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PATHOGENIC DIVERGENCE OF *Tilletia tritici* AND *T. leavis* ISOLATES - THE CAUSAL AGENTS OF WHEAT COMMON BUNT DISEASE IN IRAQ

RETRACTED MANUSCRPT

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ABSTRACT

Common bunt of wheat is one of the most serious diseases which caused heavy losses to wheat crop especially when seed of susceptible varieties were sown without treated with fungicides. The study was conducted to determine the genetic variability and pathogenicity of two *Tilletia* species *Tilletia tritici* and *T. leavis* through analyses the reaction characters of pathogenic isolates with differential varieties. Spike showing infections of common bunt were collected from the major wheat growing areas of Iraq during 2012/2013 cropping season. Based on the higher percentage of teliospores germination, 18 isolates of common bunt were selected and used for contamination the seed of differential varieties at faculty of agricultural sciences fields in Sulimania. Results of study revealed the existence of large genetic variation between *T. tritici* isolates representing different ecological areas. Among the 18 selected isolates, ten were showing similarity with the already reported world races,T1,(T1, L1, L2), T2 ,T4 ,T9, T11, T13, T12, T18, T20) while the rest 8 isolates could be new races and to best of our knowledge not recorded previously in any part of the world. It has been found that the resistance genes Bt2, Bt6, Bt14 and Bt15 were not effective against most of pathogenic isolates, while Bt1 was found non effective against 5 isolates, Bt4, and Bt8 were non-effective against 4 isolates, Bt3 and Bt5 were ineffective against 3 isolates only. All the isolates were found unable to overcome the resistance genes Bt11 and Bt12, where 2 isolates were found able to overcome the resistance genes Bt7, Bt9, Bt10 and Bt13.

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1 Introduction

Tilletia tritici (Bjerk.) Wint (*T. caries*) (Dac.) Tul.) and *T. leavis Kuhn* (*T. foetida* (*Wall.*) *Liro.* were considered as the causative agents of common bunt of wheat that distributed in western and center Asia, North Africa, China and Europ areas (Blazkova & Bartos 2002; Lipps et al., 2000). *T. tritici* is a dominant pathogen of many North and South American locations and infecting winter wheat *Triticum aestivum*, whereas *T. leavis* is showing in restricted distribution in these areas but is widely distributed in all mountain and center regions in Europe as well as eastern north of America (Jones & Cilfford 1978). In Iraq, *T.tritici* is dominant in the north areas that characterized by low temperature compared with *T.leavis* which distributed in the center and south areas (Shams-Allah, 2005). Both species had capabilities to infect all smooth and hard wheat varieties (*T.aestivum* and *T.durum*). The yield losses fluctuated from one season to another and it depends on ecological conditions, soil texture, fertilization, wheat variety, pathogenic races, inoculum potential, depth and quantity of seed sowing (Fisher & Holton, 1957). Common bunt disease are characterizes by the formation of teliospore mass balls by replacing the kernels. These balls are broken during harvesting time and liberating teliospores which cause contamination in soil and healthy seeds. Teliospores germinate within few days to few weeks, it depends on humidity and soil temperature, and the infectious hyphae penetrate in to new seedling and systemically grown in the plant (Kollmorgen et al., 1978; McManus et al., 1993).

The identification of the causal agent races of common bunt was done by inoculation of differential varieties with all the collected isolates and follows the reaction between the virulent genes of the pathogen with the resistance genes in the differential varieties (Gene for gene theory) and compared them with known races reported by Hoffman & Metzger (1976). In case of reaction between these pathogen isolates and differential varieties is different from previously known pathogen races, the isolates could be considered as new races in this area.

Several races of common bunt pathogen were identified worldwide among these L10, L20, L29, L30, L31, L32 are common in Iran (Dariaee et al., 2006) while L1, L3, L6, T1, T3 are in Turkey (Finci,1981). Two races of *T .tritici* and 3 races of *T. leavis* were named as T11and L9 which was based on the similarity of differentiation reactions with certain races from America (Ismail et al 1995).

