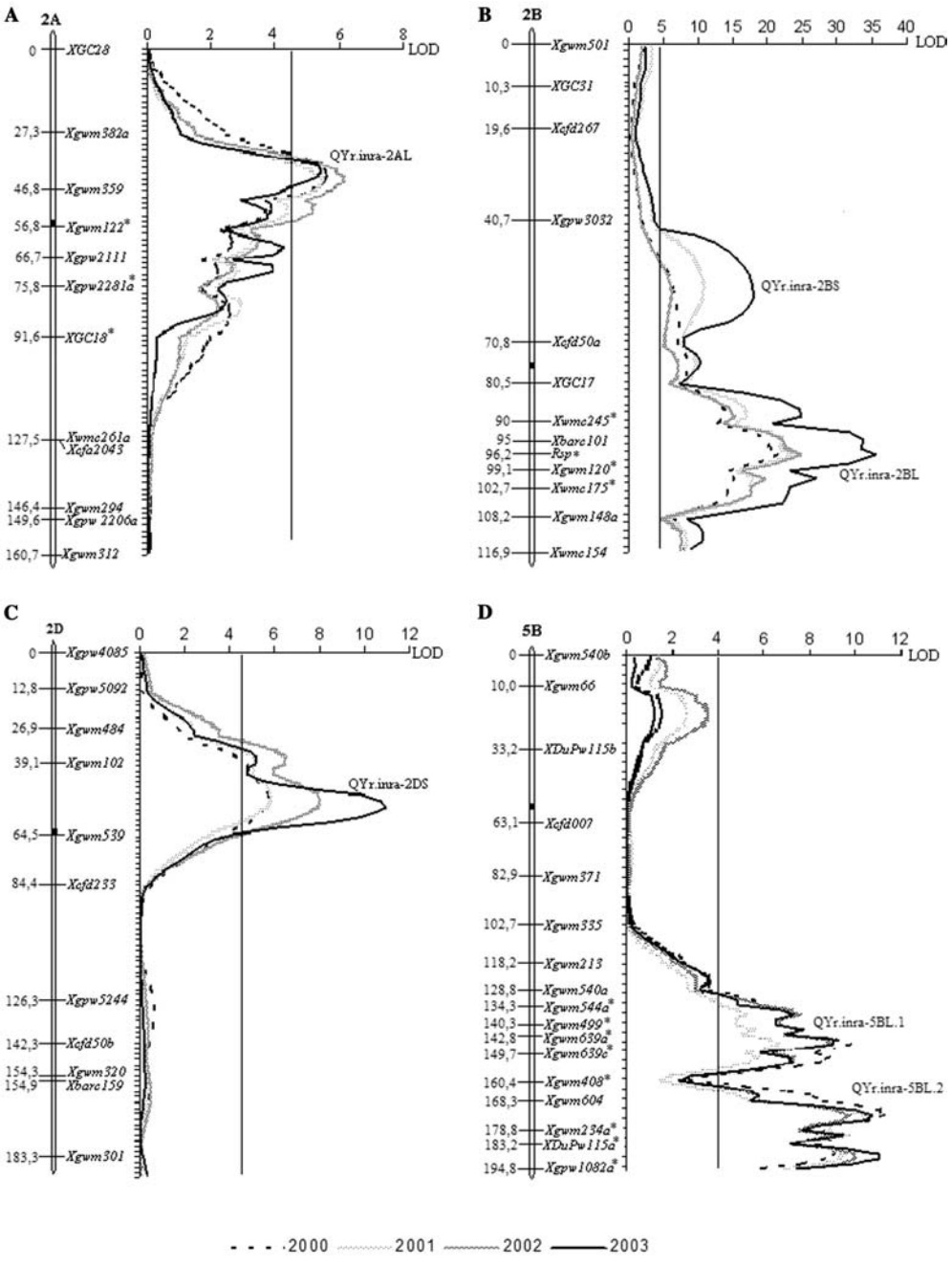


**Fig. 2** Yellow rust disease resistance loci in the CR × Récital mapping population on chromosomes 2A (**a**), 2B (**b**), 2D (**c**) and 5B (**d**). The scan of QTLs for each year (2000, 2001, 2002, and 2003) is represented separately. The positions (in centiMorgans) and names of the molecular markers are shown on the chromosome along the *horizontal axis*. The LOD score scan was obtained by CIM. Highly distorted loci ($P < 0.001$) are indicated with an *asterisk* (*)

shown) were observed. In all years, no pathotype other than the one used for inoculation was detected from pathogenicity tests on differential cultivars (data not shown).

