PHYSIOLOGICAL SPECIALIZATION OF *PUCCINIA TRITICINA* ERIKS AND EXPECTED RESISTANCE GENES IN SOME EGYPTIAN WHEAT CULTIVARS

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ABSTRACT

This study was described to demonstrate the physiologic races (pathotypes) of *Puccinia triticina* Eriks., the causal agent of brown (leaf) rust of wheat. Annual survey of 2008/2009 wheat growing season demonstrated the existence of forty three physiological races of wheat leaf rust fungus. The more prevalent race was PTTS (12.34%) followed by ·TTTS (9.87), TTTT (6.64%), PTTT (6.17%), KTTS (4.93%), PRTS (4.93%) races and the six races FTTS, KSTS, PSTS, STTP, TSTS and TTTP were recorded (2.46%) for each. The rest of races were reported by (1.23%) each. While the high efficacy leaf rust resistance genes (*Lr*'s) were *i.e.* $Lr2b$ (69.14%), $Lr36$ (64.20) and $Lr2a$ (49.39%). However, the more virulence were recorded with *Lr*'s *i.e.* 17; 10, 14b; 9, 11; 2c, 16, 21 , 30; and 3ka which were represented by (98.76%; 97.53%; 96.29%; 93.82% and 90.12% in respectively. The cultivar Giza 168 likely have *Lr*'s17, 10 and 14b. On the other hand, Gemmeiza 9 probably has $Lr146$. While, Gemmeiza 10 and Sakha 94 postulated each one of these has $Lr9$. The tested wheat cultivars were lack of the more efficacy genes in this investigate, probably at least against these pathotypes. These results would serve as a fruitful tool in the wheat breeding program for disease resistance.

INTRODUCTION

Twenty leaf rust resistant genes have described to protect wheat (*Triticum aestivum* L.) from leaf rust pathogen (*Puccinia triticina* Eriks.), expressed in the so called specific resistance (McIntosh *et al.,* 1995). To increase leaf rust resistance durability, breeders attempt to incorporate more than one of these genes in the cultivars to face the dynamic nature of the causal agent (Roelfs, 1988). These genes gives the ground to facilitate the development and improvement of resistance cultivars to manage leaf rust (McVey and Long, 1993). Meanwhile, Floor (1955) was the first to apply the gene expect technique in flax rust. On the other hand, it was developed to be used in rust diseases of small grains (Statler 1984, Modawi *et al.,* 1985 and McVey, 1992). Gene postulation used the principals of gene-for-gene, specifity to assign the most lines (Statler *et al.,* 1982; Statler, 1984; Long and Kolmer, 1989 and Kolmor, 1996).

The main objective of this investigation is :1) to survey and identify pathotypes or physiological races of wheat leaf rust in Egypt during 2009/2010 season, to expect leaf rust resistant genes at seedling stage in four commercial wheat cultivars in Egypt, through comparison with twenty near-isogenic lines (NIL'S) against eight one isolates of *Puccinia triticina*.

MATERIALS AND METHODS

Cultures of leaf rust pathogen that were collected during 2008/2009 growing season, were tested against a set of twenty near-isogenic lines of wheat and four Egyptian commercial wheat cultivars (Table 1). Infected wheat leaves with leaf rust pathogen (*Puccinia triticina*) were collected from different governorates of Egypt including rust trap and all commercial wheat cultivars. The collected samples (rusted leaves) were kept in glassine envelopes (8 × 15 cm). Rust specimens were left at room temperatures for 24 hours to remove the humidity in the specimes. Then the specimens were stored in the refrigerator at 5 °C.

Table (1). Near-Isogenic lines (NIL'S), used in the present investigation.

No.	Lr gene	Genome location	Origin of seed resource	Linkage
1	1	-	Malakof	
2	2a	5D	Webster	
3	2b	2DS	Garina	
4	2c	2DS	Brivit	
5	3a	6BL	Democrate	
6	3ka	6BL	Klien Aniversario	
7	9	6BL	Triticum umbellulatum	
8	10	1AS	Lee	
9	11	2A	Hussar	
10	14b	7BL	Bowie	-
11	15	2DS	Kenya1-12E	
12	16	2BS	Exchange	To Sr23
13	17	2AS	Klien Lucko	
14	18	5BL	T. timophevi	
15	21	1DL	T. tauchii	
16	24	3DL	A. elongatum	To Sr24
17	26	1BL	Imperial rye	To Sr31, YrA
18	30	4BL	Terenzio	
19	36	6BS	T. speltaides	
20	42	1D	-	

