Theor Appl Genet (2005) 111: 1167–1173
DOI 10.1007/s00122-005-0048-6

ORIGINAL PAPER

N. Sardesai · J. A. Nemacheck · S. Subramanyam
C. E. Williams

Identification and mapping of *H32*, a new wheat gene conferring resistance to Hessian fly

Received: 6 May 2005 / Accepted: 12 July 2005 / Published online: 6 September 2005

Abstract *H32* is a newly identified gene that confers resistance to the highly pervasive Biotype L of the Hessian fly [*Mayetiola destructor* (Say)]. The gene was identified in a synthetic amphihexaploid wheat, W-7984, that was constructed from the durum 'Altar 84' and *Aegilops tauschii*. This synthetic wheat is one of the parents of the marker-rich ITMI population, which consists of 150 recombinant inbred lines (RILs) derived by single-seed descent from a cross with 'Opata 85'. Linkage analysis of the *H32* locus in the ITMI population placed the gene between flanking microsatellite (SSR) markers, Xgwm3 and Xcfd223, at distances of 3.7 and 1.7 cM, respectively, on the long arm of chromosome 3D. The Xgwm3 primers amplified codominant SSR alleles, a 72 bp fragment linked in coupling to the resistance allele and an 84 bp fragment linked in repulsion. Primers for the SSR Xcfd223 amplified a 153 bp fragment from the resistant Synthetic parent and a 183 bp fragment from the susceptible Opata line. Deletion mapping of the flanking Xgwm3 and Xcfd223 markers located them within the 3DL-3 deletion on the distal 19% of the long arm of chromosome 3D. This location is at least 20 cM proximal to the reported 3DL location of *H24*, a gene that confers resistance to Biotype D of the Hessian fly. Tight linkage of the markers will provide a means of detecting *H32* presence in marker-assisted selection and gene pyramiding as an effective strategy for extending durability of deployed resistance.

Communicated by B. Keller

N. Sardesai
Department of Agronomy, Purdue University,
West Lafayette, IN 47907, USA

S. Subramanyam
Department of Entomology, Purdue University,
West Lafayette, IN 47907, USA

J. A. Nemacheck · C. E. Williams (✉)
Crop Production and Pest Control Research Unit
USDA-ARS-MWA and Department of Entomology,
Purdue University, West Lafayette, IN 47907, USA
E-mail: cwilliams@purdue.edu
Tel.: +1-765-4946763
Fax: +1-765-4961533

Introduction

Hessian fly, *Mayetiola destructor* (Say), is a destructive pest of wheat (*Triticum aestivum* L. em Thell.) in many wheat-growing regions worldwide. In experimental plots grown in Oregon USA, yield dropped as much as 59% in susceptible cultivars (compared to resistant), when as few as 15% of the plants were infested (Smiley et al. 2004). Despite a steady identification of resistance genes (31 genes since 1943; Noble and Suneson, 1943; Ratcliffe and Hatchett 1997 [review]; Williams et al. 2003, McIntosh et al. 2003), virulent Hessian fly genotypes continue to arise and cause major crop losses.

The center of origin and diversity for both Hessian fly and wheat is central and southwestern Asia (Harlan and Zohary 1966). The geographic distribution of Hessian fly mitochondrial haplotypes in North America supports a limited number of introductions from Europe (Johnson et al. 2004). Thus, many genes from diverse sources have provided resistance to North American fly populations even though they may no longer be effective in the center of diversity. The occurrence of numerous Hessian fly-resistance genes identified in the A, B and D genomes of wheat suggests a long co-evolutionary relationship between wild wheat species and the Hessian fly.

Hessian fly larvae are believed to inject many salivary proteins into wheat tissue (Chen et al. 2004) capable of being detected as avirulence factors in a classic gene-for-gene recognition (Hatchett and Gallun 1970; Gallun 1978) if the plant contains a corresponding resistance gene. The probable redundancy of genes encoding these salivary proteins allows for restored larval virulence if the defense-triggering elicitor is eliminated or altered. Widespread use of resistant cultivars exerts strong pressure favoring the selection of virulent genotypes

within wild Hessian fly populations (Gallun et al. 1961), thus rendering the corresponding plant resistance genes ineffective after 8–10 years of deployment (Foster et al. 1991). Continuous evolution of virulent genotypes necessitates the identification of new resistance genes from diverse origins. Fortunately, the abundance of salivary protein targets for recognition by wheat resistance genes, paired with thousands of years of co-evolution in the center of origin, has provided a vast array of potential sources of genetic resistance useful in North America where Hessian fly genetic diversity is limited.

Aegilops tauschii (formerly *Triticum tauschii*), the D-genome donor of common wheat shows considerable genetic diversity providing a rich source of disease and pest resistance genes for improvement of cultivated wheat (Cox 1991). Five independent resistance genes, *H13, H22*, *H23, H24* and *H26* (Hatchett and Gill 1981; Gill et al. 1986b; Cox and Hatchett 1994), have been previously identified from *Ae. tauschii* accessions and additional sources of resistance may be as yet undiscovered in germplasm collections of this species. One effective way of harvesting this rich source of resistance is by developing synthetic hexaploid wheats derived from crosses between tetraploid *Triticum* species and diploid *Ae. tauschii* accessions.

