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Introduction

Yellow rust, caused by *Puccinia striiformis* f. sp. *tritici*, is one of the most widespread and destructive wheat diseases in areas where cool temperatures prevail. The wheat cultivar Renan, carrying the specific gene *Yr17*, has shown effective resistance for a long period of time, even though some pathotypes overcame the *Yr17* gene. The objective of this study was to decipher the genetic architecture of the Renan durable adult-plant resistance (APR) to yellow rust.

Materials and Methods

Plant materials

A mapping population of 194 (F6 and F7) recombinant inbred lines (RILs) was developed by single-seed descent from the cross between the winter bread wheat cultivars, Renan (resistant) and Récital (susceptible).

Field assessment of resistance

Field assays were performed for 4 years (1995, 1996, 2005 and 2006). Each line was sown in a row and arranged in a completely randomized block design. Evaluations were performed in 1995 and 1996 with the *P. striiformis* 237E141 pathotype and in 2005 and 2006 with the 237E141 V17 pathotype, which contains the additional virulence to *Yr17*. Stripe rust scores were recorded on three separate occasions: at the time of rust appearance (N1) and after each cycle of pathogen multiplication on the susceptible parent Récital (N2 and N3).

QTL analysis

A genetic linkage map was constructed during a Génoplante project (Groos et al. 2002; Gervais et al. 2003). A SCAR marker of *Yr17* (Robert et al. 1999) was added to the map. Additionally, 14 PCR-based markers for NBS-LRR resistance gene analogs (RGAs) were developed and mapped in this study. QTL detection was carried out by Composite Interval Mapping (CIM) (Zeng 1993) using QTL CARTOGRAPHER software, version 2.5 (Basten et al. 1997). After 1000 permutations, critical LOD thresholds were LOD 3.5 ($p=0.05$). The epistatic effects of QTLs were evaluated using Multiple Interval Mapping (MIM) analysis.

Results

Phenotypic assessment of stripe rust infection

AUDPCs for the Renan/Récital population had a continuous distribution, suggesting a quantitative inheritance of the resistance (Figure 1). Within-year and over-years heritability values for the AUDPC were high (h^2 ranged from 0.93 to 0.97).

QTL analysis of the resistance

Three QTLs, *QYr.inra-2BS*, *QYr.inra-3BS* and *QYr.inra-6B* with resistance alleles derived from Renan were detected in 1995-1996 with the 237E141 pathotype, which is avirulent against genotypes carrying *Yr17*. These QTLs were stable and explained a major part of the phenotypic variation seen in 2005-2006, when the 237E141 V17 pathotype possessing the virulence to *Yr17* was used (Figure 2). Each of these QTLs contributed between approximately 4 and 15 % of the phenotypic variance and were effective at different adult plant stages. Interactions were observed between some markers linked to the *Yr17* gene and three Renan QTLs: *QYr.inra-2BS*, *QYr.inra-3BS* and *QYr.inra-6B*. Only one NBS-LRR-derived marker was linked to one QTL: *QYr.inra-2BS* (Figure 2).

References

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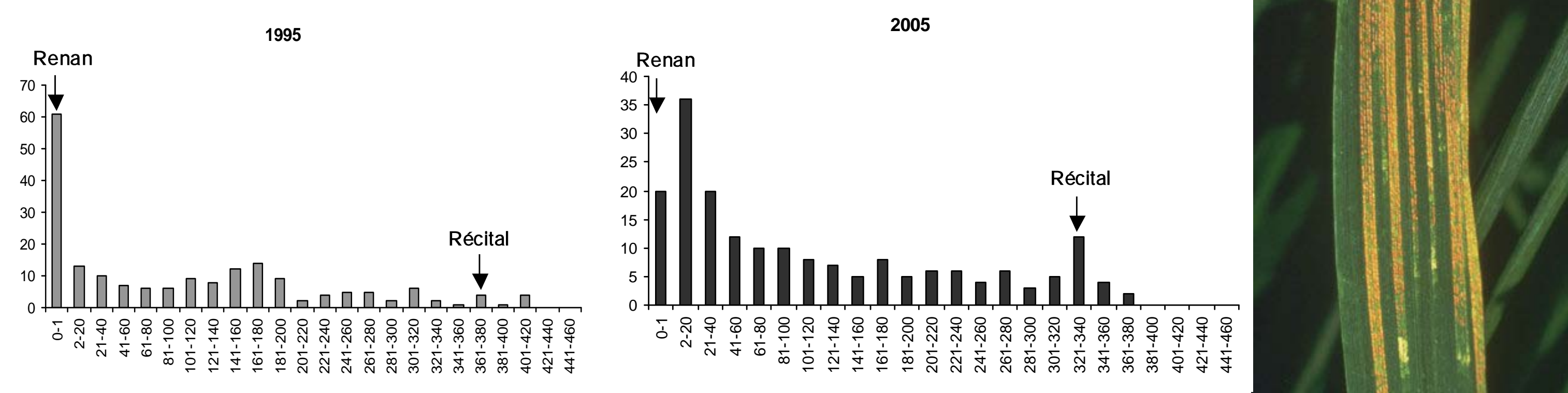


Figure 1 : Phenotypic distribution of stripe rust intensity area under the disease-progress curve (AUDPC) for the 194 RILs derived from the Renan x Récital cross in 1995 and 2005. The RILs were infected in 1995 with the *P. striiformis* 237E141 pathotype and with the 237E141 V17 pathotype in 2005.

