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Research Article

Detection of *Yr27* virulence in *Puccinia striiformis* f.sp. *tritici* populations on wheat in Iraq

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Abstract

This study was conducted to detect any possible changes in virulence spectrum of *Puccinia striiformis* f. sp. *tritici* population in Iraq. Biological trap nurseries of yellow rust were planted at different locations representing most of wheat growing areas in Iraq. The plants were exposed to the natural population of the pathogen in the fields. Disease was scored as severity and infection type on each genotype at different stages of wheat development. Yellow rust development was calculated on the local susceptible wheat cultivars Araz, and Saberbeg. yellow rust samples collected from the commercial wheat fields at different locations were sent to the Global Rust Center for race analyses. Virulence analysis of *Puccinia striiformis* f. sp. *tritici* population, revealed detection of virulence against the known resistance genes *Yr2*, *Yr6*, *Yr7*, *Yr9*, *Yr18*, *YrA*, *Yr21*, *Yr25*, *Yr27*, *Yr28*, *Yr29* and *Yr31* at adult plant stage in Sulaimania, while virulence against the known resistance genes *Yr1, Yr2*, *Yr4, Yr5*, *Yr7*, *Yr9*, *Yr SD, YrSu*, *YrND, Yr32, YrA*, *Yr21*, *Yr27*, *Yr28*, *Yr31,* were detected in the natural populations of *P. striiformis* f. sp. *tritici* at adult plant stage in Nineveh. Out of 34 rust samples sent to GRRC for race analysis, 12 samples were only recovered while 22 samples were failed to recover. Breakdown of resistant genes *Yr27* and *Yr25* was further confirmed at seedling stage. Infection rate value of yellow rust during the epidemic on Saber Beg (r- value= 0.337) was higher than Araz (0.187).

Keywords: Yellow Rust, *Triticum aestivum*, Resistance genes, Pathogen variability, Iraq

1. Introduction

The trans-boundary wheat rust diseases could have potential devastating effects on wheat production in Iraq. Stripe or yellow rust of wheat, caused by *Puccinia striiformis* f.sp. *tritici* (*Pst*), is currently considered to be the most important biotic constraint to sustainable wheat production in Iraq (Al-Maaroof *et al.*, 2003b). Yield losses of 10-60% in Iraq are common, if susceptible cultivars are grown (Al-Maaroof *et al.,* 2012). The pathogen has the ability to evolve rapidly into new races and to migrate long distances by air-borne dispersal (Chen *et al.*, 2002). Yellow rust infection was formerly restricted in some wheat fields in the northern parts and It has never been seen in the middle and South of Iraq. The disease appeared periodically both on the rain fed areas and irrigated wheat fields (Al-Beldawi *et al.*, 1993). The disease was observed for the first time in some wheat fields in the middle zone in 1988. Later on it has rapidly spread on the susceptible cultivars in this area and then moved to wheat growing area's in the South (Al-Maaroof *et a*l., 2003a). Many outbreaks of yellow rust were observed in all wheat growing areas particularly in the irrigated fields in the last decades. The most severe epidemic form was recorded in 1998, which has caused significant decline in the national grain production.

This was due to development of new virulence against *Yr9* gene, which was predominant in most of wheat cultivars as a single major gene (Al-Maaroof *et al.*, 2004). Breakdown of *Yr9* resistance gene resulted in widespread epidemics in the Middle East and the Indian Subcontinent, which caused considerable crop damage in the 1990s (Mclntosh *et al.*, 1995: Singh, 2004). The origin of this series of pandemics is considered to be mutation to virulence for *Yr9* arising in the red sea region as early as 1987, areas which have been stimulated with the recent climatic condition changes (Louwers *et al.,* 1992). Severe epidemic of yellow rust were observed in most of wheat growing area's during 2009/10 growing season particularly in the northern and central zones. The epidemic was very severe and detected in early stages of wheat developments in the irrigated fields of the Iraqi–Turkish– S*Yr*ian Triangle in Duhok (Al-Maaroof *et al.*, 2012). Kumarse (2011) indicated that stripe rust outbreak was similar to that one's which was already reported in 2010 in S*Yr*ia, Iran and Turkey (Kumarse, 2011). The recent accelerating climatic changes have already affected the complex biological interaction, climate influence the frequency and severity of disease epidemics and some studies indicates that climatic changes modify disease and pest risks and increase uncertainty in risk predictions associated with climatic changes particularly in the wheat: *P. striiformis* biological system (Chakraborty *et al.,* 2010; Newton *et al.*, 2010). The current study was conducted to detect any possible changes in *P. striiformis* population, which is responsible for yellow rust outbreak in Iraq.

