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Mapping quantitative trait loci for resistance to *Pratylenchus thornei* from synthetic hexaploid wheat in the International Triticeae Mapping Initiative (ITMI) population

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Abstract. Root-lesion nematode (*Pratylenchus thornei*) is a serious pathogen of wheat in many countries. The International Triticeae Mapping Initiative (ITMI) population of recombinant inbred lines (RILs) was assessed for resistance to *P. thornei* to determine the chromosome locations of the resistance genes. The ITMI population is derived from a cross between the resistant synthetic hexaploid wheat W-7984 and a susceptible bread wheat cultivar Opata 85. Two years of phenotypic data for resistance to *P. thornei* were obtained in replicated glasshouse trials. Quantitative trait locus (QTL) analysis was performed using available segregation and map data for 114 RILs. A QTL on chromosome 6DS showed consistent effects for reduced nematode numbers (partial resistance) across years and accounted for 11% and 23% of the phenotypic variation. A second QTL for *P. thornei* resistance on chromosome 2BS accounted for an additional 19% and 5%. Restriction fragment length polymorphism (RFLP) and simple sequence repeat (SSR) markers associated with the QTLs are physically located in regions rich in major genes at the distal ends of the short chromosome arms of 6D and 2B. SSR markers with potential for marker-assisted selection of *P. thornei* resistance effective in different genetic backgrounds have been identified.

Additional keywords: root-lesion nematodes, bread wheat, molecular markers, MAS, composite interval mapping, QTL Cartographer, W-7984, Opata 85.

Introduction

Root-lesion nematode (*Pratylenchus thornei* Sher and Allen) causes serious yield reduction in Australian wheat (*Triticum aestivum* L.) with average losses estimated at $AU36 M annually (Brennan and Murray 1998). *P. thornei* is a migratory, vermiform nematode about 0.7 mm long that penetrates the root cortex, where it feeds, reproduces, and disrupts root function (Loof 1991). The nematode passes through its lifecycle in about 6 weeks under favourable conditions. High populations of *P. thornei* (>2000/kg soil) present at sowing can severely affect plants, which may show symptoms of nutrient deficiencies and water stress.

Pratylenchus thornei can occur in large numbers throughout the soil profile, which makes nematicide application uneconomical at the rates necessary for effective control (Thompson *et al*. 1982; Vanstone *et al*. 1998). Currently, the best control strategy is an integrated approach that combines the use of tolerant and resistant crop varieties, crop rotation, and farm hygiene. Although commercial wheat varieties with superior tolerance to *P. thornei* are available, these varieties still allow nematodes to multiply in their roots and leave a residual nematode population in the soil to attack subsequent crops (Thompson *et al*. 1999). So the key component of this strategy to manage nematodes is the use of resistant varieties that prevent nematode reproduction.

Current Australian wheat varieties have poor levels of resistance to *P. thornei* and backcrossing is being pursued to incorporate resistance into commercial varieties. GS50a, a selection from the Australian bread wheat variety Gatcher, is the most widely used source of *P. thornei* resistance (Thompson *et al*. 1999). Extensive screening for new sources of resistance to *P. thornei* has identified several landrace wheats, namely AUS4930 (Iraq 48) (Nicol *et al*. 1998) and AUS13124 (Morocco 426) and AUS4926 (Iraq 43) (Seymour and Thompson 2001) with resistance levels equivalent to or better than that of GS50a (Zwart *et al*. 2004*b*). Other sources of resistance to *P. thornei* have been found in the wild grass *Aegilops tauschii* Coss. (2n = 14, DD) (Thompson and Haak 1997), in durum wheats *Triticum turgidum* L. subsp. *durum* (Desf.) Husn (2n = 28, AABB) (J. P. Thompson and M. I. Haak, unpublished data), and in synthetic hexaploid wheats produced from their hybridisation (*T. aestivum*, 2n = 42, AABBDD) (Zwart *et al*. 2004*a*).

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Recent molecular marker studies have identified putative quantitative trait loci (QTLs) for resistance to *P. thornei* on chromosomes 2B (Schmidt *et al.* 2005; Zwart *et al.* 2005), 3B (Schmidt *et al.* 2005), and 6A and 6D (Zwart *et al.* 2005), and a QTL for susceptibility to *P. thornei* on 1B (Schmidt *et al.* 2005). These mapping studies were conducted using framework maps with a minimum number of marker loci used to detect marker–trait associations. The numbers of marker loci and genome coverage of these maps were 114 markers over 1987 cM for AUS1312 × Janz, 148 markers over 3230 cM for AUS4926 × Janz (Schmidt *et al.* 2005), and 169 markers over 2570 cM for CPI133872 × Janz (Zwart *et al.* 2005).

