# Current Status of Virulence Genes and Pathogenic Races of the Wheat Bunt Fungi in the Northwestern USA

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ABSTRACT

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The classification of pathogenic races of the wheat bunt fungi is expanded to accommodate additional virulence genes and new combinations of virulence genes. Based on differential reactions conditioned by 10 bunt resistance genes, seven new pathogenic races each of *Tilletia caries* and of *T. controversa* were identified from field collections, artificially-produced hybrids, or teliospore reselections. The current race classification, comprising the three bunt pathogens on wheat, includes 39 distinct pathogenic types, combining up to eight genes for virulence. Prevalence of races of the dwarf bunt fungus having virulence genes effective against the combined 'Martin', 'Turkey', and 'Ridit' resistance and against two of the bunt resistance genes from P.I. 178383 indicates the need for a broader genetic base of bunt resistance in wheat for the northwestern USA.

*Additional key words: Triticum aestivum,* smut, host-parasite relations.

An integrated program of control using resistant cultivars and seed treatment fungicides, effective against both seed- and soil-borne inoculum, has nearly eliminated common bunt [caused by *Tilletia caries* (DC.) Tul. and *T. foetida* (Wallr.) Liro] from wheat (*Triticum aestivum* L.) in the northwestern USA. On the other hand, in the absence of effective seed treatments, broadly virulent races of the dwarf bunt fungus, *T. controversa* Kühn, continue to cause occasionally severe losses to wheat in this region.

Some time has elapsed since comprehensive reports on races of the wheat bunt pathogens have been presented (4, 6). Two new races each of the common bunt and dwarf bunt fungi were reported recently (3, 5, 9). Subsequently, additional genes for virulence have been detected in teliospore field collections and previously described races by including cultivars having new resistance genes (11, 13) in the set of wheat differentials. Likewise, new combinations of virulence genes continue to appear in the bunt populations in response to the selective action of wheats having new bunt resistance genes or gene combinations. Also, new pathogenic types have been produced by artificial hybridization and by repeated selection of teliospores from differential wheat cultivars. Here we report the current status of virulence in the bun fungi on wheat in the northwestern USA.

## MATERIALS AND METHODS

Teliospores of *T. caries, T. foetida,* and *T. controversa* were collected from commercial wheat fields and from experimental wheat nurseries in the western USA. Teliospores for subsequent tests of certain collections were obtained from appropriate differential wheat cultivars. Whenever possible, teliospores used as inoculum were from a single smutted head.

The differential wheat cultivars and the resistance gene carried by each were: Albit, C.I. 8275, *Bt1*(M1); Selection PS60-1-1075 (Elgin × Selection 1403), *Bt2*(H); Ridit, C.I. 6703, *Bt3*(rd); Turkey, C.I. 1558, *Bt4*(T); Selection R60-3432 (Elgin × Hohenheimer), *Bt5*(Ho); Rio, C.I. 10061, *Bt6*(R); Selection 50077, C.I. 13561, *Bt7*(M2); Yayla *305,* P.I. 178210, *Bt8;* Selection M69-2073 (Elgin × C.I. 7090), *Bt9;* and Selection M69-2094 (Elgin × P.I. 178383), *Bt10.* Hybrid 128, C.I. 4512, having no known bunt resistance Hybrid 128, C.I. 4512, having no known bunt resistance genes, was included as the universal susceptible host. Other cultivars and selections having the same bunt resistance genes, singly or in combination, were used in some tests. Plant Introductions P.I. 178383, P.I. 173438, and P.I. 166921 were included in most tests because they are resistant to all known bunt races.

In tests with common bunt, seed of the differential cultivars were inoculated with teliospores suspended in 5% methyl cellulose (15 centipoise). The seed were planted in fall in 1.5 m rows (100 seed/row) in duplicate nurseries at Pullman, Washington and Pendleton, Oregon.

