Phytopathol. Mediterr. (2005) 44, 247–255

Resistance of some Iraqi bread wheat cultivars to *Puccinia triticina*

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Summary. Brown rust (leaf rust) caused by *Puccinia triticina* is one of the most serious diseases of wheat worldwide. In Iraq the occurrence and distribution of brown rust is more regular and uniform than that of other wheat rusts. with yield losses as high as 44% on susceptible wheat cultivars in commercial fields. Recently several promising wheat (*Triticum aestivum*) cultivars with different levels of rust resistance have been released in Iraq. The present work was conducted to postulate the resistance genes in twenty-two Iraqi bread wheat cultivars by testing them with thirteen Mexican races of *P. triticina*. 'Thatcher' near-isogenic lines were used as testers for known resistance genes. Ten day old seedling sets were artificially inoculated with each race, and the infection type was recorded ten days later. Field reactions of the cultivars with the predominantly Iraqi races were determined under field conditions for three years. Results revealed that the Iraqi wheat cultivars possessed brown rust resistance genes *Lr1, 3, 10, 13, 16, 17, 23* and *26*, either alone or in various combinations. The presence of unknown resistance genes was also postulated in some cultivars. *Lr23*, derived from *Triticum turgidum* var. *durum*, was present in 23% of tested cultivars, whereas *Lr13* was present in 18%. The presence of *Lr26* in 'Al-Nour' and 'Hashemia' indicated that they carried the 1BL.1RS wheat-rye translocation. 'Al-Melad' displayed resistant reactions to all races used in the study. 'Tamuz 3' and 'Al-Nour' displayed high adult-plant resistance to *P. triticina* in the field.

Key words: *Triticum aestivum*, brown rust, leaf rust, *Puccinia recondita*, resistance genes.

Introduction

Bread wheat (*Triticum aestivum* L.) is one of the most important cereal crops worldwide (Gooding and Davis, 1997). The total area of wheat cultivation in Iraq is about 1.5 million hectares distributed over three agro-ecological zones or mega-environments (ME1, ME2 and ME4) (Van Ginkel *et al*., 2000). Many diseases, particularly rusts, drastically reduce the yield and quality of wheat (Al-Baldawi, 1993; Al-Maaroof *et al*., 2001). Of the three wheat rusts, brown (or leaf) rust caused by *Puccinia triticina* Ericks. is the most important. The occurrence and distribution of brown rust is more regular and uniform than that of the other rust diseases in all areas of Iraq where wheat is grown (Al-Maaroof *et al*., 1995, 2000) and epidemics have been frequent in all seasons except during 1997 and 2000 due to dry conditions (Al-Maaroof *et al*., 2002). The rust causes yield reductions as high as 44% as recorded in commercial fields (Al-Maaroof *et al*., 2001).

Deploying resistant cultivars is the most practical and economic method to control rust diseases because it is environmentally safe and does not

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require disease control inputs from the growers. This is very important, especially in areas where farmers do not have adequate resources to purchase and apply chemical agents (Browder and Eversmeyer, 1980).

The ability to diversify the genetic base of resistance depends on the availability of resistance genes in the germplasm commonly used in breeding. Genes that confer disease resistance on cultivars can be postulated if the pathogen possesses different avirulence/virulence gene combinations. This method, which is based on the gene-for-gene hypothesis, has been described and used by several workers (Browder and Eversmeyer, 1980; Statler, 1984; Singh and Rajaram, 1991). Gene postulation can be confirmed by genetic analysis.

Virulence analysis of *P. triticina* races from different locations of Iraq indicate that they are virulent against *Lr1, 2a, 2b, 2c, 3, 9, 11, 13, 14a, 14b, 18, 19, 20, 23*, *33* and *Lr*C (Al-Maaroof *et al*., 2002).

