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Field Resistance of Wheat (*Triticum aestivum* L.) Genotypes from Different Countries to Leaf Rust (*Puccinia triticina*)

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Abstract: *Puccinia triticina* causes leaf rust in wheat and results in severe yield losses in mild wheat growing areas of Turkey: Thrace, Marmara, Mediterranean, and Çukurova. A feasible way to avoid any damage is to grow slow rusting cultivars. We evaluated 94 wheat genotypes from different countries and 39 leaf rust differentials for their reactions to leaf rust field epidemics. Slow rusting, though, requires both the prediction of Lr genes in the greenhouse and an effective screening of genotypes against the leaf rust field epidemics; the evaluation at the sites regularly favoring leaf rust epidemics like Adapazarı is also possible. Various resistance genes, excluding *Lr13*, *Lr14a*, *Lr14b*, *Lr11*, *Lr30*, and *Lr32*, were still effective and many genotypes, excluding 16, 19, 49, 53, 74, 56, 61, 68, 46, 71, 5, 47, and 48, had various levels of resistance indicated by lower AUDPC%. Utilizing leaf rust effective genes in wheat breeding programs and growing resistant cultivars on a large scale would most likely decrease leaf rust related yield and quality losses.

Key Words: AUDPC, leaf rust, slow rusting, wheat, Turkey

Farklı Ülkelerde Geliştirilmiş Buğday (*Triticum aestivum* L.) Genotiplerinin Kahverengi Pasa (*Puccinia triticina*) Karşı Tarla Dayanıklılık Düzeyleri

Özet: *Puccinia triticina*, Türkiye'nin ılıman iklim buğday bölgeleri olan Trakya, Marmara, Akdeniz ve Çukurova'da kahverengi pasa ve dolayısıyla da ciddi verim kayıplarına neden olmaktadır. Hastalığın zararlarından yavaş paslanan buğday çeşitlerinin ekilmesiyle ucuz ve etkili bir şekilde sakınılabilmektedir. Çalışmamızda; 94 buğday genotipi ile 39 kahverengi pas ayırıcı hattında tarla tepkileri, yavaş paslanma düzeyleri ve ayırıcı hatlardaki olası dayanıklılık genleri araştırılmıştır. Yavaş paslanma, en doğru olarak, serada Lr dayanıklılık genlerinin tahmin edilmesi ve tarlada kahverengi pasın gözlenmesi sonucunda anlaşılır. Ancak bu değerlendirme; Adapazarı gibi kahverengi pas epidemisinin her yıl düzenli olarak etkilenen yörelerdeki tarlalarda, bir dereceye kadar, yalnızca tarla gözlemleri sonucunda da yapılabilir. Sonuç olarak; *Lr13*, *Lr14a*, *Lr14b*, *Lr11*, *Lr30*, ve *Lr32* dışındaki genlerin dayanıklılığının sürdüğü ve 16, 19, 49, 53, 74, 56, 61, 68, 46, 71, 5, 47, ve 48 numaralı genotipler dışındakilerin yavaş paslandıkları anlaşılmıştır. Anılan bu dayanıklılık genlerinin ıslah programlarında kullanılması ve bu genleri taşıyan buğday çeşitlerinin geniş alanlarda ekilmesinin sağlanması, kahverengi pasta dolaylı oluşabilecek verim ve kalite kayıplarını azaltacaktır.

Anahtar Sözcükler: HAE, kahverengi pas, yavaş paslanma, buğday, Türkiye

Introduction

Wheat (*Triticum aestivum* subsp. *aestivum*), grown both in warmer and cooler regions, is a primary food crop and an important actor in agricultural systems of developing countries around the world. Several diseases such as *Puccinia* spp. (rusts), *Ustilago* spp. (smuts), *Tilletia* spp. (bunts), and *Erysiphe* spp. (mildew)

unfortunately decrease wheat's yield and quality seriously in some years.