Here, we report the identification of *H32*, a new wheat gene residing on chromosome 3DL of a synthetic wheat derived from a cross between *Ae. tauschii* and a durum wheat. *H32* confers resistance against the highly virulent Biotype L of Hessian fly. Our objectives in this study were to design molecular markers that could be used for tracking the gene in a marker-assisted selection program, and to determine the genomic location of the gene. *H32* has potential for incorporation into a resistance gene-pyramid, along with other genes that confer resistance to Hessian fly Biotype L, in order to extend the durability of resistance.

Materials and Methods

Screening of parental lines with different Hessian fly genotypes

Wheat plants of 'Synthetic' and 'Opata' lines were grown in 4-inch pots (20 seeds/pot) in a growth chamber at 18°C and 14 h photoperiod. At the two-leaf stage (7–10 days after seed germination) each pot was covered with a plastic cup and infested with eight newly-emerged females and three males of one Hessian fly genotype; Biotype L (virulent to *H3, H5, H6* and the *H7H8* combination of Hessian fly-resistance genes), biotypes B, C, D, E and O (each virulent on plants containing one to three of the above-mentioned resistance genes; Ratcliffe and Hatchett 1997), GP (virulent on plants lacking resistance genes), *vH9* (virulent to gene *H9*) and *vH13* (virulent to gene *H13*). Flies were brushed off after 48 h of infestation and cups were removed once the eggs hatched. Two weeks after the eggs hatched, phenotypes of the plants were recorded as stunted or normal. Plants were dissected to check for the presence of live or dead larvae, which determined plant resistance genotype.

Segregation analysis of the ITMI population

We used a subset of 129 of the 150 recombinant inbred lines (RILs; derived by single-seed descent to the F8-9 generation) from a cross of a synthetic amphihexaploid wheat (W-7984; synthesized from *Ae. tauschii* accession CI 18 = WPI 219 and Altar 84 durum) with hexaploid 'Opata 85' (CIMMYT-bred spring wheat; Nelson et al. 1995). This population is referred to as the ITMI population, and the resistant and susceptible parental lines are referred to as Synthetic and Opata, respectively.

Plants were grown and infested with Hessian fly Biotype L as described above. Fifteen to thirty-eight individuals from each RIL were infested, and the phenotype of each plant in all 129 of the lines was recorded so that each RIL could be designated as resistant, susceptible or segregating.

Mapping the *H32* locus

The chromosomal location of *H32* was mapped by integrating our resistance segregation data into a file containing segregation data for a set of 1,409 RFLP and microsatellite (SSR) markers that had previously been mapped by other researchers in the ITMI population (marker data downloaded from the GrainGenes web site: http://www.graingenes.org/cgi-bin/ace/tree/graingenes?seme = 8&name = Wheat%2C%20Synthetic%20×%20Opata;class = Map_Data). This combined file of resistance data and previously mapped markers was used to determine the map position of the *H32* locus by utilizing the program MAPMAKER 3.0 (Lander et al. 1987) using a LOD 3.0 cut-off and the Kosambi mapping function (Kosambi 1944). The resulting map indicated two tightly linked SSR markers flanking the resistance locus.

SSR analysis and physical mapping of markers linked to *H32*

DNA was isolated as described earlier (Williams et al. 2003). Linkage of the SSR Xgwm3 to the *H32* resistance gene was verified on 27 resistant and 27 susceptible lines of the ITMI population. The primer sequences (Röder et al. 1998) were: Xgwm3 (L): 5′ GCAGCGGCACTGG TAAAAAAACATTT 3′, and Xgwm3 (R): 5′ AATATC GCATCACTATCCCA 3′. PCR was carried out with 50 ng of template DNA in a PTC-100 thermal cycler (MJ Research, Waltham, MA) according to the following parameters: 3 min at 94°C; 45 cycles of 1 min at 94°C, 1 min at 55°C, 2 min at 72°C; final extension of 10 min at 72°C. PCR products were resolved by poly-

acrylamide gel electrophoresis (PAGE) on a 12.5% gel and silver-stained (Blum et al. 1987). Linkage of the other flanking marker, Xcfd223, to *H32* was verified on a subset of 115 ITMI population RILs following the PCR protocol of Guyomarc'h et al. (2002). The sequences of the primers were: Xcfd223 (L): 5′ AAGAGCTACAATGACCAGCAGA 3′, and Xcfd223 (R): 5′ GCAGTGTATGTCAGGAGAAGCA 3′. Temperature conditions for the PCR were as follows: 5 min at 94°C; 32 cycles of 30 s at 94°C, 30 s at 63°C, 30 s at 72°C; final extension of 10 min at 72°C. The PCR products were visualized on a 3% (w/v) agarose gel.

