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Chromosome location of a gene conferring resistance to *Pyrenophora tritici-repentis* in Ethiopian wheat cultivars

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Abstract In this study, 118 common wheat (*Triticum aestivum*, 2n = 2× = 42, AABBDD) and durum wheat (*Triticum durum*, 2n = 4× = 28, AABB) genotypes of Ethiopian origin were evaluated for reaction to tan spot using two *Pyrenophora tritici-repentis* (*Ptr*) isolates. Among the commercial Ethiopian bread wheat cultivars HAR604, HAR2562, and Dashen were resistant to both the ASC1a and DW-16 isolates. Inheritance and allelism studies indicated that HAR604, HAR2562 and Dashen possess recessive allelic genes for tan spot resistance. Monosomic F_1 and F_2 analyses using the monosomic lines of Chinese Spring and cultivars HAR2562 and HAR604 indicated that the resistance gene temporarily designated as *TsrHar*, was located on chromosome 3B. The allelic relationship with the gene now designated *Tsr2*, formerly *tsn2*, is unknown. *TsrHar* should be useful as source of resistance for deployment in conventional breeding schemes.

Keywords Monosomic analysis · Disease resistance · *Pyrenophora tritici-repentis* · *Triticum aestivum*

Introduction

Tan spot of wheat, caused by the ascomycete *Pyrenophora tritici-repentis* (Died.) Drechs., is one of the important foliar diseases of wheat world wide and causes significant yield and grain quality losses in both durum (*Triticum durum*) and common (*Triticum aestivum* L.) wheats (Hosford 1982; Rees et al. 1988; Wolf and Hoffman 1993; Duveiller et al. 2005). The pathogen causes tan necrosis and/or extensive chlorosis depending on the specific interaction between individual isolates of the fungus and wheat genotypes (Lamari and Bernier 1989b).

Currently, at least eight races of *P. tritici-repentis* (*Ptr*) can be identified based on the type of toxin they produce and their ability to induce necrosis and/or chlorosis on a set of wheat differential cultivars (Strelkov and Lamari 2003). *Ptr* ToxA, produced from race 1 is the most well-characterized host-selective proteinaceous toxin that causes necrotic symptoms in susceptible wheat cultivars. *Ptr* ToxB, isolated from race 5 (Orolaza et al. 1995; Friesen and Faris 2004) and *Ptr* ToxC isolated from race 1 (Effertz et al. 2002) cause chlorosis symptoms. Isolates of race 3 which putatively produce only *Ptr* ToxC, are virtually non existent on hexaploid wheats and very rare (<1%) on durum wheats. According to Effertz et al. (2002), toxin insensitive wheats may be susceptible to isolates of a common race of the fungus, suggesting that breeders aiming to develop tan spot resistant wheats should not rely only on toxin reactions as they could select for toxin-insensitive wheats that are tan spot susceptible.

Resistance is the most effective, economical and environmentally friendly method of managing tan spot.

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Its success, however, depends on the availability of broad genetic diversity and continuous search for novel resistance genes in order to cope with a rapidly changing pathogen population. Reports on the inheritance and genetics of tan spot resistance varies from quantitative (Nagle et al. 1982; Elias et al. 1989; Faris et al. 1997; Friesen and Faris 2004) to qualitative (Lamari and Bernier 1989b, 1991; Gamba and Lamari 1998; Lamari et al. 2003; Singh and Hughes 2005; Tadesse et al. 2006a, b, 2007) depending on the variety and isolates used for study. Unlike the powdery mildew and rust diseases, very few genes have been identified and mapped for tan spot resistance. To-date, recessive resistance genes *Tsr1* (formerly *tsn1*) on 5BL (Faris et al. 1996), *Tsr2* (*tsn2*) on 3BL (Singh et al. 2006), *Tsr3* (*Tsn3*) on 3D (Tadesse et al. 2006a, 2007), and *Tsr4* (*tsn4*) on 3A (Tadesse et al. 2006b) have been identified.

