Egypt. J. Phytopathol., Vol. 35, No. 1, pp. 25-34 (2007)

Use of Infection Type Data to Identify Genes for Low Reaction to Wheat Leaf Rust in Gemmeiza and Sids Cultivars

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Testing 14 commercial wheat cultivars against 12 test-isolates of *Puccinia triticina* identified *Lr* resistance genes. Eleven resistance genes, representing 55% of the total *Lr* genes used, *Lr 1, 2a, 2c, 2b, 3, 3ka, 10, 11, 16, 17,* and *26* were revealed, with different frequencies. The most frequent *Lr* genes were *Lr 17* and 26 followed by *Lr 3* and *10*.

Evidence indicated that, *Lr17* and *26* were postulated in five of the 14 cultivars used and *Lr3* and *10* were present in four cultivars.

Resulting leaf rust reaction types were consistent with the presence of *Lr 1* in cultivar Gemmeiza 1, Gemmeiza 7 and Sids 3; *Lr 2a, 2b, and 2c* in Gemmeiza 1; *Lr 3* in Gemmeiza 1, Gemmeiza 7 and Sids 1 and Sids 2; *Lr 3ka* in Gemmeiza 7, Sids 1 and Sids 3; *Lr10* in Gemmeiza 1, Gemmeiza 7, Sids 1 and Sids 2; *Lr 11* in Gemmeiza 7 and Sids 3; *Lr 16* in Gemmeiza7 and *Lr 17* and *26* in Gemmeiza 1, Gemmeiza 7and Sids 1-3, Nine cultivars had none of the 20 known *Lr* genes tested, two cultivars postulated to have eight Lr genes, two cultivars had five Lr genes and one cultivar had four *Lr* genes. All these 14 cultivars also had at least one unidentified resistance gene.

Keywords: Leaf rust disease, *Puccinia triticina*, resistance genes and wheat cultivars.

Leaf rust caused by *Puccinia triticina* is one of the most common diseases of wheat occurring nearly wherever wheat is grown (Wiese, 1987). In Egypt, leaf rust can cause yield losses of 5-10% or more in wheat cultivars when they lack adequate resistance (Nazim *et al.*, 1982 and 1983).

Recently, more interest is given to horizontal resistance; however, the major control methods for wheat leaf rust centres in the use of early maturing cultivars and the incorporation of genes for low reaction into commercial cultivars. Shafer and Roelfs (1985) reported that, the high level of resistance to leaf rust are race- specific and the durability of this resistance, may depend on the number of effective resistance genes that are combined in the wheat cultivars. For this reason, when pyramiding resistance genes in cultivars, it is important to know similarities and differences in genotypes for resistance to *Puccinia triticina* in wheat (*Triticum aestivum* L.) germplasm used as parents in the breeding programs to manage the available gene resources.

The gene-for-gene relationship (Flor, 1956 and 1971) between race-specific resistance in host plant and virulence in pathogens makes it possible to postulate resistance genotypes of host cultivars based on their reaction to a selected set of

pathogen isolates, Loegering (1978 and 1985) developed the concept of interorganismal genetics of host: pathogen association in gene- for- gene interaction and used it to detect the presence of specific resistance genes in host cultivars based on their reaction to pathogen isolates with suitable combinations of avirulence and virulence alleles. Data for infection types (ITs) of the host- pathogen interaction have been used to postulate the genes present in wheat cultivars for leaf rust and stem rust resistance (Singh *et al.*, 1999; Zhemchuzhina and Kryazheva 2002; Chekowski *et al.*, 2003; Kovalenko *et al.*, 2003 and Kolomiets *et al.*, 2004). The objective of this study was to postulate the leaf rust resistance genes in commercially grown Gemmeiza (5 cultivars) and Sids (9 cultivars) cultivars of Egyptian wheat which are frequently used as donors of other agronomic and quality characteristics.

Materials and Methods

The present research was funded by the Agricultural Technology Utilization and Transfer (ATUT), Agency for International Development (AID) and carried out at Cereal Dis. Lab., USDA, St. Paul, Minnesota Univ., Minneapolis, Minnesota, USA.