We used the line 'Chinese Spring' and four Chinese Spring derivatives with terminal chromosomal deletions in the short and long arms of the group-3 chromosomes (Endo and Gill 1996). The following terminal deletion lines were used: 3DL-3, 3DS-3, −6 and 3BL-10. PCR with the Xgwm3 and Xcfd223 primers was carried out as described above.

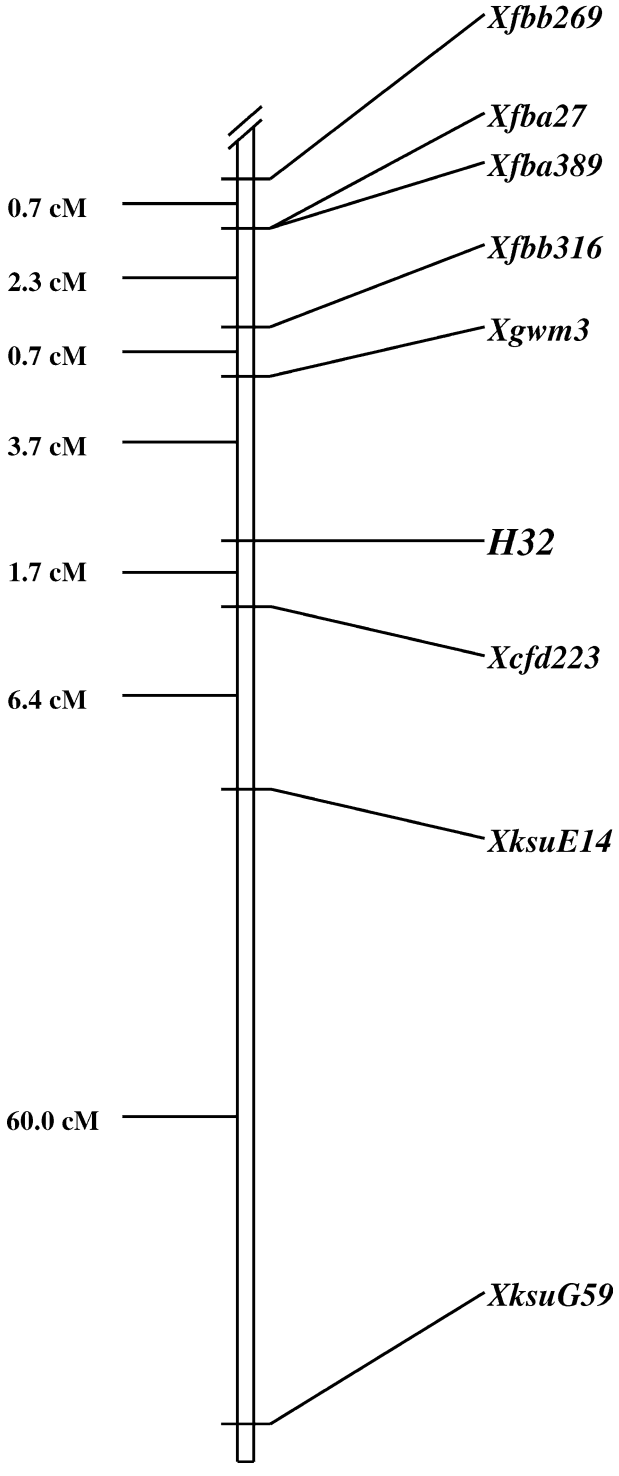


Fig. 1 Location of *H32* on the long arm of chromosome 3D. The linkage map shows SSR loci, *Xgwm3* and *Xcfd223* at a distance of 3.7 and 1.7 cM, respectively, flanking the *H32* locus. *Figures* on the left represent map units in centimorgans (cM) between adjacent markers

Results

Resistance in the Synthetic parent

The Synthetic parent of the ITMI population showed resistance to Hessian fly biotypes B, C, D, E, L, O, *vH9* and *vH13*, but was susceptible to Biotype GP. Opata, on the other hand, was susceptible to all nine of the larval genotypes tested. A subset of 129 lines of the ITMI population was infested with the highly virulent Hessian fly, Biotype L. The segregation data for these lines were consistent with the single-locus hypothesis with 54 resistant and 62 susceptible RILs ($\chi^2_{1:1} = 0.42$, 1 df, $P = 0.517$).

Identification of markers flanking the *H32* resistance locus

The map position for the *H32* locus was determined by integrating our resistance gene segregation data, obtained from the analysis of a subset of the ITMI population, with the previously generated data for the ITMI framework map comprising more than 1,400 RFLP and SSR markers. Linkage analysis of the *H32* locus placed it between two SSR markers on the long arm of chromosome 3D. The proximal marker, Xgwm3, is at a distance of 3.7 cM from the *H32* locus, whereas, the distal marker, Xcfd223, is at a distance of 1.7 cM from the resistance locus (Fig. 1).

