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ORIGINAL PAPER

Identification and molecular mapping of a Fusarium wilt resistant gene in upland cotton

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Abstract Fusarium wilt (FW) is one of the most economically damaging cotton diseases worldwide, causing yellowing, wilting, defoliation, vascular tissue damage and ultimately death. Identification of molecular markers linked to FW genes is vital to incorporate resistance into elite cotton cultivars. An intraspecific F_2 in *Gossypium hirsutum* L. was developed by crossing with a highly resistant cultivar Zhongmiansuo 35 (ZMS35) and a susceptible cultivar Junmian 1 to screen simple sequence repeats (SSRs) closely linked to the FW resistance gene. FW was identified in $F_{2:3}$ families by evaluating seedling leaf symptoms and vascular tissue damage at plant maturity under natural field infection conditions over 2 years. The results showed that FW resistance segregated in a 3:1 ratio as a simple monogenic trait in $F_{2:3}$ families. Molecular mapping identified a FW resistance gene closely linked with the SSR marker JESPR304$_{-280}$ in chromosome D3(c17). We proposed to name this gene FW^R. A composite interval mapping method detected four QTLs for FW resistance in Chr.A7(c7), D1(c15), D9(c23) and D3, respectively. Among them, one major QTL (LOD > 20) was tagged near marker JESPR304 within an interval of 0.06–0.2 cM, and explained over 52.5–60.9% of the total phenotypic variance. The data confirmed the existence of a major gene in Chr.D3. This is the first report of molecular mapping of a major gene contributing FW resistance in cotton. The present research therefore provides an opportunity to understand the genetic control of resistance to FW and conduct molecular marker-assisted selection breeding to develop FW resistant cultivars.

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Introduction

Fusarium wilt (FW) is caused by the fungal pathogen, *Fusarium oxysporum* f. sp. *vasinfectum* (*Fov*), and is a widespread soil pathogen infecting cotton world-wide. The species is subdivided into six races (1 through 6) based on the virulence level to different species and cultivars of cotton and other crops (Armstrong and Armstrong 1980). *Fov* was first reported in China in 1931, and has subsequently spread to all major cotton growing districts (Ma and Chen 1992; Huang 2007). China is the world's largest producer of raw cotton, growing a total of approximately 5.5 million hectares, and the areas infected with *Fov* cover nearly 1.5 million hectares of the total growing region (Ma et al. 2002). In Xinjiang, one of the three largest cotton-growing regions in China, a survey conducted in 1995 by the Plant Protection Station, Xinjiang Academy of Agricultural Sciences, reported that infection with FW disease had reached 46 thousand hectares. Three *F. oxysporum* f. sp. *vasinfectum* races have been reported in China, including races 3, 7 and 8. Races 7 and 8 were first reported, and race 7 is the most widely distributed in the country (Chen et al. 1985; Shi 1996).

Throughout the world, with the exception of China and Australia, FW is commonly associated with root knot nematode (RKN) infection (RKN–FW complex) (Hillocks 1984; Davis et al. 1996; Katsantonis et al. 2003;

Ogallo et al. 1997); and it is likely that some genetic studies have confused resistance to FW with resistance to RKN (Shen et al. 2006). Therefore, it is plausible the mechanisms of *Fov* pathogenicity and the inheritance patterns of FW resistance in cotton differ significantly among geographic regions. Studies of FW resistance can serve as a model system to increase our understanding of the genetic control of disease. Smith and Dick (1960) determined that two dominant genes with additive effects dictated a high degree of resistance to FW in Seabrook Sea Island cotton. One of these two dominant genes was transferred to Upland cotton. Wang et al. (2006a) and Shen et al. (2006) reported the molecular markers closely linked with gene resistance to nematodes in cotton. Furthermore, map-based cloning and characterization of FW resistance have been reported in melon (Wang et al. 2000, 2002), ginger (Swetha and Subramanian 2008), and tomato (Simons et al. 1998). However, molecular mapping of FW resistance gene(s) and/or quantitative trait locus (QTL) have not yet been reported in cotton.

