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 Sulaimania. 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 causatives 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 & Clifford 1978). In Iraq, _T.tritici_ is dominant in the north areas that characterized by low temperature and high humidity conditions 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 is characterized by the formation of teliospore mass balls by replacing the kernels. These balls are broken during harvesting 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 (Dariae et al., 2006) while L1, L3, L6, T1, T3 are in Turkey (Finck,1981). Two races of _T. tritici_ and 3 races of _T. leavis_ were named as T1 and 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, Sulaimania University, Iraq. The experimental conditions were suitable for germination of teliospores and wheat seeds and development of common bunt disease infection (Temperature 5-10°C 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 | B0 | 1 |
| M84-512 to 520, RB/WF 8 | B1 | 2 |
| M84-522 to 530, RB/Sel 1403 | B2 | 3 |
| M84-532 to 538, RB/RDT | B3 | 4 |
| M82-542 to 550, RB/TK 3055 | B4 | 5 |
| M82-34, Promote | B5 | 6 |
| M84-552 to 560, RDT | B6 | 7 |
| M82-562 to 570, RB/TK 3055 | B7 | 8 |
| M78-1996, RB PI 178210 (White Seed) | B8 | 9 |
| M84-597 to 605, RB/CI 7090 | B9 | 10 |
| M84-625, Sel, M83-162 | B10 | 11 |
| M82-2123 | B11 | 12 |
| P.I. 119333(M82-2141), BW | B12 | 13 |
| Thule III; P.I. 181463, BW Bt 13 | B13 | 14 |
| Doubi, DW | B14 | 15 |
| Carlton, DW | B15 | 16 |

*Source of differential varieties seeds is Dr. B. Goates (USDA-ARS), Aberdeen, Idaho, USA.
Pathogenic divergence of Tilletia tritici and T. leavis isolates - the causal agents of wheat common bunt disease in Iraq.

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, Sulaimania, and Duhok, tow isolates from Erbil, and one isolate from each of Salah El-Din, Baghdad, Diala, Wasit, Dewania, Garman, and Misan 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°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 isolates 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 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).

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

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 Bf1, 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, B9, 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 3 Host parasite interactions of differential varieties with 18 isolates of common bunt pathogens collected from different agro-ecological zones of Iraq.

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

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 similarity 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/Mnawia 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 Garman/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/Feshkhabor respectively were considered as new races according to non similarity in their reaction pathway with all the identified world races.

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; Oncia & 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; Liutukas & Ruzgas, 2008).
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 for them could be new races not recorded in the world. This 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 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.

**References**

Al-Maaroof EM, Hussein AK, Lateef MM, Fayyad FA(2004) Status and distribution of wheat bunt disease in Iraq. Arab Journal of Plant Protection 23: 127–131.

Al-Maaroof EM, Shams-Allah SA, Hassan MS (2006) Current status of wheat bunt disease in Iraq. Czech Journal of Genetics and Plant Breeding 42: 45-50.

Blázková V, Bartoš P (2002) Virulence pattern of European bunt samples (*Tilletia tritici* and *T. leavis*) and sources of resistance. Cereal Research Communications 30:335-342.

Chauhan RS, Sood AK, Singh BM (1994) Relative aggressiveness of new virulences of *Tilletia foetida* and *T. carries* on wheat cultivars. Indian Phytopathology 47: 232-235.

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| Location          | Isola | Germination | Pathogen          | Virulence/Avirulence against Bt | Race               |
|-------------------|-------|-------------|-------------------|--------------------------------|--------------------|
| Baghdad/Twaitha   | T1    | +++         | *T. tritici* +*T. leavis* | 1,4,6,15 / 2,3,5,7,8,9,10,11,12,13,14 | (T18) Probable new |
| Dilia/Bladroze    | T2*   | ++          | *T. tritici*      | 7,14 / 1,2,3,4,5,6,8,9,10,11,12,13,15 | (T1) Probable new race |
| Dilia/Bladroze    | T2    | +           | *T. tritici* +*T. leavis* | 5,6,14 / 1,2,3,4,7,8,9,10,11,12,13,15 | Proposes new race   |
| Wasit/Sheikh Saad | T3    | ++          | *T. tritici* +*T. leavis* | 1,3,10,14 / 2,4,5,6,7,8,9,11,12,13,15 | Proposes new race   |
| Mesan/ Ali Garubi  | T4    | ++          | *T. tritici* +*T. leavis* | 1,2,15 / 1,2,3,4,5,6,7,8,9,10,11,12,13,15 | Proposes new race   |
| Dewania/ Mhawia    | T5    | ++          | *T. tritici* +*T. leavis* | 7 / 1,2,3,4,5,6,8,9,10,11,12,13,14,15 | T1L1L2              |
| Salahdin/Bjee      | T6    | ++          | *T. tritici*      | 6,15 / (1 – 5, 7- 14) | Proposes new race   |
| 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 | T11                |
| Mosul/Rabiaa       | T8    | ++          | *T. tritici*      | 2,14,15 / 1,3,4,5,6,7,8,9,10,11,12,13,15 | (T14) Probable new |
| Mosul/Sherqat      | T9    | +++         | *T. tritici*      | 1,2,15 / 1,2,3,4,5,6,7,8,9,10,11,12,13,15 | T20                |
| Sulaimania/ Halabja| T11   | ++          | *T. tritici*      | 2,4,15 / 1,3 (4 – 14) | (T17) Probable new |
| 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*      | 6,10,15 / 1,2,3,4,5,7,8,9,11,12,13,14 | Proposes new race   |
| Erbil/ Kwsanjq     | T15   | ++          | *T. tritici*      | 5,6 / 1,2,3,4,5,7,8,9,10,11,12,13,14,15 | (T9) Probable new race |
| Duhok/ Zako        | T16   | +++         | *T. tritici*      | 1,6 / 2,3,4,5 (7 – 15 ) | (T2,T4) Probable new |
| Duhok/ Malta       | T17   | +++         | *T. tritici* +*T. leavis* | 5,8,9,14 / 1,2,3,4,6,7,10,11,12,13,15 | Proposes new race   |
| Duhok/ Feshkhabor  | T18   | +++         | *T. tritici* +*T. leavis* | 3,8,11,2,4,5,6,7,9,10,11,12,13,14,15 | Proposes new race   |
Dariaee A, Biglar HG, Haghparast R (2006) Identification of new wheat common bunt pathotypes (Tilletia laevis Kuhn.). Communications in Agricultural and Applied Biological Sciences 71:1093-1101.