Yellow rust severity was not correlated with plant height in any year ($r < 0.001$ for AUDPC in 2001, 2002 and 2003).

## The CR × Récital cross map

This map comprised 53 marker loci linked in four linkage groups assigned to chromosomes 2A, 2B, 2D, and 5B. It covered 661.6 cM, which represents an average of 18.9% of the entire wheat genome (Röder et al. 1998). EST-specific markers were located in the expected region on the basis of their location on the Chinese Spring deletion map (http://wheat.pw.usda.gov/wEST/binmaps/).

We detected two regions with distorted loci ($P < 0.001$); the first was observed on the long arm of chromosome 2B, between the marker loci *Xwmc245* and *Xwmc148*, and the second was located on the telomeric part of chromosome 5BL and included the gwm234, DuPw115a and gpw1082 markers. The gwm234 and DuPw115a markers did not amplify from the DNA of the Nulli Cappelle 5BS-7BS line, whereas the DNA amplified from CR and Cappelle Desprez using these markers exhibited the same patterns. Moreover, those

two markers are usually mapped on the 5BS arm in several wheat crosses, including Arina × Forno (Paillard et al. 2003), Renan × Récital (Dedryver, unpublished results) and the ITMI map (Röder et al. 1998), suggesting that the telomeric part of chromosome 5BL in CR is probably a 5BS region translocated at the end of the long arm of chromosome 5B.

The phenotypic marker of the seedling plant resistance gene *Rsp* was mapped on the long arm of chromosome 2B, between the *Xbarc101* and *Xgwm120* marker loci.

### QTL analysis

QTLs for yellow rust resistance were separately detected in each of the 4 years using a CIM analysis on the AUDPC data. In total, six QTLs with additive effects were detected and remained consistent over the 4-year period (Table 4). One QTL was found on each of chromosomes 2A and 2D and two on chromosomes 2B and 5B. Resistance was always conferred by alleles originating from CR, the resistant parent. The total phenotypic variance observed ranged from 0.57 in 2000 and 2002 to 0.81 in 2001 and 2003. The most consistent QTL was *QYr.inra-2BL*, which was detected on the centromeric part of the long arm of chromosome 2B and explained a large proportion of the phenotypic variance observed ($R^2 = 42$–$61\%$, depending on the year) (Table 4). The peak of *QYr.inra-2BL* was located on the seedling resistance gene *Rsp*, 1.2 cM distal from the microsatellite marker locus *Xbarc101* (Fig. 2). The *QYr.inra-2BS* QTL was located on the centromeric part of the short arm of chromosome 2B in the *Xgpw3032-Xcfd50a* interval, and it explained from 22% to 70% of the phenotypic variance observed, depending on the year. The *QYr.inra-5BL.1* QTL was located on the long arm of chromosome 5B, between the *Xgwm639a* and *Xgwm639c* marker loci. The phenotypic variance that could be explained by this QTL ranged from 18% in 2001 to 26% in 2003. *QYr.inra-2AL*, which mapped between the *Xgwm382a* and *Xgwm359* marker loci, accounted for 20% (2001) to 40% (2003) of the observed phenotypic variance. *QYr.inra-2DS*, accounting for 24–69% of the observed phenotypic variance (depending to the year), was detected on the centromeric region of the short arm of chromosome 2D in the *Xgwm102-Xgwm539* interval. The final QTL, *QYr.inra-5BL.2*, which explained 29% of the phenotypic variance observed in 2002 and an average of 32% in the other 3 years, was situated on the translocated region of the 5BS chromosome, between *Xgwm234* and *XDuPw115a*. No epistatic effects between those six QTLs were detected. ANOVA results confirmed that all markers included within the QTL intervals were highly significant ($P < 0.001$).

No QTL for plant height was detected on the four linkage groups at LOD > 2.5 determined after a permutation test.

