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Common resistance to different *Fusarium* spp. causing *Fusarium* head blight in wheat

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Abstract

Different sets of wheat genotypes were tested under field conditions by spraying inocula of isolates of seven *Fusarium* spp. and *Microdochium nivale* (formerly *F. nivale*) in the period 1998–2002. The severity of *Fusarium* head blight (FHB), *Fusarium*-damaged kernels (FDK), the yield reduction and the deoxynivalenol (DON) contamination were also measured to describe the nature of the resistance. The degrees of FHB severity of genotypes to *F. graminearum*, *F. culmorum*, *F. avenaceum*, *F. sporotrichioides*, *F. poae*, *F. verticillioides*, *F. sambucinum* and *M. nivale* were very similar, indicating that the resistance to *F. graminearum* was similar to that for other *Fusarium* spp. listed. This is an important message to breeders as the resistance relates not only to any particular isolate of *F. graminearum*, but similarly to isolates of other *Fusarium* spp. This holds true for all the parameters measured. The DON contamination refers only to DON-producers *F. graminearum* and *F. culmorum*. Highly significant correlations were found between FHB, FDK, yield loss and DON contamination. Resistance components such as resistance to kernel infection, resistance to DON and tolerance were identified in the more susceptible genotypes. As compared with western European genotypes which produced up to 700 mg kg^{-1} DON, the Hungarian genotypes produced only 100 mg kg^{-1} at a similar FDK level. This research demonstrates the importance of measuring both FDK and DON in the breeding and selection of resistant germplasm and cultivars.

Abbreviations: DON – deoxynivalenol; FHB – *Fusarium head blight;* FDK – *Fusarium-damaged kernel.*

Introduction

Fusarium head blight (FHB) is one of the most destructive diseases of small grain cereals, including wheat (Snijders, 1994; Parry et al., 1995; Miedaner, 1997; Leonard and Bushnell, 2003). The resulting yield and quality losses can be very high, but even more dangerous can be the toxin accumulation in diseased kernels following infection. As wheat can be invaded by many *Fusarium* spp., a wide range of toxins might be produced that could attain concentrations harmful for humans and animals. Surveys of *Fusarium* species occurring in infected grains or other parts of the head resulted in the detection of a number of *Fusarium* spp. (Mesterházy, 1984a; Liddell, 2003; Shaner, 2003). In most areas of the world *Fusarium graminearum* (*Gibberella zeae*) predominates, but *F. culmorum*, *F. avenaceum*, *F. sporotrichioides*, *F. poae* and *Microdochium nivale* may play significant roles in given areas. The coexistence of different *Fusarium* spp. in the same field is a normal situation. Several less aggressive species such as *F. sporotrichioides* and *F. poae*, can produce highly toxic agents; resistance to them is therefore also important (Miedaner, 1997).

Extensive studies have revealed that no true vertical races exist within *F. graminearum* and *F. culmorum* (Mesterházy, 1977, 1983, 1995; Snijders and van Eeuwijk, 1991; van Eeuwijk et al., 1995; Miedaner and Schilling, 1996; Mesterházy et al., 1999). Recent data from the USA (Bai and Shaner, 1996; Stack, 2003) support this. Most breeding programmes deal only with the dominant *Fusarium* species such as *F. graminearum* or *F. culmorum*. Therefore no detailed information is available as to whether the resistance to *F. graminearum* affords protection in their breeding material against other *Fusarium* spp. or not. The answer would influence the breeding strategy and methodology, and the reliability of resistance tests in other regions with different compositions of *Fusarium* spp. This non-specificity within *F. graminearum* and *F. culmorum* led to the idea that resistance to different *Fusarium* spp. might be common. The first indication that resistance to *F. avenaceum* (Mesterházy, 1977) could parallel that of the two main pathogens dates from 1977, but convincing evidence has not yet been achieved. For *M. nivale* the situation is the same (van Eeuwijk et al., 1995) as only one set of data was used and the correlations were only moderate. With the same genotypes as used by van Eeuwijk et al. (1995) Mesterházy (1995) found very similar resistance reactions against *F. graminearum* and *F. culmorum*; this held true for FHB, *Fusarium*-damaged kernels (FDK), yield loss and the degree of deoxynivalenol (DON) contamination.

Few studies have investigated the resistance to different *Fusarium* spp. Klechkovskaya (1997) tested the resistance to single isolates of five different *Fusarium* spp. The correlation coefficients varied between 0.48 and 0.52 for the yield, and between 0.50 and 0.65 for the infection severity, all significant at $P < 0.05$, but not sufficiently close to prove common resistance. Stack et al. (1997) demonstrated similar type II resistance to seven *Fusarium* spp. on 5 genotypes of Sumai 3 (synonyms: Sumey-3, Soo Moo 3, Sumai 3, Sumai-3) origin in the greenhouse (FHB, FDK and DON data). Resistance to *F. graminearum* also gave protection against *F. avenaceum*, *F. poae* and *F. sporotrichioides*. *F. acuminatum* and *F. equiseti* were non-pathogenic. This referred only to the Sumai 3 resistance, and it is not known whether this finding is of general significance as the number of entries was also moderate. Hollins et al. (2003) presented one-year results indicating that the resistance to different *Fusarium* spp. might be common. Diamond and Cooke (1999) reported a close relationship between the resistance of wheat heads to *F. culmorum* and the leaf resistance to *M. nivale*.