According to Vavilov (1951) and Engles and Hawkes (1991), the Ethiopian region is a centre of diversity and origin of durum wheat (*Triticum turgidum* ssp. *aethiopicum*). Common wheat (*Triticum aestivum* L.) is also found in great diversity though it is a recent introduction to Ethiopia. Owing to this diversity, Ethiopian germplasms have been utilized worldwide (Worede 1991; Tesema 1991), and many agronomically important genes have been found and incorporated into commercial cultivars (Negassa 1986; Tesema 1991; Gebremariam 1991; Zeller et al. 1998). However, Ethiopian wheat germplasm has not been evaluated for tan spot resistance. The objectives of this study were to identify sources of tan spot resistance, study the genetics of tan spot resistance and determine the chromosomal locations of resistance genes in Ethiopian wheat cultivars.

Material and methods

Plant materials

A total of 68 common wheat (*Triticum aestivum*, 2n = 2× = 42, AABBDD) and 28 durum wheat (*Triticum durum*, 2n = 4× = 28, AABB) genotypes of Ethiopian origin were obtained from the Institute of Plant Genetics and Crop Plant Research (IPK), Gatersleben, Germany. Twenty common wheat cultivars were obtained from Adet Research Centre, Bahir Dar, Ethiopia. The susceptible cultivar Glenlea was kindly provided by Dr. L. Lamari, University of Manitoba, Winnipeg, Canada. Cultivar Chinese Spring (2n = 6× = 42) and its 21 monosomic lines (2n = 6× = 41) were obtained from the late Dr. E.R. Sears, University of Missouri, USA.

Inoculum production

Two *Ptr* isolates: ASC1a (race 1) and DW-16 (unknown) were obtained from Dr L. Lamarai, University of Manitoba, Canada, and Dr S. Ali, North Dakota State University, Fargo, USA, respectively. These isolates were evaluated against differential cultivars and were found to be the most virulent (Tadesse et al. 2006b). The method of inoculum production was according to Lamari and Bernier (1989a). Mycelial plugs, 0.5 cm diameter, from the stock cultures were transferred to 10 cm petri plates containing V8 juice (150 ml), Difco PDA (10 g), $CaCO_3$ (3 g), Bacto agar (10 g) and distilled water (850 ml). These cultures were incubated in the dark at 22°C for about 8 days. The plates were then flooded with sterile distilled water, the mycellia were flattened using sterilized glass rods, and the excess water was poured off. The plates were incubated under continuous light at room temperature for 24 h to induce conidiophore production, and then for about 22 h at 16°C to induce production of conidia. Conidia were harvested by flooding the plates in sterile distilled water and gently brushing the mycelium with a camel-hair brush to dislodge the conidia from the conidiophores. Ten drops of Tween 20 (polyoxyethylene sorbitan monolaureate) per litre were added to the spore suspension, which was then adjusted to a concentration of approximately 3,000 conidia per ml.

Disease screening

One hundred and eighteen wheat cultivars were screened using *Ptr* isolates, ASC1a and DW-16. On average, five seeds per genotype were planted in a pot (10 cm diameter) containing peat moss, and placed at a temperature of 20–23°C and 16 h photoperiod on a bench in the greenhouse. Water was supplied by capillary action via holes in the base of the pot. Each cultivar was replicated two times. Seedlings were inoculated at the two leaf stage as explained in Tadesse et al. (2006b). Ratings of genotypes for reaction to tan spot were made seven days after inoculation on the first leaf using the 1 to 5 rating scale developed by Lamari and Bernier (1989a).

Table 1 Response of Ethiopian common and durum wheat genotypes for tan spot reaction to *P. tritici-repentis* isolates ASC1a and DW-16