In comparison, the International Triticeae Mapping Initiative (ITMI) population (W-7984 × Opata 85), the most densely mapped wheat population available internationally, has 1406 marker loci and a total genome coverage of 2654 cM (Song *et al.* 2005). A considerable number of marker loci on the ITMI genetic linkage map have been physically mapped into deletion bins (Erayman *et al.* 2004; Sourdille *et al.* 2004). The high marker density and extensive genome coverage of both the genetic linkage and physical consensus maps, in addition to the segregation of the recombinant inbred lines (RILs) for resistance to *P. thornei* and molecular marker data being publicly available from GrainGenes, make this population an ideal resource for the identification of QTLs associated with *P. thornei* resistance.

The objective of this study was to utilise the available information on genetic linkage and physical maps of wheat in order to locate QTLs associated with resistance to *P. thornei* in the ITMI population.

Materials and methods

Mapping population

The ITMI population consists of a total of 150 RILs derived by single-seed descent from the cross of the synthetic hexaploid wheat W-7984 (also designated M6, P. McGuire, pers. comm.) and the hard red spring wheat cultivar Opata 85 (Nelson *et al.* 1995*a*). The synthetic hexaploid wheat was originally reported as produced from the hybridisation of durum wheat cultivar Altar 84 and *Aegilops tauschii* accession CI 18 (Nelson *et al.* 1995*a*). This synthetic wheat is the same as CIMMYT Synthetic ID No. 48 and CIGM86.940 in appendix 2 of Mujeeb-Kazi (1995) in which the pedigree is given as durum Altar 84 crossed with *Ae. tauschii* (CIMMYT WX 219, syn. TA2465). However, there is uncertainty that Altar 84 is the durum component of the synthetic hexaploid (Singh *et al.* 2000). Seeds of the F_7 generation of RILs were kindly provided by Dr P. J. Sharp, University of Sydney, Australia, for phenotypic assessment of *P. thornei* resistance in 1997. The RILs were later reselected to eliminate morpho-variants and advanced to the F_{11} generation at Cornell University, USA. Seeds of the reselected ITMI population were provided by Dr K. J. Chalmers, University of Adelaide, Australia, for phenotypic assessment of *P. thornei* resistance in 2001.

Phenotypic assessment of P. thornei *resistance*

Resistance to *P. thornei* was assessed in the glasshouse using randomised block design experiments, repeated twice. In the first experiment, conducted in 1997, 3 replicates of a subset of 91 RILs were assessed for resistance to *P. thornei* using the procedure described by Thompson *et al.* (1999). In the second experiment, conducted in 2001, 4 replicates of 129 RILs were assessed for *P. thornei* resistance based on nematode counts in soil and roots as described by Zwart *et al.* (2004*a*). Count data were transformed by $\ln(x + c)$, where x is nematodes/kg soil and roots sampled, and c is constant. The value of c was optimised for each experiment using chi-squared principles to normalise the data (Proctor and Marks 1974; Berry 1987). Analysis of variance was performed using GENSTAT 6.1 (Payne *et al.* 2002) and mean squares were used to estimate heritability of *P. thornei* resistance in the ITMI population (Hartl *et al.* 1988).

Linkage map

The marker segregation data for 114 RILs was obtained from the GrainGenes database (http://wheat.pw.usda.gov). QTL analysis was performed on a subset of 537 restriction fragment length polymorphism (RFLP) markers (Van Deynze *et al.* 1995; Nelson *et al.* 1995*a*, 1995*b*, 1995*c*; Marino *et al.* 1996) and simple sequence repeat (SSR) markers (gwm, Röder *et al.* 1998; and barc, Song *et al.* 2005). The marker order in the QTL regions, initially based on the consensus map of Somers *et al.* (2004), was adjusted with Mapmaker software (Lander *et al.* 1987). The genetic linkage map was compared with the consensus wheat physical maps (Erayman *et al.* 2004; Sourdille *et al.* 2004; Shah and Hassan 2005) to reveal the physical locations of the QTLs for *P. thornei* resistance.