Lr21 = Lr40

The infected specimens were transferred on the susceptible cultivars i.e. Morocco, *Triticum spelta saharensis*, Little club and Giza 139 wheat Seedlings. Inoculation method was carried out as described by stokman *et al.*, 1962. seedling leaves were rubbed gently between moistened fingers with tap water, sprayed in the incubation chamber with water, then inoculated by shaking and brushing rusted materials over the plants and sprayed gently again with water in order to induce initial "dew" on the plants. Finally the inoculated plants were kept in moist chamber for 24 hours to allow the rust spores to germinate and cause infection. The inoculated plants were then moved to their respective benches in the greenhouse and kept for fifteen days. After developing the pustules, three single pustules were isolated separately from each specimen for reproduction on the highly susceptible wheat cultivar as previous mentioned to obtain enough urediospores yield for inoculation.

Seedling reaction were scored as susceptible or resistant depending on the infection type produced. The infection type *i.e.* 0,0;,1,2,3,4 and x were the same as described by Mains and Jackson, (1926) and Stakman *et al.*, (1962). Data were transmitted to Low (R) or High (S), since 0, 0;, 1, and 2 are considered resistance or low reaction, while 3, 4 and x are considered susceptible or high reaction.

The recent race nomenclature system.

In the present system, the sets of differential was used, the set adapted by Long and Kolmer (1989) inoculated sixteen differential host each with single gene of leaf rust resistance *i.e.* in four subset, 1) included (*Lr1, 2a, 2c, 3*), 2) (*9,16, 24, 26*), 3) (*3ka, 11, 17, 30*) and 4) (*10, 18, 21* and *2b*). The subset are indicated in (Table 2). The traditional inoculation technique, purification and race identification were above mentioned elsewhere. The *Lr's* different hosts are placed in sets of four and a letter is assigned to each of the 16 possible combination i.e. 2^4 of the interaction. The letters B through T minus vowels are used to the identified races. The results reactions were matched to those in the table aiming to reach the modern nomenclature system according the similarity with the differential hosts.

Table (2). Code for the 16 North American differential hosts for *P. triticina* (*Pt*) in ordered sets of four*.

Pt code		Infection types produced on near isogenic lines *Lr's*			
	Host set 1	1	2a	2c	3
	Host set 2	9	16	24	26
	Host set 3	3ka	11	17	30
	Host set 4	10	18	21	2b
B		L	L	L	L
C		L	L	L	H
D		L	L	H	L
F		L	L	H	H
G		L	H	L	L
H		L	H	L	H
J		L	H	H	L
K		L	H	H	H
L		H	L	L	L
M		H	L	L	H
N		H	L	H	L
P		H	L	H	H
Q		H	H	L	L
R		H	H	L	H
S		H	H	H	L
T		H	H	H	H

*Long and Kolmer, 1989.

Gene postulation in certain Egyptian wheat cultivars against leaf rust at seedling stage under greenhouse conditions.

Four commercial wheat genotypes and 20 leaf rust near-isogenic lines were tested for rust resistance using eighty one isolates of leaf rust (*P. triticina*) from collected rusted specimens of 2008/2009. inoculated and adopted according to (Stakman *et al.*, 1962) season then. Rust reaction was recorded 22 days after sowing. Genes were postulated according to the method of Statler (1984) in which, the absence of L : H or H:L reaction between the tested host (B) and the known gene host (A), indicated the presented of such gene(s) in the tested host exhibited the symbol (-0). But the presence L (in host B): H (in host A) indicated the presence of such gene in host B and it may have another ones = (0). On the other hands, the presence of pathotypes having H:L and L:H in the comparison indicates that either of hosts did not have the same gene = (+). Also, when host B proved to have H (high infection type) versus L (low infection type) in host A, this

behaviour would indicated that the absence of such gene in host B= (-).