Experimental evidence for species non-specificity originates mostly from tests in which *Fusarium* spp. or *M. nivale* were represented by single isolates; in some cases merely the results for a single year were considered and often only FHB symptoms were analysed. These results permit an hypothesis of common resistance, but do comprise experimental proof. Accordingly, a 5-year programme was started in 1998 to check the hypothesis for the common resistance of wheat against various *Fusarium* spp. The test series was made in the field; spraying inoculation was used. FHB, FDK, yield performance and DON contamination were tested. The results allow conclusions concerning the resistance types described by Schroeder and Christensen (1963) and Mesterházy (1995). They are different from the resistance types described by Browne and Cooke (2004) as they tested, among others, latent period which was not the case in the present paper. Thus a clearer picture emerged on the relations between the resistances of cultivars to various *Fusarium* species. We additionally set out to characterize the pathogenic capacities of the *Fusarium* species involved, which has not been done previously.

Materials and methods

Genotypes

Wheat (*Triticum aestivum*) genotypes with different resistance levels and background were chosen, among them the spring-type resistance sources, our resistant lines, and moderately to highly susceptible genotypes. The tables and figures detail the genotypes used. In 1998 and 1999, 16 genotypes, in 2000, 13, and in 2001 and 2002, 26 genotypes were tested; western European cultivars and lines were also included.

Isolates

In 1998 and 1999, four *F. graminearum* and four *F. culmorum* isolates were used and several other

Fusarium isolates were added. From 2000, two isolates were included for each *Fusarium* spp. The isolates were monosporic lines of the original wild-type isolates. The isolates utilized in the testing period are listed in Table 1. Isolation was carried out on Papavizas (1967) medium. The isolates were identified with the aid of the Booth (1971) manual.

Inoculum preparation

Heat-stable glass flasks (10 l) fitted with glass tubes reaching to the bottom were filled with 9 l of Czapek-Dox medium and were autoclaved at 1.5 bar for 1.5 h (Mesterházy 1978). Air was passed through a sterilized cotton filter and bubbled through the medium at room temperature for 1 week. The conidium concentration was measured in a haemocytometer (Table 1) and contained conidia and mycelium; exact control of fungal biomass in the conidial concentration was therefore not possible. The inoculum was tested for aggressiveness and stored at 4 °C when used for experiments. The original isolates were stored in test tubes, potato dextrose agar under light mineral oil (Soltral 160) at room temperature.

Aggressiveness test

Aggressiveness has been defined as 'the relative intensity of disease' (Welz and Kranz, 1997). 'Pathogenicity describes the general quality or characteristic of a pathogen genus or species able to cause disease on a given host species' (Heitefuss, 1997). In this context, we measured the disease-causing capacity of individual entities of the pathogen (isolates, mixture of isolates, natural population, etc.). The Petri dish method of Mesterházy (1977) was applied as a preselection for field use. Mesterházy (1984b) observed good agreement between the aggressiveness of *F. graminearum* isolates in a Petri dish and in a field FHB test. The inoculum was tested at the original concentration and in dilutions of 1:1, 1:2 and 1:4 with distilled water; 9 ml inoculum was uniformly distributed in Petri dishes supplied with double layer filter paper (Filtrak-6, Germany). Twenty-five wheat germs of two cultivars with somewhat differing resistances in the seedling stage (line 74.2 as moderately resistant and cv. Várkony as susceptible) were used, with seeds in a 5×5 system with the germs upward. Coleoptiles were considered healthy if there was no discolouration and mycelial

Table 1. Isolates and their conidium concentrations ($\times 10^6$) used in the resistance trials in 1998–2002