The objectives of the present study were to carry out a molecular mapping analysis to characterize genomic regions involved in resistance to FW. Ultimately, research elucidating the molecular basis of FW resistance will facilitate molecular marker-assisted selection (MAS) breeding to develop resistant cultivars and isolate the resistance genes through map-based cloning. Genetic mapping of FW resistance gene(s) in upland cotton should serve to elucidate the similarities and differences between FW and RKN resistance genes.

Materials and methods

Plant materials

Two cultivars widely planted in the northwestern cotton-growing region of China were chosen for this study. Zhongmiansuo35 (ZMS35) exhibits high resistance to FW, and is currently widely planted, and Junmian 1 was a cotton variety of choice in the past, but was replaced by ZMS35 due to its susceptibility to FW (Huang 2007). In 2003, ZMS35 and Junmian 1 were crossed in Jiangpu Breeding Station, Nanjing Agricultural University (JBS/NAU), Nanjing, China. The subsequent F_1 seeds were planted and self-pollinated in Hainan Island to produce F_2 progeny. In 2004, the F_2 seeds were planted in JBS/NAU and self-pollinated to produce 154 $F_{2:3}$ family seeds.

Concurrently, a resistant cultivar Sumian 10, formerly planted in the Yangtze River cotton region, was chosen to cross with a susceptible cultivar, Changrongmian, at JBS/NAU in 2003. The F_1 seeds were subsequently planted in Hainan Island to produce self-pollinated F_2 progeny. In 2004, the F_2 seeds were planted at the Cotton Breeding Station at Shihezhi, Xinjiang Condy Agriculture Science and Technology Development Co. Ltd (CBSS/XJ) and self-pollinated to produce 163 $F_{2:3}$ family seeds.

The parental, (ZMS35 × Junmian 1) F_1 and $F_{2:3}$ families were planted in a FW nursery at the CBSS/XJ. The FW nursery was inoculated with Race 7, a local populated race. The Fov race was made available in the Department of Plant Protection, Xinjiang Agricultural University (Xinjiang, China). The FW nursery was inoculated with wheat seed cultured with Race 7 with an amount of 0.5% of the total weight of farming soil each year until field was heavily and evenly infested with Fov. Before planting, the field was inoculated again with same amount of FW pathogen. Plant materials were planted in a randomized block design with two replications at CBSS/XJ in 2005 and 2006. (Sumian 10 × Changrongmian) F_1 and the $F_{2:3}$ families were only planted in 2006 at the same location and applying the same methodology. Each experimental replication included one 5.0 m long row; 50 cm between rows; and 12 cm between plants in each a row. The resulting sample size per each $F_{2:3}$ family line (i.e., replication) was 48 plants.

Survey of disease resistance

FW disease resistance in F_2 lines was evaluated by a $F_{2:3}$ family line survey over 2 years during different plant developmental stages. Twenty-five plants from each $F_{2:3}$ family were selected to evaluate plant resistance. Plants were scored at the seedling stage in June, approximately 5 weeks after planting. Leaf symptoms were evaluated to establish seedling resistance. In addition, vascular tissue symptoms were scored just prior to the harvesting season to investigate plant resistance at maturity. The leaf and vascular tissue damage in seedlings and at maturity were classified into five grades following China's national scoring standard for FW resistance (Wu et al. 2002). A disease grade of 0, 1, 2, 3 and 4 for leaf disease symptoms were as follows: grade 0, healthy, no disease symptoms; grade 1, ≤25.0% of the leaf surface exhibited disease symptoms; grade 2, 25.1–50.0% of the leaf surface exhibited disease symptoms or were slightly dwarfed in stature; grade 3, 50.1–90.0% of the leaf surface exhibited disease symptoms and were obviously dwarfed in stature; and grade 4, >90.0% of the leaf surface exhibited disease symptoms, and plants were completely defoliated or had died. The grade criterion for vascular disease symptoms was as follows: grade 0, healthy, absence vascular disease symptoms; grade 1, <25% of the vascular tissue area had browned; grade 2, 25–50% of the vascular tissue area had browned; grade 3, 50.1–75% of the vascular tissue area had browned; and

grade 4, >75% of the vascular tissue area had browned. A disease index was calculated by averaging the disease grade score divided by 4.