RESULTS

A virulence/virulence formulae and their frequency%:

Data presented in Table (3) demonstrated the virulence formula of forty three tested pathotypes results from eighty one isolates and their frequency. These data indicated the presence of forty three pathotypes (physiological races) *i.e.* PTTS was repeated ten times (12.34%) followed by TTTS was frequent eight (9.87), TTTT was replicated of seven (8.64%), PTTT was frequent five (6.17%), KTTS and PRTS were repeated four each (4.93%), FTTS, KSTS, PSTS, STTP and TTTP were repeated two each (2.46%), the rest of the pathotypes were repeated one each (1.235). However, the North American nomenclature method indicated that some physiological races (pathotypes) had numerous numbers of isolates *i.e.* PTTS, TTTS, TTTT,PTTT, KTTS and PRTS contained 10, 8, 7, 5; 4 and 4 respectively. It could be noticed that isolate no 43 *i.e.* TTTT is considered to be the most virulent one, since it exhibited complete virulence to either of the tested leaf rust resistance gene followed by each of isolates no. 26 (PTTT), 38 (TSTT), 40 (TTKT) and 42 (TTTS), which were avirulent to only one *Lr* gene. On the other hand, isolates no. 13 (PCKJ), 1 (DSTQ) and 6 (GSRS) were avirulent to 7 , 6 and 6 *Lr's*, respectively.

Table (3). Avirulence/virulence formulae of leaf rust pathotypes (*Pt*) identified in Egypt in 2009/2010 growing season.

No.	No. of pathotypes	Pathotypes	Avir./vir. formulae	Vir. Frequency%
1	1	DSTQ	1,2a,3,26,21,2b/	1.23
2	1	DTTS	1,2a,3,2b/	1.23
3	1	FSKS	1,2a,26,3ka,2b/	1.23
4	(2)	FTTS	1,2a,2b/	2.46
5	1	FTTT	1,2a/	1.23
6	1	GSRS	1,2c,3,26,17,2b/	1.23
7	1	KRPQ	1,24,11,21,2b/	1.23
8	(2)	KSTS	1,26,2b/	2.46
9	(4)	KTTS	1,2b/	4.93
10	1	MPSS	2a,2c,16,30,2b/	1.23
11	1	MPTS	2a,2c,16,2b/	1.23
12	1	NRTS	2a,3,24,2b/	1.23
13	1	PCKJ	2a,9,16,24,3ka,10,2b/	1.23

Cont. Table (3)

14	1	PPTS	2a,16,2b/	1.23
15	1	PQTS	2a,24,26,2b/	1.23
16	1	PRKJ	2a,24,3ka,10,2b/	1.23
17	1	PRPQ	2a,24,11,21,2b/	1.23
18	(4)	PRTS	2a,24,2b/	4.93
19	1	PRTT	2a,24/	1.23
20	1	PSKS	2a,26,3ka,2b/	1.23
21	1	PSTL	2a,26,18,21,2b/	1.23
22	(2)	PSTS	2a,26,2b/	2.46
23	1	PTKN	2a,3ka,18,2b/	1.23
24	1	PTST	2a,30/	1.23
25	(10)	PTTS	2a,2b/	12.34
26	(5)	PTTT	2a/	6.17
27	1	RPTS	2c,16,2b/	1.23
28	1	RQTS	2c,24,26,2b/	1.23
29	1	SKSP	3,9,30,18/	1.23
30	1	SSTT	3,26/	1.23
31	1	STKT	3,3ka/	1.23
32	(2)	STTP	3,18/	2.46
33	1	TKSP	9,30,18/	1.23
34	1	TQTS	24,26,2b/	1.23
35	1	TSPQ	26,11,21,2b/	1.23
36	1	TSTN	26;18,2b/	1.23
37	(2)	TSTS	26,2b/	2.46
38	1	TSTT	26/	1.23
39	1	TTJS	3ka,30,2b/	1.23
40	1	TTKT	3ka/	1.23
41	(2)	TTTP	18/	2.46
42	(8)	TTTS	2b/	9.87
43	(7)	TTTT	-/	8.64
Total	43			

Leaf rust resistance genes on the leaf side of the slash were written but those on the right were neglected as having susceptible infection type.

Leaf rust resistance gene efficacy %:

Data presented in Table (4) indicated the efficacy of leaf rust resistance genes in controlling pathotypes populations in most governorates of Egypt during 2008/2009 growing season. These data demonstated that three genes exhibited the more efficacy in resistance of leaf rust pathotypes, which were reocorded with *Lr's i.e.* 2b, 36 and 2a. Meanwhile, the lowest efficacy genes were reported with *Lr's i.e.* 17, 10, 14b, 9, 11, 2c, 16, 21 and 30. However, the rest of the leaf rust near-isogenic lines lied in between them.