QTL analysis

QTL analysis was performed on RIL means from the ANOVA of transformed nematode counts for each year, as well as on the individual replicate data. The locations and effects of QTLs were determined by composite interval mapping using the computer program QTL Cartographer v2.5 (Wang *et al.* 2005). The threshold LOD score for detection of QTL at $P = 0.05$ was calculated for each set of phenotypic data using 1000 permutations (Churchill and Doerge 1994). The proportion of observed phenotypic variation explained due to a particular QTL was estimated by the coefficient of determination (R^2). Composite interval mapping was performed using forward stepwise regression with a window size of 10 cM and background control set at 5 markers.

Results

Phenotypic assessment of P. thornei *resistance*

The 2 parents of the ITMI population differed significantly in resistance to *P. thornei* (Table 1), with the synthetic hexaploid parent, W-7984, resistant and the bread wheat parent, Opata 85, susceptible. W-7984 showed a resistance level equivalent to (1997) or better than (2001) the resistance level of GS50a, the current standard for *P. thornei* resistance. The RILs population segregated in a continuous distribution for resistance to *P. thornei* (Fig. 1). Mean nematode counts of the RILs ranged from 4850 to 120 556 *P. thornei*/kg soil and roots in 1997 and 4592 to 367 962 in 2001. The heritability estimates for *P. thornei* resistance were 0.83 and 0.84 in 1997 and 2001, respectively. There was significant correlation between nematode counts in the 2 years of phenotyping ($r = 0.37$, d.f. = 86, $P < 0.001$). In 1997, 52 RILs showed a resistance level higher than or equivalent to the resistant parent, W-7984. In 2001, 35 RILs showed a resistance level higher

Table 1. The parents, W-7984 and Opata 85, differed significantly in resistance to *P. thornei*
Mean transformed nematode counts for the 2 years of phenotyping are shown. Resistance level to *P. thornei* determined by glasshouse phenotyping trials: R, resistant; S, susceptible. Equivalent means are shown in parentheses as number of *P. thornei*/kg soil plus roots after 16 weeks growth

Genotype	Resistance category	1997 ln(*P. thornei*/kg + 3500)	2001 ln(*P. thornei*/kg + 9038)
W-7984	R	9.90 (16 398)	9.54 (4867)
GS50a	R	9.99 (18 356)	10.47 (26 204)
Opata 85	S	11.34 (84 329)	11.85 (131 046)
Janz	S	11.40 (89 618)	11.72 (113 969)
l.s.d. ($P = 0.05$)		0.76	0.84