2. Materials and methods

2.1.Biological wheat yellow rust trap nurseries: Three sets of the fourth International yellow rust biological trap nurseries *"4th IYRTN-10"* including 75 genotypes were planted in three locations representing most of wheat growing areas in Iraq. Two sites were selected in the North at Bakrajo Experimental Station, Sulymania (Latitude: N 35.32.351, Altitude: E 045.21.978), Rashidia Experimental Station, Nineveh (Latitude: N 36.19.677, Altitude: E 043.10.130), and one in the South at Shatra Experimental Station in Dhiqar. Each genotype was planted in two rows of one-meter length. The field was surrounded with a mixture of wheat cultivars with diverse response to accelerate the disease development. Virulence analyses of *Puccinia striiformis* population were conducted by exposing the yellow rust biological trap nurseries to the natural population of the pathogen in the field. Diseases scoring on the biological trap nurseries were conducted using disease severity and infection types on each genotype. Infection types were recorded for each genotype at different stages of wheat development according to Lewllen scale, whereas 0=no visible infection; R= Resistant necrotic area with or without small pustules; MR=Moderately resistant, Small pustule surrounded by necrotic area; M= Intermediate, pustules of variable size, some necrosis or chlorosis; MS = moderately susceptible, medium sized pustules, no necrosis but some chlorosis possible, S= Susceptible, large pustules, no necrosis or chlorosis (Lewllen *et al.*, 1967), while disease severity was recorded on modified Cobb's scale which depends on comparing the infected wheat leaves with a theoretical diagram showing the frequency of uredia for particular percentage disease severity (Peterson *et al.,* 1949).

2.2. Yellow rust development on wheat cultivars: The epidemic development of yellow rust disease was estimated on two local susceptible wheat cultivars Araz, and SaberBeg. Seed of each cultivar was grown in 5*5 meter square plots at Bakrajo Experimental Station, Sulaimania. Rust data were collected periodically with seven days interval by calculating the rate of rust development on each cultivars by using the below given formula (Vanderplank, 1963).

$r = 2.3/(t2 - t1) \log [X2 (1 - X1) / X1(1 - X2)]$ unit per day

Where X1, is the disease severity at time t1 and X2 at t2.

The AUDPC was calculated by the trapezoidal integration of the disease severity in time, considering the whole period evaluated as follows: -

$$AUDPC = \sum_{i-1}^{n-1} \left(\frac{Xi+(Xi+1)}{2}\right)((ti+1) - ti)$$

Where X= the disease severity (percentage of plant diseased); n = the number of evaluations, and ((ti+1)- ti) the time interval (days) between two consecutive evaluations (Cook *et. al* 2006).

Environmental data consisting of maximum and minimum air temperature, relative humidity, rainfall, wind speed and solar radiation were collected from the National Climatic Center in the region.

2.3. Race analysis and pathotype identification of P. striiformis: Rust samples were usually collected from the commercial wheat fields, biological wheat trap nurseries plots and yield trial plots. About 3-5 of infected wheat leaves were collected from each plant, the leaves were folded and put into paper envelope to avoid curling. The samples were shade dried at room temperature for about 24 h then cut into 10cm length samples. All the important information including, location, date of collection, cultivar, disease severity and incidence were recorded on each sample. The samples were kept in a paper envelope and stored in the refrigerator. The best of 34 yellow rust samples, which were collected from different locations of Iraq, were sent to the Global Rust Center, Aarhus University, Denmark for race analyses (GRRC). The samples were entered in recovery processing on the susceptible cultivars to achieve sufficient amount of spore to allow for race analysis, which were made according to the procedure described by Hovmoller and Justeen, (2007).