Three rusts: yellow rust, leaf rust, and stem rust, the most destructive wheat pathogens, reduce yield and quality by restricting the photosynthesis area on wheat leaves (Loegering, 1967; Altay, 1978; Kınacı, 1983; Onoğur, 1993; Khan et al., 1997; Sayre et al., 1998;

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Arslan et al., 2002). Each 1% increase in leaf rust severity decreases yield 40.07 kg ha⁻¹ and 1000 kernel weight 0.13 g (Arslan et al., 2002). Although some morphological markers happened to be the signs of resistance (Stakman, 1962; Singh, 1993; Ulukan and Özgen, 1999), more research is still needed to identify reliable markers apart from for leaf tip necrosis of *Lr34*.

Genetic resistance, with higher numbers of genes loaded into wheat genotypes, is the most economical and common approach to control the disease (Stubbs et al., 1986). A resistance breeding program first identifies resistance genes, and then incorporates them into cultivars of economical importance. Many scientists have identified leaf rust (*Lr*) resistance genes: Kolmer (1996) 46 *Lr* genes, Dyck et al. (1966) *Lr13* and *Lr12* genes, and McIntosh (1992) and McIntosh et al. (1995) *Lr1*, *Lr2*, *Lr3*, *Lr13*, *Lr17*, and *Lr24*.

Our main aim was to determine *Lr* resistance genes still effective and the level of slow leaf rusting in cultivars from different countries, under Adapazarı conditions, where regular leaf rust epidemics occur almost every year.

Materials and Methods

The 94 wheat genotypes (Table 1) and 39 leaf rust differentials, obtained from the International Center for Agricultural Research in Dryland Areas (ICARDA), were tested against *Lr* pathotypes at Adapazarı during the 2005-2006 season. Genotypes and leaf rust differentials were planted in two 1-m rows of 5 by 35 cm in a randomized complete block design with 3 replications. Susceptible spreaders (cv. Katia 1) were included at every 20 rows. The average rainfall, higher than the long-term average of 586.09, was 603.70 mm and the minimum temperature was 4.50 °C in January and the maximum 22.20 °C in April (Table 2). There was no need for any artificial inoculation (Arslan et al., 2002) because of optimum temperatures of 8-28 °C (Stubbs et al., 1986) with adequate rainfall just before leaf rust development in March. Leaf rust severities and responses were recorded 3 times on flag leaves at 8-13 day intervals, starting with the appearance of the first symptoms on the flag leaf. Severity estimations were according to the modified Cobb scale (Peterson et al., 1948) and growth stages to the Zadoks scale (Zadoks, 1974). The response to infection was also scored: R = resistant, smaller uredia surrounded

by necrotic tissues; MR = moderately resistant, smaller uredia surrounded by necrotic tissues; MS = moderately susceptible, moderate sized uredia without necrotic tissues; S = susceptible, large uredia without necrotic tissues. Then the area under the disease progress curve (AUDPC) over leaf rust severity scores and AUDPC% over the severity of the cultivar with the highest AUDPC value were calculated using the Excel computer program. The formula for AUDPC was $\sum ((\text{Number of days between 2 consecutive readings}) \times (\text{First leaf rust reading} + \text{Second leaf rust reading}) / 2)$, (Singh, 1993). Leaf rust data of only 1 year were reported because of uniform and abundant epidemics (susceptible spreader, cv. Katia = 80 S across all the experiment), (Singh et al., 2001; N. Bolat, pers. com.).

Results

Susceptible spreaders, cv. Katia 1, included at every 20 rows, reached up to 80 S against to leaf rust across the experiment in the study year of 2005-2006, indicating uniform and well-developed leaf rust epidemics.

Reactions by differentials

Thirty-nine leaf rust differentials varied for AUDPC and AUDPC% (Table 3). The ranges for both indicated that some resistance genes were still effective while some others, of course, were not. Lines with *Lr28*, *Lr19*, *Lr23*, *Lr24*, *Lr25*, *Lr29*, *Lr2a*, *Lr15*, and *Lr37* alone and lines with *Lr10*, *Lr2+Lr31* in combination had lower AUDPC% than 10% and were assumed to be resistant. Lines carrying *Lr13* alone were susceptible with a 100% of AUDPC. Various levels of resistance also existed in leaf rust differentials (Table 3). The reason why 2 *Lr13* differential lines (Manitou and WL711) differed in terms of their severity, infection types, and, of course, AUDPC was not clear and requires further investigation under Adapazarı conditions.

