



Yellow rust development on different wheat genotypes

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Abstract

Yellow (Stripe) rust caused by *Puccinia striiformis* Westend, is currently considered as one of the most destructive foliar diseases of wheat in many wheat-growing areas in Iraq. The recent results of rust surveillance activities efforts showed that the epidemics of the disease have occurred annually in many wheat fields and caused significant decline in the national grain production. Yellow rust development were studied in seven different wheat genotypes with diverse levels of resistance and susceptibility under natural epidemics of the disease during two successive seasons in 2009 to 2011 at Bakrajo Experimental Station, Sulaimani. Results revealed that the highest disease development curve was recorded for SaberBeg, which resulted from 100% disease severity within 24 days, while the lowest value was recorded for Al-124 and resulted from 7.2% disease severity. Significant differences between the Areas under Disease Progress Curve (AUDPCs) values were also detected among the tested genotypes. AUDPCs values ranged from 13.8 to 311. The mean infection rate value in SaberBeg (0.312) was significantly surpassed all other wheat genotypes. Variations in infection rate and disease development curves among the genotypes were mainly attributed to the characteristics of *P. striiformis* pustules and uredospore's on the tested genotype tissues. Significant differences in pustule size, number of pustules per unit area, inoculum load and dimension of uredospore were detected between the genotypes which were also reflected on the epidemic buildup of the disease on the tested genotypes.

Introduction

Stripe (yellow) rust disease incited by *Puccinia striiformis* f. sp. *tritici* Westend is currently considered as one of the most destructive foliar diseases of wheat in most wheat growing areas in Iraq. Yield losses may reach to more than 60% on the commercial susceptible wheat cultivars [1]. *P. striiformis* has the lowest temperature requirement of the three wheat rust pathogens, with minimum, optimum and maximum temperatures of 0, 11 and 23 °C, respectively [2]. EL-Naimi & Mamluk (1995) [3] stated that the early appearance of infection coupled with long wet spring mostly lead to high spread of yellow rust epidemic, which may cause high yield losses. Yield reduction reached to 108,000 tons in 1988 in Syria. The disease was most prevalent and damaging to 1.5 million tones of wheat in 1994 in Iran [4]. AL-Maarof (1997) [5] referred that yellow rust caused 29-50% reduction in grain yield in Iraq in 1994, while yield reduction was more than 33% on maxipak under natural epidemic of the disease in 1998 [6]. Serious epidemic of yellow rust were detected in most of wheat growing area in Iraq in 2010 particularly in the northern part, which

resulted in high reduction in the national grain yield [7]. Yellow rust was quite destructive in China; yield losses reached to 3.2 million tons in 1964, 1.8 million tons in 1992, and 1.3 million tons in 2002 [8].

Yellow rust distribution was formerly restricted only in Kurdistan region and other north parts of Iraq then moved to middle and southern of Iraq [9] and [10]. The disease was observed for the first time in some wheat fields in the central parts of Iraq in 1988. Later on it was appeared in different wheat fields in this region then spread to south. Many sever epidemic recorded in wheat field at the beginning of 1990. The high infection mean reached to 80% in the susceptible cultivars particularly when the environmental conditions was very suitable to spread the disease, this may be due to appearance of new races of the pathogen [5]. Al-Maarof et al. (2015) [7] referred to detection of sever epidemics of stripe rust in most of wheat fields in Iraq during 1997/98. The infection rate was ranged from 0.089 to 0.358 per unit per day. Luo and Zeng (1995) [11] conclude that yellow rust development depends on the temperature and rainfall during wheat development in winter and the lesion expansion, infection efficiency and sporulation capacity are the most important resistance components.

Gopalan & Manners (1984) [12] concluded that spores produced on old or senescent leaves would be much less effective epidemic development than spores produced on young green leaves. Kampmeijr et al. (1997) [13] showed that the same inoculums quantity randomly distributed among 400 or 16 foci produced the same rate of epidemic development, but the rate was much slower when an epidemic originated from only one focus. Eugene (2009) [14] indicated that isolates representing new strain are more aggressive than isolates representing old strain for initiating epidemics from overwintering infections and this explain why stripe rust has been more severe since 2000. Epidemiology provides the context for understanding the role and significance of pathogen and plant genes related to pathogen reproduction and also provides models for evaluating landscapes of plant phenotypes [15].

The objective of the study was to determine yellow rust development on different wheat genotypes with diverse levels of resistance and susceptibility.