Common wheat accessions						Durum wheat accessions		
IPK ACC.no.	ASC1a	DW-16	Accession	ASC1	DW-16	Accession	ASC1a	DW-16
3708/89	3	1	15831/87	4	4	14770/89	5	4
13134/88	2	3	15832/89	1	3	14771/89	5	4
14792/88	5	4	15845/87	3	2	14807/86	5	5
14797/86	4	1	16126/89	1	4	15023/99	5	5
14803/90	3	4	16131/88	1	3	15024/95	4	5
14831/86	4	5	16137/96	4	5	15061/86	5	5
15011/86	4	5	16138/97	4	2	15070/96	5	5
15012/89	4	5	16148/88	1	4	15089/86	5	4
15016/87	2	3	16152/90	2	2	15254/90	5	4
15030/86	3	4	16161/89	2	3	15360/89	2	2
15041/86	5	1	16167/88	1	2	15371/90	2	4
15043/86	1	4	16168/88	1	2	15412/89	5	4
15044/86	3	3	16192/89	1	2	15506/89	4	3
15085/86	2	2	16196/89	1	3	15519/89	3	3
15086/86	2	4	16220/89	1	4	15570/90	5	4
15087/89	4	3	16233/89	3	4	15572/88	5	5
15090/86	1	3	16240/88	4	4	15601/90	4	4
15094/86	3	2	16287/89	3	1	15657/89	3	4
15361/89	4	4	16293/90	2	2	15687/89	4	3
15440/90	2	2	16294/96	2	2	15721/89	5	4
15444/90	5	4	16300/88	1	1	15760/89	4	3
15446/87	3	5	16752/89	1	3	15838/89	5	4
15462/87	4	4	16772/89	3	4	15849/90	4	3
15476/91	5	5	17873/97	1	5	15855/89	4	4
15511/89	2	3	3427/75	4	3	15859/88	3	3
15516/90	4	2	3484/75	5	5	16278/90	3	3
15518/87	3	4	Dashen	1	1	17830/97	5	5
15525/87	1	3	ET13–02	4	5	7295/82	2	3
15527/89	3	3	HAR 1407	3	3	Mean	4.1	3.8
15538/88	4	4	HAR 1522	3	4	S.E	0.19	0.15
15540/88	2	3	HAR 1595	3	3	[a]Glenlea	5	5
15543/90	4	4	HAR 1775	4	3	[a]Chinese Spring	4	4
15572/90	3	2	HAR 1868	3	4	[a]Red Chief	1	1
15573/87	1	4	HAR 1899	3	4			
15579/87	3	2	HAR 1920	4	5			
15597/87	4	3	HAR 2029	4	4			
15608/87	4	3	HAR 2149	4	4			
15609/96	3	3	HAR 2192	3	4			
15725/92	4	3	HAR 2501	3	3			
15785/87	4	2	HAR 2536	3	4			
15805/89	4	1	HAR 2562	1	2			
15809/90	4	1	HAR 604	1	2			

Table 1 continued

Common wheat accessions						Durum wheat accessions		
IPK ACC.no.	ASC1a	DW-16	Accession	ASC1	DW-16	Accession	ASC1a	DW-16
15810/90	3	4	HAR1685	2	3			
15831/87	4	4	HAR2508	3	3			
15832/89	1	3	Paven-76	2	2			
Mean				2.8	3.1			
S.E.				0.13	0.12			

[a] Check genotypes: IPK = Institute of Plant Genetics and Crop Plant Research, Getersleben, Germany

Genetics of resistance

To study the inheritance of tan spot reaction, crosses were made between the resistant cultivars HAR604, HAR2562 and Dashen and the susceptible cultivar Glenlea. Crosses between the resistant cultivars (HAR604/HAR2562, HAR 604/Dashen, HAR2562/Dashen) were also made to check for allelism. F_1 and F_2 plants of each cross were screened using *Ptr* isolates ASC1a and DW-16 in two sets of inoculations. Evaluations were made using the Lamari and Bernier (1889a) 1–5 scale. Reaction classes 1 to 2 were grouped as resistant and 3 to 5 were grouped as susceptible. Chi-square analyses were carried out on the F_2 segregation ratios.

Monosomic analysis

Ethiopiaian common wheat lines HAR2562 and HAR604 were crossed to Chinese Spring (CS) monosomics. CS was susceptible to the *Ptr* isolates ASC1a and DW-16 (Tadesse et al. 2006b). Hybrids of CS with HAR2562 and HAR604 were also made as controls. Mitotic chromosome counts were made on squashes of root-tip cells pretreated with mono-bromnaphthaline and stained by the Feulgen method as indicated in Zeller et al. (1993). For each cross, 30 F_2 seeds (10 seeds/pot) were planted per inoculation. The 17 days old seedlings were inoculated with *Ptr* isolates ASC1a and DW-16 at different times to avoid contamination. A minimum of two sets of inoculations per isolate was made depending on the number of seeds available for each population. Evaluations were carried out seven days after inoculation following the 1–5 rating scale. The frequencies of resistant (ratings of 1–2) and susceptible (3–5) plants for each cross were subjected to χ^2 analysis.