To verify the *H32* map position that was indicated by the computer analysis, primers for the flanking SSR markers were used on our DNA samples. Primers for SSR Xgwm3 amplified alternative alleles: Xgwm3a an 84 bp Opata (susceptible parent)-specific fragment, and Xgwm3b a 72 bp Synthetic (resistant parent)-specific fragment (Fig. 2a). In a subset of the ITMI population consisting of 27 resistant and 27 susceptible plants, Xgwm3a was amplified in all the susceptible samples, and Xgwm3b in all resistant samples (data not shown). Primers for the other flanking SSR, Xcfd223, amplified a 153 bp fragment in the Synthetic parent and resistant

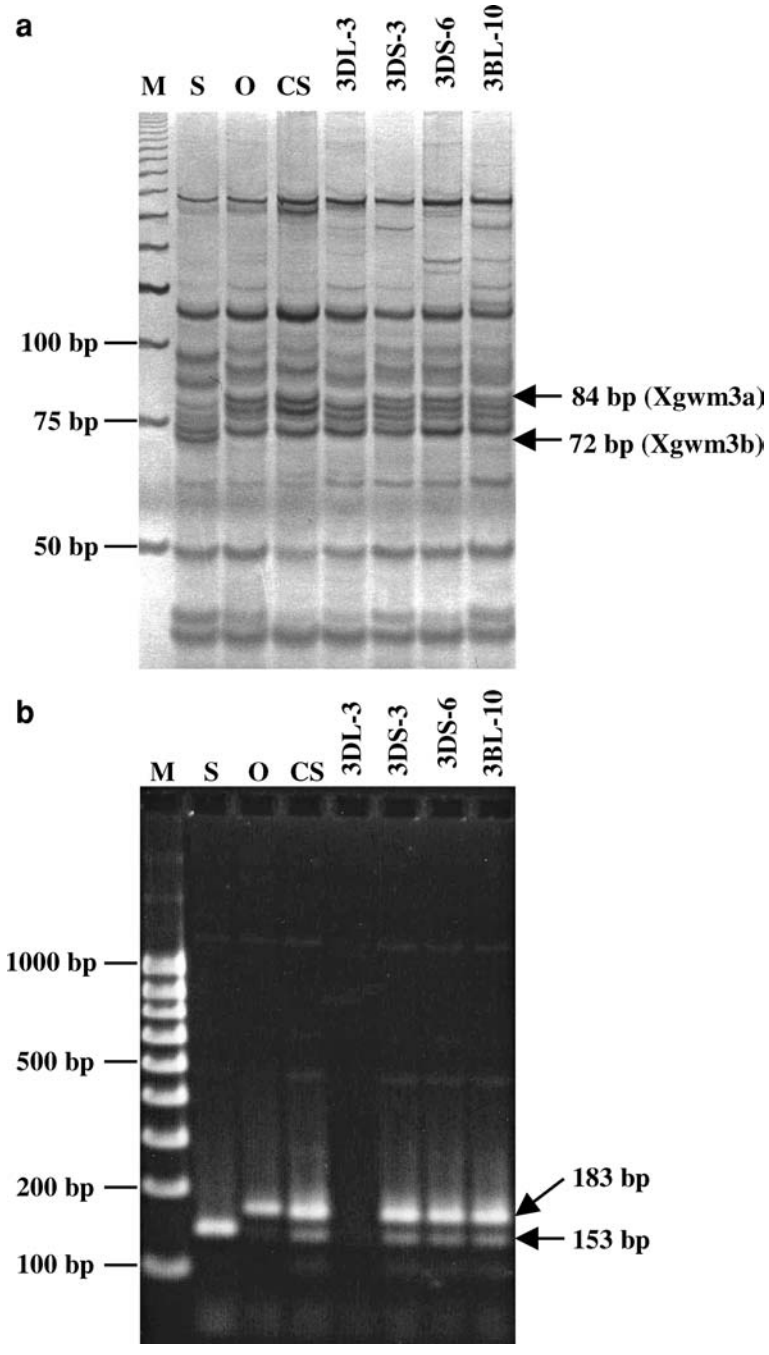


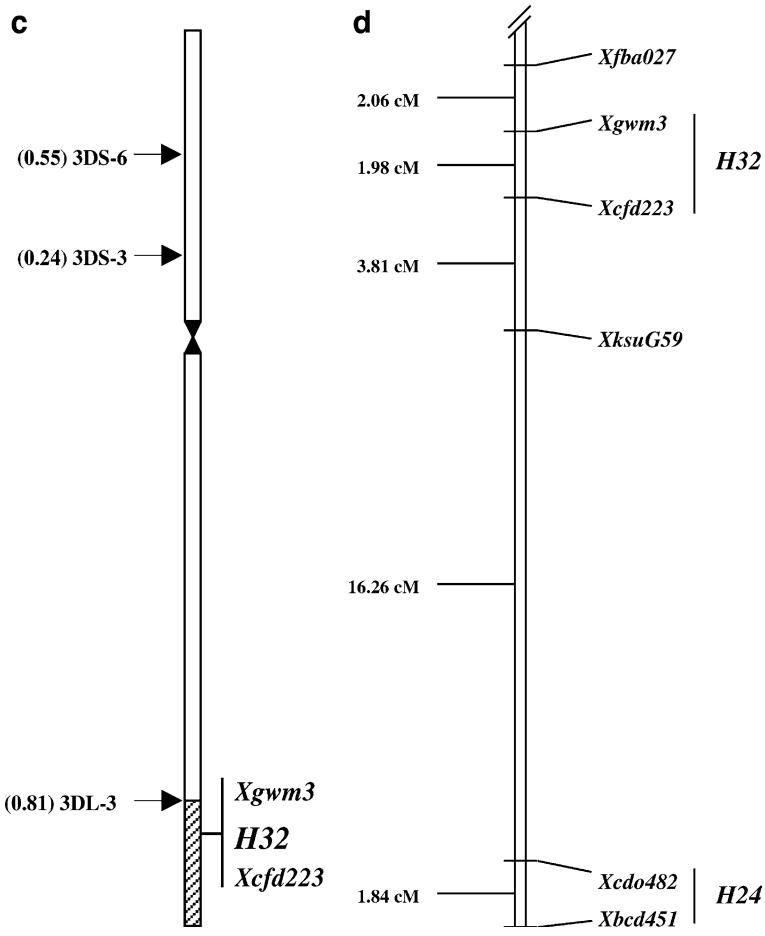
Fig. 2 Physical mapping to confirm the region flanking *H32*. Genomic DNA from Synthetic (*S*), Opata (*O*), Chinese Spring (*CS*) and the terminal deletion lines was subjected to amplification. *Figures* on the left of gel photos represent the molecular weight of the DNA ladder in bp, whereas, those on the right represent molecular weight of the phenotype-specific amplicons in bp. **a** Amplicons resulting from Xgwm3 primers. *Lane M* is a 25 bp DNA molecular-weight ladder. **b** Amplicons resulting from Xcfd223 primers. *Lane M* is a 100 bp DNA molecular-weight ladder. **c** Physical map of SSR loci flanking the *H32* resistance locus. The chromosome fraction-lengths (within parentheses) for break points of the Chinese Spring 3D terminal deletion lines are shown to the left. The *terminal shaded area* on the long arm of chromosome 3D corresponds to the region containing the *Xgwm3, H32* and *Xcfd223* loci. **d** Composite linkage map of a region of the long arm of chromosome 3D. The map shows relative positions of SSR and RFLP markers linked to *H32* and *H24*, respectively, demonstrating that they reside in distinct regions of 3DL and are not allelic. Data for the map were taken from the wheat composite map at http://www.graingenes.org. *Figures* on the left represent map units in centimorgans (cM) between adjacent markers

lines, and a 183 bp fragment in Opata and susceptible lines (Fig. 2b).

Physical mapping of the *H32* locus