3. Results and discussions

Results of virulence analyses of *Puccinia striiformis* population revealed that virulence against the known resistant genes *Yr2*, *Yr6*, *Yr7*, *Yr9*, *Yr18*, *YrA*, *Yr21*, *Yr25*, *Yr27*, *Yr28*, *Yr29* and *Yr31* were detected in the natural populations of *P. striiformis* f. sp. *tritici* at adult plant stage in the field in Sulaimania, while the known resistance genes *Yr1*, *Yr3+*, *Yr4*, *Yr5*, *Yr8*, *Yr10*, *Yr15*, *Yr17*, *YrND*, *YrCV*, *Yr32* and *YrSp* exhibited low infection type reactions at adult plant stage in the field (Table 1) . Therefore, the avirulence/Virulence pattern of *P. striiformis* in Sulaimani is *Yr1*, *Yr3*, *Yr4*, *Yr5*, *Yr8*, *Yr10*, *Yr15*, *Yr17*, *YrND*, *YrCV*, *Yr32, Yr Sp/ Yr2*, *Yr6*, *Yr7*, *Yr9*, *Yr18*, *YrA*, *Yr21*, *Yr25*, *Yr27*, *Yr28*, *Yr29*, *Yr31*. In Nineveh virulence against the known resistance genes *Yr1, Yr2*, *Yr4, Yr5*, *Yr7*, *Yr9*, *Yr SD, YrSu*, *YrND, Yr32, YrA*, *Yr21*, *Yr27*, *Yr28*, *Yr31,* were detected in the natural populations of *P. striiformis* f. sp.

Table 1. Disease Severity and infection types of Yellow rust differentials and trap nurseries at different stages and development of the host plant under natural epidemic form of *Puccinia striiformis* during 2009/10 season at Bakrajo Experimental Station, Sulaimania, Iraq

Entry No.	Cultivar/ Genotype	*Yr* genes	Disease severity and Infection type				AUDPC
			April 7	April15	April 21	May 6	
1	Morocco	-	20S	80S	100S	100S	250^{a}
2	*Yr* 1/6* Avocet S	*Yr*1	0R	0R	0R	0R	0.0^{v}
3	Chinese 166	(W; *Yr*1)	0R	3R	10R	20R	23^{pqr}
4	Kalyansona	*Yr*2	5R	10MR	15MRS	40MRS	50^{lm}
5	Heines V11	(W, *Yr*2+)	0R	0R	0R	15R	7.5^{uv}
6	Vilmorin 23	(W;*Yr*3a,4a)	0R	0R	5R	10R	10^{tuv}
7	Hybrid 46	(W;*Yr*4)	0R	0R	0R	0R	0.0^{v}
8	*Yr* 5/6* Avocet S	*Yr*5	0R	0R	0R	0R	0.0^{v}
9	Triticum spelta	*Yr*5	0R	0R	0R	0R	0.0^{v}
11	*Yr* 6/6* Avocet S	*Yr*6	0R	30MS	40MSS	90MSS	115^{gh}
12	Heine's Kolben	(S; *Yr*6+1)	0R	5MR	10MR	30MS	25^{opqr}
13	Heine's Peko	(S; *Yr*6+)	0R	5R	5R	10R	15^{stu}
14	Fielder	*Yr*6,*Yr*20	25S	50S	80S	90S	200^{b}
15	*Yr* 7/6* Avocet S	*Yr*7	20S	50S	80S	90S	195^{b}
16	Lee (S; *Yr*7)	*Yr*7	5MS	15MS	30MS	50MS	75^{jk}
17	Reichersberg 42	(W; *Yr*7+)	5R	5R	10R	15R	27.5^{nopqr}
18	Thatcher	*Yr*7	10MS	20MS	30MS	65MS	92.5^{i}
19	*Yr* 8/6* Avocet S	*Yr*8	5R	5R	5R	10RMR	20^{qrst}
21	Compair	(S;*Yr*8)	5R	5R	5R	15R	22.5^{qrst}
22	*Yr* 9/6* Avocet S	*Yr*9	15S	25MS	40MS	60MS	110^{h}
23	Fed.4/Kavkaz(*Yr*9)	*Yr*9	15S	50S	50S	80S	155^{c}
24	Clement	(W;*Yr*9+*Yr*2)	0R	5R	10R	15R	22.5^{qrst}
25	Federation		20S	25S	60S	90S	150^{cd}
26	*Yr* 10/6* Avocet S	*Yr*10	0R	0R	0R	0R	0.0^{v}
27	Moro (W; *Yr*10)	(W; *Yr*10)	5R	10R	10R	10R	30^{nop}
28	*Yr* 15/6* Avocet S	*Yr*15	0R	0R	0R	0R	0.0^{v}
29	*Yr* 17/6* Avocet S	*Yr*17	0R	0R	0R	0R	0.0^{v}
31	Strubes Dickopff	(W; *Yr*SD)	0R	10R	10R	15R	27.5^{nopqr}
32	Suwon 92*Omar	(W; *Yr*Su)	5R	5R	5R	10R	25^{opqr}