Reactions by cultivars

Genotypes 2, 7, 10, 13, 24, 36, 37, 40, 41, 54, 82, 83, 84, 85, 86, 89, 92, 12, 14, 20, 26, 29, 31, 32, 35, 38, 39, 43, 45, 51, 58, 70, 76, 87, 88, 90, 91, 11, 57, 66, 27, 6, 9, 21, 42, 55, 72, 78, 81, 94, 3, 44, 23, 26, 28, 33, 8, 22, 30, and 50 had AUDPC% lower than 10% and were assumed to be resistant. Genotypes 19, 49, 53, 74, 56, 61, 68, 46, 71, 5, 47, and 48 had AUDPC% higher than 40% and were assumed to be field

Table 1. Genotypes tested against leaf rust in Adapazarı during 2005-2006.

No.	Genotypes	Origin	No.	Genotypes	Origin
1	AGLIKA	Bulgaria	48	VERNA	Russia
2	IVETA NTA-92/89-6	Bulgaria	49	UNA	Russia
3	GANSU-1	China	50	DON 95	Russia
4	CA 9640	China	51	ELANDS	South Africa
5	ZHONGMAI 16	China	52	DEMİR	Turkey
6	YUMAR	Colorado	53	KATIA 1	Turkey
7	DECAN 4	Finland	54	AKSEL	Turkey
8	BOEMA	Finland	55	ZENCİRCİ-2002	Turkey
9	DEFENSE	France	56	BAYRAKTAR	Turkey
10	GEORGE	Georgia	57	MV-AMANDA	Turkey
11	MV22-2000	Hungary	58	LAU/AGD/3/ODESSKAYA 95//OLVIYA/B 16	Turkey
12	MV18-2000	Hungary	59	MIR32/5/LOV29/3/FTG/SPWX	Turkey
13	MV14-2000	Hungary	60	PEHL//RPB8-68/CHRC	Turkey
14	MV10-2000	Hungary	61	PEHLI/504-14-3/6/KNR79/SPRINT/4/LOV29	Turkey
15	TODORA	Hungary	62	TE 3904-313110/FTM 2	Turkey
16	FATİMA – 2	Hungary	63	TEKİRDAĞ	Turkey
17	TURAN 2000	Hungary	64	GEREK 79	Turkey
18	MARTAR	Hungary	65	KIRGIZ	Turkey
19	SABALAN	Iran	66	AYTİN	Turkey
20	C73-20	Iran	67	SÖNMEZ 2001	Turkey
21	ES14/SITTA//AGRI/NAC	IWWIP*	68	ÇETİNEL	Turkey
22	DMN//SUT/AG(ES86-7)/3/OPATA	IWWIP	69	ALPU 2001	Turkey
23	VORONA/OPATA//PYN/BAU	IWWIP	70	BAĞCI 2002	Turkey
24	AU/3/MINN//HK/38MA/4/YMH/34A	IWWIP	71	KONYA 2002	Turkey
25	F4141-W-1-1/PASTOR//PYN/BAU	IWWIP	72	DAĞDAŞ 98	Turkey
26	F1502-W9-01//KS82W409/STP	IWWIP	73	EKİZ	Turkey
27	KS82W409/SPN//TAM106/TX78V3630	IWWIP	74	AHMETAGA	Turkey
28	AU/3/MINN//HK/38MA/4/YMH/ERA	IWWIP	75	KINACI 97	Turkey
29	PIOPIO/ATTILA/4/YMH/TOB//MCD/3/LIRA	IWWIP	76	TAHİROVA 2000	Turkey
30	F130-L-1-12*2/MILAN	IWWIP	77	PAMUKOVA 97	Turkey
31	94.43591/CHOIX	IWWIP	78	BANDIRMA 97	Turkey
32	YE2453//PPBB68/CHRC	IWWIP	79	META 2002	Turkey
33	DJAMIN	Kyrgyzstan	80	KAŞİFBEY 95	Turkey
34	8023.16.1.1//KAUZ	Mexico	81	ZİYABEY 98	Turkey
35	TAM106 RESEL/TX69D4819/6/WRM	Mexico	82	CEYHAN 99	Turkey
36	BONITO-36	Mexico	83	CANİK	Turkey
37	BONITO-44	Mexico	84	BITARAP	Turkmenistan
38	PONY/OPATA//PSN/BOW	Mexico	85	GUNDJA	Turkmenistan
39	CAPUZ	Moldova	86	NIKONIYA	Ukraine
40	DED 598/95	Poland	87	SIRENA	Ukraine
41	DESTIN	Romania	88	SELYANKA	Ukraine
42	EXPRES	Romania	89	TX96V2427	USA
43	F95948G1-4	Romania	90	OK101	USA
44	BUCUR	Romania	91	OR 942496	USA
45	ZIMORODOK	Russia	92	NE93496	USA
46	L 4224 K 12	Russia	93	JAGGER	USA
47	POBEDA 50	Russia	94	X87581L-1-1/KS84063-9-39-3-27	USA