PCR analysis of DNA from Chinese Spring and the terminal-deletion lines 3DS-3, 3DS-6 and 3BL-10 with the Xgwm3 primers revealed amplification of Xgwm3a, the 84 bp susceptible parent-specific fragment (Fig. 2a). However, Xgwm3a did not amplify from the 3DL-3 terminal-deletion line DNA, confirming the location of the target locus in 3DL. Primers for the distal flanking SSR, Xcfd223, amplified both the resistance-specific 153 bp fragment and the susceptible-specific 183 bp fragment from DNA of Chinese Spring and the terminal-deletion lines, 3DS-3, 3DS-6 and 3BL-10, but did not amplify from the terminal-deletion line 3DL-3 (Fig. 2b). The amplification of both fragments from Chinese Spring and lack of amplification of both from the 3DL-3 deletion DNA suggests that the target loci occur in coupling within the region of chromosome 3DL that is covered by the deletion, whereas Synthetic and Opata each possess only one of the two alternative target loci. The localization of two linked and flanking SSR markers of the *H32* locus in the 3DL-3 deletion confirms

Fig. 2 (Contd.)



the location of the *H32* gene in the distal 19% of the long arm of chromosome 3D (Fig. 2c).

Discussion

Resistance in Synthetic

The Synthetic parent of the ITMI population was generated via a cross of *Ae. tauschii* (DD) with Altar 84 durum (AABB) wheat (Nelson et al. 1995). Since *Ae. tauschii* is an immediate progenitor of common wheat, this species is considered an important potential source of Hessian fly-resistance (Hatchett and Gill 1981; Hatchett et al. 1981). Being the D-genome donor to common wheat, genetic transfer of resistance is very efficient (Gill and Raupp 1987; Raupp et al. 1993). Several Hessian fly-resistance genes from *Ae. tauschii* have been introgressed into wheat (Martin et al. 1982; Gill et al. 1986a, 1991a, 1991b), and a number of synthetic hexaploid wheats have been evaluated for resistance to Hessian fly (Hatchett et al. 1981; SS Xu and MO Harris, personal communication). Other synthetic hexaploid wheats have been evaluated for resistance to pathogens such as *Fusarium graminearum* but not for Hessian fly. Because these synthetic wheats were derived from *Ae. tauschii* accessions that have resistance to Hessian fly biotypes D and L (Berzonsky et al. 2004), they are as yet untapped sources of resistance to Hessian fly.