Isolates	Origin	Years					Author
		1998	1999	2000	2001	2002	
F. acuminatum No. 14563[a]	wheat grain, Szeged, 1997		0.62				Á. Mesterházy
F. avenaceum a-17 , 1	wheat grain				0.43	1.76	T. Yli-Mattila
F. avenaceum No. 14683, 2	wheat grain, Szeged, 1999				1.11	0.78	Á. Mesterházy
F. culmorum, No. 12375	wheat root, Szeged, 1978	0.01	0.01		0.05	0.5	Á. Mesterházy
F. culmorum, No. 12551	wheat stalk base, Szeged, 1978	0.15	0.73	0.28	0.13	1.2	Á. Mesterházy
F. culmorum, No. 207/1	Suttgart			0.21			C. Kling
F. culmorum, No. 223[a]	Suttgart	0.15	0.01				C. Kling
F. graminearum No. 12377	Maize, Vésztő, 1977	0.01	0.20	0.15	0.28	0.36	Á. Mesterházy
F. graminearum No. 207	Suttgart	0.01	1.07				C. Kling
F. graminearum No. 40	IFA Tulln	0.05	0.67				P. Ruckenbauer
F. graminearum No. 44	IFA Tulln	0.01	0.27	0.01	0.01	0.01	P. Ruckenbauer
F. moniliforme No. 14585[a]	corn seed, Szeged, 1997	0.31					Á. Mesterházy
F. poae 14151, 1	1997			0.20	0.38		Á. Mesterházy
F. poae No. 14169, 2	1997			0.18	0.18		Á. Mesterházy
F. sambucinum No. 14593	1997	0.01	0.01		0.05	0.38	H. Kwasna
F. sporotrichioides No. 14593, 1	wheat grain, Szeged, 1997	0.38	0.68	0.58	0.68	0.8	Á. Mesterházy
F. sporotrichioides No. 15123, 2	wheat grain, Szeged, 1998			0.48	0.43	0.45	Á. Mesterházy
F. verticillioides No. 14598[a]	maize seed, Szeged, 1997	0.28	0.77				Á. Mesterházy
M. nivale 1/99, 1	Dublin			0.16			M. Cooke
M. nivale B6, 2	Dublin			0.83			M. Cooke

[a] These isolates were not evaluated together as no two years data were present in 1998/1999 and 2001/2002.

Table 2. Resistance of wheat genotypes against isolates of different *Fusarium* spp., field FHB severity data, means for 1998 and 1999

Genotype	*Fusarium* spp. and isolates										Mean
	*Fg*44	*Fg* 207	*Fc* 223	*Fc* 12551	*Fc* 12375	*Fg* 40	*Fg* 12377	*F. samb.*	*F. spor.*	*F. vert.*	
Sumai-3	0.00	0.00	0.00	0.58	0.00	0.00	0.00	0.17	0.00	0.00	0.08
Sgv-NB*MM-Sum3,A	1.50	3.42	4.17	0.00	0.33	0.08	0.92	0.25	0.00	0.00	1.07
Sgv-NB*MM-Sum3,B	2.25	0.92	6.25	1.92	2.83	1.25	1.25	4.33	0.67	0.50	2.22
Wuhan2	7.00	6.25	3.42	3.67	0.33	1.92	4.92	0.00	0.00	0.00	2.75
Sum3-81.60*Kõ	12.75	8.58	4.33	0.33	0.00	0.83	0.33	3.00	0.25	0.00	3.04
Bence	24.25	24.08	21.67	11.67	7.67	0.33	6.00	7.25	2.42	1.00	10.63
Sum3-81.60*Kõ	26.75	29.25	19.92	12.17	18.00	0.83	16.92	8.58	6.00	0.33	13.88
Sámson	50.92	38.92	33.83	24.75	22.58	17.17	18.00	23.08	7.00	2.00	23.83
Kende	45.75	37.42	34.50	29.58	28.75	11.83	11.67	26.92	13.58	7.00	24.70
Öthalom	43.83	34.50	20.75	33.92	19.67	39.92	31.25	16.42	5.92	4.42	25.06
Zu*Ré-NB	51.08	46.67	34.08	36.58	32.83	21.08	16.00	24.92	17.67	8.17	28.91
Rába	58.00	37.92	34.50	35.00	25.67	39.50	20.83	14.67	9.17	16.00	29.13
Jbj-50	58.42	52.50	37.92	40.33	23.92	36.08	20.08	14.58	18.25	17.33	31.94
Zugoly	54.17	50.42	36.08	46.50	35.08	23.83	32.92	30.83	20.00	18.17	34.80
Kalász	64.00	55.17	43.33	53.33	41.17	52.50	36.25	14.67	14.92	21.12	39.65
Góbé	65.67	66.67	48.75	45.33	30.17	41.67	41.33	22.17	27.92	15.08	40.48
Mean	35.40	30.79	23.97	23.48	18.06	18.05	16.17	13.24	8.98	6.94	19.51
LSD 5% genotypes											0.83
LSD 5% isolates											0.65

Table 3. ANOVA of the data of FHB values for Table 2 (MS values)

Source of variance	FHB	FDK	Yield loss	df	DON	DON df
Cultivar A	12681.9*	25145.2*	19711.8*	15	1096.9*	15
Isolate B	7882.4*	22155.1*	11086.2*	9	1962.5*	9
Year C/Repl.DON	5832.0*	42599.0*	1126.0ns	1	1862.4*	1
A × B	352.82ns	1226.4ns	519.2ns	135	107.39ns	135
A × C	768.3**	2377.5***	790.7ns	15	–	–
B × C	6368.0*	10513.8*	5755.1*	9	–	–
A × B × C	317.7	852.7	540.7	135	–	–
Error	5.4	45.4	116.3	640	147.3	159

*$P = 0.001$, **$P = 0.05$, ***$P = 0.01$.

coverage. Control seeds were germinated in distilled water. After field inoculations, the aggressiveness was again tested in the laboratory. In all 5 years no loss of aggressiveness was observed from the beginning of May to the beginning of June, and thus the results from different inocula were not modified by the possible loss of aggressiveness during this period (data not shown).