Material and Methods

Field experiments

All the experiments were conducted at Bakrajo Research Station Directorate, 10km south of Sulaimania (Lat.N35.32.375, Long.E045.73.825, Elev.703) during the growing seasons 2009/11, which is rain feed area.

Monitoring of the pathogen arrival

To determine the first arrival time of the primary inoculums of *P. striiformis*, its sedimentation and development on wheat cultivars in the field, six microscopic slides covered with 4-5 ml layer of water agar were distributed vertically and horizontally on particular stand at three different locations within the plots of susceptible cultivars. The slides were located at the same level of plant high. Each slide was left for 24 hours in the field then taken to the laboratory; uredospore numbers of *P. striiformis* were calculated in one-centimeter square area of each slid. Uredospore counting was repeated each 10 days from March to April, 2011.

Yellow rust development on different wheat genotypes

Origin of the tested genotypes and experimental design

Seven different wheat genotypes with diverse response to yellow rust were used in this experiment. Four of the genotypes (Al-8/70, Al-84, Al-8/172 and Al-124) representing the advance promising resistance wheat lines of our breeding program for improving rust resistance of wheat in Iraq. While Araz, Tamuz 2 and SaberBeg are registered and released as wheat cultivars. The tested genotypes explored diverse range of wheat reaction to yellow rust disease started from resistant reaction (R) in genotype AL-124 to highly susceptible reaction (HS) in SaberBeg, while Araz and AL-8/70 were susceptible (S), Tamuz 2 was moderately susceptible to susceptible (MS-S), AL-84 was moderately resistant to moderately susceptible (MR-MS), and Al-8/172 was moderately resistant (MR) [7]. A total of 120g seeds from each genotype were grown in 25m² plots on 28thDecember 2010 using RCBD with three replications with one-meter interval

between two plots and two meter between blocks. The field was entirely cultivated with the boarder susceptible cultivars particularly SaberBeg as a trap and spreader of *P. striiformis* inoculums in the field.

Disease scoring and data analyses.

Monitoring of yellow rust primary infection and development of the disease were started by the first of March to the end of May with 7 days interval period during the natural epidemic of the disease in 2011. Infection types and disease severities were assessed at different wheat growth stages using Lewellen score [16], where 0= no visible infection; R= Resistant, necrotic area with or without small pustules; MR= moderately resistant, small pustules surrounded by necrotic areas, 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. While disease severities were estimated by using the modified Cobb scales [17], which depends on comparing the infected wheat leaves with the theoretical diagram showing the frequency of uredia for particular percentage disease severity. Data were randomly collected from 30 plants/plot including lines no. 2, 5 and 7 in each plot. The infection rate of yellow rust (r) on each genotype was calculated at any two times of disease development by using Vander Plank equation, [18] as indicated in the following formula: -

$$r = 2.3 / (t_2 - t_1) \log_{10} [X_2(1 - X_1) / X_1(1 - X_2)] \text{ Per unit per day}$$

Where r=rate of disease development per unit per day

t₁ and t₂=the time of first and second reading of disease severity

X₁ and X₂ =disease severity at first and second reading for each time.

The coefficient of infection (C.I) of yellow rust on each cultivar was calculated by multiplying the severity times with a constant values given to the host response; where immune (I) =0.0, R=0.2, MR =0.4, M= 0.6, MS =0.8 and S=1.0. This makes it easy to rank or statistically compare between genotypes or nurseries [19].

$$C. I = DS * IT$$

Where DS=Disease severity· IT=Infection type

Infected leaves/ genotype were collected to measure pustule size, number of spore/pustule, spore size and number of pustules /cm².

Laboratory experiments.

Characteristics of *P. striiformis* pustules and urediospores on different genotypes

Infected leaves from different wheat genotypes with diverse response to yellow rust disease (Al-124, Al-8/172, Al-84, Al-8/70, Tamoz 2, Araz and SaberBeg) were collected separately, from flag and second leaf at adult plant stage and transferred carefully to the laboratory for further studies and calculations as follows.

Pustule size.

Pustule dimensions (length and width) of *P. striiformis* f. sp. *tritici* from different wheat genotypes were measured with the micrometer and simple ruler.

Number of spores in each pustule.

The number of spores in each pustule was calculated for each genotype by using the haemocytometer.

Spore dimension.

Dimensions(length and width) of each uredospore of *P. striiformis* from different genotypes were calculated by using the micrometer. Five pustules were used for each genotype, and 10 uredospore's from each pustule were used to calculate the means.

Number of pustules per unit area.

Number of pustules per unit area was calculated by counting number of pustules in one centimeter.