The ITMI Synthetic showed a broad range of resistance to the Hessian fly genotypes we tested. This is not unexpected, since biotypes A through O have evolved through sequential selection against the four R genes, *H3, H5, H6* and *H7H8* combination (Gallun 1977), each biotype having an altered allele for one to four of the avirulence genes. Thus, the *H32* line likely recognizes a larval salivary protein that is distinct from those recognized by any of the other resistant wheat lines. Interestingly, *H32* does not confer resistance against Biotype GP, the least virulent biotype identified so far. To our knowledge there is no published literature available on any other wheat line having a Hessian fly resistance gene while also being susceptible to Biotype GP. However, one unrelated line of wheat has been observed to be resistant to Biotype E, but susceptible to Biotype GP (RH Ratcliffe and SE Cambron, personal communication). Our data suggest that Biotype GP lacks the avirulence factor that is recognizable by *H32* wheat and that is present in Biotypes B, C, D, E, O plus *vH9* and *vH13* flies. Prior to this report, Biotype GP was often described as the ''avirulent biotype'' (example: Ratcliffe

et al. 2000) implying that it was avirulent to any wheat containing a Hessian fly-resistance gene.

Chromosomal location of *H32*

The genomic location of the *H32* resistance locus was determined, in the highly polymorphic and marker-rich ITMI mapping population, to be on the long arm of chromosome 3D. The *H32* locus is flanked by SSR markers, Xgwm3 and Xcfd223, that respectively are only 3.7 and 1.7 cM from the resistance locus. Multiple bands visible in the PAGE profiles for the SSR Xgwm3 are caused by the presence of 'stutter products' where a change in the number of repeating units is a result of *Taq* DNA polymerase slippage (Levinson and Gutman 1987; Shinde et al. 2003). However, the original 84 bp fragment reported in Röder et al (1998) was robust and linked to the *H32* resistance locus. Through monosomic analysis, one other Hessian fly-resistance gene, *H24*, was reported to be located on chromosome 3D (Raupp et al. 1993). Subsequently, RFLP loci, *Xbcd451* and *Xcdo482*, were identified to be linked to the *H24* resistance gene (Ma et al. 1993). These markers have been mapped to the terminal end of chromosome 3DL on the ITMI mapping population (http://www.graingenes.org/). Analysis of the composite wheat genetic map (at http://www.graingenes.org/) reveals that markers flanking the *H32* locus and those linked to the *H24* locus are in distinct regions of 3DL that are separated by at least 20 cM (Fig. 2d), with *H32* being proximal to the terminally located *H24*. Although order and distance between tightly linked markers can be imprecise on composite linkage maps, markers linked with *H24* and *H32* are clearly separated by a large genetic distance. Thus, the *H32* resistance gene is a new gene and is not allelic to the *H24* resistance locus.

Potential application of synthetic in gene-pyramiding programs

It has been postulated that the durability of resistance can be increased up to 20-fold by developing and releasing elite cultivars that contain multiple resistance genes to a single biotype of Hessian fly (Gould 1986). However, the success of any breeding program in developing pyramided cultivars will depend on the ability to ensure the presence of all desirable genes in the elite cultivar. This can be achieved by identification of molecular markers linked to the genes of interest and monitoring their presence during the construction of the gene pyramid. Molecular markers have been identified for several Hessian fly resistance genes in wheat (Dweikat et al. 1994, 1997, 2002; Ma et al. 1993; Najimi et al. 2002; Williams et al. 2003, Liu et al. 2005). Molecular markers have also been identified for resistance genes to the closely-related dipteran, gall midge, in rice (Sardesai et al. 2001, 2002; Jain et al. 2004). Molecular markers for the *H31* gene (Williams et al. 2003), as well as those for *H9* and *H13* genes (Dweikat et al. 1997), are being used in the construction of breeding lines containing all three resistance loci. The newly found source of resistance to Biotype L in Synthetic can potentially contribute in strengthening the field resistance of such a pyramided cultivar. Marker-assisted selection can help in following the introgression of the resistance loci and prevent their inadvertent unstacking during development of the elite cultivar.

Acknowledgements We thank Sue Cambron (USDA-ARS Crop Production and Pest Control Research Unit) for maintenance of the Hessian fly stocks, Cal Qualset (University of California, Davis) for ITMI lines, and the Wheat Genetics Resource Center at Kansas State University for Chinese Spring and deletion lines. Mention of a proprietary product does not constitute an endorsement or a recommendation for its use by USDA. This is a joint contribution by the USDA Agricultural Research Service and Purdue University. The work was supported by funding from USDA CRIS number 3602-22000-014-00D. Purdue University Agricultural Experiment Station Journal Article No. 2005–17640.