Field experiments

The experimental design was as that described by Mesterházy (1995). Each genotype was sown in a single 5×1 m^2 plot in the autumn at a sowing rate of 550 seeds per m^2; 1998 and 1999 were *Fusarium* years, and therefore natural background infection could occur and was tested via DON contamination of the DON non-producing isolates. Each genotype was inoculated at full flowering (Zadok's scale 65) in the early morning. As the genotypes had different flowering times, the inoculation period lasted 10–14 days. Each isolate was used independently (not mixed). Three replicates of groups of spikes containing 15–25 ears per isolate were selected for inoculation and three control samples of ears were left without inoculation at the end of the plot. The heads were sprayed uniformly with a 1-l hand sprayer from all sides. As the

Table 4. Correlation coefficients between genotype reactions of wheat to *Fusarium* spp.; means for 1998 and 1999

	F. graminearum	*F. culmorum*	*F. sambucinum*	*F. sporotrichioides*
FHB%				
F. culmorum	0.9709*			
F. sambucinum	0.7781*	0.8569*		
F. sporotrichioides	0.8731*	0.8992*	0.8070*	
F. verticillioides	0.8680*	0.8748*	0.6132**	0.8206*
FDK%				
F. culmorum	0.9622*			
F. sambucinum	0.8721*	0.9310*		
F. sporotrichioides	0.2518ns	0.2700ns	0.3183ns	
F. verticillioides	0.6420**	0.6663**	0.7838**	0.3737ns
Yield loss%				
F. culmorum	0.9610*			
F. sambucinum	0.7317*	0.7845*		
F. sporotrichioides	0.8236*	0.8442*	0.8575*	
F. verticillioides	0.8288*	0.8581*	0.7010**	0.6888**
DON mg kg^{-1}				
F. graminearum		0.6846**		
F. sambucinum	0.5761***	0.6326**		
F. sporotrichioides	0.1346ns	0.2557ns	−0.0299ns	
F. verticillioides	0.6640**	0.3253ns	0.5113***	0.1841ns

*$P = 0.001$, ** $P = 0.01$, *** $P = 0.05$, ns = not significant.

Table 5. Correlations between resistance of genotypes to *Fusarium* spp., 2000

	F. graminearum	*F. culmorum*	*F. sporotrichioides*	*F. poae*
F. culmorum	0.9236*			
F. sporotrichioides	0.8689*	0.8770*		
F. poae	0.7793**	0.6490***	0.6745***	
M. nivale	0.8625*	0.8683*	0.9927*	0.6126+

*$P = 0.001$, **$P = 0.01$, ***$P = 0.02$, +$P = 0.05$.

genotypes had different head sizes, the amount of inoculum for a group of heads varied (15–20 ml). Control groups of heads were sprayed with distilled water. Following inoculation, each group of spikes was covered with a polyethylene bag. Bags were removed after 24 (1998–2000) or 48 h (2001–2002). The plants were loosely bound at half plant height to allow the leaves to assimilate freely and to avoid increased infection severity. The data for 1998 and 1999 were analysed together, as were those for 2001 and 2002. Isolates used in only one of the years were not considered in the common analysis. The data for 2000 were evaluated separately, as the isolates and cultivars differed from those applied earlier and later; 1998 and 1999 were epidemic years, while 2000 was very dry. The 24 h bagging of inoculated ears was not long enough to enhance significant epidemic enhancement; 2001 and 2002 were again dry, but the 48 h bagging helped to initiate a significant epidemic level.

Disease evaluation

The disease severity (% diseased spikelets: 0 = no spikelets infected, 100 = all spikelets infected) was evaluated 10, 14, 18, 22 and 26 days (in some years also 30 days) after inoculation (Mesterházy, 1977). Ratings were made until the control heads started to turn yellow. In the statistical analyses, the mean values of the readings were considered. After harvesting, 10 similarly infected heads from each group were taken for further evaluations; the heads were threshed at low wind speed to save infected and shrivelled grains. The grain mass was measured

Table 6. Resistance of wheat genotypes to isolates of five *Fusarium* spp. FDK values (%), 2001 and 2002