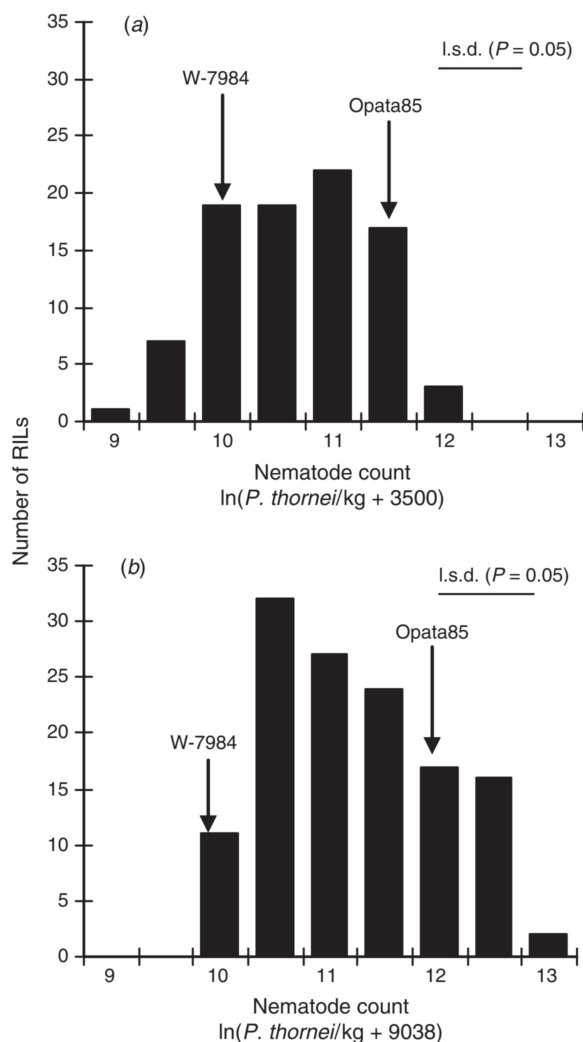

Fig. 1. Resistance to *P. thornei* in W-7984 × Opata 85 ITMI population is continuously distributed. (*a*) Frequency distribution of recombinant inbred lines (RILs) for resistance to *P. thornei* in the 1997 glasshouse experiment. The horizontal bar represents l.s.d. ($P = 0.05$) of 0.76. The transformed nematode count for the resistant parent, W-7984, was 9.90 and for the susceptible parent, Opata 85, was 11.38. The population mean was 10.44 and variance was 0.45. (*b*) Frequency distribution of RILs for resistance to *P. thornei* in the 2001 glasshouse experiment. The horizontal bar represents l.s.d. ($P = 0.05$) of 0.84. Transformed nematode count for W-7984 was 9.54 and for Opata 85 was 11.85. The population mean was 10.95 and variance was 0.61.

than or equivalent to W-7984. Of these lines 17 RILs showed a resistance level consistently higher than or equivalent to the resistance level of W-7984 in both years of *P. thornei* phenotyping.

QTL analysis

QTL analysis on individual replicate data resulted in similar QTL peak locations and the results of the pooled means for each year are reported. Significant QTL associations with resistance to *P. thornei* were detected on the short arms of chromosomes 6D and 2B (Fig. 2). In both cases, the marker alleles conferring resistance in these QTL regions were inherited from the synthetic hexaploid parent, W-7984.

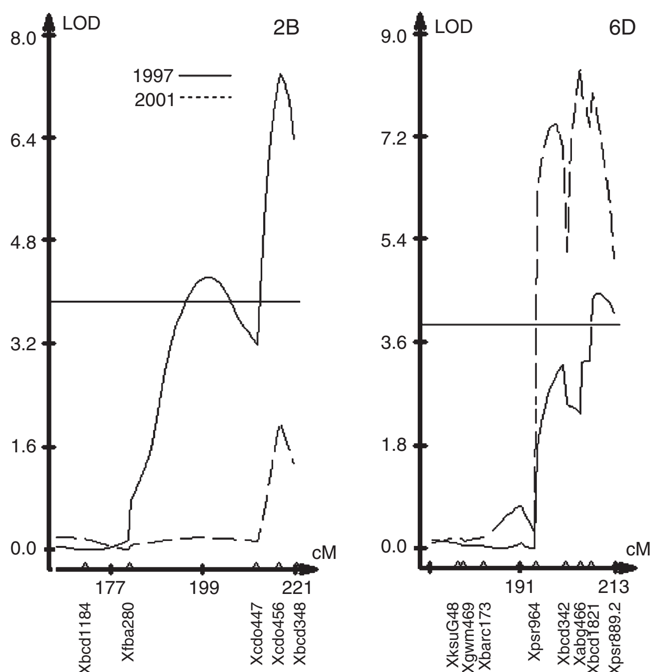

Fig. 2. Composite interval mapping (CIM) contours showing QTL for resistance to *P. thornei* in W-7984 × Opata 85 ITMI population on the short arms of chromosomes 2B and 6D. Numbers on horizontal axes represent distances in cM from the end of the long arms of the chromosomes. The threshold line set by permutation tests for significance (95%) is shown at LOD 3.8 in both plots.

The QTL on the distal end of chromosome arm 6DS explained 11% and 23% of the phenotypic variation for resistance to *P. thornei* in 1997 and 2001, respectively. One-LOD confidence intervals for the 2 years overlap. Based on the large 2001 effect, the QTL lies near RFLP marker locus *Xbcd1821* (Fig. 2). The closest SSR marker locus, *Xbarc183*, is about 6 cM proximal but could not be reliably (LOD 2) placed.

A second QTL on chromosome 2BS explained 19% and 5% of the phenotypic variation for resistance to *P. thornei* in 1997 and 2001, with the peak LOD value for the QTL found at RFLP marker locus *Xcdo456* and the effect falling short of the LOD significance cut-off in 2001. A 1-LOD confidence interval localised the QTL in the 1997 data in a 3-cM region that encloses SSR marker locus *Xgwm210*, a marker not used in the QTL scan because it could not be reliably (LOD 2) placed on the map. The group of markers on the distal end of chromosome 2BS, initially reported as linked to the distal end of chromosome 2AS (Nelson *et al.* 1995*b*), was later mapped to the distal end of chromosome 2BS (Röder *et al.* 1998; Langridge *et al.* 2001).