Entry No.	Cultivar/ Genotype	*Yr* genes	Disease severity and Infection type				AUDPC
			April 7	April15	April 21	May 6	
33	Nord Desprez	(W; *Yr*ND)	0R	0R	TR	TR	0.0^{v}
34	*Yr* CV/6* Avocet S	*Yr* CV	0R	0R	0R	0R	0.0^{v}
35	Carstens V	(W; *Yr*32)	0R	0R	0R	0R	0.0^{v}
36	*Yr* SP/6* Avocet S	*Yr*SP	0R	0R	10R	15R	17.5^{rstu}
37	Spaldings Prolific	(W; *Yr*SP)	TR	TR	TR	0R	0.0^{v}
38	Avocet 'R'	*Yr*A	10S	40S	90S	100S	190^{b}
39	Inia 66	*Yr*A	5MS	10MS	20MS	30MS	50^{lm}
41	Avocet 'S'	-	15S	30S	40S	85S	127.5^{efg}
42	Tres/6* AVS	-	0R	0R	0R	0R	0.0^{v}
43	*Yr* 18/3* Avocet S	*Yr*18	0R	10MR	10MRMS	20MRMS	30^{nov}
44	Jupateco 'R' (S)	*Yr*18+	5MR	5MR	10MR	15MR	27.5^{nopqr}
45	Jupateco 'S'	-	5MS	10MS	15MS	20MS	40^{mn}
46	Anza	*Yr*A, *Yr*18	5MR	10MR	20MR	30MR	50^{lm}
47	Cook (S)	APR	0R	10R	10MR	25MR	32.5^{nop}
48	Lemhi	*Yr*21	25S	70S	70S	75S	202.5^{b}
49	TP 981	-	10MS	15MS	30MS	50MS	80^{ij}
51	TP 1295	*Yr*25	5MS	15MS	30MS	40MS	70^{jk}
52	*Yr* 27/6* Avocet S	*Yr*27	10MS	20MS	40MS	80S	110^{h}
53	Ciano 79	*Yr*27	20MS	25MS	30MS	30MS	90^{i}
54	ATTILA CM85836-50Y	*Yr*27+?	5MS	10MS	15MS	40MS	50^{lm}
55	OPATA 85	*Yr*27+*Yr*18	15MSS	20MSS	60MSS	85MSS	138.5^{de}
56	Avocet-*Yr*A*3/3/	*Yr*28	15MSS	30MSS	50MSS	85MSS	138.5^{de}
57	Lal Bahadur/Pavon1BL	*Yr*29	5MR	10MR	10MRMS	20MRMS	35^{nop}
58	Avocet-*Yr*A*3/PASTOR	*Yr*31	5MR	20MR	25MRMS	65MRMS	82.5^{ij}
59	PASTOR	*Yr*31+APR	5R	10R	10MR	15MR	32.5^{nop}
61	Polimer 2.1.1(Triticale)		5R	10R	10MR	25MR	37.5^{mno}
62	Cham 1	DW	0R	0R	5R	10R	10^{tuv}
63	Cham 4		10MS	10MS	20MS	45MS	62.5^{kl}
64	Cham 6		10MS	20MS	30MS	60MS	90^{i}
65	Cham 8		15MS	30MS	70S	100S	125^{i}
66	Gobustan		0R	0R	5R	8R	9.0^{uv}
67	Sardari		20S	30S	40S	80S	130^{ef}
68	Alamout		10S	40S	50S	75S	137.5^{def}
69	Bohouth 6		0R	5R	10R	15R	22.5^{qrst}
70	Gereck 79		5R	10R	10R	20R	35^{nop}
71	Kinaci 97		5R	5R	10R	20R	30^{nop}
72	Gun 91		--	--	--	--	--
73	Dustlik		0R	5R	10R	15R	22.5^{qrst}
74	TATARA	*Yr*3	0R	10MR	10MR	25MR	32.5^{nop}