Data in Table (5) showed that forty three pathotypes (physiologic races) of *P. triticina* were identified from eighty one isolates by four sets of leaf rust resistance genes. However, the matching of twenty leaf rust near-isogenic lines and four Egyptian commercial wheat cultivars against forty three pathotypes of leaf rust *P. triticina*. Data in Table (6) showed the presence of high infection type : low infection type in the (near-isogenic lines : commercial cultivar) demonstrated the inclusion of Lr within genetic makeup of commercial cultivar, this was assigned by the symbol (0). Other group of comparison indicated the existence of low infection type : high infection type and high infection type : low infection type between near isogenic lines and commercial cultivar against the tested types, the cultivar may have gene(s) not involved in the other. It was assigned by the symbol (+). On the other hand, the symbol (-) and (-0) were lack in this study.

Table (4). Virulence frequency (%) of wheat leaf rust *P. triticina* isolates and *Lr's* genes efficacy (%) during 2009/2010 growing season.

No.	Lr's	No. of virulent isolates	No. of a avirulent isolates	total number of isolates	Vir. Frequency%	Vir. frequency
1	1	67	14	81	82.71	17.29
2	2a	41	40	81	50.61	49.39
3	2b	25	56	81	30.86	69.14
4	2c	76	05	81	93.82	06.18
5	3a	72	09	81	88.88	11.12
6	3ka	73	08	81	90.12	09.88
7	9	78	03	81	96.29	03.71
8	10	79	02	81	97.53	02.47
9	11	78	03	81	96.29	03.71
10	14b	79	02	81	97.53	02.47
11	15	71	10	81	87.65	12.35
12	16	76	05	81	93.82	06.18
13	17	80	01	81	98.76	01.24
14	18	72	09	81	88.88	11.12
15	21	76	05	81	93.82	06.18
16	24	68	13	81	83.95	16.05
17	26	63	18	81	77.77	22.23
18	30	76	05	81	93.82	06.18
19	36	29	52	81	35.80	64.20
20	42	58	23	81	71.60	28.40

*high efficacy gene.
"high virulence gene

Data in Table (7) indicated the probable resistance genes may be existence within the genetic back ground of certain wheat cultivars as derived from that table *i.e. Lr14* probably found in

Gemmeiza 9 and Giza 168. Also, *Lr9* likely presented in Gemmeiza 10 and Sakha 94. While, *Lr 17* and *Lr10* postulated excised in Giza 168.

Table (5). Infection types produced by 43 pathotypes of *P. triticina* (*Pt*) isolated from most governorates of Egypt against leaf rust resistance genes and differential sets samples under greenhouse conditions at seedling stage in 2009/2010 growing season.

No	Pathotypes	Infection type produced on near-isogenic lines *Lr's* *															
		1	2a	2c	3	9	16	24	26	3ka	11	17	30	10	18	21	2b
1	DSTQ	L	L		L				L							L	L
2	DTTS	L	L		L												L
3	FSKS	L	L					L	L								L
4	(2)FTTS	L	L														L
5	FTTT	L	L														
6	GSRS	L		L	L				L			L					L
7	KRPQ	L						L			L					L	L
8	(2)KSTS	L							L								L
9	(4)KTTS	L															L
10	MPSS		L	L		L							L				L
11	MPTS		L	L		L											L
12	NRTS		L		L			L									L
13	PCKJ		L		L	L	L			L				L			L
14	PPTS		L			L											L
15	PQTS		L					L	L								L
16	PRKJ		L					L		L				L			L
17	PRPQ		L					L			L					L	L
18	(4)PRTS		L					L									L
19	PRTT		L					L									
20	PSKS		L						L	L							L
21	PSTL		L						L					L		L	L
22	(2)PSTS		L						L								L
23	PTKN		L							L				L			L
24	PTST		L										L				
25	(10)PTTS		L														L
26	(5)PTTT		L														
27	RPTS			L		L											L
28	RQTS			L				L	L								L
29	SKSP				L	L							L		L		
30	SSTT				L				L								
31	STKT				L					L							
32	(2)STTP				L										L		
33	TKSP					L							L		L		
34	TQTS							L	L								L
35	TSPQ								L		L					L	L
36	TSTN								L						L		L
37	(2)TSTS								L								L
38	TSTT								L								
39	TTJS									L			L				L
40	TTKT									L							
41	TTTP														L		
42	TTTS																L
43	TTTT																

Leaf rust resistance genes.

