

Wheat screening for resistance to common bunt and dwarf bunt

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Abstract

A wide range of wheat genotypes were tested from 2009 to 2012 for resistance to common and dwarf bunt in artificial inoculation tests. Highly resistant (0% diseased spikes) and resistant (<10% diseased spikes) common wheat varieties were identified. The highest incidence of common bunt was observed in 2009 for variety Pitbull (86% diseased spikes). Dwarf bunt infection was low in all years. No infection was observed in genotypes carrying *Bt11*, *Bt12* and *Bt13*.

Keywords

Tilletia controversa, *Tilletia laevis*, *Tilletia tritici*, *Triticum aestivum*

Introduction

Common bunt and dwarf bunt are cereal fungal diseases causing severe economic losses worldwide, whenever use of resistant varieties, seed treatment or bunt limiting cultural practices are abandoned. Chemical treatment of the seed is widely used to control bunt, however, it is not allowed under organic farming conditions. Resistant varieties may reduce the losses due to bunt drastically. Infection levels in highly susceptible cultivars exceed 80% of diseased spikes, while in highly resistant cultivars only 0% diseased spikes are observed.

Common bunt is caused by *Tilletia caries* (D. C.) Tul. & C. Tul. (*syn. T. tritici* (Bjerk.) G. Winter and *T. laevis* J. G. Kühn (*syn. T. foetida* (Wallr.) Liro, dwarf bunt by *T. controversa* J. G. Kühn. The three morphologically distinct bunt species are genetically very closely related, to the extent that genes for bunt resistance are identical. Although numerous studies did not succeed to differentiate the three species at the genetic level using molecular techniques, GAO et al. (2011) distinguished *T. controversa* based on a diagnostic molecular marker generated from intersimple sequence repeat (ISSR). Despite the close relationship of the three pathogens, common bunt and dwarf bunt diseases differ in etiologies and climatic requirements.

Material und Methods

A wide range of wheat varieties (Tables 1-3) were tested from 2009 to 2012. Common bunt inoculation was done by shaking seed with a surplus of teliospores in Erlenmeyer flasks for 1-2 min. Inoculation and sowing (1 m long rows, 4 replications) was carried out in early October.

For dwarf bunt tests 1 m long rows with 8-10 replications were sown. Teliospores were evenly spread on the soil surface after sowing. In absence of a snow cover the plots in covered with straw or white nonwoven fabric.

Results and Discussion

Common bunt

The inoculum was a mixture of Czech isolates. Its virulence to *Bt1*, *Bt2*, *Bt7* genes was determined on a set of differential varieties carrying individual *Bt* genes. Genotypes with *Bt9*, *Bt10*, *Bt11* and *Bt12* genes were resistant to the inoculum mixture.

Different isolates were used occasionally. They can have different reactions on the same varieties and enable to estimate resistance genes as was proved in the test for Euris, 'Bussard', 'Nela', 'Mv 25' and Hadmerslebener 20037-88 using the *T. laevis* isolate from Praha-Ruzyně and the *T. tritici* isolate from Kroměříž. These genotypes were resistant to *T. tritici* and susceptible to *T. laevis*. They may carry gene(s) *Bt1* and/or *Bt2*, because the *T. laevis* isolate used in the test is virulent to *Bt1* and *Bt2*, whereas the *T. tritici* isolate is avirulent to *Bt1* and *Bt2*. Other differences between the isolates were not determined.

In our experiment including 9 isolates mostly of European provenience virulence to *Bt2* and *Bt7* prevailed, virulence to *Bt1* was frequent as well. In the Czech Republic virulence appeared also to *Bt3*, in Bulgaria to *Bt4* and *Bt6*, in Syria and Sweden to *Bt10*. *Bt* genes effective against the isolates tested were *Bt5*, *Bt8*, *Bt11* and *Bt12* (BLAŽKOVÁ and BARTOŠ 2002). Similar results were obtained by KUBIAK and WEBER (2008) and HUBER and BUERSTMAYR (2006). However, WÄCHTER et al. (2007) discovered additionally virulence to *Bt9* and *Bt13*. In comparison, in the USA a high number of international isolates as well as products of hybridization were studied in extensive tests. Recently, new races reached unusual high number of genes for virulence, even 9 genes for virulence may cumulate in a single race. Thus, only *Bt11* remains effective against all known races of common bunt to date, whereas a dwarf bunt race virulent to *Bt11* was already detected (GOATES 2012).

The majority of varieties registered in the Czech Republic showed to be susceptible to common bunt. Table 1 summarizes cultivars with a high bunt incidence (>40% diseased spikes). Table 2 comprises varieties with up to 40% diseased spikes. The highest bunt incidence was recorded in 2009 for 'Pitbull' (85.9%), 2010 for 'Aladin' (77.4%), 2011 for

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Table 1: Varieties with a high bunt incidence (>40% diseased spikes) in 2009-2012. Mean values are presented in brackets; genotypes in decreasing order.