Results and Discussion

Monitoring of yellow rust arrival

Figure 1 indicates that *P. striiformis f. sp. tritici* inoculum were arrived to Bakrajo wheat fields at mid of March 2011. About 1.25 uredospore's were caught in one centimeter square during 24 hour on vertical trap stands on 20 March, 2011 and 1.75 uredospore/cm² were caught by horizontal trap stands, but no germinated spores were detected on wheat leaves and the primary infection not appear in March due to unfavorable environmental conditions to uredospore germination and infection process. The first infection on the susceptible cultivars was appeared on 24th April. Number of uredospores, which were detected on vertical and horizontal trap, was gradually increased with the development of the disease. The highest number of uredospores reached to 36.5spores/cm² on the vertical traps and 67.5spores/cm² on the horizontal stands when the disease severity on SaberBeg reached to 100% at milky stage on 23th May, 2011. Number of uredospores decreased with increasing of the disease and plant maturity due to decrease in the RH% and the amount of precipitation coupled with increasing in temperature degree by the end of April (Fig. 3)

Decreasing the RH to 50% and increasing the temperature over 25°C lead to decrease in uredospore number, which produced on the susceptible tissues [20]. Pustules continue producing uredospore to one week and the highest production happened in the fourth day at a moderate temperature [21] and [22]. The highest number of uredospore may not be coupled with increasing in the infection rate because some of the uredospore's may be unviable or physiologically incompatible with the tested host plants. They also might affected by the change of daily temperature and relative humidity since germination of uredospore's and invasion of host tissues needs 100% humidity for three to four hours [21] and [23]. Iraqi Kurdistan region is lying in the epidemiological zone (Zone7), which is the source of spreading of the new races of the fungi in Asia. Many epidemics of yellow rust disease were detected in most of wheat field in Iraq during the last decades. Sever epidemics of the disease were observed in some wheat field, in the northern part particularly Nineveh, Kirkuk and Salahaddin in 1995, where high infection were conducted on the commercial wheat cultivars in the fields [5]. While the first epidemics of yellow rust was observed in 1998 in some wheat field in the middle zone particularly Babylon and Baghdad. The infection rate was ranged from 0.089 to 0.348 per unit per day during the epiphytotic of the disease [6]. Recently, the disease appeared annually in different locations during 1997 to 2010. The highest epidemic was detected in 2010 in most wheat fields, in different agrological zones when the infection rate was very high during the season 2010 [7], [24] and [25]. The infection rate and the epidemic period of yellow rust during 2010 in the same field and area was higher than the one's which we detected during our study in 2011 [25]. Distribution of yellow rust disease in the middle zones of Iraq in last years may be due to appearance of new biotype or races of *P. striiformis f. sp. tritici* in this region or due to the recent climatic condition changes. Two new races, 230 E150 and 6E 16 were identified in 1998 [26] and new virulence against *Yr27* and *Yr25* were identified in *P. striiformis* population [7]. We believe that uredospores of *P. striiformis f. sp. tritici* reach to Kurdistan region from other neighboring countries such as Turkey, Iran, Syria, and other, countries, since it is capable to pass a long distance due to its air borne characters and resistance to short wave rays and unfavorable conditions [19] and [27]. Furthermore the absence of the alternate host in Iraq [5], countries with the epidemiological zone 7 are the source of spreading of new races of the fungi in Asia particularly Iraq, north eastern Syria, south eastern Turkey, Iran, north western Afghanistan and south western of former united soviet [28], this explain why the spore found during February to March while the diseases not appear on the susceptible cultivar SaberBeg until 24th April, Figure (1).