Results

Screening of germplasm for tan spot resistance

Of the common wheat genotypes, 32 (36.4%) and 26 (29.6%) were resistant (scores 1–2) to *Ptr* isolates

Table 2 Reaction of F_1 and F_2 plants to *Ptr* isolates ASC1a and DW-16, and chi-squared tests of F_2 segregation ratios

Cross	Isolate ASC1a				$\chi 2$ for F_2	Isolate DW-16				$\chi 2$ for F_2
	No. F_1 plants		No. F_2 Plants			No. F_1 plants		No. F_2 plants		
	R	S	R	S	1:3 ratio	R	S	R	S	1:3 ratio
HAR 604/Glenlea	0	12	60	150	1.427	0	9	40	130	0.196
HAR 2562/Glenlea	0	11	42	145	0.642	0	8	56	128	2.894
Dashen/Glenlea	0	10	37	122	0.253	0	11	62	143	3.01
					7:9 ratio[a]					7:9 ratio
HAR 604/HAR 2562	12	0	180	0	231.8[b]	10	0	200	0	257.1[b]
HAR 604/Dashen	11	0	175	0	225.3[b]	10	0	190	0	281.3[b]
HAR 2562/Dashen	10	0	200	0	257.1[b]	10	0	185	0	238[b]

R = resistant, S = susceptible

[a] Ratio expected if resistance genes were recessive and independent, [b]significant at $P = 0.01$

ASC1a and DW-16, respectively (Table 1). HAR604, HAR2562 ACC. 16300/88 and Dashen were the most resistant genotypes to both isolates. The durum wheat genotypes showed disease reactions ranging from 2 to 5 with mean values of 4.1 and 3.8 for the *Ptr* ASC1a and DW-16 isolates, respectively. The cultivars Chinese Spring and Glenlea were susceptible to both isolates showing both necrosis and chlorosis symptoms.

Inheritance of tan spot resistance

The F_1 and F_2 results for crosses of resistant and susceptible genotypes are shown in Table 2. F_1 plants were susceptible and the F_2 populations segregated in 1 resistant:3 susceptible ratios indicating that resistance was controlled by a single recessive gene in each instance. All F_1 and F_2 plants of the three possible Resistant/Resistant crosses (HAR604/HAR2562, HAR604/Dashen, HAR2562/Dashen) were resistant (Table 2) to both isolates indicating the resistance genes were allelic or tightly linked.

Chromosomal location of the resistance gene

Monosomic F_1 and F_2 analyses of resistance in cultivars HAR604 and HAR2562, are shown in Tables 3 and 4, respectively. The F_1 crosses were tested only with isolate ASC1a. The cross mono5A/HAR604 failed. All CS mono/HAR604 and CS mono/HAR2562 F_1 hybrids, except CS mono3B/HAR604 and CS mono3B/HAR2562, which both segregated into resistant and susceptible plants, were susceptible

Table 3 Segregation for seedling reaction to *Ptr* isolate ASC1a and DW-16 in monosomic F_1 and F_2 populations from crosses of 21 'CS' monosomics with common wheat cultivar HAR604

Monosomic chromosome	ASC1a				$\chi 2_{1:3}$ in F_2	DW-16		$\chi^2_{1:3}$
	Segregation in F_1		Segregation in F_2			Observed segregation in F_2		
	R	S	R	S		R	S	
1A	0	7	12	30	0.285	15	42	0.052
2A	0	8	10	28	0.034	22	50	1.186
3A	0	12	13	29	0.793	11	30	0.072
4A	0	9	14	35	0.333	12	34	0.028
5A	–	–	–	–	–	–	–	–
6A	0	11	13	27	1.20	13	35	0.108
7A	0	11	7	25	0.165	10	37	0.347
1B	0	10	14	34	0.444	16	41	0.287
2B	0	12	15	35	0.667	20	49	0.584
3B	7	3	70	9	170.45**	60	8	145.01**
4B	0	9	10	36	0.26	9	28	0.01
5B	0	9	11	29	0.133	13	33	0.26
6B	0	8	13	33	0.26	17	47	0.082
7B	0	8	12	31	0.214	10	29	0.012
1D	0	10	10	28	0.036	14	43	0.01
2D	0	11	17	45	0.193	19	48	0.403
3D	0	12	20	48	0.705	12	36	0.529
4D	0	10	18	44	0.537	10	27	0.08
5D	0	8	9	22	0.268	14	37	0.162
6D	0	8	11	31	0.033	16	44	0.089
7D	0	9	13	38	0.01	10	28	0.667
Disome	0	20	30	84	0.104	35	93	0.374