Genotypes	Isolate									
	Fc 12551	*Fc* 12375	*Fg* 12377	*F. samb.*	*Fg* 44	*F. av.* 1	*F. av.* 2	*F. spor.* 2	*F. spor.* 1	Mean
Sumai-3	15.17	2.00	1.50	2.17	0.00	0.17	0.00	1.33	0.17	2.50
Wuhan 6B	19.67	4.33	1.83	3.17	0.33	0.50	1.17	0.50	3.83	3.93
Nobeoka Bozu	17.50	6.83	7.50	3.00	6.00	2.00	0.50	1.50	5.17	5.56
Sgv/NB//MM/Sum3	32.17	3.83	10.00	3.50	1.33	0.67	0.42	0.42	0.83	5.91
RSt//MM/NB	30.00	12.17	3.67	17.50	1.42	5.00	3.42	2.67	1.00	8.54
Frontana	35.00	27.50	10.92	15.83	1.83	0.17	0.17	0.67	0.25	10.26
Esprint	23.33	26.67	26.67	10.50	15.00	5.50	6.83	1.67	1.50	13.07
Sgv/NB//MM/Sum3	46.67	17.50	31.67	30.83	16.50	25.00	2.67	2.83	1.33	19.44
RSt//MM/NB	59.17	52.50	40.00	29.17	35.00	5.83	3.17	1.58	4.33	25.64
Arina	68.33	60.00	42.50	24.17	24.17	5.92	2.92	2.33	1.83	25.80
Jbj-50	60.42	79.58	55.00	37.50	16.75	4.92	2.25	2.83	1.83	29.01
Furore	78.00	63.33	69.17	45.83	29.17	20.00	9.42	2.33	1.25	35.39
P 7318	67.50	55.00	47.50	50.00	33.00	28.50	25.08	5.28	10.25	35.79
Ludwig	79.17	66.67	65.00	47.50	50.00	20.00	3.75	1.83	3.00	37.44
Zugoly	85.83	90.83	85.00	35.00	71.67	1.67	1.67	5.83	8.17	42.85
Kimon	91.00	79.17	72.50	58.33	57.50	30.17	17.75	4.50	9.33	46.69
Aristos	78.33	77.50	70.00	63.33	48.33	33.58	18.58	16.50	17.17	47.04
Öthalom	94.00	88.33	60.00	78.33	54.17	7.50	3.00	28.17	20.67	48.24
P 8635	82.83	87.50	84.17	61.67	69.17	20.08	17.08	13.17	25.00	51.19
Contra	94.17	90.83	73.33	65.00	80.00	26.75	8.00	7.33	15.83	51.25
Ritmo	89.67	85.67	74.17	76.67	55.83	30.25	24.42	33.83	13.17	53.74
Carlos	90.67	80.83	79.17	64.17	65.00	35.00	37.50	17.58	20.00	54.44
LW 90.Z 11.1	93.50	83.33	73.33	73.33	64.17	55.83	48.50	5.33	6.17	55.94
SJ981249	100.00	91.67	74.17	78.33	64.17	32.00	35.58	27.83	25.42	58.80
Biscay	97.17	95.00	86.67	77.50	87.50	42.67	37.75	15.17	13.33	61.42
Pentium	96.67	89.17	79.17	81.67	66.67	46.33	45.00	45.83	40.17	65.63
SJ981153	97.50	98.83	90.00	90.00	75.00	37.83	15.67	54.83	40.25	66.66
Mean	67.53	59.87	52.39	45.33	40.36	19.40	13.79	11.25	10.79	35.63
LSD 5% genotypes										2.65
LSD 5% isolates										1.56

and the percentage of scabby (tombstone) kernels was rated visually. The results allowed the evaluation of resistance components. In Table 10, the R–S classification of the genotypes is given according to the FHB values. Tolerance was found in genotypes where the yield loss was significantly less than in others with very similar FHB values. Grain samples inoculated with DON non-producers and containing DON, were subjected to reisolation.

Toxin extraction

The grains of the three replicates per isolate were pooled. The samples were milled and 5 g of meal was extracted in polypropylene centrifuge tubes according to Lauren and Greenhalgh (1987); 20 ml of acetonitrile/water 84/16 (v/v) were used for extraction using a vertical shaker at room temperature for 2 h. Samples were centrifuged at $5000 \times g$ for 10 min and 2 ml of supernatant was cleaned by solid-phase extraction on an extraction column filled with 1.0 g neutral alumina activated charcoal (20/1 m/m). The eluents were evaporated to dryness under N_2 (Jouan RC 10.22 centrifugal evaporator) and redissolved in 0.75 ml of acetonitrile/water 80/20 (v/v). Finally, the samples were membrane filtered into HPLC autosampler vials. The chemicals used were DON, neutral alumina and activated charcoal (Darko G60) from Sigma-Aldrich Ltd. (Budapest, Hungary), and HPLC grade acetonitrile from Farmitalia Carlo Erba (Milan, Italy). HPLC grade water with 18 MΩ resistivity was produced with Nanopure II cartridge-type water purification equipment (Bornstead/Thermolyne Co., Dubique IA, USA).

Table 7. Resistance of wheat genotypes to isolates of five *Fusarium* spp. DON values (mg kg^{-1}), 2001 and 2002