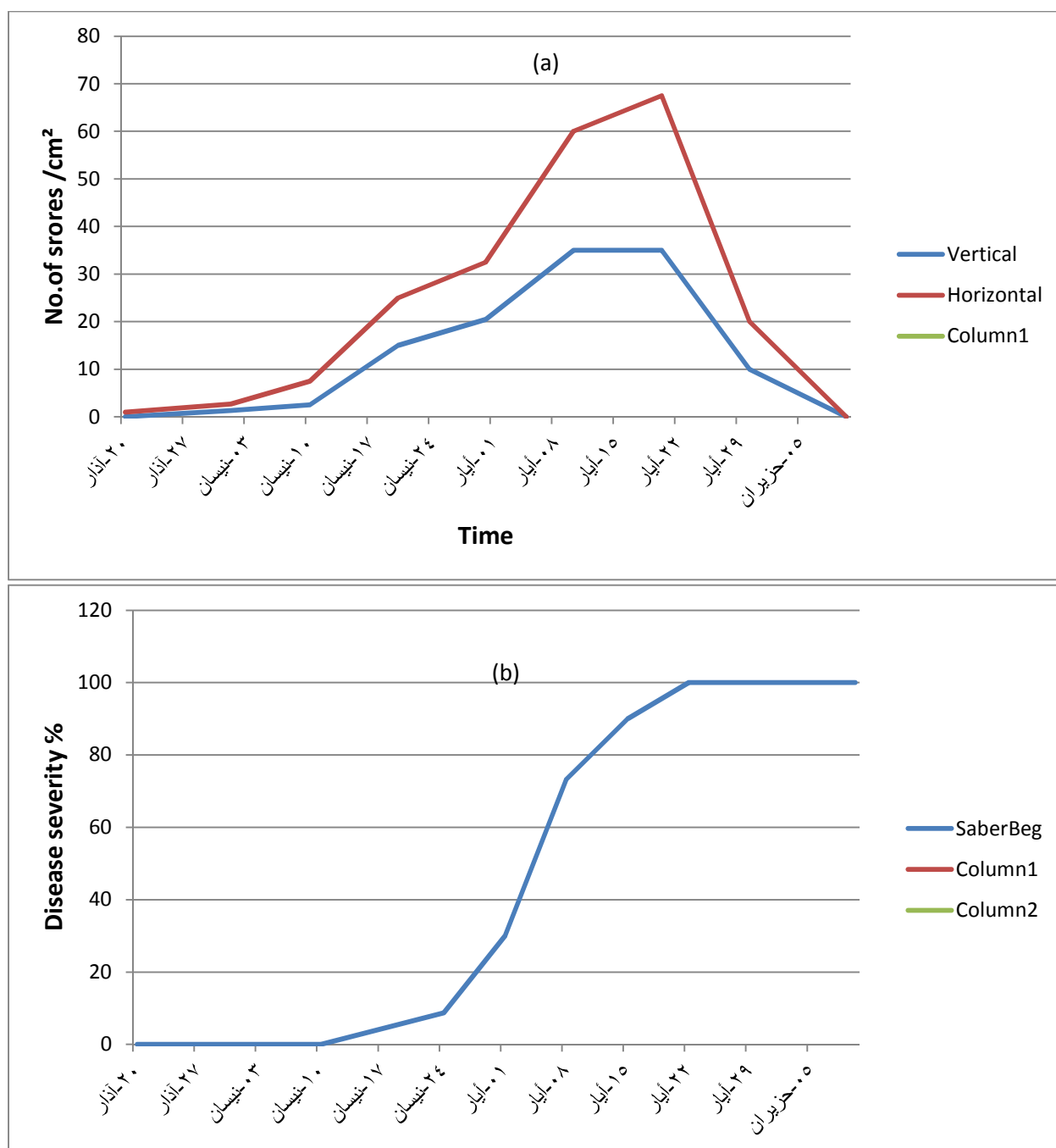


Figure-1: Number of uredospores of *Puccinia striiformis* f. sp *tritici* collected on vertical and horizontal traps (a) during yellow rust development on SaberBeg (b) at Bakrajo Experimental Research Directorate Field in 2011.

Development of stripe rust disease on some wheat genotypes

Figure 2 presents yellow rust development curve on seven different wheat genotypes, each one has different response to the disease. SaberBeg, Tamuz 2 and Araz are local commercial wheat cultivars sown on a large scale in the rain fed area, while Al-8/70, Al-84, Al-8/172, and Al-124 are advanced promising resistant lines developed by breeding program and ready to release in the same area. Results revealed that the highest disease development curve was observed on the highly susceptibility cultivar SaberBeg which was resulted from 100% disease severity, during 28 days study period under the epidemics of yellow rust at Bakrajo experiment station in 2011 followed by the susceptible genotypes Al-8/70 and Araz which were resulted from 82.7% and 77.5 % disease severity respectively. While the lowest disease development curve was recorded for the resistant genotype Al-124 and resulted from 12.3%, the rest genotypes have divers disease