Nongerminated teliospores of the dwarf bunt fungus applied to seed usually do not produce infection. Therefore, in tests with dwarf bunt, seed were inoculated with germinating teliospores and subsequently handled as described previously (4). Following inoculation and vernalization, seedlings were transplanted in the spring to replicated field nurseries at Pullman, Washington.

Percentages of bunt infection were determined by head counts. In terms of virulence of the pathogen, infection percentages from 0-10 were classified as avirulent; those

from 11-100 were classified as virulent.

## RESULTS

**Common bunt.**—Seven new races of *T. caries* were identified (Table 1). Four of the new races were obtained from artificially produced hybrids. One race, designated T-21, originated from a pairing of compatible, haploid cultures of races T-18 and T-8 inoculated onto Red Bobs, C.I. 6255. The $F_1$ teliospores were used as inoculum to produce $F_2$ teliospores on the differential wheat cultivars. Teliospores from a single smutted head of Albit were used to inoculate the differential cultivars to produce $F_3$ teliospores. Subsequent reselection of teliospores from Selection 50077 and Omar, C.I. 13072, yielded a stable segregate having virulence effective against *Bt1, 2, 4, 6,* and *7*. Thus, this new race of *T. caries* is like race T-18 except that race T-21 has virulence, presumably derived from the T-8 parent, effective against the 'Hussar' resistance gene, *Bt2*.

Another new race of *T. caries,* presumed to have originated from the cross T-18 × T-8, is designated T-22. As with T-21, $F_1$ spores were produced on Red Bobs and subsequent spore generations obtained by repeated inoculation of and reselection from the differential cultivars. Like race T-21, T-22 has virulence effective against *Bt2, 4, 6,* and *7*. But, unlike T-21 and both parent races (T-18 and T-8), T-22 lacks virulence on cultivars having the *Bt1* gene. Its origin as a hybrid of T-18 × T-8, therefore, seems questionable.

A new pathogenic race of *T. caries* was recovered from inoculation with paired monosporidial lines of T-13 and L-8. This race, designated T-23, has virulence effective against the *Bt1, 2, 4, 6, 7,* and *9* resistance genes. Thus, pathogenically, it is like race L-8 except for having the additional virulence effective against *Bt1,* which was presumably derived from the T-13 parent.

A new race of *T. caries,* designated T-24, was recovered from an inbred line of race T-16. This race has virulence effective against *Bt2, 3, 4, 6,* and *7*. It is the first race of the common bunt fungi to be identified that attacks cultivars having the combined 'Ridit' and 'Turkey' resistance genes, *Bt3* and *4,* respectively.

Inoculation of Selection W62-35, a derivative of Hohenheimer × Elgin, with race T-5 produced teliospores which, following subsequent inoculation of and reselection from the differential cultivars, yielded two new races of *T. caries*. One of these races, designated previously as race X-1 (9), is here redesignated race T-25. Like race T-5, it has virulence effective against *Bt1* and *7* but, in addition, it is virulent also on cultivars having the *Bt10* gene. The second new race derived from T-5 is designated T-26. Its virulence is like that of race T-25, except that T-26 is virulent also on cultivars having the *Bt2* gene.

Inoculation of Selection W62-35 with a mixture of teliospores of races T-25 (X-1) and L-16 resulted in a new race of *T. caries* designated T-27. This race has virulence effective against *Bt1, 2, 4, 6, 7,* and *10,* and thus combines virulence genes from both T-25 and L-16.