The disease indices of parents and hybrid F_1 and $F_{2:3}$ families were slightly higher in 2006 than 2005 at the mature stage, so the relative FW disease indices of the $F_{2:3}$ family lines in 2006 were used. The relative FW disease index in 2006 was calculated by multiplying its original disease index of the $F_{2:3}$ family lines in 2006 with the relative disease index ratio (the average disease index of $F_{2:3}$ family lines in 2005/the average disease index of $F_{2:3}$ family lines in 2006).

DNA extraction and PCR reaction

Cotton DNA was extracted following Paterson et al. (1993) and modified by excluding DIECA from the DNA extraction buffer. Simple sequence repeat (SSR) PCR amplifications were performed in the following reaction mixture: 67 mM Tris–HCl (pH 8.8), 16 mM $(NH_4)_2SO_4$, 2.5 mM $MgCl_2$, 0.2 mM dNTPs, 0.6 M primers, 0.25–0.5 U of Taqase (Sangon, Shanghai, China) and 20 ng of genomic DNA per 10 µl of reaction mixture using a Thermal Cycler 9600 (Perkin-Elmer). PCR conditions included one cycle of 95°C for 2 min; 30 cycles of 94°C for 45 s, 57°C for 45 s and 72°C for 60 s; and one cycle of 72°C for 7 min. A non-radioactive protocol to detect SSR polymorphisms was conducted as reported by Zhang et al. (2002).

Data analysis

We used 5400 SSR primers which are available from CMD (http://www.cottonssr.org). Markers were first screened from the parents and their F_1 progeny to detect polymorphisms, and subsequently used to genotype the F_2 plants. A chi-square analysis was used to analyze marker data to test for goodness-of-fit to an expected segregation ratio. Linkage maps were constructed by MAPMAKER/Exp Version 3.0b Software (Lander et al. 1987); and linkage groups were identified by pairwise comparisons. Marker groupings were determined using the "Group" command at a maximum recombination fraction of 50 cM and a minimum LOD score greater than 4.0. Marker order was confirmed with the "ripple" command. Recombination frequencies were converted into map distances (cM) using the Kosambi mapping function. Windows QTL Cartographer 2.5 (Basten et al. 2001) was applied to identify QTLs by composite interval mapping (CIM) (Zeng 1994). QTLs were determined significant if the corresponding likelihood ratio (LR) score was greater than 11.5 (equal to a LOD score of 2.5). The percent phenotypic variance (PV) explained by a QTL was estimated at the highest probability peaks.

Chromosome assignments

The SSR loci chromosome assignments were used as bridge loci with existing linkage maps (Han et al. 2006; Wang et al. 2006b; Guo et al. 2007) by checking the molecular sizes of the parental alleles. QTL nomenclature was adapted from McCouch et al. (1997) in rice, which begins with "*q*", followed by an abbreviation of the trait name, the name of the chromosome or linkage group, and then followed by the QTL number affecting the trait on the chromosome or linkage group (QTL + trait + number). For example, qFW-1 designates the identified FW resistance QTL.