Discussion

Two QTLs for resistance to *P. thornei* in the ITMI population have been detected on chromosomes 6DS and 2BS and their locations tightly defined by flanking molecular markers. Both showed strong effects in one evaluation year and strong or suggestive effects in a second year. For both regions, the QTL resistance alleles were inherited from the synthetic hexaploid parent, W-7984. This indicates that both the B genome of the durum and the D genome of the *Ae. tauschii* parent of the synthetic hexaploid contributed to its *P. thornei* resistance.

Both QTLs lie in regions rich in major genes on the distal end of short chromosome arms. RFLP marker loci *Xpsr964* and *XksuG48* (Erayman *et al.* 2004) and SSR marker loci *Xbarc173*, *Xbarc183*, and *Xgwm496* (Sourdille *et al.* 2004) have been physically mapped using deletion lines to the 10-Mb deletion bin 6DS-0.99-1.00, which contains 2 putative R genes (Dilbirligi *et al.* 2004). RFLP marker locus *Xbcd348* (Erayman *et al.* 2004) and SSR marker locus *Xgwm210* (Sourdille *et al.* 2004) have been physically mapped to the 39-Mb deletion bin 2BS-7-0.89-1.00, which contains 7 putative R genes (Dilbirligi *et al.* 2004). Genes for other agronomically important traits, including stem rust resistance (*Sr5*, *Sr29*), are located on chromosome 6DS (McIntosh *et al.* 2003). It is interesting that the 6DS *P. thornei* resistance QTL lies very near a defence response gene locus encoding a ribosome-inactivating protein (*Rip*) isolated from maize (Li *et al.* 1999). Ribosome-inactivating proteins have been reported to act on a number of plant pests and pathogens and are presumed to play an important role in defence against pathogens (Krawetz and Boston 2000).

Comparison of maps using common markers revealed that SSR marker locus *Xbarc183* on chromosome 6DS was associated with resistance to *P. thornei* in this study as well as in a second mapping study in which resistance was contributed by a different synthetic hexaploid parent, CPI133872 (Zwart *et al.* 2005). The 6DS QTL contributed by CPI133872 explained 24% of the variation in resistance, which is similar to the value reported here. QTLs on chromosome 2B for resistance to *P. thornei* have been detected in other mapping populations (Schmidt *et al.* 2005; Zwart *et al.* 2005). However, due to a lack of common markers on the 2BS region, comparisons between the location of the QTL identified in this study and other *P. thornei* resistance QTL studies could not be made. *QRlnt.lrc-2B.1* was associated with SSR marker locus *Xwmc025b* and explained 7% of the variation for *P. thornei* resistance in a CPI133872 × Janz population (Zwart *et al.* 2005). In an AUS13124 × Janz population a QTL on chromosome 2B, flanked by SSR marker loci *Xgwm319* and *Xgwm494.2*, explained 14% of the variation for *P. thornei* resistance (Schmidt *et al.* 2005).

The synthetic hexaploid wheat lines W-7984 and CPI133872, which both have QTLs for *P. thornei* resistance in similar locations on chromosomes 6DS and 2BS, were originally developed at the International Maize and Wheat Improvement Centre (CIMMYT), Mexico. The 6DS and 2BS QTLs in W-7984 may be allelic to *QRlnt.lrc-6D.1* and *QRlnt.lrc-2B.1* identified in CPI133872, although there is no direct evidence for this as the wheat lines were derived from different durum and *Ae. tauschii* lines. The pedigree of CPI133872 (CIMMYT Synthetic ID No. 177 and CIGM89.576 in appendix 2, Mujeeb-Kazi 1995) is durum 68.111/Rugby//Ward/3/Flamingo/4/Rabicorno (syn. CPI 133821) crossed with *Ae. tauschii* (CIMMYT WX 949; syn. TA2525 and AUS24199).

The consistency and size of the heritability estimates indicate that much of the phenotypic variation for resistance to *P. thornei* in the ITMI population is accounted for by genetic variation. Together with the QTL mapping results, this suggests that much of the resistance to *P. thornei* is controlled by a few loci with relatively large effects in this population. The coincidence in locations of QTLs associated with SSR marker loci *Xbarc183* on chromosome 6DS, found in different mapping studies, suggests the utility of this marker for use in marker-assisted selection with material from different genetic backgrounds. It also suggests the importance of investigations for alternative sources of resistance to *P. thornei* so that resistance genes conferring partial resistance can be pyramided.

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