A collection of *T. caries* from South Dakota (from V. A. Johnson) was determined to be a new pathogenic race and is designated race T-28. It is virulent on cultivars carrying genes *Bt2, 4, 6, 7,* and *9* and is thus

TABLE 1. Percentages of infection[a] produced by new pathogenic races of *Tilletia caries* on 10 differential wheat cultivars carrying single bunt resistance genes

| | | Resistance genes | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Race | Origin | *Bt1* | *Bt2* | *Bt3* | *Bt4* | *Bt5* | *Bt6* | *Bt7* | *Bt8* | *Bt8* | *Bt10* |
| T-21 | T-18 × T-8 | 60 | 90 | 0 | 85 | 0 | 95 | 90 | 0 | 0 | 5 |
| T-22 | T-12 × T-8 ? | 3 | 98 | 2 | 95 | 0 | 95 | 97 | 0 | 0 | 3 |
| T-23 | T-13 × L-8 | 95 | 98 | 0 | 75 | 0 | 90 | 90 | 0 | 97 | 0 |
| T-24 | T-16 inbred line | 0 | 95 | 50 | 95 | 0 | 95 | 97 | 0 | 0 | 0 |
| T-25 | (X-1) T-5 reselection | 85 | 5 | 5 | 0 | 0 | 0 | 90 | 5 | 0 | 90 |
| T-26 | T-5 reselection | 95 | 90 | 5 | 3 | 0 | 0 | 65 | 0 | 0 | 75 |
| T-27 | X-1 + L-16 | 95 | 90 | 2 | 95 | 1 | 95 | 97 | 0 | 1 | 95 |
| T-28 | South Dakota (CT804) | 0 | 95 | 6 | 95 | 5 | 97 | 90 | 2 | 90 | 6 |

[a]0-10% infection is considered an avirulent reaction; 11-100% infection is considered a virulent reaction.

TABLE 2. Percentages of infection[a] produced by new pathogenic races of *Tilletia controversa* on 10 differential wheat cultivars carrying single bunt resistance genes

| | | Resistance genes | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Race | Distribution | *Bt1* | *Bt2* | *Bt3* | *Bt4* | *Bt5* | *Bt6* | *Bt7* | *Bt8* | *Bt9* | *Bt10* |
| D-1 | Nephi, UT[b]; Preston, ID; Bozeman, MT | 0 | 5 | 6 | 4 | 80 | 1 | 80 | 3 | 0 | 70 |
| D-11 | Southeastern WA; Camas Co., ID | 0 | 8 | 3 | 35 | 80 | 80 | 80 | 2 | 5 | 90 |
| D-12 | Southeastern WA; northwestern ID; northeastern OR | 75 | 2 | 8 | 85 | 70 | 98 | 85 | 2 | 1 | 65 |
| D-13 | Southeastern WA; northeastern ID; northeastern OR | 85 | 90 | 10 | 75 | 80 | 95 | 85 | 5 | 4 | 90 |
| D-14 | Southeastern ID; northern UT | 5 | 95 | 95 | 95 | 70 | 98 | 85 | 4 | 6 | 55 |
| D-15 | Southeastern ID; northern UT; Moffat Co., CO | 85 | 98 | 75 | 80 | 60 | 95 | 90 | 10 | 3 | 75 |
| D-16 | Southeastern ID; northern UT | 90 | 98 | 75 | 5 | 10 | 6 | 85 | 7 | 95 | 8 |
| D-17 | Southeastern ID; northern UT | 0 | 95 | 90 | 3 | 35 | 3 | 85 | 1 | 95 | 3 |

[a]0-10% infection is considered an avirulent reaction; 11-100% infection is considered a virulent reaction.
[b]U.S. postal zip code letter pairs CO, ID, OR, UT, and WA refer to Colorado, Idaho, Oregon, Utah, and Washington, respectively.

pathogenically like race L-8 of *T. foetida*.

**Dwarf bunt.**—Results obtained from inoculation of the expanded set of differential cultivars have identified seven new races of *T. controversa* (Table 2) and have necessitated the revision of the virulence pattern of one race reported previously.

All teliospore collections of the dwarf bunt fungus previously (5) assigned to race D-1 were determined (Table 2) to express virulence also against *Bt10*. Therefore the virulence pattern of race D-1 is revised to include virulence against *Bt10* in addition to *Bt5* and *7*. Race D-1 has been collected infrequently near Nephi, Utah, Preston, Idaho, and Bozeman, Montana.