Pathogen isolates:

A set of 12 isolates of *Puccinia triticina* with different combinations of avirulence and virulence genes were selected for the test (Table 5). The isolates used in this study are representative of the predominant leaf rust races in Egypt. The isolates were assigned five- letters race designations based on high and low infection types to 16 Thacher differential lines (Long and Kolmer, 1989). Supplemental near-isogenic lines containing resistance genes *Lr14b, 15, 36,* and *42* were also inoculated at the same time as differential set (ATUT-W4 Project, ARC, Tables 1 and 2). Each isolate was tested on a set of 20 differential wheat lines, each with a different single *Lr* gene for resistance to leaf rust. The 20 differential lines and their low infection types (LITs) were shown in Table (3). The IT is indicated according to Stakman *et al.* (1962) scale of 0 (no visible reaction); (hypersensitive necrosis or chlorotic flecks), 1 (small uredinia often surrounded by necrosis), 2 (small to medium uredinia often surrounded by chlorosis), or various combinations of these reactions. In a few cases, IT3 (medium-size uredinia without chlorosis or necrosis) which is regarded as a high infection type (HIT), was seen in combination with a low IT. Each isolate was increased on a selected *Lr* lines susceptible to that isolate but not to other, to avoid potential combination of cultures used in subsequent tests.

Inoculation procedures:

The 20 near-isogenic *Lr* lines and the 14 cultivars (Table 4) to be tested were planted in vermiculite in 5.3 cm square plastic pots. Four entries were planted per pot with 10 to 15 seeds per entry planted in each corner of the pot. Pots were grouped in plastic trays; each is designed to hold six pots. Plants were grown in a rust-free greenhouse until inoculation. Plants were fertilized with a water-soluble fertilizers (23:19:17 NPK) at a rate of 2.5g per tray of six pots.

Table 1. Code (Pt) for the 16 North American differential hosts for *Puccinia triticina* in ordered sets of four and an additional set of five

		Infection type[b] produced on near isogenic *Lr* lines:			
	Host set 1:	1	2a	2c	3a
	Host set 2:	9	16	24	26
	Host set 3:	3ka	11	17	30
	Host set 4:	B	10	14a	18
Pt code[a]	Host set 5: #	14b	15	36	2b
B		Low	Low	Low	Low
C		Low	Low	Low	H
D		Low	Low	High	Low
F		Low	Low	High	High
G		Low	High	Low	Low
H		Low	High	Low	High
J		Low	High	High	Low
K		Low	High	High	High
L		High	Low	Low	Low
M		High	Low	Low	High
N		High	Low	High	Low
P		High	Low	High	High
Q		High	High	Low	Low
R		High	High	Low	High
S		High	High	High	Low
T		High	High	High	High

[a] Pt code consists of the designation for set 1 followed by that for set 2, etc. For example, race MGB: set 1 (M) - virulent to Lr1, 3a; set 2 (G) - virulent to Lr16; set 3 (B) - avirulent. [b] Low infection type (avirulent pathogen); High infection type (virulent pathogen), see Table 2. # ATUT-W4 Project, ARC.

Table 2. Description of infection types and symptoms

Infection type*		Symptoms
0	Low	No uredinia or other macroscopic sign of infection
0;	Low	Few faint flecks
;	Low	No uredinia, but hypersensitive necrotic or chlorotic flecks present
1	Low	Small uredinia often surrounded by a necrosis
2	Low	Small to medium uredinia often surrounded by chlorosis
Y	Low	Ordered distribution of variable-sized uredinia with largest at leaf tip
X	Low	Random distribution of variable-sized uredinia
3	High	Medium-sized uredinia without chlorosis or necrosis
4	High	Large uredinia without chlorosis or necrosis

* The infection types are often refined by modifying characters as follows:
=, uredinia at the lower size limit for the infection type,-, uredinia somewhat smaller than normal for the infection type,+, uredinia somewhat larger than normal for the infection type, ++, uredinia at the upper size limit for the infection type, C, more chlorosis than normal for the infection type, N, more necrosis than normal for the infection type.