Table (6). The LIT:HIT (low infection type : high infection type) of twenty near-isogenic lines of leaf rust (Lr's) and four Egypt wheat cultivars against eighty one pathotypes of *Puccinia triticina* (Pt) at seedling stage in 2009/2010 growing season.

Cultivars	1	2a	2c	3	9	16	24	26	3ka	11	17	30	10	18	21	2b	14b	15	36	42
Gem.9	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	0	+	+	+
Gem.10	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
G.168	+	+	+	+	+	+	+	+	+	+	0	+	0	+	+	+	0	+	+	+
Sakha 94	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

*(+) = indicated that the absence of such gene in commercial wheat cultivar.

**(0) = indicated that the presence of such gene in commercial wheat cultivar and it may have another ones's.

Table (7). Probable resistance genes for leaf rust in some Egyptian wheat cultivars at seedling stage in 2009/2010 growing season.

No.	Cultivar	Probably Lr's gene(s)
1	Gemmeiza 9	14b,
2	Gemmeiza 10	9,
3	Giza 168	17,10,14b
4	Sakha 94	9,

Data in Table (8) showed frequency of leaf rust resistance genes within commercial cultivars at seedling stage *i.e.* Lr9 and Lr14b exhibited a frequency reached to 50%. While, Lr10 and Lr17 have a frequency approached to 25%. On the other hand, sixteen Lr's out of twenty were not represent in that cultivars, due to the presence of symbol (+).

Data presented in Table (9) showed that comparison of four Egypt commercial wheat cultivars within each other for searching the common gene(s). the commercial cultivars may have another genes different than those tested genes and would assigned by the symbol (+).

Table (8). The frequency of identified *Lr's* genes within Egyptian wheat cultivars at seedling stage in 2009/2010.

No.	*Lr's*	No. of cultivars carrying *Lr's* gene	Percent (%)
1	1	0	0
2	2a	0	0
3	2b	0	0
4	2c	0	0
5	3	0	0
6	3ka	0	0
7	9	2	50
8	10	1	25
9	11	0	0
10	14b	2	50
11	15	0	0
12	16	0	0
13	17	1	25
14	18	0	0
15	21	0	0
16	24	0	0
17	26	0	0
18	30	0	0
19	36	0	0
20	42	0	0

*Lr's : Leaf rust resistance genes.

Table (9). Incidence of cultivar in (LIT:HIT) comparison between cultivars inoculated with eighty one isolates of *P. triticina* at seedling stage in 2009/2010 growing season.

Cultivars	Gemmeiza 9	Gemmeiza 10	Giza 168	Sakha 94
Gemmeiza 9		+	+	+
Gemmeiza 10	*+		+	+
Giza 168	+	+		+
Sakha 94	+	+	+	

LIT=Low infection type.
HIT=High infection type.
*(+)=Indicated either of cultivars did not have the same gene.

DISCUSSION

Wheat leaf rust caused by *Puccinia triticina* Eriks (*Puccinia recondite* Rob. ExDesm. f. sp. *tritici* Eriks. & Henn.) was the first factor in failure of such cultivars, which was mainly due to the dynamic nature in population of the causal organism which produces new virulences having the ability to breakdown their resistance.

Environment is a complex term that includes many factors which must be beyond a minimum threshold for disease development to occur. A change in one environment factor may alter the effect of other environmental factors on disease development. For example, precipitation washes inoculums from the air, reduce leight intensity, lower temperature and increases the probability of dew formation for several succeeding days. Dew usually occurs more frequently than rain in the great plains, and hr of free moisture proved to be the most accurate measure of moisture available for leaf rust development, as shown by the increased precision in predicting leaf rust severities with hr of free moisture rather than days of precipitation. However, inclusion of precipitation variable increased the accuracy of prediction over that with no moisture variable according to Eversmyer and Burleigh (1970). Also dew usually occurs when daily temperature are at or near the minimum, minimum temperature may provide the most accurate measure of temperature affecting disease development. in the early part of the growing season (tillering to bott.), minimum temperatures are often below or fluctuating across the temperature threshold for leaf rust development. But maximum temperatures are at or near the development optimum and therefore do not limit disease development Therefore, the critical period for rust control occurs early in the growing season (Eversmeyer and Burleigh 1970).