Due to the importance of wheat as main food crop as well as the wide distribution of common bunt disease in all its cultivated areas and absence of studies concerning genetic variation in *T.tritici* and *T. leavis* in Iraq, this study has been conducted to determine genetic variations in pathogen isolates and determine the pathogenic races.

2. Materials and Methods

The experiment was carried out in the cropping season of 2012/2013 at experimental field of Faculty of Agricultural Sciences, Sulimania University, Iraq. The experimental conditions were suitable for germination of teliospores and wheat seeds and development of common bunt disease infection (Temperature 5-10° and humidity 41%).

Table 1 Different variety and their resistant genes used for identification pathogen races.

Differential variety*	Resistant gene	No.
M84-504 to 510, Red Bobs	Bt0	1
M84-512 to 520, RB/WF 8	Bt1	2
M84-522 to 530, RB/SEL 1403	Bt2	3
M84-532 to 538, RB/RDT.	Bt3	4
M82-542 to 550, RB/TK 3055	Bt4	5
M82-34, Promose	Bt5	6
M84-552 to 560, RDT.	Bt6	7
M82-562 to 570, RB/TK 3055	Bt7	8
M78-9496 , RB/PI 178210 (White Seed)	Bt8	9
M84-597 to 605, RB/CI 7090	Bt9	10
M84-625 , SEL M83-162	Bt10	11
M82-2123	Bt11	12
P.I. 119333(M82-2141), BW	Bt12	13
Thule III; P.I. 181463, BW Bt 13	Bt13	14
Doubi, DW	Bt14	15
Carlton, DW	Bt15	16

*Source of differential varieties seeds is Dr. B. Goates (USDA-ARS), Aberdeen, Idaho, USA.

2.1 Collection of common bunt samples

Common bunt infected wheat spike were collected from major wheat growing areas of Iraq during 2012/2013 seasons. Three isolates of the causal agent were selected from each of Mousl, Sulimania, and Duhok, tow isolates from Erbil, and one isolate from each of Salah El-Din, Baghdad, Diala, Wasit, Dewania, Garmian, and Mesan areas according to 5 level of teliospores germination. The isolation of teliospores was done by rupturing bunt balls center with the help of sterile needle and distribution them on 2% agar in 9cm petriplates with 3 replications. The plates were incubated at 15^{o}C for 3-4 days and the percentages of teliospores germination of various isolates were recorded. The isolates with more than 50% germination percentages were selected and used for races identification by using differential varieties as shown in table 1.

2.2 Differential varieties inoculation

Each infected spike considered as a pathogenic isolate representing determined area. The bunt balls of 18 selected isolate were separately ground in porcelain mortar and passed through a sieve of 500 µm. Seed of the differential varieties were mechanically inoculated with the powder of each isolates with continual agitation for homogenous distribution of teliospores on seed. The contaminated seeds were sown in lines of 1.5 m length (2 lines/isolate; 15seeds/line) in the soil which is not used for the wheat cultivation from several years. The virulence of the various isolates against different varieties was recorded at maturity stage. The isolate with infection percentages between 0-10% were considered as avirulent while those have infection percentages between 11-100% were considered as virulent (Hoffman & Metzger, 1976).

3 Results and Discussion

Results of the study have been shown in table 2 and 3; these results revealed the variation in the virulence between collected pathogenic isolates from different areas of Iraq. From the collected isolates, the isolates T1, T6, T8, T9, T10, T11, T14 were virulent against the resistance gene Bt15 while the isolates T7, T8, T9, T10, T11, T12, T13 were reported virulent against resistance gene Bt2. Similarly the available isolates T2, T3, T4, T7, T9, T17 were showed virulence against resistance gene Bt14 and the isolates T1, T3, T8, T10, T16 were showing virulence against resistance gene Bt1, T1, T11, T12, T13 were virulent against resistance gene Bt4.