Table 3. Low infection types* conferred by near-isogenic lines possessing 21 genes for resistance to *Puccinia triticina*

Lr gene	Isoline	Low IT	*Lr gene*	Isoline	Low IT
1	RL6003	0;	*15*	RL6052	;c,;1c
2a	RL6000	O;,;1c	*16*	RL6005	1-2cm
2b	RL6019	;1,;1+	*17*	RL6008	;12,23c
2c	RL6047	;1c	*18*	RL6009	2
3	RL6002	O;c	*21*	RL6043	O;,12-
3ka	RL6007	12	*24*	RL6064	O;,o;1-
9	RL6010	O;	*26*	RL6076	O;
10	RL6004	O;,2c	*30*	RL6049	O;,;1
11	RL6055	23	*36*	ER84018	O;,21c
14b	RL6006	x	*42*	WGRC11	O;,;1c

* low infection types at 18°C, may vary at other temperatures (Luig and Rajaram, 1972). 0=no flecks or uredinia, 0;=faint hypersensitive flecks, ;= hypersensitive flecks, 1-small uredinia with necrosis, 2= small uredinia with chlorosis, 3= moderate size uredinia, 4= large uredinia, +indicates slightly larger uredinia, -=indicates slightly smaller uredinia, C, more chlorosis than normal for the infection type, N, more necrosis than normal for the infection type, s =susceptible.

At 7 days after planting, when the first leaves were fully expanded, the seedlings were inoculated by spraying them with a suspension of urediniospores in a light mineral oil carrier. The oil was allowed to evaporate from the leaves for 30-60 min and the seedlings were placed overnight in a dew chamber at 17°C. They were then transferred to a greenhouse with mean temperature approximately 20 to 21°C and light supplemented with 100-200 mol m^{-2} s^{-1} photo flux fluorescent light. At 14 days after inoculation, plants were scored for IT according to Stakman *et al.* (1962) scale.

The same procedure was used to test cultivars against each of *P. triticina* isolates, but the inoculation with different isolates was done at different times provided that cross contamination is avoided.

Postulation of resistance genotypes:

Reactions of the 20 near- isogenic lines to the 12 isolates are shown in Table (5). The 14 commercial wheat cultivars and their pedigrees are shown in Table (4). The presence of specific genes for resistance in cultivars was detected as previously described by McVey (1993). Briefly, cultivars that are susceptible to isolates possessing avirulence to a given monogenic tester line must lack the gene for resistance present in that tester line. Cultivars that are resistant only to which the monogenic tester lines are also resistant, were postulated to have the gene for resistance of that line, if the LITs were similar.

Table 4. Egyptian wheat cultivars tested for the postulation of genes for resistance to leaf rust

Cultivar	Pedigree
Gemmeiza 1	Maya "s"/On//1160 147/3/Bluebird/Gui 1/4/Chat "s"
Gemmeiza 3	Bb/Siete Cerros//Yaqui 50/Kal*3//Sakha 8/4/Prv/WW/3/3/Bg"s"ON CGM-4024-1-GM-2GM-0GM
Gemmeiza 5	Vee"s"/SWM6525CGM4017-1GM-6GM-3GM-0GM
Gemmeiza 7	Vee"s"/SWM6525CGM4017
Gemmeiza 9	Vee"s"/SWM6525CGM4017
Sids 1	HD2172/Pavon "s"//1158.57/Maya74 "s"
Sids 2	HD2206/Hork "s"/3/Nappo63/Inia 66//Wren "s"
Sids 3	Sakha 69/Giza 155
Sids 4	Maya "s"/Mon "s"/CMH74.A592/3/Giza 157*2
Sids 5	Maya "s"/Mon "s"/CMH74.A592/3/Giza 157SD10001-7sd-4SD-2SD-0SD
Sids 6	Maya "s"/Mon "s"/CMH74.A592/3/Sakha 8*2 SD10002-4SD-3SD-1SD-0SD
Sids 7	Maya "s"/Mon "s"/CMH74.A592/3/Sakha 8*2 SD10002-8SD-1SD-1SD-0SD
Sids 8	Maya "s"/Mon "s"/CMH74.A592/3/Sakha 8*2 SD10002-14SD-3SD-1SD-0SD
Sids 9	Unknown

Table 5. Seedling infection types* of Thatcher near-isogenic lines inoculated with 12 virulent phenotypes of the leaf rust fungus *Puccinia triticina*