Results

FW resistance performance and its inheritance analysis

Plant disease response in both the parental and hybrid progeny (F_1 and $F_{2:3}$) families was evaluated for leaf symptoms in seedlings and vascular tissue evaluation in mature individuals (Table 1). In 2005, the ZMS35 disease index was 5.3 at the seedling stage, and 6.3 and 10.3 at plant maturity in 2005 and 2006. These data indicated that the genotype

Table 1 Basic statistic characteristics of FW resistance in parents, F_1 and $F_{2:3}$ in 2 years

Parents and populations	Index of disease %		
	The seedling stage (2005)	The mature stage (2005)	The mature stage (2006)
Junmian1	45.20	75.50	80.40
ZMS35	5.30	6.30	10.30
F_1	7.30	8.20	15.40
$F_{2:3}$	17.50	20.80	27.76
Changrongmian			78.10
Sumian 10			4.70
F_1			6.90
$F_{2:3}$			28.80

exhibited significant disease resistance. The disease index for parents and families at the 2006 seedling stage was not obtained due to dry conditions unsuitable for FW growth. The disease index for Junmian 1 (45.2–80.4) and its hybrid F_1 (7.3–15.4) showed high susceptibility in Junmian 1, while the F_1 hybrid was highly resistant. A dominant FW pattern of inheritance may be operating at the seedling and mature stages in *G. hirsutum*. The average disease index in vascular tissue for the $F_{2:3}$ was 20.8 and 27.7 in the respective study years. The disease index distribution for these populations is shown in Fig. 1. Those plants with the standard disease index of ≤15.4 were classified as resistant, and a disease index of >15.4 as susceptible since the F_1 disease index was 7.3–15.4 in 2005. Following this classification standard, the $F_{2:3}$ family populations (ZMS35 × Junmian1) fit a 3:1 (113R:40S, $\chi^2 = 0.027$) resistance and susceptible segregation ratio.

Results found the disease index in the parents and hybrid F_1 and $F_{2:3}$ families was slightly higher in 2006 than that in 2005. The average disease index of $F_{2:3}$ family lines in 2006 were higher by 25% than that in 2005, so the relative FW disease indices of the $F_{2:3}$ family lines in 2006 were used in order to make the same level analysis between 2 years. The relative FW disease indices of the $F_{2:3}$ family lines in 2006 is calculated by multiplying its original disease index with the relative FW disease index ratio 0.75. Based on the $F_{2:3}$ adjusted data sets for the 2006 $F_{2:3}$ family lines, resistance also fit a 3:1 (115R:38S, $\chi^2 = 0.005$) segregation ratio following the same classification standard as that in 2005.

(Sumian 10 × Changrongmian) $F_{2:3}$ families produced the same 3:1 segregation pattern (119R:41S, $\chi^2 = 0.891$). These data further supported results from the present study suggesting that a single dominant gene governs resistance. We proposed to name this dominant resistance gene as FW^R (Kohel 1973).