Great importance is given to selection for disease resistance in Iraqi wheat breeding programs. Recently a number of promising wheat cultivars with varying host reactions against rust diseases were released in this country (Ministry of Agriculture, 1992, 1994). The resistant cultivars were obtained by different breeding methods. Selection for resistance was based on field observations against the prevalent local races of *P. triticina* in the absence of information about the exact gene constitution of the cultivars. Therefore, the postulation of resistance genes can assist Iraqi breeding programs in developing cultivars that have more effective resistance genes against prevalent races of the pathogen.

The objective of the current study was to determine the genetic basis of brown rust resistance in Iraqi bread wheat cultivars. This information will be useful for national breeding strategies to improve brown rust resistance in wheat.

Materials and methods

Twenty-two Iraqi spring bread wheat (*Triticum aestivum*) cultivars and a set of testers, mostly 'Thatcher' near-isogenic lines carrying specific genes for resistance, were included in the study (Tables 1 and 3). Pure cultures of thirteen Mexican *P. triticina* races, designated according to Long and Kolmer (1989) and Singh (1991) were used. Between 8 and 10 seeds of the cultivars and testers were sown as hills in 30×23×7-cm plastic trays with a pasteurized mixture of soil and compost. Fourteen sets, each consisting of cultivars and 'Thatcher' near-isogenic lines were used separately in three replicates. Seedlings were grown under greenhouse conditions at 18–22°C. Ten days later each set of seedlings (with newly emerged second leaves) was artificially inoculated separately with each of the thirteen races. For one set the seedlings were allowed to grow for 14 days (fully expanded second leaves) before being inoculated. Inoculation was carried out by uniformly spraying the seedlings with suspensions of urediniospores of each race in light-weight mineral oil (Soltrol 170, Philips 66 Co, Bartlesville, OK, USA) using a fine atomizer. Inoculated seedlings were left in an open area for one to two hours and were then placed overnight in a humidity chamber set at 18–20°C. After incubation the seedlings were placed in a greenhouse at 20–25°C. Disease reactions or the infection types displayed by seedlings were recorded ten days later according to the 0–4 scale described by Stakman *et al*. (1962). The presence of brown rust resistance genes (*Lr* genes) in the seedlings of the cultivars were postulated by comparing the low and high infection types displayed by them with the infection type of known *Lr* genes in the testers (Singh and Rajaram, 1991).

Field experiments were carried out for three seasons at Al-Twaitha Experimental Station located 30 km southeast of Baghdad, Iraq during the 5-year period 1997–2001. The cultivars and 'Thatcher' near-isogenic lines were planted in three rows two meter in length and 30 cm apart using a randomized complete block design with three replicates. A bulk population of *P. triticina* urediniospores, collected from naturally infected fields at various locations in the previous season, was multiplied to obtain fresh urediniospores for inoculation. Artificial inoculation was conducted at the stem elongation stage by spraying with the urediniospore-water suspension, supplemented with four drops of Triton per liter of water to break the surface tension of the water and thus allow the spores to be suspended. The inoculation was repeated two weeks later (Zadoks *et al*., 1974). The host response

to infection was evaluated following the scale described in Roelfs *et al*. (1992), in which 0, no visible infection; R, resistant, yellow chlorotic or necrotic area with or without small pustules; MR, moderately resistant, small pustules surrounded by chlorotic or necrotic areas; M, intermediate (mesothetic) resistance, pustules of variable size with some chlorosis or necrosis; MS, moderately susceptible, medium sized pustules, no necrosis but some chlorosis possible; and S, susceptible, large pustules, no necrosis or chlorosis. Disease severity was estimated using the modified Cobb scale giving the percentage of rusted leaf tissues (Peterson *et al*., 1948).

Table 1. Host tester series with named *Lr* genes for brown (leaf) rust resistance and their chromosomal location.