*Note: * Different at p means followed by the same letters in the same column are not significantly =0.05*

tritici at adult plant stage in the field, while the known resistance genes *Yr6*, *Yr8*, *Yr10*, *Yr15*, *Yr17*, *YrCV* and *YrSp* produced low infection type reactions at adult plant stage in the field (Table 2). Therefore, the avirulence/ Virulence pattern of *P. striiformis* in Nineveh is *Yr6*, *Yr8*, *Yr10*, *Yr15*, *Yr17*, *YrCV*, *YrSp / Yr1,Yr2*, *Yr4, Yr5*, *Yr7*, *Yr9*, *Yr SD, YrSu*, *YrND, Yr32, YrA*, *Yr21*, *Yr27*, *Yr28*, *Yr31*. Yellow rust disease did not develop at Dhiqar due to the unfavorable environmental conditions during this season**.** Virulence analysis reveals difference in *P. striiformis* virulence spectrum between Sulaimania and Nineveh, particularly on *Yr1, Yr4, Yr5*, *YrSD, YrSu*. This variation may be due to the difference in the predominant races of the pathogen population in each location (Kumarse, 2011). Virulence against *Yr5* is not known in the region. It is quite clear that there is a shift in virulence pattern of *P. striiformis* population in Iraq. Particularly the resistance gene *Yr27* that was previously highly effective against the population of *P. striiformis*. This may be due to the appearance of new races of *P. striiformis* in Iraq. The high infection of yellow rust on Sham 8 variety with *Yr27* (100S) is a very good indication of virulence on *Yr27*, which is also confirmed on the biological trap nursery response at adult plant stage in both locations (Table 1,2),

Table 2. Disease Severity and infection types of Yellow rust differentials and trap nurseries at different stages and development of the host plant under natural epidemic form of *Puccinia striiformis* during 2009/2010 season at Rashidia Experimental Station, Nineveh, Iraq

Entry No.	Cultivar/Genotype	*Yr* genes	Disease severity and Infection type					AUDPC
			Mar20	Mar27	April3	April17	May2	
1	Morocco	-	25S	50S	80S	90S	90S	290^{fg}
2	*Yr* 1/6* Avocet S	*Yr*1	20R	30R	40MR	50MS	50MS	170^{p}
3	Chinese 166	(W; *Yr*1)	50MS	70S	80S	95S	95S	342.5^{b}
4	Kalyansona	*Yr*2	10MR	20MR	10MR	10MR	10MR	55$^{a'b'c'}$
5	Heines V11	(W, *Yr*2+)	70MS	80MS	80MS	90S	90S	365^{a}
6	Vilmorin 23	(W;*Yr*3a,4a)	50MS	60MS	70MS	80MS	80MS	300^{bc}
7	Hybrid 46	(W;*Yr*4)	60MS	70MS	75MS	90S	90S	340^{b}
8	*Yr* 5/6* Avocet S	*Yr*5	20MR	30MR	40MR	40MR	40MR	150^{rs}
9	Triticum spelta	*Yr*5	50S	70S	75S	90S	90S	330^{bc}
11	*Yr* 6/6* Avocet S	*Yr*6	10MR	10MR	10R	10R	10R	45$^{b'c'd'}$
12	Heine's Kolben	(S; *Yr*6+1)	60MS	70MS	80MS	85MS	90MS	340^{b}
13	Heine's Peko	(S; *Yr*6+)	60MS	70MS	75MS	80MS	90MS	330^{bc}
14	Fielder	*Yr*6,*Yr*20	5R	10MR	15MR	15MR	15MR	52.5$^{a'b'c'}$
15	*Yr* 7/6* Avocet S	*Yr*7	5R	10R	10R	15R	15R	45.5$^{b'c'd'}$
16	Lee (S; *Yr*7)	*Yr*7	20R	30R	30MR	30MR	40MR	130^{tuv}
17	Reichersberg 42	(W; *Yr*7+)	30MS	50MS	70MS	75MS	80MS	270^{gh}
18	Thatcher	*Yr*7	40MS	60MS	70MS	80MS	80MS	290^{ef}
19	*Yr* 8/6* Avocet S	*Yr*8	20MR	20MR	20MR	10MR	10MR	75^{yz}
21	Compair	(S;*Yr*8)	25R	30R	40MR	40MR	40MR	155^{nop}
22	*Yr* 9/6* Avocet S	*Yr*9	20MR	30MR	35MR	20MR	20MR	115^{stuv}
23	Fed.4/Kavkaz(*Yr*9)	*Yr*9	30MS	30MS	40MR	30MR	30MR	155^{nop}
24	Clement	(W;*Yr*9+Y2+)	50MS	60MS	70S	80S	80S	300^{e}
25	Federation		20R	30MR	30MR	15MR	10MR	100^{vwx}
26	*Yr* 10/6* Avocet S	*Yr*10	10MR	10MR	20MR	10MR	10MR	110^{tuvw}
27	Moro (W; *Yr*10)	(W; *Yr*10)	40MS	50MS	60S	75S	80MS	265^{ghi}
28	*Yr* 15/6* Avocet S	*Yr*15	10MR	20MR	30MR	40MR	40MR	120^{stu}
29	*Yr* 17/6* Avocet S	*Yr*17	20R	30MR	40MR	40MR	30MR	145^{pq}
31	Strubes Dickopff	(W; *Yr*SD)	50MS	70MS	80MS	90S	95S	337.5^{b}