development curves and severities as showed in Figure 2. Table 1 shows the highest value in Area Under Disease Progress Curve (AUDPC) appeared for SaberBeg (252.0) which was significantly surpassed all other genotypes followed by AI-8/172 (158.0). While the lowest AUDPC value appeared for AI-124 (7.9), there were obvious significant differences between all the genotypes in AUDPC-values. This value mostly depends on disease severity and infection rate of the disease of the genotypes. The high disease development curve on SaberBeg resulted from high infection rate (r) of yellow rust on this genotype during the epidemic of the disease in 2011 (Table 2). The mean infection rate value of SaberBeg, which was 0.247 per unit per day significantly surpassed all other genotypes followed by the susceptible cultivars AI-8/70 (0.179) and Araz (0.155). While the lowest r -value, (0.076) was recorded for the resistant genotype AI-124 and 0.084 for the moderately resistant genotype AI-8/172. No significant differences were observed between AI-124 and AI-8/172 in r -value but AI-124 had significant differences with all other wheat genotypes. Although AI-84 showed moderately resistant to moderately susceptible reaction with yellow rust during this season, r -value on this genotype was low (0.096) and not differs significantly with the moderately resistant genotype AI-8/172. Also no significant difference in r -value was found between the susceptible cultivar Araz and the moderately susceptible cultivar Tamuz 2. Although yellow rust uredospores were found in the air from the end of February to march, but the primary infection of the disease did not appear until April, this was because of the unfavorable environmental conditions requirement for yellow rust development, which is obvious in Figure 3. Metrological data of Sulaimania province in 2011 revealed that during March the precipitation was very rare except at 8th March, which was resulted from low relative humidity 56% and temperature (18.2°C). This condition was not suitable to development of the disease. While the frequent precipitation and amount of rain by the beginning of April, 19th April resulted in raising the relative humidity to above 63% in 19th April and to 84% on 21th April. Also the mean temperature increased to 22°C on 19th April. All these factors stimulated uredospore germination and appearance of the primary infection of yellow rust in the susceptible cultivars on 24th April. This indicates that the environmental conditions are the most critical factor for the host-pathogen interaction system in the presence of pathogen virulence and the susceptible tissue [20]. We were not able to predict the epidemiology of yellow rust because it depends particularly on the environmental conditions made the disease very dangerous, beside the ability of the pathogen to develop it self or arrive new races from other countries. All these factors complicated yellow rust control. Hogg et al. (1969) [2] stated that *P. striiformis* has the lowest temperature requirement of the three wheat rust pathogens, with minimum, optimum and maximum temperature of 0, 11 and 23°C respectively, while Loladze et al. (2009) [29] explained the adaptation of new fungus strains by its shorter latent periods and faster rate in germination of uredospores. The researcher also suggested that post 2002 stripe rust population in Australia may be potentially better adapted to higher temperatures, thus it possess a superior environmental fitness. This was confirmed in the North-west Europe and Mediterranean area including Iraq [6] and [30]. The high resistance of AI-124 may be due to it's incompatibility with the pathogen community in the country or its mechanical or chemical resistance mechanism against the disease due to the high amount of wax layer around the pores and garden cell. The structure of cell wall or the high number of leaf hair content might be another possible reason, besides increasing in the rate of hydrogen peroxide or other phenolic compounds which inhibits the growth of pustules inside the leaf [31], [32] and [33]. Other varieties have different response to the pathogen population, which clearly reflects the range of infection development and disease severities. These results are similar to Kolmer (1996) [34] who found variation in disease development on different cultivars; the genetic component of each cultivar and the type of resistant genes are mainly depends on.