Cross[a]	Tester	*Lr* genes	Chromosome location
Thatcher	Thatcher	*Lr22b*	2DS
TC×6/Centenario	RL6003	*Lr1*	5DL
TC×6/Webster	RL6016	*Lr2a*	2DS
TC×6/Carina	RL6019	*Lr2b*	2DS
TC×6/Loros	RL6025	*Lr2c*	2DS
TC×6/Democart	RL6002	*Lr3*	6BL
TC×6/Klein Aniversario	RL6007	*Lr3ka*	6BL
Bage/8×TC	RL6042	*Lr3bg*	6BL
Transfer/6×TC	RL6010	*Lr9*	6BL
TC×6/Exchange	RL6004	*Lr10*	1AS
Hussar W976	RL6053	*Lr11*	2A
Exchange/6×TC	RL6011	*Lr12*	4B
Manitou	Manitou	*Lr13*	2BS
Selkirk/ 6×TC	RL6013	*Lr14a*	7BL
TC×6/Maria Escobar	RL6006	*Lr14b*	7BL
TC×6/ Kenya W1483	RL6052	*Lr15*	2DS
TC×6/ Exchange	RL6005	*Lr16*	2BS
Klein Lucero/ 6×TC	RL6008	*Lr17*	2AS
TC×7/S. Africa 43	RL6009	*Lr18*	5BL
TC×7/TR	RL6040	*Lr19*	7DL
Thew W203	Thew	*Lr20*	7AL
TC×6/RL5406	RL6043	*Lr21*	1DL
TC×6/RL5404	RL6044	*Lr22a*	2DS
Lee FL 310/6×TC	RL6012	*Lr23*	2BS
TC×6/Agent	RL6064	*Lr24*	3DL
TC×7/Transec	RL6084	*Lr25*	4BS
TC×6/ST-1-25	RL6078	*Lr26*	1BL.1RS
Gatcher (W3201)	Gatcher	*Lr10,27+31*	3BS, 4BS
CS 2D-2M	RL6079	*Lr28*	4AL
TC×6/CS7D/AG# 11	RL6080	*Lr29*	7DS
TC×6/Terenzio	RL6049	*Lr30*	4AL
TC×7//R.L.5497-1/Marquis-K	RL6086	*Lr32*	3DS
TC×6/P158548	RL6057	*Lr33*	1BL
TC×6/P158548	RL6058	*Lr34*	7DS
Marquis-K×8/R.L.5347	RL5711	*Lr35*	2B
E 84018	E84018	*Lr36*	6BS
TC×8/VPM1	RL6081	*Lr37*	2AS
TC×6//Carina	RL6051	*LrB*	–
WL 711	WL711	*Lr13*	2BS
Gaza (W277)	Gaza	*Lr23,+*	2BS

[a] Seed source: International Maize and Wheat Improvement Center (CIMMYT), Mexico D.F., Mexico.

Results and discussion

The host-parasite interactions of the 37 tester lines with the 13 *P. triticina* races are given in Table 2. Resistance genes *Lr3ka, 9, 16, 21, 25, 29* and *Lr30* displayed low infection types (ITs) with all races. Postulation of the resistance genes *Lr12, 14b, 20, 22a, 22b, 35* and *37* was not possible because of the high infection types displayed by them with all races either due to the absence of avirulence in the races used (for genes *Lr14b* and *20*) or because

Table 2. Seedling reactions (infection types) displayed by known *Lr* gene testers when inoculated with 13 races of *Puccinia triticina*.