Genotype	*Fusarium* spp./isolates									Mean	
	Fc 12551	*Fc* 12375	*Fg* 12377b	*Fg* 44	*F. samb*	*F. spor.* 1	*F. av.* 2	*F. av.* 1	*F. spor.* 2	Mean	*Fg* + *Fc*
Sgv/NB//MM/Sum3	7.79	4.21	0.00	3.39	0.00	0.00	0.00	0.00	0.00	1.71	3.85
Sum3/81.60//Kő	9.23	3.60	3.67	0.50	0.29	0.00	0.00	0.00	0.00	1.92	4.25
Sumai 3	13.12	3.20	1.50	0.46	0.00	0.00	0.00	0.15	0.00	2.05	4.57
Wuhan 6B	11.24	4.91	3.93	1.14	0.00	1.36	0.00	0.47	0.37	2.60	5.31
RSt//MM/NB	11.53	8.32	0.80	3.50	0.00	0.00	0.00	0.00	0.00	2.68	6.04
Nobeoka Bozu	8.21	4.23	11.13	0.71	0.00	0.38	0.00	0.82	0.00	2.83	6.07
Frontana	13.29	12.20	7.61	1.87	0.16	0.00	0.00	0.00	0.00	3.90	8.74
Sgv/NB//MM/Sum3	21.93	12.46	12.36	14.53	0.00	0.38	0.34	0.50	0.00	6.94	15.32
Öthalom	47.34	40.73	17.75	11.74	0.47	3.22	0.47	0.00	0.00	13.52	29.39
Arina	57.38	25.63	27.66	12.93	0.26	0.00	0.00	0.00	0.39	13.80	30.90
P7318	55.75	32.03	21.52	25.03	0.67	0.00	0.00	0.31	0.00	15.03	33.58
RSt /MM/NB	62.94	34.99	32.30	44.13	0.31	0.00	2.65	0.17	0.18	19.74	43.59
Zugoly	56.11	61.13	27.83	35.94	1.23	0.25	0.00	0.00	0.00	20.27	45.25
SJ 89.81249	118.78	75.99	47.46	41.41	0.00	1.53	0.32	0.00	0.24	31.75	70.91
Kimon	143.75	64.93	38.27	58.08	2.31	1.11	0.76	0.00	0.25	34.38	76.26
Furore	138.62	74.35	82.96	56.21	2.59	0.63	0.36	0.25	0.42	39.60	88.03
Ludwig	159.64	96.78	64.26	48.89	0.40	0.66	0.10	0.08	0.09	41.21	92.39
LW 90Z11.1	133.27	97.71	83.44	89.37	0.55	1.67	0.71	0.08	0.00	45.20	100.94
Cardos	141.57	98.01	92.41	102.95	1.39	0.00	0.00	0.54	0.00	48.54	108.73
Aristos	233.69	147.10	59.63	67.79	3.73	0.87	0.32	0.00	0.50	57.07	127.05
Ritmo	228.83	125.53	97.83	92.86	0.53	0.25	0.13	0.20	0.00	60.68	136.26
P8635	284.17	146.99	78.26	56.23	1.60	1.20	0.37	0.40	0.73	63.33	141.41
Pentium	334.31	90.04	132.33	47.19	2.80	1.28	2.12	0.50	0.00	67.84	150.97
Biscay	261.68	196.18	155.84	87.61	2.64	0.00	0.32	1.55	0.76	78.51	175.32
Contra	409.14	144.12	119.95	91.40	0.40	0.64	0.11	0.97	0.10	85.20	191.15
SJ 9811.53	432.91	250.43	145.40	110.13	3.20	2.13	0.28	0.15	0.00	104.96	234.71
Mean	130.62	71.37	52.54	42.54	0.98	0.67	0.36	0.27	0.15	33.28	74.27
LSD 5% genotypes										28.45	64.70
LSD 5% isolates										11.84	17.60

Below the line at cultivar Zugoly are the western European genotypes.

Toxin analysis

For DON, HPLC separation was performed on a Hewlett-Packard HP 1090 M liquid chromatograph equipped with a diode-array UV detector (Agilent, Waldbronn, Germany); 5 µl aliquots were injected onto a Hypersil ODS microbore column (200 × 2 mm × 5 µm) . A binary gradient of water and acetonitrile (flow rate 0.5 ml min^{-1}) was applied for separation. Before analysis, the solvent was membrane-filtered through a PTFE membrane and degassed continuously by a vacuum degasser. Detection was performed at 220 nm; the minimum detectable amount of DON at a signal to noise ratio of 3:1 was 0.2 mg kg^{-1}. To confirm DON, the UV spectra were recorded continuously from 190 to 400 nm with the built-in diode array detector. Quantitative evaluations were performed by use of external standards (in the range 1–1000 ng). DON concentrations below 0.2 mg kg^{-1} could not be detected in the grains and are noted by zero in the variance analyses. HPLC measurements on the same extraction had a standard deviation within 1%. To check the accuracy of toxin determinations, nine average samples were subjected to two independent parallel clean-ups. The linear regression function was $y = 0.9549x + 0.3127$, with $r = 0.9906$, significant at $P > 0.001$. This confirmed that the DON data were reliable.

Statistical analysis

Data were evaluated using analyses of variance, correlation and regression (Microsoft Excel 7.0). Three-way analyses of variance were performed according to the functions given by Sváb (1985) and Weber (1972). Main effects (genotype, year

Table 8. General means across isolates for 2001 and 2002 FHB resistance tests for five *Fusarium* spp.