The isolates T2 and T5 were characterized by their virulence against resistance gene BT7, and T12, T17 against resistance gene Bt9, T12, T13 against Bt13 while the isolates T3 and T14 overcome resistance gene Bt10, while all the isolates were unable to overcame the resistance gene Bt11 and Bt12, which indicate the possibility of using these varieties as source of resistance genes to hybridization process with susceptible varieties possessing high productivity. These resistance genes were followed by the genes Bt7, Bt9, Bt10, Bt13, for controlling the virulence of *T. tritici* and *T.leavis* isolates distributed in the wheat field in Iraq which were in accordance with several previous studies to the world (Metzger & Hoffmann, 1978; Blazkova & Bartos, 2002; Gaudet et al., 2006; Noruzi et al., 2012). The importance of other resistance gene came from the number of isolates possessing virulence gene against them, lesser the number of isolates that overcome host resistance genes become preferable for including in breeding programs to control disease.

Table 2 Virulence of common bunt pathogen isolates against the known resistant genes (Bt genes) of the differential wheat varieties

Resistant genes	Number of Isolates	Resistant genes
T14 ; T11 ; T10; T9; T8 ;T6; T1	7	**Bt15**
T13; T12 ; T11 ; T10; T9; T8; T7	7	**Bt2**
T17 ; T9; T7 ;T4; T3 ;T2	6	**Bt14**
T16 ; T15; T14 ;T6;T4 ; T1	6	**Bt6**
T16 ;T10;T8 ;T3 ;T1	5	**Bt1**
T13;T12 ;T11 ; T1	4	**Bt4**
T18 ;T17;T12 ;T7	4	**Bt8**
T18 ;T8 ;T3	3	**Bt3**
T17 ;T15 ;T4	3	**Bt5**
T5 ;T2	2	**Bt7**
T17 ;T12	2	**Bt9**
T13 ;T12	2	**Bt13**
T4 ;T13	2	**Bt10**
0	0	**Bt11**
0	0	**Bt12**

Table 3 Host parasite interactions of differential varieties with 18 isolates of common bunt pathogens collected from different agro ecological zones of Iraq.

Isolate	Location	Resistant gene															
		Bt0	Bt1	Bt2	Bt3	Bt4	Bt5	Bt6	Bt7	Bt8	Bt9	Bt10	Bt11	Bt12	Bt13	Bt14	Bt15
T1	Baghdag/Twaitha	S	S	R	R	S	R	S	R	R	R	R	R	R	R	R	S
T2	Diala/Bladroze	S	R	R	R	R	R	R	S	R	R	R	R	R	R	S	R
T3	Wasit/Sheikh Saad	S	S	R	S	R	R	R	R	R	R	S	R	R	R	S	R
T4	Mesan/ Ali Gharbi	S	R	R	R	R	S	S	R	R	R	R	R	R	R	S	R
T5	Dewania/Mhnawia	S	R	R	R	R	R	R	S	R	R	R	R	R	R	R	R
T6	Salahdhin/Bejee	S	R	R	R	R	R	S	R	R	R	R	R	R	R	R	S
T7	Mosul/Hamdania	S	R	S	R	R	R	R	R	S	R	R	R	R	R	S	R
T8	Mosul/Rabiaa	S	S	S	S	R	R	R	R	R	R	R	R	R	R	R	S
T9	Mosul/Sherqat	S	R	S	R	R	R	R	R	R	R	R	R	R	R	S	S
T10	Garmian/ Kalar	S	S	S	R	R	R	R	R	R	R	R	R	R	R	R	S
T11	Sulaimania/	S	R	S	R	S	R	R	R	R	R	R	R	R	R	R	S
T12	Sulaimania/Bakrajo	S	R	S	R	S	R	R	R	S	S	R	R	R	S	R	R
T13	Sulaimania/	S	R	S	R	S	R	R	R	R	R	R	R	R	S	R	R
T14	Erbil/ Rania	S	R	R	R	R	R	S	R	R	R	S	R	R	R	R	S
T15	Erbil/ Kwesanjaq	S	R	R	R	R	S	S	R	R	R	R	R	R	R	R	R
T16	Duhok/ Zakho	S	S	R	R	R	R	S	R	R	R	R	R	R	R	R	R
T17	Duhok/ Malta	S	R	R	R	R	S	R	R	S	S	R	R	R	R	S	R
T18	Duhok/ Feshkhabor	S	R	R	S	R	R	R	R	S	R	R	R	R	R	R	R