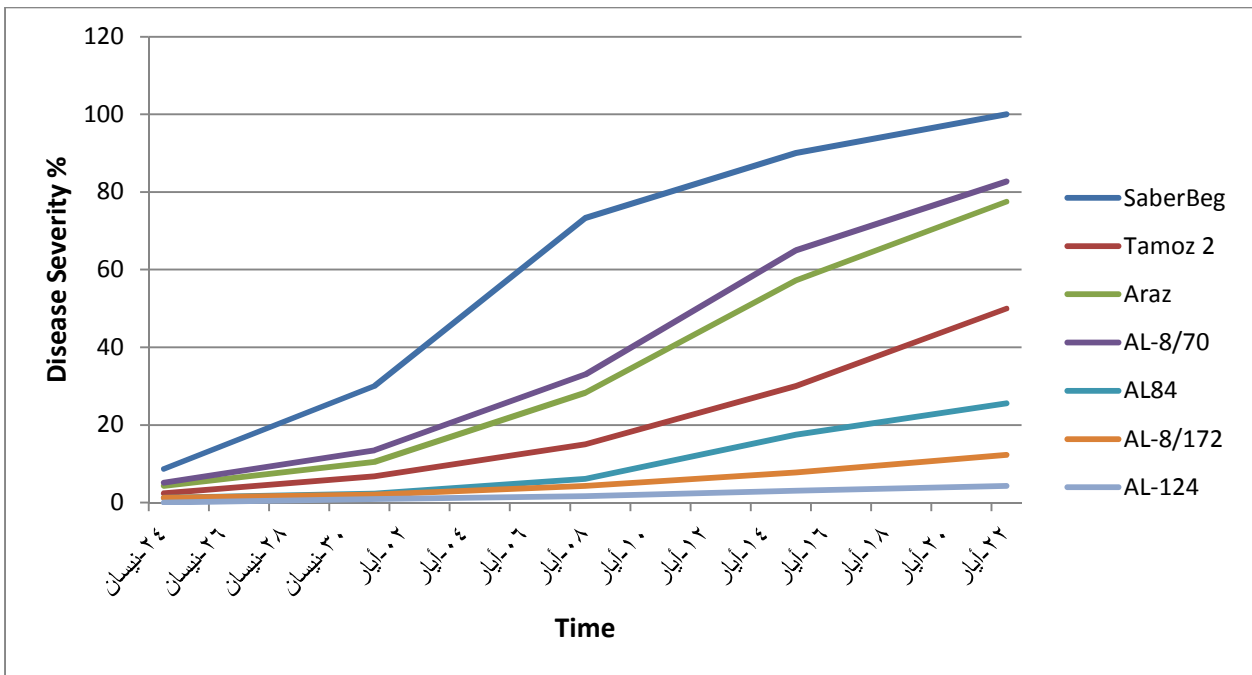


Figure-2: Yellow rust development on different wheat genotypes during 2010/11 season at Bakrajo experimental Directorate, Sulaimani, IKR, Iraq.

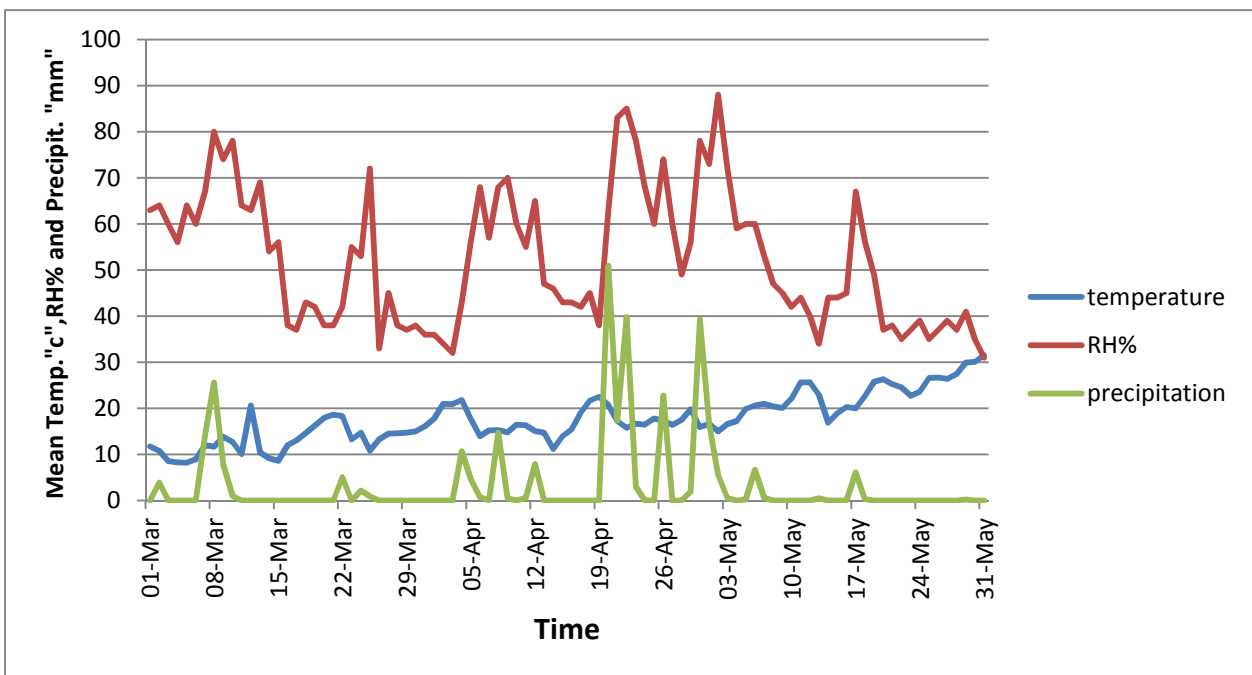


Figure-3: Mean Temperature “°C”, relative humidity “RH%” and precipitation “mm” from the 1st march to the end of May, 2011.