Resistance gene	Race												
	1 BBB/BB	2 BBG /BN	3 CBJ/QB	4 CBJ/QL	5 CBJ/QQ	6 CCJ/SP	7 NCJ/BN	8 MFB/SP	9 TBD/TM	10 TCB/TD	11 MCJ/QM	12 MCJ/SP	13 MBJ/SP
Lr22b	3+	3+	3+	3+	3	3	4	3+	3+	3+	3+	3	3+
Lr1	0;	0;	0;	0;	0;	;	4	3+	3+	4	3+	3+	3+
Lr2a	;	;	;	0;	0;	0;	1	;	3	3+	0;	0;	0;
Lr2b	;1-	1+	1	0;	;	;	1+	;	3+	3+	0;	;	;
Lr2c	;1-	3+c	1+3c	;1-	;1-	;	3+	;1-	3+	3+	;	;	;1-
Lr3	;1-	0;	3+	12	3+	23c	;	3	3+	3+	3+	12-	3+
Lr3ka	;12	0;	12	;	12	;12	12	;1	12	12	;12	;1-	22+
Lr3bg	;1	0;	3	3+	3+	;12	0;	3	3+	3+	3+c	23c	3
Lr9	0;	0;	0;	0;	0;	;	0;	0;	0;	0;	0;	0;	0;
Lr10	;1-	3	;1-	3+	3+	3+	3+	3+	3+	;1-	3+	3+	3+
Lr11	1+3c	3+4	4	3+	4	3+	3+	3+	3+c	3+	3+	3+	3+
Lr12	4	3+	3+	3+	3+	3+	4	3+	3+	3+	3+	3+	3+
Lr13	X+	X	3+	3+	3+	3+	X+	3+	3+	3+	3+	3+	3+
Lr14a	X+	XX+	3+	3+	4	3+	4	3+	3+	3+	3+	3+	3+
Lr14b	3+	3+	3+	3+	3+	3+	4	3+	3+	3+	3+	3+	3+
Lr15	;1-	;1-	0;	0;	0;	3+	1	3+	3+	3+	;1-	3+	3+
Lr16	1+	1	1	1+	;1-	1	1+	1-	1	1	;1-	1	1+
Lr17	1-	0;	3+	3+	3+	3+	3+	;	3+	;	3+	3+	3+
Lr18	2+3	3+	3+	_	2+3c	3	3+	3+	3+	3+	3	3	2+3
Lr19	0;	0;	;	0;	3+	0;	0;	0;	0;	0;	0;	0;	0;
Lr20	3+	3+	3+	3+	3+	4	4	3+	3+	3+	3+	3+	3+
Lr21	2	12-	1+2	12	12	12	12	;1	;1	12	1	12-	12
Lr22a	3+	3+	3+	3+	3+	3	3+	3+	3+	3+	3	3	3
Lr23	12	3+	;1-	;1-	11+	3+	3+	3+	12	3+	;	3	3+
Lr24	;	;	;	;	;	;1-	;	3+	12	;12	;12	;	;1-
Lr25	0;	0;	0;	0;	;	0;	0;	0;	0;	0;	0;	0;	;
Lr26	11+	0;	1	;	0	3	4	3c	;	3+	3c	3	12
Lr10, 27+31	;1	;1	;	X	XX+	3c3	X	3	3+	;	3	3	4
Lr28	0;	X-	0;	0;	0;	0;	4	3+	3+	3+	0;	0;	0;
Lr29	;1	;1-	;1	;1-	;1-	;	;12	1	1	;1	;1-	;1	;1
Lr30	;	12	23c	_	23c	;	3c	12	23c	12	;1	;	23-
Lr33	3	3	2+3	3+	3c3	23c	3+	12	12	12	12	12	3+
Lr34	3	3	3	3	3-3	3-3	3	3c	3	3	3c3	3	3
Lr35	3+	3c	3c3	3c	3c3	3c3	3+	3c3+	3+c	–	3	3+c	3+
Lr36	;1-	;1	;1	1	1+3c	12	;1-	;	1	1	1	1+	12
Lr37	3+4	3+	3+	3	3+	4	3+	3+	3+	3+	3	3+	4

[a] Infection types follow a 0 to 4 scale (Stakman *et al.*, 1962); + and – sign following the infection type indicate a larger or smaller size than normal for uredinia; indicates presence of hypersensitive necrotic or chlorotic flecks of varying size; X, indicates random distribution of variable size; XX, indicates random distribution of variable-sized uredia on single leaf with a pure culture; c, indicats uredinia surrounded with chlorosis.

of their adult-plant nature (*Lr12, 22a, 22b, 35* and *37*) (Singh and Huerta, 1995). The resistance gene *Lr34* could be detected with these races only if it was tested at low temperatures and with low light intensity (Singh and Chen, 1999).