Thatcher line-*Lr* gene	SCPCS	MHPLQ	DBLLL	MBBLB	TCDML	MCNLQ	TKPMR	MCSLQ	TCSMQ	MCSLL	THDML	FCPQL
RL 6001-*Lr1*	S	S	;	S	S	S	S	S	S	S	S	S
RL 6001-*Lr2a*	S	;	1C	1C	S	;	S	;	S	;	S	;1C
RL 6001-*Lr2c*	S	;1	S	;1C	S	;	S	;1C	S	;1C	S	S
RL 6001-*Lr2b*	S	;	2C	;1C	S	;1C	S	;	S	;	S	1C
RL 6001-*Lr3*	;1	S	2+	S	S	S	S	S	S	S	S	S
RL 6001-*Lr3ka*	S	S	S	1+	23C	S	S	S	S	S	2+C	S
RL 6001-*Lr9*	;	;	;	;	;	;	;	;	;	;1	;1C	;
RL 6001-*Lr10*	12	S	S	S	S	S	S	S	S	S	S	S
RL 6001-*Lr11*	23	2+	12C	12=	2C	23	23C	S	S	S	2+C	12C
RL 6001-*Lr14b*	S	S	S	2+3	S	S	S	S	S	S	S	S
RL 6001-*Lr15*	S	S	;1C	;1C	1C	S	S	S	S	;1C	;	2+C
RL 6001-*Lr16*	2	2+3	2+	;1C	2	2	S	23+C	23C	12C	S	12C
RL 6001-*Lr17*	S	S	2+3	2=	S	S	S	S	S	S	S	S
RL 6001-*Lr18*	12	1	;1	;1C	1C	12C	2C	;1C	;1C	;1C	;1C	S
RL 6001-*Lr21*	2+	1	12-C	;	;1C	;1C	;1C	12C	;12C	;12C	;1C	12C
RL 6001-*Lr24*	;	12	;1C	;	;	;	S	;	;1	;	;	;
RL 6001-*Lr26*	S	S	;	;	S	S	S	S	S	S	S	S
RL 6001-*Lr30*	S	S	2+C	12=	;1C	;1C	S	12-C	;1C	;1C	12	S
RL 6001-*Lr36*	S	2	12C	;1+	1C	;1C	1C	12-C	2C	;1C	;1C	1C
RL 6001-*Lr42*	1	1	;1	;	;	;1C	S	;1C	;	;1C	;1C	;1C

* Infection types: 0=no flecks or uredinia, 0;=faint hypersensitive flecks, ;= hypersensitive flecks, 1-small uredinia with necrosis, 2= small uredinia with chlorosis, 3= moderate size uredinia, 4= large uredinia, +indicates slightly larger uredinia, -=indicates slightly smaller uredinia, C, more chlorosis than normal for the infection type N, more necrosis than normal for the infection type, s =susceptible.

Results

Infection types of the 14 commercial wheat cultivars to the 12 leaf rust isolates are shown in Table (6), with the postulated leaf rust resistance genes for each cultivar. Sids1 illustrates the process by which the presence of *Lr* genes was postulated. Sids1 is susceptible to isolates MCNLQ, TKPMR, MCSLQ, TCSMQ, and MCSLL (Table 6), which indicates that, Sids1 does not have *Lr2a, 2b, 2c, 9, 11, 14b, 15, 16, 18, 21, 24, 30, 36* and *42*, all of which condition a LITs to one or more of these isolates. The LITs of Sids1 to isolates SCPCS, MHLPQ, DBLLL, MBBLB, TCDML, THDML, and FCPQL are consistent with the LITs conferred by *Lr3, 3ka, 10, 17* and *26* against avirulent isolates of leaf rust. Also, isolates TCDML and FCPQL are avirulent to Sids1, but virulent to *Lr3, 10, 17* and *26*. This indicates that, Sids1 has at least one other identified leaf rust resistance gene.

By a similar process, the leaf rust resistance genotypes of the other wheat cultivars were postulated. The data indicated the probable presence of *Lr1, 2a, 2b, 2c, 3, 10, 17* and *26* in Gemmeiza1 (Table 6). Gemmeiza7 probably has *Lr3, 3ka, 10, 11, 16, 17* and *26*. Sids2 and Sids3 were postulated to have 4 and 5 *Lr* genes, respectively. Three Gemmeiza cvs. (Gemmeiza3, Gemmeiza5 and Gemmeiza9) and six Sids cvs. (Sids4, Sids5, Sids6, Sids7, Sids8 and Sids9) have none of the 20 *Lr* genes tested, but each of these cultivars appears to have an unidentified gene(s) for leaf rust resistance (Table 6).