## Discussion

### Phenotypic distribution

In 2002, the frequency distribution of yellow rust scores exhibited more classes of AUDPC, which exceeded 350 in some lines. These higher scores were probably not due to any greater susceptibility of the lines but rather to a longer interval between scoring. In contrast, the AUDPC distribution based on a phenotypic assessment of resistance in 2003 showed a higher proportion of susceptible lines, with AUDPC values exceeding 300. This result was probably not due to the extra virulence (V17) in pathotype 237 E141V17 because neither CR nor Récital is known to possess the *Yr17* race-specific gene. It is possible that this pathotype possesses other virulence genes that were not detected by our set of differential lines. In addition, the possibility of a race effect was supported by the increased number of lines that were totally resistant in 2003 and by the increase in that part of the phenotypic variation which could be explained by all QTLs except *QYr.inra-5BL.1*. Given that the total number of QTLs remained unchanged, it is probable that this increase was also due to favourable environmental conditions for yellow rust development in 2003 that resulted in highly susceptible lines being infected.

### The seedling resistance gene

The *QYr.inra-2BL* QTL corresponds to the QTL reported by Boukhatem et al. (2002). During our study, *QYr.inra-2BL* explained 42–61% of the phenotypic variation observed each year and corresponded to a major resistance gene. This gene probably explained the stability of the rust scores during the 4 years in two environments (Le Rheu, France and Gembloux, Belgium), in two populations (Récital and Michigan Amber) and with three pathotypes of *P.striiformis*. The allele was dominant because all of the $F_1$ generation plants were totally resistant. *QYr.inra-2BL* corresponded to a seedling-stage resistance gene and co-located with the seedling gene *Rsp*, which accounted for more than 30% of the phenotypic variation observed.

Cytogenetic studies have shown that the seedling resistance gene *Yr7* is located on the long arm of chromosome 2B (Macer 1966). *Yr7*, located 15 cM from the centromere in different populations by Hart et al. (1993) and Bariana et al. (2001), corresponds to the *QYr.inra-2BL* interval. The *Yr5* resistance gene has also been mapped to the same position (Hart et al. 1993). It is therefore possible that *Yr7* and *Yr5* are allelic. *QYr.inra-2BL* probably corresponds to *Yr7* since the variety Thatcher, an ancestor of CR, possesses this gene (McIntosh et al. 1995). Yellow rust scores for CR and Lee, the differential host possessing *Yr7*, produced the same score of 2N (= 2 with necrosis). De Vallavieille-Pope et al. (1990) suggested the presence of another

seedling gene in CR because of the long-term effectiveness of CR seedling resistance. Other genes may also be present, but they were not detected because the map presented in this study is only a partial one. However, the total phenotypic variation observed which could be explained by the QTL reached 81% in 2001 and 2003, suggesting that nearly all of the genetic variability could be attributed to *QYr.inra-2BL*. It is therefore unlikely that another major QTL will be found in the remaining genome. We thus propose that the *QYr.inra-2BL* interval detected may contain a series of tightly linked resistance genes. However, because all of the markers located between WMC245 and WMC154 exhibited a highly segregated distortion ($P < 0.05$) in favour of the CR allele, it remains difficult to satisfactorily interpret this "enormous" QTL. This region of chromosome 2B has been shown to contain several disease resistance genes, including the *Sr9* gene for stem rust resistance that is also present in var. Thatcher, and is closely linked to *Yr7* (Hart et al. 1993). Recently, a QTL involved in partial leaf resistance to nodorum blotch was detected near the gwm120 marker in a population derived from the cross Liwilla × Begra (Czembor et al. 2003).

## Adult plant resistance

*QYr.inra-2AL* is the only QTL we found in common with those obtained by Boukhatem et al. (2002), although one of the reasons for this could be that these authors constructed only a partial genetic linkage map of the wheat genome. In our study, *QYr.inra-2AL* was detected at a LOD score that was greater than 4.5, and it explained a larger proportion of the total phenotypic variation than reported by Boukhatem et al. (2002). The map constructed by Boukhatem et al. (2002) may have had an insufficient marker density to detect other QTLs, which may also explain the lower $R^2$ observed for this QTL in their study, because the linkage group was constructed with only two microsatellite markers.

*QYr.inra-2BS* was detected on the centromeric part of chromosome 2B at a distance of 45 cM from *QYr.inra-2BL*. It was seen more consistently in 2001 and 2003, together with a marked increase in phenotypic variation, which could be explained by an increased detection of *QYr.inra-2BS* following inoculation with the 237 E141V17 pathotype. Therefore, although this QTL was detected throughout the year, environmental conditions may affect its detection, and thus explain why it was not found by Boukhatem et al. (2002).