The virulence formulae were designated by a four letter code (Long and Kolmer 1989). The present study showed that eighty one leaf rust isolates gives forty three virulence formulae (Physiologic races). Races PTTS was the more dominance one and comprised (12.43%) of the races, followed by TTTS, TTTT, PTTT, KTTS and PRTS were frequent repeated by (9.87%), (8.16%), (6.17%), (4.93%) and (4.93%), respectively. Also, the races FTTS, KSTS, PSTS, STTP, TSTS and TTTP each one of these represented by (2.46%). The rest of physiologic races were frequent repeated by (1.23%) each. Similar results were recorded by Najeeb *et al.*, (2005) Youssef (2006) and Youssef *et al.*,(2010)

On other hand, gene efficacy (%) of the tested *Lr*'s, the results showed the presence of high efficacy which were reported with *i.e. Lr*'s 2b, 36 and 2a which percent about 69.14%, 64.20% and 49.39%, respectively. While, high virulence which were recorded with *i.e. Lr*'s 17; 10, 14b, 9, 11, 2c, 16, 21 and 30 were represented by 98.76%; 97.53%; 96.29%; 93.82% and 93.82%; respect. The rest of *Lr*'s genes were lied in between them. The expected genes of leaf rust near-isogenic lines

within the coomercial wheat cultivars *i.e. Lr14b* probably found in Gemmeiza 9 and Giza 168, Lr9 was postulated to be excistend in Gemmeiza 10 and Sakha 94. While, Giza 168 likely included *Lr17* and Lr10 added to *Lr14b*. On the other hand, the rest of the leaf rust near-isogenic lines probably couldn't be detected in the commercial wheat cultivars tested in this work, similar results were recorded by (Mc Vey, 1989, Naseebetal., 2005 and Youssef *et al.*,2010)

The second likely is the presence of such genes in tested cultivars but the presence of suppressors or modifiers prevented their expression (Knott, 1989). This postulation may be the reason behind the genetic behaviour of certain Egyptian wheat cultivars toward the tested races. These results are not considered and indication to lack of the tested wheat cultivars from high efficacy resistance genes, may be they have genes other than those tested (category +).

Acknowledgements

REFERENCES

Eversmyer, M.G. and j.R. Burleigh (1970). A method of predicting epidemic development of wheat leaf rust. *Phytopath.* 60(5). 805-811.

Floor, H.H. (1955). Host parasite interaction in flax rust, it's genetics and after implications. *Phytopath.* 45:680-685.

Knott, D.R. (1989). The Wheat Rusts-breeding for Resistance. Pb. Springier Verlag London, Paris and Tokyo. PP.201.

Kolmer, J.A. (1996). Genetic of resistance to wheat leaf rust. Ann. Rev. *Phytopath.* 34:435-455.

Long, D.L. and J.A. Kolmer (1989). A north America system of nomenclature for *Puccinia recondica* ERIKS *Phytopath.* 79: 525-529.

Main, E.B. and H.S. Jackson (1926). Physiologic specialization In the leaf rust of wheat *Puccinia triticina* Eriks. *Phytopath.* 16:89-120.

McVey, D.V. (1989). Verification of Infection type data for identification of genes for resistance to leaf rust in some hard red spring wheats, Crop Sci. , 29:304-307.

McVey, D.V. (1992). Genes for rust resistance in international wheat nurseries. XII through XVII. Crop Sci., 32:891-895.

McVey, D.V. and D.L. Long (1993). Genes for leaf rust resistance in hard red winter wheat cultivars and parental lines. Crop Sci. 33:1373-1387.

McIntosh, R.A.; S.C.R. Welling and R.E. Park (1995). Wheat Rusts, An Atlas of Resistance Genes. Kluwer Academic Publishers, London PP.200.

Modawi, R.S.; L.E. Browder and E.G. Heyne (1985). Use of Infection-type data to identify genes for low reaction to *Puccina recondita* in several winter wheat cultivars Crop. Sci., 25:9-13.