Eight known brown-rust resistance genes, *Lr1, 3, 10, 13, 16, 17, 23* and *26*, could be postulated either alone or in various combinations in Iraqi spring bread wheat cultivars (Table 3). It is likely that some cultivars carried additional resistance genes that conferred resistance against some races; however such resistance may not be useful because these unknown resistance genes were not effective to several other races. Gene *Lr23*, derived from *Triticum turgidum* var. *durum*, was the most frequent, being identified in 23% of cultivars. It was followed by gene *Lr13,* present in 18% of cultivars. Six cultivars, 'Iratom', 'Al-Ize', 'Maxipak', 'Telafar 2', 'Telafar 3' and 'Saber Beg' were susceptible to all races in seedling tests; therefore we could not postulate any resistance gene in them.

Lr1 displayed very low infection types (0; or ;) with six of the thirteen races (Table 2) and was postulated in two cultivars, 'Rabia' and 'Al-Kaed' (Table 3). Low IT with race CCJ/SP and high infection type with race MCJ/SP confirmed the presence of *Lr1* because these two races differed only in their avirulence or virulence to *Lr1*. Gene *Lr3* could be present in 'Al-Nour' due to its low infection type (0; and ;) with race NCJ/BN, which is avirulent to *Lr3* but virulent to the other genes, *Lr17, 23* and *26*, postulated in this cultivar. Low infection types with races MFB/SP and TCB/TD could indicate the presence of *Lr17* in 'Al-Nour'. Infection type 3+ with race MCJ/SP and IT X with MBJ/SP almost certainly indicated the presence of *Lr26*. High virulence frequencies are known to occur for *Lr1* and *Lr3* in the Iraqi *P. triticina* pop-

Table 3. Seedling reactions (infection types) displayed by the Iraqi wheat cultivars when inoculated with 13 *Puccinia triticina* races and postulated *Lr genes*.

Cultivar	Race[a]														Postulated
	1 BBB/BB	2 BBG /BN	3 CBJ/QB	4 CBJ/QL	5 CBJ/QQ	6 CCJ/SP	7 NCJ/BN	7 [b] NCJ/BN	8 MFB/SP	9 TBD/TM	10 TCB/TD	11 MCJ/QM	12 MCJ/SP	13 MBJ/SP	*Lr* genes
Abu-Ghraib	X	;1	1	1+3c	;1	1+	1+	1+	1	1	1	;1-	;1-	1+3c	*16*
Al-Kaed	0;	;	0;	0;	0;	0;	3+	3+	3c3	12	4	;	3+	_	*1,23*
Al-Khair	1	4	1+	1+	1	3+	3+	3+	3c3	23c	X	;1	;1	X	*23,+*
Al-Nour	0;	0;	0;	;	;1	3c3	;	0;	1+	;	;1	;1	3+	X	*3, 17, 23, 26*
Al-Melad	1+	1	1	1+	;	1+	1	1	;1-	1	1	;1-	1+	1+	*16*
Al-Neda	;1	X	;1-	3+	X	3	3+	4	3c3	3+	;	3+	X-	3+	*10,+*
Al-Hashemia	;	0;	;1-	0;	0;	3+	3+	4	3c3	;	3c3	;1-	3c3	X	*23, 26*
Intsar	;1	;1	3+	3+	3	3+	X+	X+	3	3+	4	3+	3+	3+	*13*
Iratom	3+	4	3+	3+	3+	4	4	4	3+	3+	4	3+	3+	3+	None
Al-Ize	3	4	3+	3+	3+	4	4	4	3+	3+	4	3+	3+	3+	None
Latifia	;1	3	12	3+	3+	3+	4	4	3c3	3+	;1-	3+	3+	3+	*10*
Maxipak	3+	3+	3+	3+	3+	3+	4	4	3c3	3+	4	3+	3+	3+	None
Rabia	0;	0;	0;	0;	0;	0;	4	4	3+	3+	4	4	4	3+	*1*
Sali	1+	;1-	3+	3+	3	3+	X+	X+	3+	3+	4	3+	3+	3+	*13*
Tahadi	0;	X	;1-	2+3	3c3	3	3+	4	23c	3+	;	3+	_	3	*10,+*
Tamuz 2	3+	;1	12	3+	3c3	3c3	3+	33+	3c3	4	3+	3+	12	3+	+
Tamuz 3	;12	;1	3+	3+	3	3	X	X+	;1+	3+	3+	3	3	3	*13,+*
Telafar 2	3	4	3+	4	3	3+	3+	3+	3	3+	3+	3+	3+	4	None
Telafar 3	3	3+	3+	3+	3+	3+	3+	3+	3c3	3+	4	3+	3+	3+	None
Al-Zehra	0;	0;	0;	;	0;	3	3+	3+	;	;1	;	;	3+	3+	*17,23*
77M	X	X	3	3	3	3+	X+3	XX+	3c3	3+	4	3	3+	3+	*13*
Saber Beg	4	3+	3+	3+	3+	4	4	4	3+	4	4	4	3+	3+	None