* IWWIP: International Winter Wheat Improvement Program.

Table 2. Monthly and long-term rainfall and temperature in Adapazari.

Months	2005-2006		Long-term average	
	Temp. (°C)	Rainfall (mm)	Temp. (°C)	Rainfall (mm)
November	11.00	165.7	11.13	79.26
December	9.50	76.60	8.05	100.02
January	4.50	77.50	5.74	88.30
February	6.20	98.60	6.34	75.07
March	10.10	67.20	8.03	69.37
April	13.30	3.30	12.49	59.16
May	18.10	13.80	16.97	49.00
June	22.20	101.00	21.02	65.91
Total		603.70		586.09
Mean	11.86		11.22	

susceptible to Adapazari leaf rust races. The differences among cultivars for AUDPC% were significant ($P \leq 0.01$). Most cultivars having slow rusting or partial resistance (Caldwell, 1968; Parlevliet, 1975), a type of long lasting resistance (Figure), reflected durable field resistance in the cultivars. Slow rusting, as already described, develops slowly on wheat plants after leaf rust infection either because of a longer latent period or because of fewer and smaller uredia (Singh and Gupta, 1991; Singh, 1995; Kolmer, 1996; Singh et al., 1998; Singh, 1999; Singh et al., 2001; Singh et al., 2004).

Leaf tip necrosis

Genotypes 7, 10, 12, 26, 32, 39, 9, 8, 22, 80, 73, and 34 showed (Table 4) clear signs of leaf tip necrosis (Ltn), linked to *Lr34*, a long lasting resistance gene against leaf rust (Singh, 1993). *Lr34*, which can be easily determined via Ltn even when no leaf rust epidemics occur, appears to be a preferred resistance gene of leaf rust.

Discussion

Leaf rust development on 39 leaf rust differentials indicated that resistance genes *Lr28*, *Lr19*, *Lr23*, *Lr24*, *Lr25*, *Lr29*, *Lr2a*, *Lr15*, and *Lr37* were still effective while *Lr13* alone and *Lr10*, *Lr2+Lr31* in combination were not. Since adult plant responses of the differentials are indicators for the occurrence of the pathotypes (Singh and Gupta, 1991), the reactions on differential genotypes

could reflect the possible leaf rust resistant genes in the genotypes against the pathotypes in Adapazari. Prevalence of resistance genes against the pathotypes under field conditions, when supported, of course, by the seedling genes, could guide breeders to improve leaf rust resistant wheat genotypes. Therefore, gene determinations in the seedling stage too should accompany field studies for leaf rust, in the infrastructures built in Turkey as early as possible.

Sixty genotypes out of 94 had lower than 10% AUDPC% and were considered resistant to leaf rust pathotypes prevalent in Adapazari. Only 12 genotypes in the study were susceptible with 40% or more AUDPC percentage. The higher number of resistant genotypes with slow leaf rusting and the significant differences among genotypes for resistance indicated either the prevalence of *Lr34* or other unidentified genes effective at around 10 °C, or additional partially effective adult plant resistance genes, which might vary from year to year with slight ranking changes (Singh and Gupta, 1991; Singh, 1993). Singh (1993) also recommended to make crosses between genotypes with *Lr34* and genotypes with lower AUDPCs but without *Lr34* and expected transgressive segregants with increased resistance if adult plant genes were additive. The different sources of resistance utilized by breeders do not, of course, decrease the value of a good build-up of resistance, but durable ones should be preferred.