Table 6. Seedling infection types* of the 14 wheat cultivars inoculated with 12 virulent phenotypes of the leaf rust fungus *Puccinia triticina*

Cv.	Postulated *Lr* gene(s)	SCPCS	MHPLQ	DBLLL	MBBLB	TCDML	MCNLQ	TKPMR	MCSLQ	TCSMQ	MCSLL	THDML	FCPQL
1	*Lr3ka,11,17,19,21,24,26,30*	;	;	;	;	2+C	;1	;1	;1	;1	;1C	S	12C
2	*Lr1?,2a,2b,2c,3ka,16,17,18, 26,30*	;	;	;	;	S	;	;	;	;	;	;	;
3	*Lr1?,3ka,17,26*	;	;	;	;	;	;	;	;1	;	;	;1C	S
4	*Lr2a,2b,3ka,11,16,17,26*	;1	2+	;	;	12C	1+C	S	S	S	S	12C	;1CN
5	*Lr3ka,17*	;1	12	;	;	;1C	1+C	12	12	;1	2C	;1C	12C
6	*Lr3ka,17*	;	12	;1	;1C	12CN	S	S	S	S	S	;1C	23C
7	*Lr1,3ka,17*	;1	1	;1	;1+C	12C	S	S	S	S	S	;1C	23C
8	*Lr1,2a,2b,16*	S	;1C	;	;1C	12C	S	S	S	S	S	;1C	;1C
9	*Lr1,2a,2b,11,16,17*	;1C	;	;1C	12C	12C	12C	S	2	2	;1C	12C	1C
10	*Lr1,2a,2b,16*	;1C	;	;1	;1C	2	12C	2	2	2	;1C	2C	1C
11	*Lr1,2a,2b,3ka,11,16,17,26*	12C	;1	;1	12C	;1	12C	2	2	12	;1C	23C	;1C
12	*Lr1,2a,3ka,11,16,17,26,30*	;1	;	;	1+	21C	12C	1	;1	2C	2C	12C	;1C
13	*Lr1,2a,3ka,11,16,17,26,30*	;	;	;	;1C	;	;1	1	;1	2C	;	2C	;

* Infection types: as described in footnote of Table (5).
1=Gemmeiza 1, 2=Gemmeiza 3,3= Gemmeiza 5, 4=Gemmeiza 7, 5=Gemmeiza 9, 6= Sids 1, 7= Sids 2, 8= Sids 3,9= Sids 4, 10=Sids 5, 11=Sids 6,12= Sids 7, 13=Sids 8, 14=Sids 9.

Discussion

The most frequent *Lr* genes postulated to be present in the commercial wheat cultivars were *Lr3* (36%), *Lr10* (36%), *Lr17* (45%), and *Lr26* (45%), followed by *Lr1* and *Lr3ka* (26%, each). This pool of resistance genes is not effective against most of the isolates used in Egypt (Imbaby *et al.*, 2006) and would not be effective in many areas of the world (Huerta-Espino, 1992). Gemmeiza1, Gemmeiza7, Sids1, Sids2, and Sids3, were resistant to most of the tested isolates, but their postulated *Lr* genes do not account for their resistance. Moreover, eight of the 14 cultivars appear to have none of the known *Lr* genes. Therefore, these cultivars appear to have unidentified gene(s) for leaf rust resistance. This is indicated by the presence of isolates in high: low infection types in monogenic lines to the cultivars comparisons. For example, the unidentified resistance gene(s) present in combination with *Lr3, 10, 17,* and *26* in Sids2 accounts for resistance to the isolates used because all of these postulated genes do not confer resistance to most, if no all, the 12 isolates used. Nine Lr genes, representing 45% of the 20 resistance genes used, could not be postulated in the cultivars used. Six of them (*Lr9, 18, 21, 24, 36* and *42*), were effective against all or most of the *P. triticina* isolates used, one (*Lr14b*) was susceptible and two (*Lr14, 30*) have intermediate reaction. The unidentified genes may be masked by the presence of another gene or genes that conditioned the same or lower ITs (McVey, 1993). Furthermore, Leonard and Czochar (1980) have pointed out that, a gene for resistance can be recognized only when compared with corresponding allele for susceptibility (Person, 1959). More diverse isolates of *P. Triticina* were needed to allow postulation of these genes. Generally, the maximum number of *Lr* genes that could be identified in a host cultivar is limited by the number of the isolates and available tester lines carrying known genes.