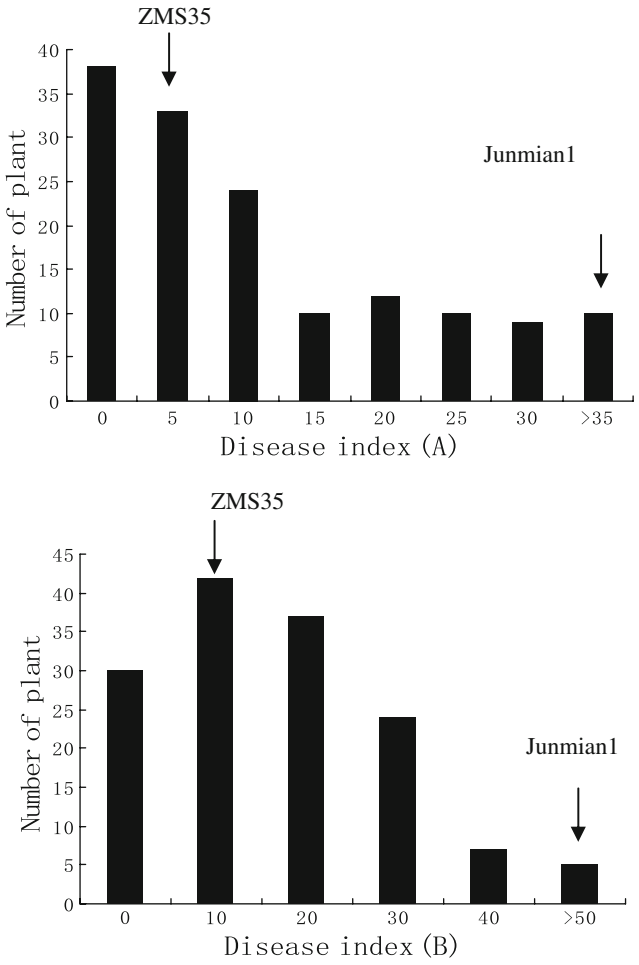

Fig. 1 The frequency distribution of disease index of FW in 2005 (**a**) and 2006 (**b**) in ZMS35 × Junmian 1 $F_{2:3}$ family lines Note: Average disease index values for parents (ZMS35 and Junmian1) are indicated by *arrows*

Molecular mapping of the major FW resistance gene FW^R

Polymorphisms between the two parents were screened using 5400 SSR primer pairs, which detected 124 polymorphic loci. Segregation at each locus was tested for deviations from expected Mendelian segregation ratios for 1:2:1 or 3:1, and 11 of 124 significant deviations were identified by chi-square tests. FW^R and the 124 polymorphic loci were used to construct a genetic linkage map based on 154 (ZMS35 × Junmian1)F_2 individuals. Twenty linkage groups were constructed with 75 loci, and covered 743.1 cM, with an average distance between two markers of 9.91 cM. Based on our saturated linkage map (Guo et al. 2007), the 20 linkage groups could be assigned to their corresponding chromosome except Chr.A4, A10, A11, A13, D16, D10 and D22. The FW^R gene was assigned to Chr. D3 with an interval genetic distance of 5.30 cM from JESPR304, which amplified a 280 bp band (Fig. 2).

The $JESPR304_{-280}$ SSR marker was subsequently used to conduct a linkage test based on (Sumian 10 × Changrongmian)$F_{2:3}$ families. The analysis confirmed the linkage relationship between JESPR304 SSR and FW^R with a genetic distance of 0.80 cM.

QTL mapping for the FW resistance gene in Upland cotton

The observed resistance level frequencies under natural *Fov* fungus infection pressures did not conform to a normal distribution. Therefore, the disease index data were transformed using square root transformation (SQRT) to meet a normal distribution pattern. Based on a CIM analysis of the 2005 and 2006 data sets of adjusted $F_{2:3}$ measures of disease reaction, six FW resistance QTL were detected and located on Chr.D1, A9, D9, and D3. The significant LOD score peak values, peak position, the percent PV explained

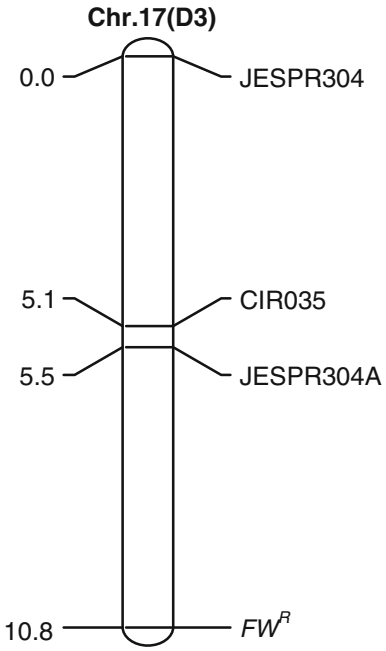

Fig. 2 Position of FW^R resistance gene in Chr.D3

and the estimate of QTL effects are summarized in Table 2 and Fig. 3. The LOD thresholds for the four QTL ranged from 3.89 to 22.17 and PV from 15.9 to 60.9%. Four FW resistance-QTL were detected and located on different chromosomes at the seedling stage in 2005. Two of the same QTL were detected in mature cotton in both years at the same location. A major resistance QTL (*qFW-D3-1*) was detected tightly linked to $JESPR304_{-280}$. The QTL exhibited a genetic distance from 0.06 to 0.22 cM and explained 52.5–60.9% of the phenotypic variance from three different developmental stages. The resistance QTL originated from the resistant parent (ZMS35). This major QTL was reproducible and detected independently in the same genetic region, therefore it exhibited increased reliability. The major QTL may represent FW^R because both were detected in the same region.