Table 3. Genes that existed, area under the disease progress curve (AUDPC), and AUDPC% over the most susceptible genotype of 39 leaf rust differentials, Adapazarı, 2005-2006.

No	Genotypes	Gene	Final		AUDPC	AUDPC%
			Severity	Infection		
1	THATCHER	Lr22b	20	MS	297	21
2	TC*6/CENTENATRIO (RL6003)	Lr1	10	MR	152	11
3	TC*6/WEBSTER (RL6016)	Lr2a	5	MR	73	5
4	TC*6/CARINA (RL6019)	Lr2b	10	MR	152	11
5	TC*6/LOROS (RL6047)	Lr2c	30	MS	442	31
6	TC*6/DEMOCRAT (RL6002)	Lr3	30	MS	442	31
7	TC*6/ANIVERSARIO (RL6007)	Lr3Ka	10	MR	152	11
8	BAGE/8*TC (RL6042)	Lr3Bg	10	MS	272	19
9	TRANSFER/6*TC (RL6010)	Lr9	10	MR	152	11
10	TC*6/EXCHANGE (RL6004)	Lr10	10	MS	432	30
11	KUSSAR (W976)	Lr11	40	MS	587	41
12	EXCHANGE/6*TC (RL6011)	Lr12	10	MR	145	10
13	MANITOU	Lr13	20	MS	297	21
14	SELKIRK/6*TC (RL6013)	Lr14a	40	MS	587	41
15	TC*6/MARIA ESCOBAR (RL6006)	Lr14b	30	MS	642	45
16	TC*6/KENYA 1483 (RL6052)	Lr15	5	MR	79	6
17	TC*6/EXCHANGE (RL6005)	Lr16	10	MR	152	11
18	KLEIN LUCERO/6*TC (RL6008)	Lr17	10	MR	152	11
19	TC*7/AFRICA 43 (RL6009)	Lr18	10	MR	152	11
20	TC*7/TR (RL6040)	Lr19	1	MR	7	0
21	THEW (W203)	Lr20	5	MR	178	13
22	TC*6/RL5406 (RL6043)	Lr21	5	MR	73	5
23	TC*6/RL5404 (RL6044)	Lr22a	5	MR	73	5
24	LEE 310/6*TC (RL6012)	Lr23	1	R	15	1
25	TC*6/AGENT (RL6064)	Lr24	5	R	73	5
26	TRANSEC (AWNED)	Lr25	5	R	73	5
27	TC*6/ST-1-25 (RL6078)	Lr26	10	MR	152	11
28	GATCHER (W3201)	Lr10, Lr27+Lr31	5	MR	79	6
29	CS2D-2M	Lr28	0	R	0	0
30	TC*6/CS7AG#11 (RL6080)	Lr29	5	MR	73	5
31	TC*6/TERENZ10 (RL6049)	Lr30	20	MS	537	38
32	TCLR32 (RL5497)	Lr32	20	MS	537	38
33	TC*6/PI58548 (RL6057)	Lr33	20	MS	297	21
34	TC*6/PI58548 (RL6058)	Lr34	20	MS	297	21
35	RL5711	Lr35	10	MR	145	10
36	E84018	Lr36	30	MS	442	31
37	TC*6/VPM (RL6081)	Lr37	5	MR	79	6
38	TC*6//CARINA (RL60510)	Lr B	30	MS	468	33
39	WL711	Lr13	80	S	1420	100

Table 4. Final infection severity, infection type, leaf tip necrosis, area under the disease progress curve (AUDPC), and AUDPC% of 94 genotypes from different countries, Adapazarı, 2005-2006.