References

Berzonsky WA, Hartel KD, Kianian SF, Leach GD (2004) Registration of four synthetic hexaploid wheat germplasm lines with resistance to Fusarium head blight. Crop Sci 44:1500–1501

Blum H, Beier H, Gross HJ (1987) Improved silver staining of plant proteins, RNA and DNA in polyacrylamide gels. Electrophoresis 8:93–99

Chen M-S, Fellers JP, Stuart JJ, Reese JC, Liu X (2004) A group of cDNAs encoding secreted proteins from Hessian fly [*Mayetiola destructor* (Say)] salivary glands. Insect Mol Biol 13:101–108

Cox TS (1991) The contribution of introduced germplasm to the development of U.S. wheat cultivars. In: Use of plant introductions in cultivar development, Part 1 (Spec Publ no. 17) Madison, Wisconsin, Crop Science Society of America, pp 24–47

Cox TS, Hatchett JH (1994) Hessian fly-resistance gene *H26* transferred from *Triticum tauschii* to common wheat. Crop Sci 34:958–960

Dweikat I, Ohm H, Mackenzie S, Patterson F, Cambron S, Ratcliffe R (1994) Association of a DNA marker with Hessian fly resistance gene *H9* in wheat. Theor Appl Genet 89:964–968

Dweikat I, Ohm H, Patterson F, Cambron S (1997) Identification of RAPD markers for 11 Hessian fly resistance genes in wheat. Theor Appl Genet 94:419–423

Dweikat I, Zhang W, Ohm H (2002) Development of STS markers linked to Hessian fly resistance gene *H6* in wheat. Theor Appl Genet 105:766–770

Endo TR, Gill BS (1996) The deletion stocks of common wheat. J Hered 87:295–307

Foster JE, Ohm H, Patterson F, Taylor P (1991) Effectiveness of deploying single gene resistance in wheat for controlling damage by the Hessian fly (Diptera: *Cecidomyiidae*). Environ Entomol 20:964–969

Gallun RL (1977) Genetic basis of Hessian fly epidemics. Ann NY Acad Sci 287:223–229

Gallun RL (1978) Genetics of biotypes B and C of Hessian fly. Ann Entomol Soc Am 71:481–486

Gallun RL, Deay HO, Cartwright WB (1961) Four races of Hessian fly selected and developed from an Indiana population. Purdue University Agriculture Experiment Station Research Bulletin 732, West Lafayette, Indiana

Gill BS, Raupp WJ (1987) Direct genetic transfers from *Aegilops squarrosa* L. to hexaploid wheat. Crop Sci 27:445–450

Gill BS, Hatchett JH, Raupp WJ, Sears RG, Martin TJ (1986a) Registration of KS85WGRC01 Hessian fly-resistant hard red winter wheat germplasm. Crop Sci 26:1266–1267

Gill BS, Sharma HC, Raupp WJ, Browder LE, Hatchett JH, Harvey TL, Moseman JG, Waines JG (1986b) Resistance in *Aegilops squarrosa* to wheat leaf rust, wheat powdery mildew, greenbug, and Hessian fly. Plant Dis 70:553–556

Gill BS, Wilson DL, Raupp WJ, Hatchett JH, Cox TS, Amri S, Sears RG (1991a) Registration of KS89WGRC3 and KS89WGRC6 Hessian fly-resistant hard red winter wheat germplasm. Crop Sci 31:245

Gill BS, Wilson DL, Raupp WJ, Hatchett JH, Cox TS, Amri S, Sears RG (1991b) Registration of KS89WGRC4 hard red winter wheat germplasm with resistance to Hessian fly, greenbug, and soil-borne mosaic virus. Crop Sci 31:246

Gould F (1986) Simulation models for predicting durability of insect-resistant germ plasm: Hessian fly (Diptera: Cecidomyiidae)-resistant winter wheat. Environ Entomol 15:11–23

Guyomarc'h H, Sourdille P, Charmet G, Edwards KJ, Bernard M (2002) Characterisation of polymorphic microsatellite markers from *Aegilops tauschii* and transferability to the D-genome of bread wheat. Theor Appl Genet 104:1164–1172

Harlan JR, Zohary D (1966) Distribution of wild wheats and barley. Science 153:1074–1080

Hatchett JH, Gallun RL (1970) Genetics of the ability of the Hessian fly, *Mayetiola destructor*, to survive on wheats having different genes for resistance. Ann Entomol Soc Am 63:1400–1407

Hatchett JH, Gill BS (1981) D-genome sources of resistance in *Triticum tauschii* to Hessian fly. J Hered 72:126–127

Hatchett JH, Martin TJ, Livers RW (1981) Expression and inheritance of resistance to Hessian fly in synthetic hexaploid wheats derived from *Triticum tauschii* (Coss) Schmal. Crop Sci 21:731–734