Certain collections of *T. controversa* from southeastern Washington and Camas County, Idaho, originally identified as race D-2 (5), also exhibit virulence against *Bt10*. Other collections assigned to race D-2 are avirulent on cultivars with this gene. To distinguish between these different reaction patterns, a new race category, D-11, is assigned to those collections having virulence effective against *Bt10*. Likewise, some collections of races D-5, D-6, D-7, and D-9 were determined (Table 2) to have virulence effective against *Bt10*. New races are designated to accommodate these types: collections having virulence like D-5 and, in addition, expressing virulence against *Bt10* are designated D-12; those having virulence like D-6, D-7, or D-9, and in addition, expressing virulence against *Bt10,* are designated D-13, D-14, or D-15, respectively.

Certain teliospore collections of races D-8 and D-10 were found to express virulence against *Bt9*. These collections having virulence like D-8 or D-10 and also expressing virulence against *Bt9* are designated D-16 or D-17, respectively.

Based on frequency of collection, race D-13 is the predominant race of *T. controversa* in the northwestern USA. It is particularly prevalent in southeastern Washington and in the contiguous areas of Idaho and Oregon. Races D-5, D-6, and D-12 are frequently collected in the same areas. Races D-7, D-8, D-9, D-10, D-14, D-15, D-16, and D-17 occur mostly in southeastern Idaho, and northern Utah. Races D-7 and D-15 have been collected also in Moffat County, Colorado.

## DISCUSSION

Control of the bunt diseases of wheat through use of resistant cultivars requires a sustained and systematic breeding program. Such a program depends on the continuous acquisition of information concerning the current and potential pathogenic capabilities of the bunt pathogens.

An up-to-date classification of races of the bunt fungi on wheat in the northwestern USA is presented in Table 3. This classification, patterned after systems adapted for the rust fungi (2, 8) uses standard race designations augmented by race formulae emphasizing virulence of the pathogen. It now comprises 39 distinct reaction types and includes races that combine up to eight genes for virulence.

The use of cultivars that combine several genes for resistance or that utilize new sources of resistance present new thresholds upon which new pathogenic types can be expected to arise (1). This is illustrated in the ascendance of races virulent against cultivars with combinations of the *Bt1* ('Martin'), *Bt4* ('Turkey'), and *Bt3* ('Ridit') resistance genes. Winter-wheat cultivars utilizing this resistance and still grown in the northwestern USA include Nugaines, Paha, Hyslop, Wanser, Bridger, and Colorow. All are now susceptible to several races of the bunt pathogens.

Recently developed wheat cultivars resistant to all known bunt races are Luke, Ranger, Franklin, Jeff, Cardon, and Hansel. All of these derive their bunt resistance from P.I. 178383, a wheat selection from Turkey (7). This selection contains *Bt9* and *Bt10* and probably a third unidentified bunt resistance gene (10).

Results presented here and elsewhere (11, 13) indicate that virulence genes effective against *Bt9* and *Bt10* were present in the bunt population even before these resistance genes were utilized in the wheat breeding

TABLE 3. Races of *Tilletia caries* (T-races), *T. foetida* (L-races), and *T. controversa* (D-races) determined by pathogenicity on 10 bunt resistance *(Bt)* genes in wheat