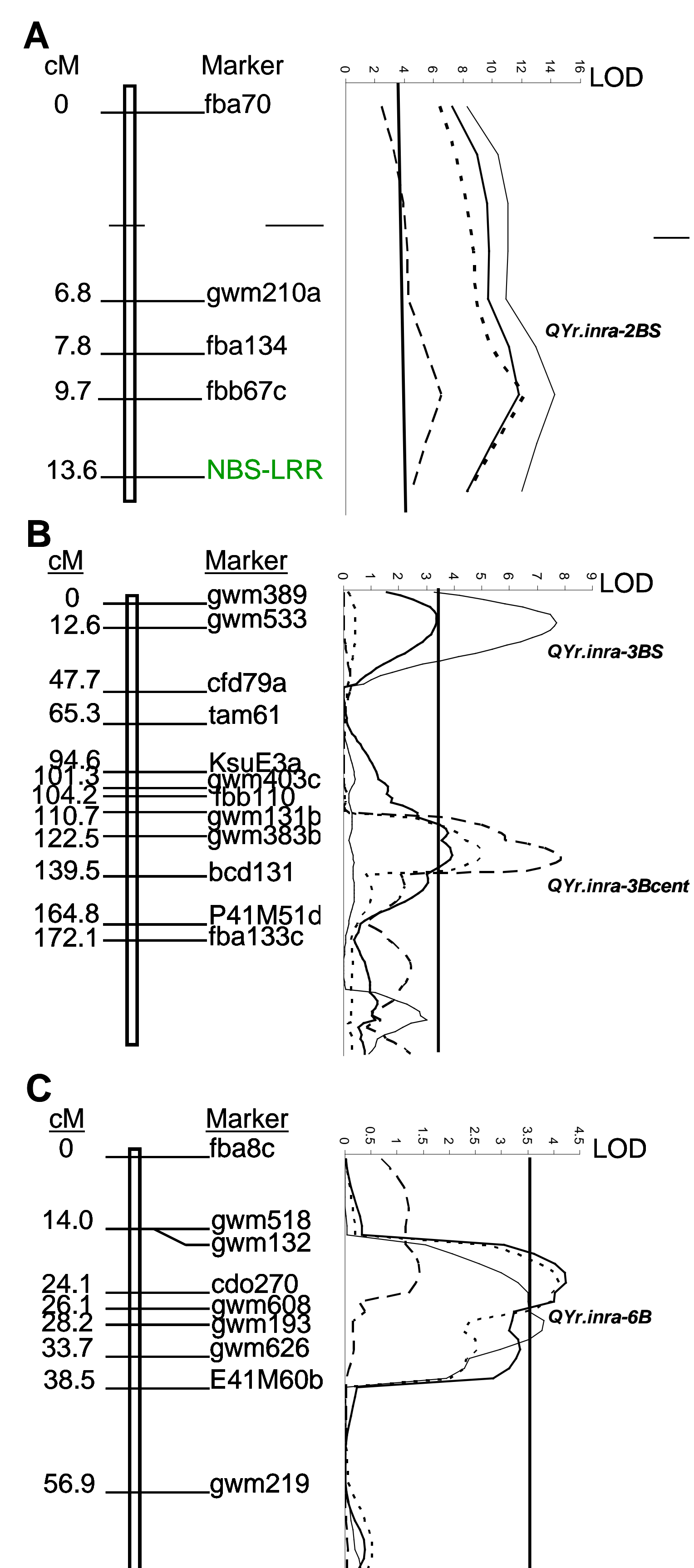


Figure 2 : Scan for stable Renan QTLs, *QYr.inra-2BS*, *QYr.inra-3BS*, and *QYr.inra-6B* detected in 2005-2006 with the *P. striiformis* 237E141 V17 pathotype and mapped on linkage groups corresponding to chromosomes A, 2BS; B, 3B; and C, 6B and using three scoring times N1, N2, and N3 and AUDPC. Vertical bold line indicated significant LOD thresholds (3.5).

Discussion

Our results suggest that resistance against stripe rust of the cultivar Renan could not be easily overcome by the rust pathogen since it would be confronted with different target gene(s) at different infection cycles (Dedryver et al. 2009). Mallard et al. (2005 and 2008) showed that cv. Camp Rémy, another durable source of resistance to stripe rust, modifies some defense-related genes expression between two different infection cycles of the pathogen.

A marker assisted selection strategy can be used to accelerate the incorporation of these APR genes into adapted genotypes.