2009	2010	2011	2012
Pitbull (85.9)	Aladin (77.4)	JB Asano (64.8)	Magister (66.3)
Barryton (85.5)	Federer (77.2)	Batis (63.1)	Citrus (65.6)
Federer (85.3)	Batis (66.8)	Athlon (61.7)	JB Asano (59.4)
Megas (80.4)	RW Nadal (64.5)	Jindra (61.6)	Caroll (58.3)
Bakfis (80.3)	Preciosa (64.4)	Federer (59.0)	Athlon (57.3)
Kodex (80.1)	Brentano (64.0)	Preciosa (54.3)	Henrik (52.9)
Raduza (79.7)	Magister (55.5)	Brentano (51.6)	Evina (51.6)
Mladka (73.8)	Fortis (54.5)	Altigo (49.5)	Fortis (51.4)
Hermann (73.0)	Henrik (54.4)	Graindor (48.3)	Altigo (49.4)
Bagou (71.9)	Seladon (48.6)	Salut (48.3)	Preciosa (46.4)
Kerubino (71.2)	Jindra (45.5)	Bodyček (47.8)	Elly (44.2)
Anduril (68.2)	Bagou (44.2)	Aladin (44.0)	Hewitt (44.1)
Batis (66.5)	Elly (43.2)	Magister (42.8)	Aladin (43.0)
Sakura (62.0)		Henrik (41.9)	Batis (42.2)
Sultan (61.8)			Graindor (41.9)
Seladon (61.5)			WO 4458 A-2 (41.4)
Manager (59.7)			Dulina (41.2)
Baletka (57.2)			
Bohemia (50.9)			
Orlando (45.7)			
Mulan (42.6)			

Table 2: Varieties with a low to medium incidence (<40% diseased spikes) in 2009-2012. Mean values are presented in brackets; genotypes in decreasing order.

2009	2010	2011	2012
Nikol (31.7)	Brilliant (39.4)	Caroll (38.8)	Salut (38.5)
Helmut (28.9)	Bodyček (38.2)	Bagou (36.8)	20817-3 (38.2)
Secese (26.6)	Secese (38.0)	Potenzial (34.9)	Bodyček (37.5)
Brilliant (19.8)	Iridium (35.2)	RW Nadal (34.6)	Potenzial (33.9)
Bill (8.1)	Hermann (31.0)	Manager (34.4)	Turandot (33.9)
Globus (4.1)	Salut (28.7)	Matylda (32.4)	Cimrmanova raná (32.6)
	Graindor (23.9)	WO 4458A-2 (30.6)	Chevalier (32.4)
	Manager (15.7)	Fortis (30.0)	Beduin (32.2)
	Globus (1.9)	Elly (28.8)	Hermann (30.0)
	Bill (1.5)	Elan (28.7)	Brentano (29.8)
		Seladon (25.1)	RW Nadal (29.1)
		20817-3 (24.0)	Jindra (29.0)
		Secese (23.9)	Fermi (25.8)
		Feria (22.7)	Feria (25.1)
		Sorrial (22.3)	KWS Ozon (23.5)
		Beduin (21.9)	Dagmar (22.7)
		Iridium (21.5)	Sj. 07-042 (22.1)
		Brilliant (20.2)	Sorrial (21.1)
		Hermann (12.0)	Elan (20.7)
		Globus (2.0)	Princeps (19.9)
		Bill (0.2)	Matylda (19.5)
			Tiguan (19.4)
			Iridium (19.2)
			Golem (10.5)
			Manager (10.4)
			Sailor (5.4)
			Bill (0.0)
			Globus (0.0)

‘JB Asano’ (64.8%) and 2012 for ‘Magister’ (66.3%). The susceptible check ‘Batis’ had 66.5%, 66.8%, 63.1% and 42.2% in 2009, 2010, 2011 and 2012, respectively. High resistance was detected in ‘Globus’ and ‘Bill’, however, relevant resistance genes were not characterized yet. The low bunt incidence on ‘Sailor’ in 2012 has to be still verified, to exclude underestimation of its bunt incidence due to a relatively weak infection pressure in 2012.

The presence of effective genes for resistance to common bunt in Czech varieties is rare. Therefore other materials were screened. In total, 46 of the varieties tested since 1995 in Praha-Ruzyně displayed high resistance to common bunt (no bunt incidence), 13 were resistant (0-10% of spikes diseased) (Table 3). Furthermore, results of 6 years of the European *Tilletia* cooperative test from Praha-Ruzyně may provide some additional potential sources of resistance.