A second QTL (*qFW-D9-1*) explained 15.9% PV, and was located on Chr.D9(c9). *qFW-D1-1* was located on Chr.D1, explained 25.5% of the PV and the resistance allele originated from the susceptible parent (Junmian 1). The fourth QTL (*qFW-A7-1*) was located on Chr.A7 between NAU474 and NAU1048 markers, and explained 25.4% of the PV. It is interesting that the QTL conferring resistance to nematodes was also located between these same two markers (Shen et al. 2006).

Discussion

Fusarium wilt is one of the most economically damaging agricultural crop diseases worldwide. Control measures applied to affected fields are often impractical and expensive. However, natural resistance to FW has been well documented in many crops. The mechanism conferring FW resistance has been intensively examined in cotton. Smith and Dick (1960) determined that two dominant genes with additive effects dictated FW resistance in Seabrook Sea Island cotton, and one gene was transferred to Upland cotton. Successive genetic analyses suggested that FW resistance in cotton was determined by one or two major genes with incomplete dominance (Feng et al. 1996; McPherson et al. 2004; Ulloa et al. 2006). Molecular tagging of gene(s) or QTL conferring FW resistance has not been previously reported in cotton, and marker assisted selection is a reliable and faster method than classical field screening. Furthermore, screening for disease resistance genes using molecular markers offers the additional advantage of permitting selection for resistance in the absence of the pathogen. Therefore, the availability of PCR-based markers will allow MAS breeding to efficiently develop FW resistant cultivars. Given the presumed major gene control of FW resistance in cotton species, introgressing FW resistance genes into cotton cultivars can be easily achieved. In the present study, our objectives were to determine the mode of

Table 2 Estimates of QTL positions and effects of FW resistance in ZMS35 × JunMian1 population

QTL	Periods	Left mark	Chr.	Map position (cM)[a]	LOD score[b]	Additive effect	Dominant effect	Variance explained[c] %
qFW-D1-1	June2005	BNL830	D1	0.520	4.91	14.37	−14.98	25.5
qFW-A7-1	June2005	NAU474	A7	2.138	7.24	17.31	−16.63	25.4
qFW-D9-1	June2005	NAU423	D9	0.133	3.89	−13.9	−14.65	15.9
qFW-D3-1	June2005	JESPR304	D3	0.220	20.62	−16.07	−18.79	58.3
qFW-D3-1	Nov2005	JESPR304	D3	0.120	21.43	−13.20	−18.20	52.5
qFW-D3-1	Nov2006	JESPR304	D3	0.060	22.17	−18.72	−17.04	60.9