G.* No.	Final		Ltn	AUDPC	AUDPC%	G.* No.	Final		Ltn	AUDPC	AUDPC%	
	Severity	Infection					Severity	Infection				
2	0			0	0	78	5	MR		73	5	
7	0		1	0	0	81	5	MR		73	5	
10	0		1	0	0	94	5	MS		73	5	
13	1	MR		4	0	3	5	MS	1	79	6	
24	1	MR		4	0	44	5	MS		79	6	
36	0			0	0	23	10	MSMR		93	7	
37	0			0	0	25	10	MS		93	7	
40	1	R		4	0	28	10	MS		93	7	
41	0			0	0	33	10	MS		93	7	
54	0			0	0	8	20	MS	1	133	9	
82	0			0	0	22	10	MS	1	145	10	
83	1	R		4	0	30	10	MRMS		145	10	
84	0			0	0	50	10	MS		145	10	
85	0			0	0	80	10	MSMR	1	152	11	
86	0			0	0	34	30	MSS	1	173	12	
89	0			0	0	17	20	MS		185	13	
92	0			0	0	18	20	MS		185	13	
12	1	MSMR	1	15	1	62	20	MS		185	13	
14	1	MR		15	1	65	20	S		185	13	
20	1	R		15	1	59	30	MS		225	16	
26	1	R	1	15	1	60	30	S		225	16	
29	1	R		15	1	1	20	MS		290	20	
31	1	R		15	1	52	20	MS		290	20	
32	1	R	1	15	1	63	20	MS		290	20	
35	1	R		15	1	75	20	MSMR		290	20	
38	1	R		15	1	93	20	MS		290	20	
39	1	R	1	15	1	69	20	MS		297	21	
43	1	MR		15	1	79	20	MS		297	21	
45	1	R		15	1	67	50	S		312	22	
51	1			15	1	73	30	MSS	1	337	24	
58	5	MS		20	1	15	40	S		370	26	
70	1	R		15	1	64	40	S		370	26	
76	1	R		15	1	77	30	MS		442	31	
87	1			15	1	4	40	S		475	33	
88	1	R		15	1	16	80	S		537	38	
90	1	MR		15	1	19	100	S		617	43	
91	1	MS		15	1	49	60	S		660	46	
11	5	MSMR		31	2	53	60			660	46	
57	5	MSMR		31	2	74	60	S		667	47	
66	5	MS		31	2	56	80	S		747	53	
27	10	MS		51	4	61	80	S		747	53	
6	5	MR		73	5	68	80	S		747	53	
9	5	MSMR	1	73	5	46	80	S		852	60	
21	5	MSMR		73	5	71	80	S		852	60	
42	5	MS		73	5	5	60	S		877	62	
55	5	MS		73	5	47	60	S		1000	70	
72	5	MS		73	5	48	80	S		852	85	
										F	1.95**	2.02**
										LSD	19.04	25.6

* G. No. = Genotype number; Ltn = Leaf tip necrosis; AUDPC = Area under the disease curve; AUDPC% = Area under the disease curve%; R = resistant; MR = moderately resistant; MS = moderately susceptible; S = susceptible.

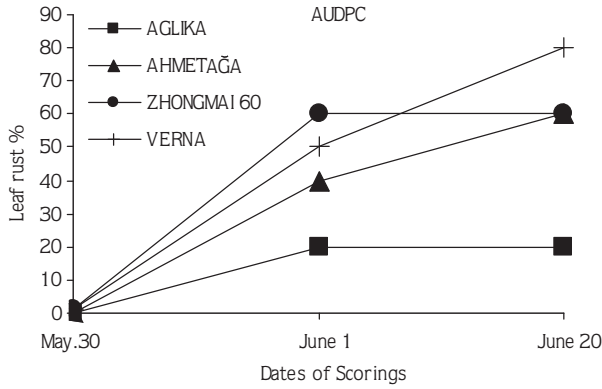


Figure. Area under the disease progress curve (AUDPC) of some genotypes.

Those susceptible cultivars, fortunately, did not seem to create a problem in wheat production areas of Turkey, because those released approximately 10-15 years ago still carry some sort of slow rusting resistance genes against leaf rust pathotypes. Kaşifbey 95 (Genotype 80), Pamukova 97 (Genotype 77), and META 2002 (Genotype 79), for instance, still had 11%, 31%, and 21% lower AUDPC percentages, respectively (Table 4).

Although either most cultivars carry resistance or even susceptible ones have slow rusting against leaf rust in the study, the building up of more leaf rust genes in cultivars still remains a necessity because of the fast, unexpected, and higher molding capacity of the pathotypes, where the leaf rust disease triangle in the nature is completed, regardless of whether it is in Turkey or somewhere else in the world.

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