Table-1: Disease severity, infection rate (r-value) and area under disease progress curve (AUDPC) of yellow rust for different wheat genotypes during 2011 epidemics at Bakrajo Experimental Directorate, Sulaimani, IKR, Iraq.

Genotype	Disease Severity (%)	Infection rate (r)	AUDPC
SaberBeg	100.0	0.247	252.0
Araz	77.5	0.155	129.1
Tamuz 2	50.0	0.143	79.2
Al-8/70	82.7	0.179	158.0
Al-84	25.6	0.096	40.0
Al-8/172	12.3	0.084	21.6
Al-124	4.3	0.076	7.9
Mean	50.3	0.140	98.3
LSD 0.05	6.3	0.019	9.1

Host reaction of different wheat genotypes with *Puccinia striiformis*

Table 2 results represent a wide range of host reaction between the tested genotypes with the pathogen population of *P. striiformis* started from high resistance reaction in Al-124 to high susceptibility in SaberBeg, susceptible in Araz and Al-8/70, moderate susceptible to susceptible in Tamuz 2, moderate resistance to moderate susceptibility in Al-84, and moderate resistance in Al-8/172. Furthermore, high significant differences between the tasted genotypes in coefficient of infection value (C.I), The highest value of (C.I) was recorded for SaberBeg which was 100.0 followed by 82.7 in Al-8/70, while the lowest value of (C.I) was recorded for Al-124 which was 0.9 followed by 4.9 in Al-8/172. Coefficient of infection value in SaberBeg significantly surpassed all other tested genotypes. We can also clearly observe significant differences between all other genotypes in this value except Al-124 and Al-8/172, (Table 2). The coefficient of infection value facilitates the statistical ranking or comparison between the genotypes with different responses to the disease. Adding two separate factors in a single value results in nearly equal coefficient but from different disease score. The differences in genetic background of resistance reflect the differences in the infection type toward the disease. The infection type in some cultivars may be changed by time due to appearance of new virulence's in the pathogen population. Some cultivar may stay resistant to many years but after a period it will be susceptible such as mentioned by Al-Maarouf (1995)[5] that the infection type of Al-Eze was moderately resistant at the beginning of its release in 1995 while after a decade the infection type of the same cultivar was recorded to be changed to susceptible type [21]. The ability of the pathogen to change itself and its virulence and appearance of more aggressive pathogen might happened by the sexual reproduction, combination or a crossing methods and other mechanisms that the pathogen could develop itself. Hence, it is very important to study the pathogen population annually to recognize the new virulence that may come from other countries particularly the disease is air borne, which make it very difficult to control, and it may overcome resistance of some cultivars after some time from their release as a resistant cultivars which is clear in boom and burst cycle [19], and many researchers confirmed this fact [36], [37] and [38]. They mentioned to the ability of rust pathogens to develop and produce new and unique races. The resistant reaction of Al-124 and Al-8/172 may be due to presence of more than one resistant gene in their genetic structure but not in other susceptible cultivar. Possessing more resistant genes increased the time of resistance stability in each cultivar, also the pathogen needs more time to develop virulence against different resistant genes, as it is mentioned by many researchers [35], [39], [40] and [41].

Characteristics of *P. striiformis* pustules and uredospores on different genotypes

Result of Table 3 shows number of *P. striiformis* pustules in each unit area (pustules/cm²) in different wheat genotypes with diverse infection types. The highest pustules number (131 pustule/cm²) was found in the highly susceptible wheat cultivar SaberBeg, which was significantly, surpassed all other genotypes, while the lowest number of pustules was found in the resistant genotype Al-124, with 2 pustules/cm². No significant differences between the susceptible cultivars Araz and Al-8/70 were detected in number of pustules, which

Table- 2: Host reaction of different wheat genotypes with *P. striiformis* f. sp. *tritici* population during 2011 epidemics at Bakrajo Experimental Directorate, Sulaimani, Iraq.

Genotype	Infection Type	Disease Severity %	C.I
SaberBeg	HS	100.0	100.0
Araz	S	77.5	77.5
Tamuz 2	MSS	50.0	45.0
Al-8/70	S	82.7	82.7
Al-84	MR-MS	25.6	20.5
Al-8/172	MR	12.3	4.9
Al-124	R	4.3	0.9
Mean	-	50.3	23.7
LSD 0.05		6.3	4.3

Table- 3: Characteristics of *P. striiformis* pustules, uredospores and inoculum load on different wheat genotypes during 2011 at Bakrajo, Sulaimani, IKR, Iraq.