Genotype	Traits				DON/FDK ratio[a]	Resistance components[b]
	DON mg kg^{-1}	FDK%	Yield loss%	FHB%		
Sumai 3	2.05	2.50	3.64	0.04	0.98	R
Wuhan 6B	2.60	3.93	−2.35	0.19	0.82	R
Nobeoka Bozu	2.83	5.56	8.23	0.38	0.76	R
Sum3/81.60//Kő	1.92	4.49	18.19	0.54	0.85	R, T+,
RSt//MM/NB	2.68	25.64	22.09	1.14	0.16	R, KI+, T+
Sgv/NB//MM/Sum3	1.71	5.91	8.39	1.37	0.33	R
Sgv/NB//MM/Sum3	6.94	19.44	11.48	2.25	0.64	R, KI+, T+
Frontana	3.90	10.26	20.22	4.53	0.48	R, KI
Arina	13.80	25.80	14.69	10.51	0.72	MR, T
P7318	15.03	35.79	26.56	10.76	0.79	MR
Öthalom	13.52	48.24	25.51	11.32	0.49	MR, KI+
Zugoly	20.27	42.85	43.59	19.68	0.69	MS, KI+
RSt /MM/NB	19.74	8.54	18.88	23.95	3.80	MS, KI, T
Furore	39.60	35.39	22.64	24.59	1.67	MS,T
Kimon	34.38	46.69	40.82	26.66	1.26	MS
Cardos	48.54	54.44	39.36	27.94	1.74	MS, KI
Ludwig	41.21	37.44	30.08	29.76	1.75	MS, KI, T
P8635	63.33	51.19	30.66	31.30	2.22	S, T
LW 90Z11.1	45.20	55.94	38.11	33.48	1.61	S, D
SJ 9811.53	104.96	66.66	40.23	36.19	0.99	S, KI+, D+
Aristos	57.07	47.04	35.15	38.07	2.25	S, KI
Ritmo	60.68	53.74	40.30	40.62	2.18	S
Biscay	78.51	61.42	42.86	40.93	2.52	S, D+
Pentium	67.84	65.63	46.29	43.36	2.28	S, KI+, D+,
Contra	85.20	51.25	43.20	44.38	2.96	S,KI, D+
SJ 89.81249	31.75	58.80	39.02	45.55	3.53	S, D
Mean	33.28	35.56	27.22	21.13	1.68	
LSD 5%	28.45	2.65	6.4	0.51		

[a]Only for means of *F. graminearum* and *F. culmorum* isolates.
[b]R = resistant, MR = moderately resistant, MS = moderately susceptible, S = susceptible, T = tolerance, T+ = extreme yield loss, KI+ = extra susceptibility to kernel infection, KI= resistance to kernel infection, D+ = extreme toxin production, D = resistance to toxin accumulation.

and isolate), interactions and in some cases replicate effects were computed. When the three-way interactions were significant, the *F* values were computed not only against error, but also against the three-way interaction. For ANOVAs, only MS values are presented (except for 2000). To evaluate the correlations between resistances of genotypes to different *Fusarium* spp. the data for the isolates of a *Fusarium* spp. were nested (see Tables 4 and 9).

Results

The FHB values for 1998 and 1999 (Table 2) revealed clear resistance and aggressiveness differences. For the most resistant genotypes, there was either no disease or only traces of infection; here, no differences in aggressiveness were observed. For the more susceptible or very susceptible cultivars, however, the differences in aggressiveness were striking. The most aggressive isolates were found for *F. graminearum* and *F. culmorum*. The correlations between the reactions to the isolates (above $r > 0.80$; $P = 0.001$) were highly significant, indicating very similar reactions to the different *Fusarium* spp. and their isolates. Variance analyses for all parameters (Table 3, significance tested against the three-way interaction) demonstrated no significant interaction between the isolates and the genotypes, supporting the very similar reactions to the different *Fusarium* spp. The isolate/year interaction was very high for all parameters, indicating strong differences in aggressiveness between different inocula of the

Table 9. Correlation coefficients between five *Fusarium* species to wheat lines ($n = 26$), means for 2001 and 2002

	F. culmorum	*F. graminearum*	*F. sambucinum*	*F. sporotrichioides*
FHB%				
F. graminearum	0.9865*			
F. sambucinum	0.9453*	0.9296*		
F. sporotrichioides	0.8421*	0.8261*	0.9084*	
F. avenaceum	0.6826*	0.6535*	0.7144*	0.5893**
DON mg/kg^{-1}				
F. graminearum	0.9231*			
F. sambucinum	0.6933*	0.6537*		
F. sporotrichioides	0.4106***	0.2925ns	0.3569***	
F. avenaceum	0.3353ns	0.4071***	0.2927ns	0.1230ns
FDK%				
F. graminearum	0.9746*			
F. sambucinum	0.9296*	0.9061*		
F. sporotrichioides	0.6601*	0.6514*	0.7879*	
F. avenaceum	0.7006*	0.7166*	0.8111*	0.5846**
Yield loss%				
F. graminearum	0.9671*			
F. sambucinum	0.9269*	0.8929*		
F. sporotrichioides	0.6322*	0.5730**	0.6268*	
F. avenaceum	0.4549***	0.3558ns	0.4732***	0.7296*

*$P = 0.001$, **$P = 0.01$, ***$P = 0.05$, ns = not significant.

same isolate in different years. However, this did not significantly influence the cultivar ranking as the genotype/isolate interaction was not significant in these tests. As some of the 1998 data were published earlier (Mesterházy, 2002), and the test was repeated in 1999, only the mean data are presented here. The year effect revealed significant differences in the FHB and FDK values, but differences between the means were not significant for yield loss or DON contamination. It is important that the year/isolate interactions were not significant at any of the traits, the cultivar/year interaction was not significant for the yield loss and DON concentrations, as this indicates that the similarity between the two data sets was high. For FHB and FDK, the interactions were just significant. However, their value was less than 10% of the value of the main cultivar effect, and therefore only a slight moderating effect was experienced.