Finci S (1981) Studies on the Pathogenic Races of Tilletia foetida and Tilletia caries and their Pathogenicity on Some Wheat Varieties in Turkey. EPPO Bulletin 11: 77–82. doi: 10.1111/j.1365-2338.1981.tb01769.x

Fisher GW, Holton CS (1957) Biology and control of smut fungi. Ronald press, New York. Pp 622.

Gaudet DA, Leggett F, Lu ZX, Frick M, Puchalski B, Despins T, laroche A (2006) Compatible and incompatible interactions involving the Bt10 gene in wheat for resistance to Tilletia tritici, the common bunt pathogen. Proceeding of the 15th biennial Workshop on the smut fungi held on June 11-14, 2006 at Prague, Czech republic.

Goates BJ (1996) Common bunt and dwarf bunt. In: Wilcoxon RD, Saari EE (Eds.) Bunt and smut diseases of wheat: Concepts and methods of disease management. CIMMYT, Mexico DF., Pp 12-25.

Hoffman JA, Metzger RJ (1976) Current status of virulence genes and pathogenic races of wheat bunt fungi in north western USA. Phytopathology 66: 657-660.

Ismail SF, Mamluk OF, Azmeh MF (1995) New pathotypes of common bunt of wheat from Syria. Phytopathologia Mediterranea 34:1-6.

Jones GD, Clifford BC (1978) Cereal Diseases: Their Pathology and Control. BASF United Kingdom Limited, Agrochemical Division Ipswich, Suffolk. Pp 279.

Kayali M, Al-Ahmad A, Nachit M, Yahyaoui A (2011) Determining Pathogen Races and Resistant Genes According to Reaction on Differential Varieties under Syrian Conditions. Journal of Jordan in Agricultural Sciences 7:711-720.

Kollmorgen JF, Allen JV, Hess WM, Trione EJ (1978) Morphology of primary spordial development in Tilletia caries. Transactions of the British Mycological Society 71:223-229.

Liatukas Z, Ruzgas V (2008) Resistance genes and sources for the control of wheat common bunt (Tilletia tritici (DC.) Tul.). Biologija 54: 274-278.

Lripp PE, Dorrance AE., Rhods LH., La Barge G (2000) Seed and soil-borne diseases of field crop. Seed treatment for agronomic crops, The Ohio State University Bulletin Pp 639-98.

Braun HJ, Altay F, Kronstad WE, Beniwal SOS, McNab A (1997) Wheat: Prospects for global improvement. Proceedings of the 5th International Wheat Conference, 10–14 June, 1996, Ankara, Turkey, Kluwer Academic Publishers, Dordrecht, Netherlands.

Mamluk OF, Nachit MM (1994) Sources of Resistance to Common Bunt (Tilletia foetida and T. caries) in Durum Wheat. Journal of Phytopathology 142: 122–130. doi: 10.1111/j.1439-0434.1994.tb04522.x

Ittu M, Saulescu NN, Ittu G (2006) Latest in breeding for resistance to common bunt in Romania. Proceeding of the XVth Biennial Workshop on the Smut Fungi held on June 11-14, 2006 at Prague, Czech Republic available on http://www.cazv.cz/service.asp?act=print&val=58193.

McManus PS, Ravencroft AY, Fulbright DW (1993) Inhibition of Tilletia laevis Teliospore germination and suppression of common bunt of wheat by Pseudomonas fluorescens 2-79. Plant Disease 77:1012-1015.

Metzger RJ, Hoffman JA (1978) New Races of Common Bunt Useful to Determine Resistance of Wheat to Dwarf Bunt. Crop Science 18: 49-51 doi:10.2135/cropsci1978.0011183X001800010013x.

Noruzi Z, Mahinpoor V, Fayazi F, Mohamedi M., Jalali F, Sohilinejad A, Darvishnia M (2012) Identification of pathogenic races of wheat common bunt using differential lines in Lorestan province. Agricultural Science and Technology 4:154-158.

Oncica F, Saulescu N (2008) Potentially new sources of genes for resistance to common Bunt (Tilletia spp) in winter wheat (Triticum aestivum L.) proceedings of the Romanian Academy, series B.1 :97-100.

Shams-Allah SA (2005) Biological and protectional studies on covered smut disease of wheat in Iraq. MSc Thesis submitted to the council of the College of Agriculture-University of Baghdad Pp 93.

Wang S, Knox RE, DePauw RM, Clark JM (2006) Markers to and chromosomal location of the Bt12 common bunt resistance gene in common wheat. Proceeding of the XVth Biennial Workshop on the Smut Fungi held on June 11-14, 2006 at Prague, Czech Republic available on http://www.cazv.cz/service.asp?act=print&val=58193