Najeeb, M.A.; O.A. Boulat; M.M. Mousa and S.S. Negam (2005). Physiological specialization in *Puccinia triticina* and postulated genes of resistance in certain Egyptian wheat cultivars. Ann. Of Agric. Sci., Moshtohor, 43 (1):265-278.

Roelfs, A.P. (1988). Resistance to leaf and stem rusts of wheat. Breeding strategies of resistance to rusts of wheat N.W. Semmounds, and S. Rajaram eds CIMMYT Mexico DF. : CIMMYT.

Stakman, E.C.; D.M. Stewart and W.Q. Loegering (1962). Identification of physiologic races of *Puccinia graminis tritici*. A. R. S., U.S. Dept. Serv. Bull. E617.53pp.

Statler, G.D. (1984). Probably genes for leaf rust resistance in several hard red spring wheat. Crop Sci. 24:883-886.

Statler, G.D.; JD. Miller and S. Leben (1982). Wheat leaf rust in North Dakota during 1979-1981. Plant Dis. 66:1174-1176.

Youssef, I.A.M. (2006). Physiological specialization in *Puccinia triticina*, and genes conditioning resistance to wheat leaf rust disease in Egypt. J. Agric. Sci. Mansoura Univ., 31(4):2057-2071.

Youssef, I.A.M.; Doaa, R. El-Naggar; Gamalat, Hermas and S.S. Negam (2010). Identification of physiologic races of *Puccinia triticina* and postulated genes of resistance in certain Egyptian commercial wheat cultivars. J. of Plant Protection and Pathology, vol. 1(2):75-85.

الملخص العربي

التخصص الفسيولوجى لفطر بكسينيا تريتيسينا والجينات المتوقعة لمقاومتة فى بعض أصناف القمح المصرية

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اجريت هـذه الدراسـة مـن اجـل تعريـف السـلالات الفسـيولوجية (المسـببات المرضية) لفطـر Eriks. *Puccinia triticina* العامـل المسـبب للـصدأ البنـى أو صـدأ الأوراق فى القمح. حيـث أوضـح الحـصر السـنوى فى موسـم نمـو القمـح ٢٠٠٩/٢٠٠٨ وجود ثلاثـة وأربعـون سـلالة فـسيولوجية (مسبب مرضـى) مـن فطـر صـدأ الأوراق ز حيـث ان السـلالة PTTS كانـت اكثـر سـيادة (٣٤٫١٢%) يليهـا TTTS (٩٫٨٧%) ، TTTT (٨٫٦٤%) ، PTTT (٦٫١٧%) ، و KTTS (٤٫٩٣%) و PRTS (٤٫٩٣%) وسـتة سـلالات كـلا منهـا يمثـل (٢٫٤٦%) وهـى TTTP و TSTS و STTP و PSTS و KSTS و FTTS .

بـاقى السـلالات سـجلت (١٠٫٢٣%) . بينمـا جينـات المقاومـة لـصدأ الاوراق الأكثـر كفـاءة كـان الجـين *Lr2a* قـد سـجلت (٤٩٫٣٩%) والسـلالة *Lr36* (٦٤٫٢٠%) والسـلالة *Lr2b* (٦٩٫١٤%) . مـع ذلـك الجينـات الاكثـر مرضـية تـم تـسجيلها مـع *Lr17* (٩٨٫٧٦%) و *Lr14b* ، *Lr10* (٩٧٫٥٣%) ، *Lr9* ، *Lr11* (٩٦٫٢٩%) ، *Lr2c* ، *Lr16* ، *Lr21* ، *Lr30* (٩٣٫٨٢%) *Lr 3Ka* ، (٩٠٫١٢%).

وقد اظهرت النتائج احتمـال وجود الجينات *Lr's 14b ,10 ,17* فى الصنف جيزة ١٦٨ ، امـا الـصنف جميـزة ٩ يفتـرض أنـه يحتـوى علـى الجـين *Lr14b* بينمـا جميـزة ١٠ وسـخا ٩٤ يفترض ان كـلا منهمـا يحتـوى علـى الجـين *Lr9* وفـى هـذه الدراسـة تـم ملاحظـة نقـص فـى الجينـات الأكثـر كفـاءة وذلـك فـى الأصـناف التجاريـة المختبـرة علـى الأقـل ضـد تلـك المسـببات المرضية . وتخدم هذه النتائج فى برامج تربية القمح من أجل مقاومة المرض .