Jain A, Ariyadasa R, Kumar A, Srivastava MN, Mohan M, Nair S (2004) Tagging and mapping of a rice gall midge resistance gene, *Gm8*, and development of SCARs for use in marker-aided selection and gene pyramiding. Theor Appl Genet 109:1377–1384

Johnson AJ, Schemerhorn BJ, Shukle RH (2004) A first assessment of mitochondrial DNA variation and distribution of haplotypes in Hessian fly (Diptera: Cecidomyiidae). Ann Entomol Soc Am 97:940–948

Kosambi DD (1944) The estimation of map distances from recombination values. Ann Eugen 12:172–175

Lander ES, Green P, Abrahamson J, Barlow A, Daly MJ, Lincoln SE, Newburg L (1987) MAPMAKER: an interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics 1:174–181

Levinson G, Gutman GA (1987) Slipped-strand mispairing: A major mechanism for DNA sequence evolution. Mol Biol Evol 4:203–211

Liu XM, Gill BS, Chen M-S (2005) Hessian fly resistance gene *H13* is mapped to a distal cluster of R genes in chromosome 6DS of wheat. Theor Appl Genet 111:243–249

Ma Z-Q, Gill BS, Sorrells ME, Tanksley SD (1993) RFLP markers linked to two Hessian fly-resistance genes in wheat (*Triticum aestivum* L.) from *Triticum tauschii* (coss.) Schmal. Theor Appl Genet 85:750–754

Martin TJ, Harvey TL, Hatchett JH (1982) Registration of greenbug and Hessian fly resistant wheat germplasm. Crop Sci 22:1089

McIntosh RA, Yamazaki Y, Devos KM, Dubcovsky J, Rogers J, Appels R (2003) Catalogue of gene symbols for wheat. Available on the Internet at http://www.grs.nig.ac.jp/wheat/komugi/genes/download.jsp

Najimi B, Boukhatem N, Jaafri SE, Jlibène M, Paul R, Jacquemin J-M (2002) Amplified fragment length polymorphism (AFLP) analysis of markers associated with *H5* and *H22* Hessian fly resistance genes in bread wheat. Biotechnol Agron Soc Environ 6:79–85

Nelson JC, Van Deynze AE, Autrique E, Sorrells ME, Lu YH, Negre S, Bernard M, Leroy P (1995) Molecular mapping of wheat. Homoeologous group 3. Genome 38:525–533

Noble WB, Suneson CA (1943) Differentiation of two genetic factors for resistance to the Hessian fly in Dawson wheat. J Agric Res 67:27–32

Ratcliffe RH, Hatchett JH (1997) Biology and genetics of the Hessian fly and resistance in wheat. In: Bondari K (ed) New developments in entomology. Research Signpost, Trivandrum, India, pp 47–56

Ratcliffe RH, Cambron SE, Flanders KL, Bosque-Perez NA, Clement SL, Ohm HW (2000) Biotype composition of Hessian fly (Diptera: Cecidomyiidae) populations from the southeastern, mid-western, and northwestern United States and virulence to resistance genes in wheat. J Econ Entomol 94:1319–1328

Raupp WJ, Amri A, Hatchett JH, Gill BS, Wilson DL, Cox TS (1993) Chromosomal location of Hessian fly-resistance genes *H22, H23* and *H24* derived from *Triticum tauschii* in the D genome of wheat. J Hered 84:142–145

Röder MS, Korzun V, Wendehake K, Plaschke J, Tixier M-H, Leroy P, Ganal MW (1998) A microsatellite map of wheat. Genetics 149:2007–2023

Sardesai N, Rajyashri KR, Behura SK, Nair S, Mohan M (2001) Genetic, physiological and molecular interactions of rice with its major dipteran pest, gall midge. Plant Cell Tiss Org 64:115–131

Sardesai N, Kumar A, Rajyashri KR, Nair S, Mohan M (2002) Identification and mapping of an AFLP marker linked to *Gm7*, a gall midge resistance gene and its conversion to a SCAR marker for its utility in marker aided selection in rice. Theor Appl Genet 105:691–698

Shinde D, Lai Y, Sun F, Arnheim N (2003) *Taq* DNA polymerase slippage mutation rates measured by PCR and quasi-likelihood analysis: $(CA/GT)_n$ and $(A/T)_n$ microsatellites. Nuc Acid Res 31:974–980

Smiley RW, Gourlie JA, Whittaker RG, Easley SA, Kidwell KK (2004) Economic impact of Hessian fly (Diptera: Cecidomyiidae) on spring wheat in Oregon and additive yield losses with Fusarium crown rot and lesion nematode. J Econ Entomol 97:397–408

Williams CE, Collier CC, Sardesai N, Ohm HW, Cambron SE (2003) Phenotypic assessment and mapped markers for *H31*, a new wheat gene conferring resistance to Hessian fly (Diptera: Cecidomyiidae). Theor Appl Genet 107:1516–1523