Table 3: Genotypes with effective genes for resistance to common bunt

Highly resistant (0% diseased spikes)		Resistant (0-10% diseased spikes)	
Blizzard	Lewjain	Rio	Cardon
Amigo	M82-2123	Sel. M72-1250	Trintella
Bold	Magnifik	Sel. M72-345	Wasatch
Bonneville	Manning	Sel. R63-6982	Hildebrands Weissweizen B
Bruehl	Meridian	Sel. M65-2124	Weston
CI 14106	PI 119333	Sprague	Sel. M72-2501
Crest	PI 166910	Stava	Madsen
Deloris	PI 178383	SW 51136	Mikon
DW Red	PI 560601	Thule III (PI181463)	Šechurdinovka
Franklin	PI 178201	Tjelvar	Nebred
Gary	PI 178383	Tommi	Yayla 305 (PI 178210)
Golden Spike	PI560795 Sel. BCO	Turkey (CI1558)	Cardos
Hansel	PI 560841 Sel. BCL	Ute	Dobrovická přesívka
Hohenheimer	PI 560841 Sel. WCO	Winridge	
KW9403	Promontory		
KW9410	Ridit		

The European winter wheat variety ‘Trintella’ was investigated for the location of bunt resistance genes using a doubled haploid mapping population of a cross with the susceptible variety ‘Piko’. The population was scored for bunt infection in the field for two years following inoculation with a mixture of teliospores of *T. tritici* and *T. laevis*. A genetic map of 29 linkage groups was constructed using polymorphic simple sequence repeat (SSR) markers. This map was used for QTL analysis and in both years it was evident that resistance to common bunt was due to a gene on chromosome 1BS, near the centromere, close to marker *Xgwm273*. Additionally, in 2008, small effects on resistance were ascribed to chromosomes 7A and 7B. Another small effect was ascribed to chromosome 5B in 2009 only (DUMALASOVÁ et al. 2012). ‘Winridge’ and PI 166910 from the group of highly resistant varieties were further evaluated in the field for *Fusarium* head blight (FHB) resistance under high infection pressure. Data on deoxynivalenol (DON) content were supplemented by symptom scores and the determination of the percentage of *Fusarium* damaged grains and the relative reduction of grain weight per spike. Both genotypes showed reaction similar to the moderately resistant checks ‘Arina’ and ‘Petrus’, however, with a significantly lower DON content (CHRPOVÁ et al. 2012). Common bunt resistance was also tested in durum, spelt and emmer wheat. Materials that seemed to be resistant in the field often showed susceptibility in the greenhouse under optimal conditions for infection. However, susceptible durum, spelt and emmer wheats did not reach as high bunt incidence as susceptible spring wheat check ‘Vinjett’. Dehulling of spelt and emmer wheat increased the level of infection after inoculation (DUMALASOVÁ and BARTOŠ 2010).

Dwarf bunt

Seven genotypes carrying different *Bt* genes effective to common bunt were tested in seedbeds. Dwarf bunt usually contains a higher number of genes for virulence than common bunt. This is why varieties resistant to dwarf bunt have to contain more resistance genes, corresponding to virulence genes, than varieties resistant to common bunt. Therefore, varieties resistant to dwarf bunt are usually resistant also to common bunt.

The level of dwarf bunt infection was very low in the test, 4.9% *Bt7*, 0.3% *Bt8*, 0.1% *Bt9*, 0.7% *Bt10*. It is impossible to draw conclusions from these data, though we noticed no infection on genotypes carrying *Bt11*, *Bt12* and *Bt13*.

Several authors described ‘Blizzard’, ‘Bonneville’, ‘Golden Spike’, ‘Gary’, ‘Winridge’, ‘Wasatch’, ‘Weston’ and ‘Sprague’ to carry effective resistance to dwarf bunt. These varieties proved to be resistant also to common bunt in our trial. Moreover, ‘Winridge’ is moderately resistant to FHB. Genotypes carrying *Bt11*, which is effective against the majority of common bunt and dwarf bunt races, were not used in resistance breeding programs so far (GOATES 2012).

Triticale is supposed to be a dwarf bunt host. In our tests with artificial soil infection, six triticale cultivars appeared to be resistant compared to the susceptible check ‘Batis’. Only one variety was infected with dwarf bunt, and even in this case the infection did not exceed 10%. Triticale varieties showed resistance to common bunt in our field tests. In total 17 varieties were tested.

Conclusions

Deliberate breeding for bunt resistance was performed in the USA, Canada, Ukraine, Sweden, Romania and other countries. Moreover, some level of resistance to common bunt occurred in European varieties (e.g. Tommi) unintentionally, which indicates, that it is possible to combine resistance and agronomically important traits.

Sources of resistance may be found also in wild relatives of wheat (e.g. *Aegilops* spp., *Agropyron intermedium*, e.g. the *BtZ* gene). Resistance is based on major (*Bt* genes) and minor genes for quantitative resistance (QTL).

Up to now screening of genotypes for bunt resistance is mainly done in infection tests with artificial inoculation. Besides, molecular techniques were already employed, e.g. in the USA, Canada and Romania.

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