[a] See Table 2.
[b] 14–days old seedlings inoculated for detecting the presence of *Lr13*.
+, presence of additional unknown gene.

ulation, therefore these genes are not likely to be useful in controlling brown rust if deployed alone.

Gene *Lr10* was postulated in 'Al-Tahadi', 'Al-Neda', and 'Latifia' due to the low infection type that these cultivars displayed with the *Lr10*-avirulent races BBB/BB, CBJ/QB and TCB/TD (Tables 2 and 3). Low and high infection types with the almost identical races CBJ/QB and CBJ/QL respectively gave a definite postulation for *Lr10*. Virulence for *Lr10* was low in the past and hence this gene it may play a role in Iraq if deployed in combination with other genes. It is worth mentioning that virulence for this gene is common in many other countries (McIntosh *et al*., 1995).

The low infection types conferred by *Lr13* could be observed in 'Intsar', 'Sali', 'Tamuz 3', and '77M' with the avirulent races BBB/BB, BBG/BN and NCJ/BN (Table 3). The variation in the low infection types conferred by this gene on the seedlings depended on various factors including temperature, plants growth stage, homozygosity or heterozygosity of avirulence in the pathogen and the genetic background (Singh and Rajaram, 1991; McIntosh *et al*., 1995). The variable infection type thus complicated the postulation of *Lr13*. Earlier studies in Mexico (Singh and Rajaram, 1991) have shown that inoculating 14-day-old plants often gives desirable results, therefore we inoculated 14-day-old seedling of the cultivars with the *Lr13*-avirulent race NCJ/BN. The intermediate or mesothetic infections produced indicated the presence of *Lr13*. This gene was described as a gene that confers resistance only on adult plants by McIntosh *et al.* (1995); however, earlier research and the present study show that it can also be detected in the seedlings. Virulence frequency for this gene was low in the past in Iraq, and it must therefore be deployed carefully in combination with other genes.

'Abu-Ghraib' and 'Al-Melad' were postulated to carry *Lr16* due to their characteristic infection types, about 1 with almost all races. Even with the avirulent races, this gene does not confer adequate resistance in the field; however, it interacts with other, unknown resistance genes to enhance the level of resistance (Singh and Huerta-Espino, 1995).