11 – 100% =S *R=0 -10% , infection

The resistance gene Bt11 and Bt12 showed high efficiency against virulence isolates of the pathogen in most of the wheat growing areas of the world (Blazkova & Bartos, 2002; Wang et al., 2006). Mamluk & Nachit (1994), Braun et al. (1997) have indicated to the efficiency of Bt5, Bt8, Bt9, Bt10, Bt11 which exist in differential varieties in Syria. While some studies reported that Bt1, Bt2, Bt3, Bt4, Bt6, Bt7 were overcome by the virulence strains of the pathogen in Syria ,and Bt1, Bt2, Bt7 in Lebanon Bt1, Bt2, Bt3, Bt4, Bt7 in Turkey, whereas the Iranian isolates showed virulence against Bt4, Bt7, Bt9 (Mamluk & Nachit, 1994). It was reported that the virulence of fungal isolates has dominated the resistance genes, Bt2, Bt5, Bt7, Bt8, Bt9, Bt10 in India (Chauhan et al., 1994).

High efficiency in controlling virulent pathogen isolates was obtained by using the resistance genes Bt9 and Bt10 in Europe, followed by the gene Bt5, Bt6, Bt8 (Ittu et al., 2006; Oncica & Saulescu, 2008). The genetic variety PI178383 (Turkey origin) which possess the resistance genes Bt8, Bt9, Bt10 and another unidentified gene was adopted in breeding program to control common bunt disease in USA. The same genes were used in Russia and Australia (Goates, 1996; Blazkova & Bartos, 2002; Liatukas & Ruzgas, 2008).

Results presented in table 4 showed the presence of genetic variations between the various isolates of common bunt pathogen as proved by their reaction nature with the differential varieties viz Bt1-Bt15,compared with those between world identified races with the deferential varieties by Hoffman & Metzger (1976). T1 isolate (Baghdad/Twaitha) has Bt1, Bt4, BT6 & Bt15 as dominated resistance genes, and in this respect it showed similarity with the world race T18 except the presence of Bt15 which may probable a new race. T2 isolate (Diala/Bladroze) showed similarty with the world race T1 except the presence of the gene Bt14 which may probable a new race, in the same way T9, T11, T13 may probable a new race.

Results also showed similarity between T5 and T8 collected from Dewania/Mhnawia and Mosul/Rabiaa, with the world race T1, L2, L1 and T13 in their virulence against Bt17 and (Bt2, Bt3, Bt15) respectively and isolate T10 from Garmian/Kalar, with the world race T20 in its virulence against resistance gene , Bt11, Bt2, Bt15. The isolates T3, T4, T6, T7, T12, T14, T17, T18 from Wasit/Sheikh Saad, Mesan/Ali Gharbi, Mosul/Hamdania, Sulaimania/Bakrajo, Erbil/Rania, Duhok/Malta and Duhok/Feshk- habor respectively were considered as new races according to non similarity in their reaction pathway with all the identified world races.

Table 4 Host parasite interaction of common bunt differential set with *Tilletia spp* isolates.