[a] Position of peak LOD score

[b] Peak LOD score in the QTL

[c] Percentage of variance explained at peak LOD score

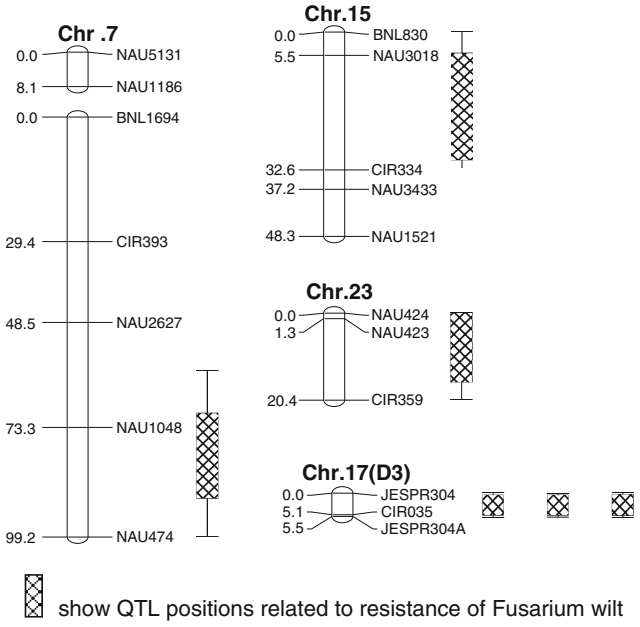

Fig. 3 Significant QTL related to FW resistance (LOD > 3.0)

inheritance and genomic location of genes or QTL conferring FW resistance. Resistance segregation was based on a simple trait in two mapping populations. The FW^R gene was detected on Chr.D3 with a genetic distance 0.8–5.30 cM from $JESPR304_{-280}$. One major QTL associated with FW resistance was found also on Chr.D3, and consistently identified in seedlings with leaf symptoms and in vascular tissue of mature plants from both study years (Fig. 3). This QTL explained approximately 52.5–60.9% of the phenotypic variance, providing additional evidence of one major gene coding for FW resistance. The present genetic and molecular mapping research confirmed previous genetic analyses of FW resistance in cotton. The resistance mechanism discerned in cotton is consistent with the mode of inheritance in other crop species, including melon (Brotman et al. 2005); tomato (Hemming et al. 2004), *Lens* sp.(Hamwieh et al. 2005), chickpea (Sharma et al. 2004), and eggplant (Mutlu et al. 2008). It is not known whether the basis of resistance observed in the above crops is a model that can be applied to *G. hirsutum*, but it is possible related genes are involved. It is likely the genetics of resistance in one species may provide insights to unravel the mode of resistance in other species.

The RKN is the most damaging endoparasitic pest of cotton. Its feeding causes root damage and extensive galling, resulting in reduced water and nutrient translocation (Bridge 1992). It is estimated that RKN reduces cotton yield by 10.7% worldwide. In addition to the direct damage from RKN, the nematode forms a disease complex with the FW pathogen and greatly increases the incidence and severity caused by FW. The role of RKN in the establishment of *Fusarium* has been known for many years. Reports by Hyer et al. (1979) and Shepherd (1970) suggested the gene(s) conferring resistance to FW and RKN are different. For instance, 'N6072' cotton line is susceptible to FW but resistant to RKN, while 'Delcot 277' is wilt tolerant but susceptible to root-knot. Furthermore, nematode resistance is more important than fungal resistance in controlling the nematode–fungus disease complex. RKN resistance in cotton exhibits simple inheritance and is governed by one to a few major genes (Bezawada et al. 2003; McPherson et al. 2004). It has been reported that a RKN resistance gene (rkn1) in Acala Nem X and/or a major QTL accounted for 63.7% of the total phenotypic variation in Auburn 623; which was anchored on Chr.A11 at a distance of about 2.1–3.3 cM from the CIR316 SSR marker (Wang et al. 2006a; Shen et al. 2006). The results of the present study identified the FW resistance gene closely linked with the SSR marker $JESPR304_{-280}$ on Chr.D3. The present study further suggests previous reports that FW, and RKN resistance is conferred by different genes or QTL on different chromosomes in cotton. The availability of genetic markers associated with FW and RKN resistance will enable two resistance genes to be pyramided in one genotype or individually deployed.

We detected one QTL (*qFW-A7-1*) located between NAU474 and NAU1048 on Chr.A7, which explained 25.4% of the PV. Shen et al. (2006) also reported a minor QTL for RKN resistance located in the same interval between NAU474 and NAU1048 on Chr.A7. This minor QTL identified in the PimaS-6 parent contributed to increased RKN resistance. These two QTL conferring disease resistance mapped to the same location, which suggests common resistance genes exist that control FW and RKN disease traits and can be used effectively in cotton MAS breeding.

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