Entry No.	Cultivar/Genotype	*Yr* genes	Disease severity and Infection type					AUDPC
			Mar20	Mar27	April3	April17	May2	
32	Suwon 92*Omar	(W; *Yr*Su)	50MS	65MS	75S	80S	90S	325^{bc}
33	Nord Desprez	(W; *Yr*ND)	20MS	30MS	50MS	60MS	80MS	200^{l}
34	*Yr* CV/6* Avocet S	*Yr* CV	T	5R	5R	5R	5R	18.5^{f}
35	Carstens V	(W; *Yr*32)	10MR	20MS	30MS	20MS	20MS	90^{xyz}
36	*Yr* SP/6* Avocet S	*Yr*SP	15MS	40MS	25MR	30MR	30MR	125^{rst}
37	Spaldings Prolific	(W; *Yr*SP)	30MR	50MR	60MS	70MS	75MS	247.5^{ij}
38	Avocet 'R'	*Yr*A	20MS	40S	60S	75S	80S	235^{jk}
39	Inia 66	*Yr*A	10R	20MR	20MR	25MR	25MR	137.5^{pqrs}
41	Avocet 'S'	-	10MR	20MS	30MS	40MS	50MS	125^{rst}
42	Tres/6* AVS	-	10MR	20MR	20MS	30MS	30MS	95^{wx}
43	*Yr* 18/3* Avocet S	*Yr*18	5MR	10MR	10MR	10MR	10MR	40$^{c'd'e'}$
44	Jupateco 'R' (S)	*Yr*18+	10MR	10MR	15MR	20MR	20MR	65$^{za'}$
45	Jupateco 'S'	-	10MR	10MR	15MR	20MR	20MR	65$^{za'}$
46	Anza	*Yr*A, *Yr*18	10MR	10MR	20MR	20MR	30MR	75^{yz}
47	Cook (S)	APR	10MR	20MR	20MR	30MR	30MR	95^{wx}
48	Lemhi	*Yr*21	60MS	70MS	80MS	90S	90S	335^{b}
49	TP 981	-	20MS	40MS	40MS	50MS	50MS	175^{m}
51	TP 1295	*Yr25*	10MR	20MR	30MR	30MR	30MR	105^{uvwx}
52	*Yr* 27/6* Avocet S	*Yr*27	30MS	50MS	70S	80S	90S	275^{fg}
53	Ciano 79	*Yr*27	30MS	40MS	50MS	60MS	61MS	210.5^{l}
54	ATTILA CM85836-50Y	*Yr*27+?	10R	20R	20MR	30MR	20MR	90^{xyz}
55	OPATA 85	*Yr*27+*Yr*18	20R	30MR	40MR	40MR	30MR	145^{pq}
56	Avocet-*Yr*A*3/3/	*Yr*28	5R	10R	10R	5R	5R	32.5$^{d'e'f'}$
57	Lal Bahadur/Pavon 1BL	*Yr*29	15MR	20R	30MR	30MR	30MR	110^{tuvw}
58	Avocet*Yr*A*3/PASTOR	*Yr*31	10MR	20MR	35MR	50MR	40MR	155^{nop}
59	PASTOR	*Yr*31+APR	5R	20MR	30MR	50MR	50MR	130^{qrs}
61	Polimer2.1.1 (Triticale)		5R	10R	15MR	20MR	20MR	60$^{za'b'}$
62	Cham 1	DW	5R	5R	5R	5R	5R	22.5$^{e'f'}$
63	Cham 4		5R	10R	5R	5R	5R	27.5$^{d'e'f'}$
64	Cham 6		40MR	50MR	75MR	75MS	80MS	280^{fg}
65	Cham 8		10MS	15MS	20MS	30MS	50MS	100^{vwx}
66	Gobustan		10R	15MR	20MR	30MR	35MR	92.5^{wxyz}
67	Sardari		15MR	20MS	30MS	40MS	50MS	130^{qrs}
68	Alamout		50S	60S	70S	80S	95S	307.5^{cde}
69	Bohouth 6		10R	20R	20R	30MR	30MR	95^{wx}
70	Gereck 79		20MS	30MS	40MS	50MS	50MS	165^{mno}
71	Kinaci 97		50S	60S	70S	80S	90S	305de
72	Gun 91		30S	40S	50S	60S	75S	217.5^{kl}
73	Dustlik		30S	50S	60S	80S	80S	255^{hi}
74	TATARA	*Yr*3	20MR	30MR	30MR	40MR	40MR	140^{pqr}