Location	Isola	Germination	Pathogen	Virulence/Avirnlence against Bt	Race
Baghdag/Twaitha	T1	+++	*T.tritici+laevis*	1,4,6,15 / 2,3,5,7,8,9,10,11,12,13,14	(T18) Probable new
Diala/Bladroze	T2*	++	*T.tritici*	7,14 / 1,2,3,4,5,6,8,9,10,11,12,13,15	(T1) Probable new race
Diala/Bladroze	T2	+	*T.tritici+laevis*		
Wasit/Sheikh Saad	T3	++	*T.tritici+laevis*	1,3,10,14 / 2,4,5,6,7,8,9,11,12,13,15	Proposes new race
Mesan/ Ali Gharbi	T4	++	*T.tritici+laevis*	5,6,14 / 1,2,3,4,7,8,9,10,11,12,13,15	Proposes new race
Dewania/ Mhnawia	T5	++	*T.tritici+laevis*	7 / 1,2,3,4,5,6,8,9,10,11,12,13,14,15	T1,L1,L2
Salahdhin/Bejee	T6	++	*T.tritici*	6, 15 / (1 –5, 7- 14)	Proposes new race
Mosul/Hamdania	T7	++	*T.tritici*		
Mosul/Hamdania	T7*	+++	*T.tritici*	2,8,14 / 1,3,4,5,6,7,9,10,11,12,13,15	Proposes new race
Mosul/Rabiaa	T8*	+++	*T.tritici*	1,2,3,15 / 1,4,5,6,7,8,9,10,11,12,13,14	T13
Mosul/Rabiaa	T8	++	*T.tritici*		
Mosul/Sherqat	T9	+++	*T.tritici*	2,14,15 / 1,3,4,5,6,7,8,9,10,11,12,13	(T11) Probable new
Garmian/ Kalar	T10	+++	*T.tritici*	1,2,15 / 3,4,5,6,7,8,9,10,11,12,13,14	T20
Sulaimania/ Halabja	T11	+++	*T.tritici*	2,4,15 / 1, 3 (5 – 14)	(T17) Probable new
Sulaimania/ Bakrajo	T12	++	*T.tritici*		
Sulaimania/ Bakrajo	T12	+++	*T.tritici*	2,4,8,9,13 / 1,3,5,6,7,10,11,12,14,15	Proposes new race
Sulaimania/ Penjween	T13	+++	*T.tritici*	2,4,13 / 1,3,5,6,7,8,9,10,11,12,14,15	(T17) Probable new
Erbil/ Rania	T14	++	*T.tritici*		
Erbil/ Rania	T14	+++	*T.tritici*	6,10,15 / 1,2,3,4,5,7,8,9,11,12,13,14	Proposes new race
Erbil/ Kwesanjaq	T15	+++	*T.tritici*	5,6 / 1,2,3,4,7,8,9,10,11,12,13,14,15	(T9) Probable new race
Duhok/ Zakho	T16	+++	*T.tritici*	1,6 / 2,3,4,5 (7 – 15)	(T2,T4) Probable new
Duhok/ Malta	T17	+++	*T.tritici+laevis*	5,8,9,14 / 1,2,3,4,6,7,10,11,12,13,15	Proposes new race
Duhok/ Feshkhabor	T18	+++	*T.tritici*	3,8 /1,2,4,5,6,7,9,10,11,12,13,14,15	Proposes new race

higher germination level of teliospores Selection; * higher germination) more than 50%. +++(moderate germination) 25 - 49%. ++ (low germination) 1- 24%. + (infection (0 -10%) avirulent , (11-100) virulent (Hoffmann& Metzeger,1976).

These results explained the reasons of migration common bunt disease from northern to central and southern areas of Iraq as represented by Wasit and Mesan governorates as a result of appearing new races of disease causal agent as reported by Al-Maaroof et al. (2004) in a survey of the disease in wheat field during 2002-2003 and proved by next studies (Shams-Allah, 2005; Al-Maaroof et al., 2006).

The results of this study showed the presence of 18 races of *T.tritici* and *T. leavis* causing common bunt disease in Iraq. Among these races, 10 were found similar to world races T1, (T1, L1, L2), T2, T4, T11, T9, T13, T17, T18, T20 and 8 of them could be new races not recorded in the world . This number could be increase or decrease in case of more than one species are present in the one kernel (Fisher & Holton, 1957), or in case of the presence of mechanical or genetic mixture in the differential varieties seed which may affect the reaction between virulence genes in the pathogen with the resistance genes in the differential varieties. Therefore the experiment needs some more exploration of the available results. In addition the isolates should be confirmed at molecular level and compared with known world races.

Noruzi et al. (2012) reported that the races L19, T1, T2 of common bunt causal agent were dominant in Lrstan/Iran. Whereas T1, T11, L4, L9 were dominant in Syria as well as 5 new races were recorded by their reaction with the differential varieties referred as T17, T20, L13, L14, L15 (Kayali et al., 2011). In Turkey the races T1, T3, L1, L3, L6 were the more distributed (Finci, 1981). The results demonstrated the presence of relationship between common bunt causal agent races of Iraq with those of Syria and Iran larger than with those of Turkey.

Conflict of interest

The authors declare no conflict of interest.

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