Gene *Lr17* displayed low infection types with four races: BBB/BB, BBB/BN, MFB/SP and TCB/TD (Table 2), and its presence in combination with *Lr23* could be postulated in 'Al-Zehra' and, in combination with *Lr3, 23* and *26*, in 'Al-Nour' (Table 3). The presence of *Lr23* in 'Al-Zehra' was inferred from its low infection types with *Lr23*-avirulent races CBJ/QB, CBJ/QL, CBJ/QQ, TBD/TM and MCJ/QM. Low IT with race MCJ/QM and high infection with MCJ/SP in the absence of gene *Lr15* gave a clear indication of the presence of *Lr23* due to the high similarity of these two races. Because virulence for *Lr17* is not known in Iraq (Al-Maaroof *et al*., 2002), this can be a useful resistance gene it must be used in combination with other effective resistance genes to enhance its longevity. Both 'Al-Zehra' and 'Al-Nour' were resistant in the field trials (Table 4).

Gene *Lr23*, derived from *Triticum turgidum* var. *durum*, was postulated in five cultivars: 'Al-Kaed', 'Al-Khair', 'Al-Nour', 'Al-Hashemia' and 'Al-Zehra' either alone or in combination with other genes. Virulence against this gene was first detected (Al-Maaroof *et al*., 2002) in Iraq in 1994. Cultivars possessing *Lr23* in combination with other genes can provide adequate resistance in warmer areas, as it is more effective at temperatures over 20°C (Dyck and Johnson, 1983).

Lr26 was present in two CIMMYT-derived cultivars: 'Al-Nour' and 'Hashemia'. This gene is known to be located on the wheat-rye translocation 1BL.1RS, which is present in many recent spring wheat cultivars derived from CIMMYT germplasm (Rajaram *et al*., 1997). The 1BL.1RS translocation is of special interest because besides *Lr26* it also carries genes *Sr31*, *Yr9* and *Pm8*, which confer resistance to black rust, yellow rust and powdery mildew respectively (McIntosh *et al*., 1995).

Adult-plant field responses of Iraqi cultivars to the populations of *P. triticina* are presented in Table 4. Three cultivars, 'Al-Nour', 'Al-Zehra' and 'Tamuz 3', showed high levels of resistance during all three seasons (group 1). Five cultivars 'Al-Kaed', 'Al-Khair, 'Al-Melad', 'Hashemia' and '77M', displayed fair resistance (resistant-moderately resistant) during the first two years and moderate resistance in 2001 (group 2). Six cultivars; 'Abu-Ghraib', 'Intsar', 'Iratom', 'Rabia', 'Sali' and 'Tamuz 2' showed moderate resistance in 1998 and 1999 but were moderately susceptible in 2001. The remaining eight cultivars belonged to the susceptible category (group 4): 'Al-Neda', 'Al-Ize', 'Latifia', 'Maxipak', 'Tahadi', 'Telafar 2', 'Telafar 3' and 'Saber Beg'. The higher responses of the cultivars in group 1

Table 4. Disease severity and reaction caused by *P. triticina* on Iraqi wheat cultivars at the adult-plant stage during 1998 to 2001 at the Twaitha Experimental Station, Baghdad, Iraq.

Cultivar	Postulated *Lr* genes[a]	Disease severity and infection type[b]		
		1998	1999	2001
Abu-Ghraib	*Lr16*	55 MS	35 MS	70 MS
Al-Kaed	*Lr1,23*	20 MR	10 MR	35 MR
Al-Khair	*Lr23,+*	30 MR	15 MR	50 MS
Al-Nour	*Lr3,17,23,26*	5 R	4 R	10 R
Al-Melad	*Lr16*	20 MS	15 MS	45 MS
Al-Neda	*Lr10,+*	80 S	45 S	85 S
Hashemia	*Lr23,26*	20 MR	10 MR	40 MS
Intsar	*Lr13*	20 MS	15 MS	70 MS
Iratom	None	30 MS	18 MS	65 S
Al-Ize	None	65 S	46 S	85 S
Latifia	*Lr10*	72 S	47 S	83 S
Maxipak	None	85 S	53 S	87 S
Rabia	*Lr1*	25 MR	17 MR	75 S
Sali	*Lr13*	25 MS	20 MS	65 MS
Tahadi	*Lr10,+*	85 S	55 S	90 S
Tamuz 2	+	35 MS	25 MS	65 S
Tamuz 3	*Lr13,+*	3 R	2 R	15 R
Telafar 2	None	87 S	47 S	90 S
Telafar 3	None	70 S	43 S	85 S
Al-zehra	*Lr17,23*	5 R	5 R	15 MR
77m	*Lr13*	15 MR	10 MR	25 MS
Saber Beg	None	92 S	57 S	95 S