*Note: * Means followd by the same letters in the same column are not significantly different at p=0.05*

Table 3. GRRC race analysis of *P. striiformis* f.sp. *tritici* samples collected from different locations in Iraq during 2010

Locations	No. of Samples		Pathotype code*	No. of isolate	Aggressiveness
	Failed	recoverd			
Duhok	2	2	-,2,-,-,-,6,7,8,9,-,-,-,-,25,27,-,-,AvS,-	2	+
Sulaimania	6	6	-,2,-,-,-,6,7,8,9,-,-,-,-,25,27,-,-,AvS,-	2	+
Nineveh	3	1	-,2,-,-,-,6,7,8,9,-,-,-,-,25,27,-,-,AvS,-	1	+
Erbil	5	1	-,2,-,-,-,6,7,8,9,-,-,-,-,25,27,-,-,AvS,-	1	+
Garmian	6	2	-,2,-,-,-,6,7,8,9,-,-,-,-,25,27,-,-,AvS,-	1	+
ltaTo	22	12		7	+

*Note: *The figures correspond to virulence matching YRresistance genes.*

(Al-Maaroof *et al.*, 2011, 2012). The highest AUDP value was detected on Morroco (250) which was significantly surpassed all other genotypes in Sulaimania followed by Lemhi and Fielder while the lowest values ranged between 0-23 on the resistant genotypes (Table 1). AUDP values reached to 365 on Heines V11, which was significantly higher than all other genotypes in Nineveh followed by Chinese 166, Hybrid 46 and Heine's Kolben, while the low values ranged between 18.5- 45 on the resistant genotypes (Table2)

Out of 34 yellow rust samples sent to GRRC for race analysis, 12 samples were only recovered while 22 samples were failed to be recovered (Table 3). The low recovery rate of the pathogen is ascribed to a relative long time between sampling and receipt of the samples at GRRC. Race analysis of yellow rust samples at seedling stage indicated the presence of virulence for *Yr2*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr25*, and *Yr27*. This race is similar to the dominant race identified in S*Yr*ia during the 2010 yellow rust outbreak particularly in northeast regions close to S*Yr*ia – Iraq – Turkish triangle borders (Al-Chaabi and Abu Fadel, 2012). ICARDA reports also confirmed distribution of *Yr27* virulence in the Middle East particularly in S*Yr*ia, Turkey, Iraq and also Azerbaijan and Uzbekistan (Solh *et al.*, 2012). Yellow rust resistance genes *Yr1*, *Yr3*, *Yr4*, *Yr5*, and *Yr15* were affective against the yellow rust race identified in Iraq. Based on race and previous studies of aggressiveness (Miles *et al.*, 2009), a high frequency of aggressive strains of yellow rust are spreading further to Africa and West Asia including Iraq and Afghanistan (Hovemoler *et al.*, 2011), The combination of virulence for *Yr27* and aggressiveness may initiate devastating yellow rust epidemics including areas where *Yr27*- resistant cultivars are commonly grown. Loss of effectiveness of *Yr27* in cvs PBW343, Inquilab 91 and Chamran in India, Pakistan and Iran, respectively were first reported during 2002-2004. While the unfavorable environmental conditions possibly restricted rapid increase of the *Yr27*-virulent pathotype until 2009 but conducive conditions resulted in severe epidemics in a number of CWANA countries like Pakistan, Morocco, Algeria, Tunisia, Uzbekistan, Turkey, Iran, Yemen, Azerbaijan, Georgia and Afghanistan. Environmental conditions favoring rust development continued until 2010, with a mild winter and adequate rainfall in several CWANA countries resulting in early stripe rust outbreaks. The consequence was the 2010 stripe rust pandemic throughout the major wheat growing areas in CWANA and Caucasus countries, causing very high yield losses due to the absence of resistance sources in the major wheat cultivars.(Wellings, 2011, Solh *et al.*, 2012). Yellow rust development on the commercial susceptible wheat cultivars was high and resulted from severe epidemic in wheat fields. The primary infection was detected on the highly susceptible cultivar SaberBeg plots at Stem Extension stage at Bakrajo experimental station. Disease severity and infection type was 5S on March 31,2010 then reached to 100S after 16 day on April 15th. Infection rate value (r-value) on SaberBeg (0.337) was higher than Araz (0.187) (Fig 1). The high infection rate and early disease incidence in the season also reflect the aggressiveness of the new *Yr27* virulent race. This also may be due to the favorable environmental conditions including high precipitation in the beginning of