| Race designation | Virulence formula (virulence/avirulence against *Bt* genes) |
|---|---|
| T-14 | *1/2,3,4,5,6,7,8,9,10* |
| T-10 | *5/1,2,3,4,6,7,8,9,10* |
| T-1, L-1, L-2 | *7/1,2,3,4,5,6,8,9,10* |
| T-20 | *1,2/3,4,5,6,7,8,9,10* |
| T-2,T-4, T-6, L-4 | *1,7/2,3,4,5,6,8,9,10* |
| T-11 | *2,3/1,4,5,6,7,8,9,10* |
| T-3, L-3 | *2,7/1,3,4,5,6,8,9,10* |
| T-9 | *5,7/1,2,3,4,6,8,9,10* |
| T-13, L-9 | *1,2,3/4,5,6,7,8,9,10* |
| T-5, T-7, T-8, L-5, L-6, L-7 | *1,2,7/3,4,5,6,8,9,10* |
| T-12 | *1,5,7/2,3,4,6,8,9,10* |
| T-25 | *1,7,10/2,3,4,5,6,8,9* |
| L-10 | *2,3,7/1,4,5,6,8,9,10* |
| T-17 | *2,4,6/1,3,5,7,8,9,10* |
| D-1 | *5,7,10/1,2,3,4,6,8,9* |
| T-19, D-8 | *1,2,3,7/4,5,6,8,9,10* |
| T-15, D-4 | *1,2,5,7/3,4,6,8,9,10* |
| T-26 | *1,2,7,10/3,4,5,6,8,9* |
| T-18 | *1,4,6,7/2,3,5,8,9,10* |
| D-10 | *2,3,5,7/1,4,6,8,9,10* |
| T-22 | *2,4,6,7/1,3,5,8,9,10* |
| D-2 | *4,5,6,7/1,2,3,8,9,10* |
| D-16 | *1,2,3,7,9/4,5,6,8,10* |
| T-21, L-16 | *1,2,4,6,7/3,5,8,9,10* |
| D-5 | *1,4,5,6,7/2,3,8,9,10* |
| T-24, D-7 | *2,3,4,6,7/1,5,8,9,10* |
| D-17 | *2,3,5,7,9/1,4,6,8,10* |
| T-16 | *2,4,5,6,7/1,3,8,9,10* |
| T-28, L-8 | *2,4,6,7,9/1,3,5,8,10* |
| D-11 | *4,5,6,7,10/1,2,3,8,9* |
| D-3 | *1,2,3,4,6,7/5,8,9,10* |
| D-6 | *1,2,4,5,6,7/3,8,9,10* |
| T-23 | *1,2,4,6,7,9/3,5,8,10* |
| T-27 | *1,2,4,6,7,10/3,5,8,9* |
| D-12 | *1,4,5,6,7,10/2,3,8,9* |
| D-14 | *2,3,4,6,7,10/1,5,8,9* |
| D-9 | *1,2,3,4,5,6,7/8,9,10* |
| D-13 | *1,2,4,5,6,7,10/3,8,9* |
| D-15 | *1,2,3,4,5,6,7,10/8,9* |

program. Several races have now been identified that attack cultivars with *Bt9* or *Bt10* individually. Moreover, unconfirmed results suggest the existence of races which have virulence effective against the combined *Bt9* and *Bt10* resistance. Thus, genes other than *Bt9* and *Bt10* will likely determine the continued usefulness of P.I. 178383 as a source of bunt resistance. In any case, it would seem prudent that new, diverse sources of bunt resistance be used in the wheat-breeding programs in the northwestern USA.

Studies on the genetics of pathogenicity of the bunt fungi have not kept pace with similar studies in other host-parasite systems. This is due, in part, to the failure of most laboratory-produced bunt hybrids to sporulate profusely in either the universal-susceptible or differential host cultivars. Assuming that the gene-for-gene concept is applicable (12), it would be expected that any two races virulent on the same host differential would carry the critical gene for virulence in common. An $F_1$ hybrid and subsequent generations should be virulent on the universal susceptible host and on the differential hosts that are susceptible to both parental races. Failure of artificially produced hybrids to sporulate suggests a lack of vigor or aggressiveness rather than a lack of virulence. Consequently, large numbers of hybrid combinations must be tested to find the few vigorous hybrids with the desired virulence.

In spite of increased virulence, new races of *T. caries* or *T. foetida* are not likely to limit wheat production as long as effective seed treatment chemicals remain available and are judiciously used. On the other hand, dwarf bunt is not controlled by presently registered seed treatment chemicals. Consequently, new and more broadly virulent races of *T. controversa* pose serious threats to the production and marketing of wheat in the northwestern USA.

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