**Significant at $P = 0.01$

Table 4 Segregation for seedling reaction to *Ptr* isolates ASC1a and DW-16 in monosomic F_1 and F_2 populations from crosses of 21 'CS' monosomics with common wheat cultivar HAR2562

Monosomic chromosome	ASC1a				$\chi^2_{1:3}$ in F_2	DW-16		
	F_1 segregation		F_2 segregation			F_2 segregation		$\chi^2_{1:3}$
	Resistant	Susceptible	Resistant	Susceptible		Resistant	Susceptible	
1A	0	10	18	42	0.80	11	30	0.072
2A	0	12	15	36	0.529	10	36	0.26
3A	0	8	10	42	0.922	14	34	0.319
4A	0	11	10	45	2.239	15	38	0.308
5A	0	9	8	30	0.314	10	28	0.034
6A	0	8	13	29	0.793	14	35	0.333
7A	0	8	9	26	0.01	12	33	2.917
1B	0	10	12	39	0.1	17	40	0.706
2B	0	11	16	40	0.38	19	45	0.749
3B	12	4	62	6	158.8**	69	7	175.35**
4B	0	10	15	40	0.15	10	36	0.26
5B	0	8	14	34	3.19	13	29	0.793
6B	0	7	14	41	0.01	16	40	0.38
7B	0	10	17	39	0.856	11	29	0.133
1D	0	11	10	34	0.12	13	37	0.03
2D	0	10	12	32	0.11	18	40	1.12
3D	0	9	18	40	1.12	16	41	0.287
4D	0	10	14	36	0.24	11	29	0.133
5D	0	8	8	25	0.01	10	26	0.148
6D	0	8	10	38	0.444	13	36	0.06
7D	0	10	15	40	0.151	15	35	0.666
Disomic	0	25	25	85	0.302	30	98	0.167

**Significant at $P = 0.01$

to both ASC1a and DW-16 isolates. All F_2 monosomic populations, except CS mono3B/HAR604 and CS mono3B/HAR2562, segregated 1 resistant:3 susceptible. Similarly, the disomic CS/HAR604 and CS/HAR2562 F_2 populations segregated 1:3 indicating that resistance in both cultivars, HAR604 and HAR2562, to both isolates was controlled by single recessive genes. The F_2 population CS mono3B/HAR604 segregated 70 resistant and 9 susceptible plants for isolate ASC1a and 60 resistant and 8 susceptible plants for isolate DW-16 (Table 3). The CS mono3B/HAR2562 F_2 population segregated in a similar manner (Table 4), deviating very significantly ($P < 0.001$) from the expected 1 resistant:3 susceptible ratios obtained for other monosomic populations and the control crosses. Thus the monosomic analyses and allelism tests indicated that the resistances in the two Ethiopian cultivars were controlled by a common recessive gene located on chromosome 3B. This gene is temporarily designated as *TsrHar*.

Discussion

The disease evaluation data of both durum and common wheat genotypes against the *Ptr* ASC1a (race 1) and DW-16 isolates (Fig. 1) could suggest that common wheat genotypes are better sources of tan spot resistance than durum wheat genotypes, although the number of durum wheat genotypes tested was very limited. Similar results were reported by Lamari and Berneir (1989a) and Singh and Hughes (2005). Furthermore, most of the tan spot resistance genes reported to-date, namely *Tsr1*

on 5BL (Faris et al. 1996), *Tsr3* on 3D (Tadesse et al. 2006a), and *Tsr4* on 3A (Tadesse et al. 2006b), are from hexaploid wheat. The major reported QTLs (Faris et al. 1997; Cheong et al. 2004; Faris and Frieson 2005) were also from hexaploid wheats. Singh et al. (2006) identified *Tsr2* on the long arm of chromosome 3B in tetraploid wheat using a *Ptr* race 3 isolate.