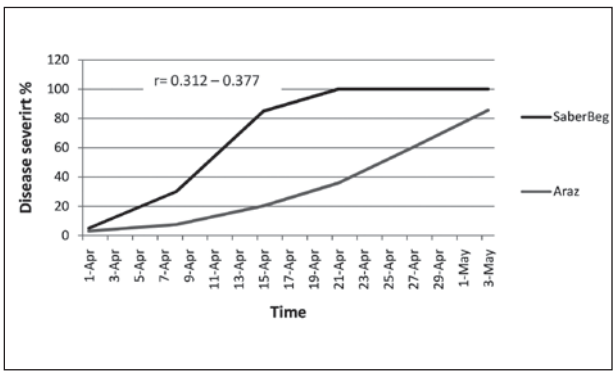

Fig 1. Graphical representation of **y**ellow rust development on the local wheat cultivars at Bakrajo experimental station, Sulaimania during 2010.

the disease onset, which resulted in high relative humidity, an important factor in the establishment and progress of yellow rust on wheat. Furthermore, the dominant mean temperature was around 20C during the epidemic period in this area that is also more suitable for disease development (Rapilley, 1979) (Fig 2). *Yr27* virulence r-value on the susceptible cultivar (0.337) was higher than *Yr9* virulence infection rate by race 230E150 on Maxipak in 1998 (Al-Maaroof *et al.*, 2003c, Al- The aggressive race showed more spore production, shorter latent period when evaluated at high (12 to 28°C) temperature regimes as compared to the one at low temperature regimes (10 to 18°C). Lesion length, lesion width and lesion area became larger when the isolates evaluated under the same regimes of hot temperature comparing with cold regimes (Maaroof *et al.*, 2012) .

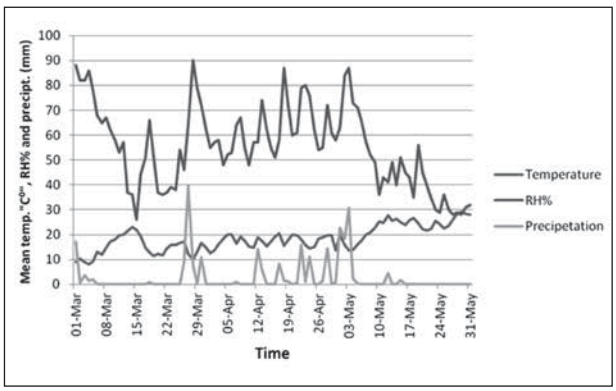

Fig 2. Average daily temperature's, relative humidity (RH) and precipitation (mm) during yellow rust development on wheat during March to May, 2010 in Sulaimania.

Aggressive isolates showed more severity on the differential lines at high temperature regime than at cold temperature regime. Climate change now appears to play a major role in *P. striiformis* population dynamics in CWANA. Direct multiple affect of climatic changes on epidemiology of rust pathogens is expected, including the survival of primary inoculum, the rate of disease development and distribution of rust populations. Emergence of yellow rust in non-traditional areas, changes in the frequency of new race evolution, early infection shift in predicted pathway of rust migration, and finally wide spread epidemics of yellow rust in warmer area's as a potential indicator of adaptation to high temperature are considered as possible consequences of climatic changes (Nazari *et al.*, 2011)

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