[a] Following Table 3.
[b] R, resistant; MR, moderately resistant; MS, moderately susceptible; S, susceptible.

and 2 during 2001 may have been due to a more favorable climate for disease development, or to a change in the virulence pattern of the *P. triticina* population as observed on known *Lr* gene testers (Al-Maaroof *et al*., 2002). The high infection types displayed with all races by 'Saber Beg', 'Maxipak', 'Iratom', 'Al-Ize', 'Telafar 2' and 'Telafar 3' at the seedling stage was associated with a high susceptibility at the adult-plant stage in the field (Table 4). This was due to the absence of any resistance genes in these cultivars, which were also reported to be susceptible to brown rust in previous studies (Al-Baldawi, 1993; Al-Maaroof *et al*., 2000). In contrast, 'Iratom' was susceptible to all races at the seedling stage but displayed moderate resistance at the adult-plant stage, indicating that it might carry unknown adult resistance gene(s). As reported earlier (Singh and Huerta, 1995; Singh *et al*., 1999), gene *Lr16* present in 'Abu-Ghraib' and 'Al-Melad' made these cultivars resistant to all races at the seedling stage but did not confer adequate resistance in the field. Therefore, this gene must be combined with additional adult-plant resistant genes in wheat cultivars. The presence of *Lr10* alone in 'Latifia', or in combination with an unidentified gene in 'Tahadi' and 'Al-Neda', was not useful due to the high susceptibility of these cultivars in the field (Table 4).

An unknown gene in 'Tamuz 2' probably conferred some resistance on the adult plants (Table 4). Cultivars that carried *Lr13* varied in their susceptibility in the field; 'Tamuz 3' and '77M' were most resistant, while 'Sali' and 'Intsar' were moderately resistant in the first two years of testing and moderately susceptible in the third year. While 'Tamuz 3'seedlings carried an additional unknown resistance gene that was effective against some races, additional adult-plant resistance genes may

also be present in 'Tamuz 3' and '77M'. The resistance of 'Tamuz 3' could be of a durable nature as it has remained effective despite its cultivation on a large scale in different areas since its release in 1992 (Ministry of Agriculture, 1992, 1994; Ibrahim *et al*., 1998; Al-Maroof *et al*., 2000). 'Al-Nour' and 'Al-Zehra', which carry *Lr17*, were highly resistant in the field. This was expected as virulence for *Lr17* is absent in Iraq (Al-Maaroof *et al*., 2002).

Gene *Lr23* conferred effective resistance on several cultivars during the first two years of field testing but was less effective in the third year (Table 4). Since *Lr26* always occurred in combination with *Lr23* or other effective genes, it may not confer adequate resistance to Iraqi races of *P. triticina*. With avirulent races, *Lr26* displayed immunity (McIntosh *et al.*, 1995).

In conclusion, Iraqi bread wheat cultivars have narrow genetic diversity for genes that confer resistance to brown rust. In total, eight known genes (*Lr1, 3, 10, 13, 16, 17, 23* and 26) and one or more unknown genes were postulated in the Iraqi cultivars. These findings should be useful to national wheat breeding programs in order to improve brown rust resistance. The incorporation of additional effective resistance genes in wheat germplasm currently used in the WANA (West Asia and North Africa) region will be extremely important to increase the genetic diversity of cultivars in the future.

Acknowledgements

The first author thanks the International Maize and Wheat Improvement Center (CIMMYT) for supporting this study.

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Accepted for publication: October 1, 2005