The absence of resistant plants in the F_1 disomic crosses of resistant cultivars HAR604, HAR2562, and Dashen with the susceptible cultivar Glenlea and the segregation of the corresponding F_2 crosses into 1 resistant:3 susceptible ratios indicated that the resistances in these cultivars were controlled by single recessive genes. The monosomic and disomic F_1 and F_2 crosses of these resistant cultivars with the susceptible CS monosomic and disomic CS showed the same results, which were in agreement with previous reports (Singh and Hughes 2005; Lamari et al. 2003; Lamari and Bernier 1989b, 1991; Gamba and Lamari 1998; Lee and Gough 1984; Tadesse et al. 2006a; b). On the other hand, quantitative inheritance of tan spot resistance was reported by Elias et al. (1989), Faris et al. (1997), Friesen and Faris (2004). Comparison of the studies, however, are difficult due to the variations in the methods of inoculation, rating scales, symptoms studied, isolates used, and the environmental conditions for disease development.

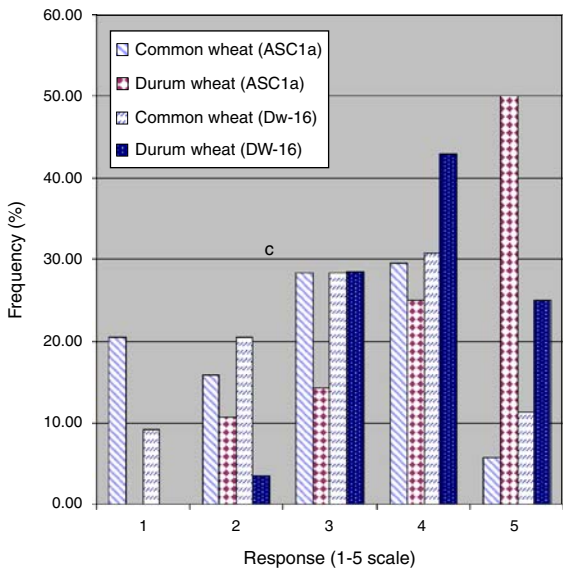

Fig. 1 Distribution of 118 Ethiopian wheat genotypes to two Pyrenophora tritici-repentis isolates

In monosomic analysis, when resistance is governed by a single hemizygous-effective recessive gene, the F_1 plants of all 20 non-critical monosomic crosses should be heterozgous and susceptible, but the critical cross should segregate into susceptible disomic and resistant monosomic F_1 plants. In the F_2, the 20 non-critical crosses segregate into a 1 resistant:3 susceptible ratios, whereas the critical cross should deviate significantly from this ratio (Knott 1989). In the present investigation, only CS mono 3B crosses segregated into resistant and susceptible F_1 plants and their F_2 segregation deviated significantly from the 1:3 ratios, indicating that the recessive resistance gene was located on chromosome 3B. The susceptible plants in the critical crosses were expected to be nullisomic ($2n = 6\times = 40$), but such confirmations were not made in this study.

The lack of segregation among the F_2 crosses HAR604/HAR2562, HAR604/ Dashen and HAR2562/Dashen, indicated that all three cultivars posses the same gene or, less likely, tightly linked genes. The temporary gene designation *TsrHar* was applied assuming there was a single gene. Singh et al. (2006) identified resistance gene *Tsr2* (*tsn2*) on chromosome 3B from durum line PI 352519 using an isolate of *Ptr* race 3. The gene in the Ethiopian common wheats is not expected to be the same as *Tsr2* because isolates of race 3, to which the T*sr2* confers resistance, are virtually never isolated from hexaploid wheats (Effertz et al. 2002). Furthermore, *Ptr* race 3 isolates also cause necrosis in durum wheats, but not in hexaploid wheats (Singh et al. 2006). However, the genes could be located at the same locus. The temporary designation *TsrHar* is therefore used to describe the gene identified in the present study.

The highly resistant cultivars identified in the present study are recommended for use in breeding programs aimed at improving tan spot resistance in common wheat.

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