Genotype	Infection type	Number of pustules/ Cm ²	Pustule dimension (μ)		Number of spores / Pustule	Spore dimension (μ)	
			Length	Width		Length	Width
SaberBeg	HS	131	540.0	268.8	2010	37.0	34.3
Araz	S	56	517.5	248.8	1600	25.8	20.3
Tamuz 2	MSS	35	360.0	180.0	1200	25.4	20.7
Al-8/70	S	66	472.0	202.5	2100	33.8	29.6
Al-84	MR MS	28	438.8	180.0	900	26.3	19.4
Al-8/172	MR	13	300.0	150.0	500	24.1	16.9
Al-124	R	2	0.00	0.00	0.00	0.00	0.00
L.S.D. 0.05	-	7.9	52.3	27.5	97.2	3.6	2.4

was significantly higher than other genotypes. Number of pustules per unit area was highly reduced by 99% in the resistant genotype Al-124 and by 79-90% in the moderately resistant to moderately susceptible genotypes Al-8/127 and Al-84 in compare with the susceptible cultivars. The high numbers of pustules on the susceptible cultivars clearly explain the high losses in grain yield, since these pustules destroy large leaf area's in the susceptible cultivar than the resistant ones. Results of Table 3 also state high significant differences in pustules dimension (length and width) between the genotypes. SaberBeg had the largest pustules size (540*268.8μm) which significantly different from all other genotype except Araz. While the resistant genotype Al-124 explores only chlorosis area on the leaf, which is not real pustules, this is part of resistant reaction that the plant defense against the disease. Significant differences also found among other genotypes with diverse categories of resistance. Numbers of spores/pustules, which represent the inoculum load, were significantly different among all the tested genotypes. The highest number of uredospore (2100) was found in Al-8/70 followed by SaberBeg in 2010. No any spore found in the resistant genotype Al-124, while 500 spores in each pustule was found in the moderately resistant genotype Al-8/172. The inoculum load of the susceptible cultivars explains the high infection rate of yellow rust and development of the disease in the susceptible plots comparing with the resistant ones (Fig. 4). Data of Table 3 also revealed that there are no significant differences in the uredospore length between SaberBeg and Al-8/70 but there are

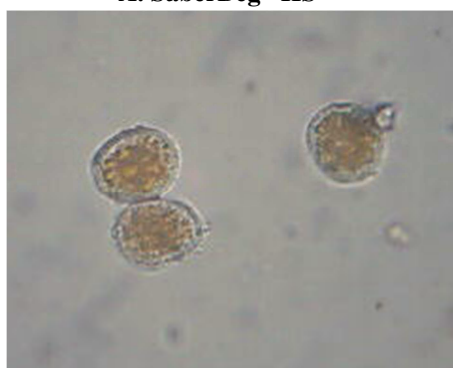
significant differences with other cultivars, meanwhile no significant differences in width of spores were detected among Araz, Tamuz2, Al-84, and Al-8/172, which was significantly lower than SaberBeg spores (Fig. 4). No uredospore was found in the pustules of Al-124 only, which explore resistance reaction as necrotic area on the leaf surface. We can conclude that the highly susceptible cultivar presented in SaberBeg had biggest pustules, highest numbers of uredospore and the largest spore dimensions, that mean there are a positive relation between the infection type and the pathogen development. Furthermore the pustules on the susceptible tissues extended semi systematically in line to about 8cm, while the extension of pustule lines was determinate with the host plant response. If this compared with the resistant cultivar Al-124 we can see that the pathogen inoculum could not developed on resistant tissues because of the necrotic or chlorotic areas will kill the plant cells around the spore to prevent germination or any development of the spore and at the end the spore will die and the pustule extension is extremely determinate [42].



A. SaberBeg "HS"



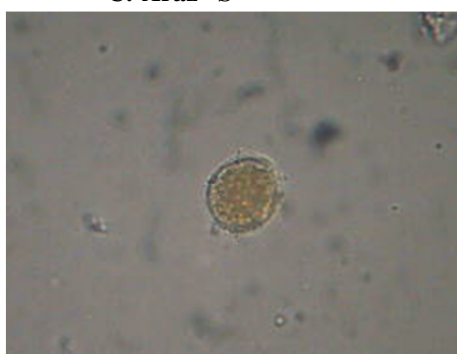
B. Al-8/70 "S"



C. Araz "S"



D. Tamuz2 "MS"



E. AL-84 "MRMS"



F. AL 8/172 "MR"

Figure- 4: Characteristics of *Puccinia striiformis* uredospore on different wheat genotypes

(A. SaberBeg, B. Al-8/70, C. Araz, D. Tamuz 2, E. Al-84, F. Al-8/172).

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