*QYr.inra-2DS* may be the qualitative *Yr16* gene from Cappelle Desprez. This gene, which confers partial adult plant resistance to yellow rust, is located on the centromeric region of chromosome 2D (Worland and Law 1986). In the Catalogue of Gene Symbols (McIntosh et al. 1995), *Yr16* was mapped at 9 cM from the centromere (Hart et al. 1993), which corresponds to the interval for *QYr.inra-2DS*. Polymorphism patterns for the gwm102 and gwm539 markers were identical for CR and Cappelle Desprez, suggesting that this part of the chromosome was inherited from Cappelle Desprez. A durable leaf rust resistance QTL has also been observed within the *Xgwm102-Xgwm539* interval (Schnurbusch et al. 2004). This resistance was conferred by an allele in the leaf rust-susceptible variety Arina, which is also derived from Cappelle Desprez. Thus, this chromosome interval, which corresponds to *QYr.inra-2DS*, probably contains the genetic factors responsible for the durable rust resistance observed in CR and Cappelle Desprez.

The *QYr.inra-5BL.1* QTL, which accounts for 25% of the phenotypic variation observed, was detected on chromosome arm 5BL in the *Xgwm499-Xgwm639c* marker loci interval. Another major QTL involved in nodorum leaf blotch resistance, explaining 27% of the observed phenotypic variation, was also mapped on chromosome arm 5BL, proximal to the gwm499 marker, by Czembor et al. (2003). Schnurbusch et al. (2003) detected a QTL for resistance to Stagonospora glume blotch peaking at the gwm639 marker in a cross derived from Forno × Arina. To date, several major race-specific genes have been mapped in this region, including *Lr18* for leaf rust resistance, which is situated 45 cM distal from the centromere in the consensus wheat map (Hart et al. 1993) and corresponds to the location of *QYr.inra-5BL.1*. This chromosomal region also corresponds to one of the five major resistance gene cluster regions described by Dilbirligi et al. (2004).

*QYr.inra-5BL.2*, located on the translocated region of chromosome 5BS in the long arm of chromosome 5B, may contain resistance genes inherited from the cultivar Cappelle Desprez because the 5BS-7BS translocation is involved in the yellow rust resistance of this variety (Law and Worland 1997). These authors suggest that the genes involved in resistance to yellow rust are most probably located on the short arm of chromosome 5B and may be closely linked to the break point of the translocation. Dilbirligi et al. (2004) also physically mapped a putative nucleotide binding site-leucine-rich repeat (NBS-LRR) gene (sequence BM136556a) in the 5BS-5 bin where the gwm234 marker is located (Sourdille et al. 2004).

No association was detected between resistance to yellow rust and morphological traits. With respect to plant height, Récital, the susceptible parent, possesses a reduced height gene, *Rht-1*, located on homeologic group 4. Camp Rémy is not known to possess any *Rht* gene, but Cappelle Desprez is endowed with *Rht8*, located on chromosome 2D and tightly linked to *Xgwm261*, which is approximately 20 cM distal from *Xgwm484*. If we refer to the ITMI map and the location of the *Xgwm484* locus, *Rht8* should be located on the telomeric part of linkage group 2D. However, no QTL for plant height was detected on this chromosome, or on the other three. We can therefore conclude that this trait does not affect resistance.

Factors for durability

The race-specific resistance gene *Yr7* probably did not explain the durability of yellow rust resistance in CR because this gene has already been overcome by other pathotypes. However, no pathotype containing *Yr7* virulence has been reported to overcome CR seedling resistance in France (de Vallavieille-Pope, personal communication). It is possible that resistance genes linked to *Yr7* may also be involved in seedling resistance. This set of seedling resistance genes may be crucial to explaining the durable resistance of CR during the juvenile stage.

We propose that several factors inherited from Cappelle Desprez are responsible for the durability of yellow rust resistance in CR and that this is a form of adult plant resistance. Since the *Yr16* gene has been overcome by some pathotypes, durability is probably due to other factors located on chromosome 5BS and activated by the translocation in 5BL, or a combination between these factors and the gene *Yr16*. Moreover, it is possible that other regions of the genome involved in resistance and not inherited from Cappelle Desprez may also provide some important resistance factors. Therefore, durability in the CR variety may be due to a set of qualitative and quantitative resistance genes located on chromosomes 2B, 2D, and 5B for which no yellow rust pathogen has yet evolved the necessary combination of virulence genes.

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