Acknowledgements

The ATUT-W4 project, ARC, Egypt acknowledges and thanks Dr. McVey, D.V., Cereal Diseases Lab., St. Paul, USDA, Minnesota Univ., Minneapolis, Minnesota, USA for cooperation and assistance to achieve and carry out this research.

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(Received 20/02/2007;
in revised form 10/03/2007)

استخدام بيانات طرز الاصابه فى تعريف جينات المقاومه لمرض صدأ الاوراق فى اصناف قمح الجيزه وسدس

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قسم بحوث امراض القمح- مركز البحوث الزراعيه- الجيزه.

ينتشر مرض صدا الاوراق المتسبب عن الفطر بكسينيا تريتيسينا تقريبا فى معظم مناطق زراعه القمح فى مصر والعالم. وتعتبر زراعه الاصناف المقاومه من اهم طرق مقاومه هذا المرض. ولذا تهدف هذه الدراسه الى توصيف وتعريف جينات المقاومه فى طور البادره والموجوده فى بعض اصناف من القمح الجيزه (5 اصناف) والقمح سدس (9 اصناف) باستخدام طرز الاصابه. هذا وقد تم التعريف لجينات المقامه بمقارنه طرز الاصابه (Infection types) على الاصناف المستخدمه مع طرز الاصابه على مجموعه من اصناف العائل (20 عامل وراثى) والحامله لجينات المقاومه بصوره فرديه (*Lr* genes) وذلك باستخدام 12 عزله فسيولوجيه لفطر صدا الاوراق وقد اسفرت النتائج المتحصل عليها على مايلى:-

1- تم تعريف 8 جينات مقاومه فى كل من الصنفين: جميزه1 (*Lr1,2a,2b,2c,3,10,17and26*) وجميزه7 (*Lr1,3,3ka,10,11,16,17,and 26*) .
2- تم تعريف 5 جينات مقاومه فى الصنف سدس 1 (*Lr3,3ka,10,17,* and *26*) والصنف سدس 3 (*Lr1,3ka,11,16, 17,*and *26*).
3- تمكنت الدراسه من تعريف 4 جينات فقط فى الصنف سدس 2 (*Lr3,10,17* and *26*).
4- لم تتمكن الدراسه من تعريف اى من الجينات المختبره فى الاصناف جميزه3 وجميزه 5 وجميزه 9 وسدس 4 وسدس 5 وسدس6 وسدس 7 وسدس 8 وسدس 9.
5- امكن تعريف 11 جين تمثل 55% من مجموع الجينات المختبره فى حين لم تتمكن الدراسه من تعريف 9 جينات تمثل 45% وذلك فى الاصناف التجاريه المستخدمه.
6- كانت الجينات *Lr17* and *26* هى الاكثر تكرارا فى الاصناف التجاريه (معدل تكرار 45%) تليهم الجينات *Lr3* and *10* (معدل تكرار 36%) ثم الجينات *Lr1* and *3ka* (معدل تكرار 27%).
7- تشير مقارنه طرز الاصابه بين الجينات المختبره مع الاصناف التجاريه المستخدمه الى وجود طراز قابل للاصابه: مقاوم (LIT:HIT) مما يرجح وجود عوامل وراثيه اخرى غير المختبره مرتبطه مع تلك الجينات التى تم تعريفها فى الاصناف التجاريه .
8- تشير طرز الاصابه الجينات التى تم تعريفها فى الاصناف التجاريه الى انها قابله للاصابه لمعظم السلالات المستخدمه مما يرجح ان صفه المقاومه التى تبديها الاصناف التجاريه للعزلات المستخدمه لاترجع الى تلك العوامل وانما قد ترجع الى عوامل اخرى والتى لم تتمكن الدراسه من تعريفها.