The FDK data proved similar (not shown in detail); the difference from the FHB data was that *F. sporotrichioides* gave lower and less consistent FDK values. The yield reactions (not shown) displayed the same pattern as found for the FHB values; all correlations were highly significant, except for *F. sporotrichioides* (although here too $r = 0.68$). The DON content was 30–50 mg kg^{-1} for the DON-producing isolates on susceptible genotypes, but only 0.2–8 mg kg^{-1} for the resistant ones. The DON non-producing *Fusarium* spp. contained only traces of DON (mostly lower than 0.5 mg kg^{-1}), reflecting a low level of background infection. Table 4 shows the correlations for all parameters according to the *Fusarium* spp. The correlations were high and significant for FHB, FDK and yield loss. *Fusarium sporotrichioides* was an exception, with very low FDK values as compared with other *Fusarium* spp. For DON only the *F. graminearum*/*F. culmorum* correlation was close. *Fusarium sambucinum* is a low DON-producer, whereas the other *Fusarium* spp. are not DON-producers. Three genotypes exhibited resistance to kernel infection, while two indicated resistance to DON accumulation and two showed tolerance. The ratio of DON/FDK ratio was calculated. At 1% FDK higher DON values were observed for the more resistant materials and less for the susceptible genotypes. The correlations between the reactions to different traits were very close, ranging from 0.8359 to 0.9559; $P < 0.001$, indicating that, in spite of the influence of resistance components, the level of FHB clearly determined the other parameters in this test.

Table 10. Aggressiveness of the *Fusarium* spp. for the four parameters tested in 2001 and 2002

Traits	FHB%	FDK%	DON mg kg^{-1}	Yield loss%
F. graminearum	42.01	46.03	47.54	34.80
F. culmorum	30.72	62.84	101.00	45.37
F. sambucinum	18.10	45.73	0.98	25.93
F. sporotrichioides	10.17	11.33	0.41	14.73
F. avenaceum	3.15	16.95	0.32	14.65
Mean	20.83	36.58	30.05	27.09
LSD 5%	0.26	1.10	22.85	2.66

Figure 1 depicts the pathogenic characteristics of the *Fusarium* spp. For *F. graminearum* and *F. culmorum*, high FHB values and even higher FDK were characteristic, and the level of DON contamination was also high. For *F. sambucinum*, the FHB and FDK values were similar, but the DON content was negligible. For *F. sporotrichioides* and *F. verticillioides*, the FHB values were low, their FDK values were even lower, and DON contamination was present only in traces. This was supported by reisolations from the non-inoculated control and the infected grains.

Due to the hot and dry weather the severity of infection was low in 2000. Each *Fusarium* spp. was represented by two isolates. The FHB results were very similar to those found in 1998 and 1999. The correlations (Table 5) among the cultivar responses to the *Fusarium* spp. were similar to those determined in the previous two years, but the relation between *F. poae* and *M. nivale* was weak (low infection severity). The data showed that the disease severity was lower than in the previous years; the less aggressive *Fusarium* spp. caused considerable disease only in the most susceptible genotypes. ANOVA (not shown in detail) demonstrated highly significant isolate and genotype differences. The interactions (apart from the three-way interaction) for all parameters were significant, but less than 10% of the MS values of the main effects of the genotype or isolate. Accordingly, only some modification of the responses was detected.

The FHB data for 2001 and 2002 clearly revealed the reaction differences for the cultivars and *Fusarium* spp. The means of five readings were up to 80% for the most susceptible cultivars for *F. culmorum*, and 70% for *F. graminearum*. *Fusarium sambucinum* exhibited medium values, those for *F. sporotrichioides* were lower, and for *F. avenaceum* showed the least pathogenicity. The most resistant genotypes remained symptomless in response to all pathogen strains, or only traces of infection were recorded. The correlations between the cultivar reactions to the isolates were very close. The FDK means rose from 2.5 to 67%. Isolates ranked from 10.8 to 67% (Table 6). The correlations between reactions to the different isolates were normally higher than $r = 0.90$; a low (about $r = 0.50$) value was recorded only for low aggressive isolate *F. avenaceum* a-17.

The yield loss data were very similar. The yield loss ranged from 11 (*F. avenaceum* a-17) to 48% (*F. culmorum* No. 12551) as the mean across the cultivars. The DON data (Table 7) displayed extreme

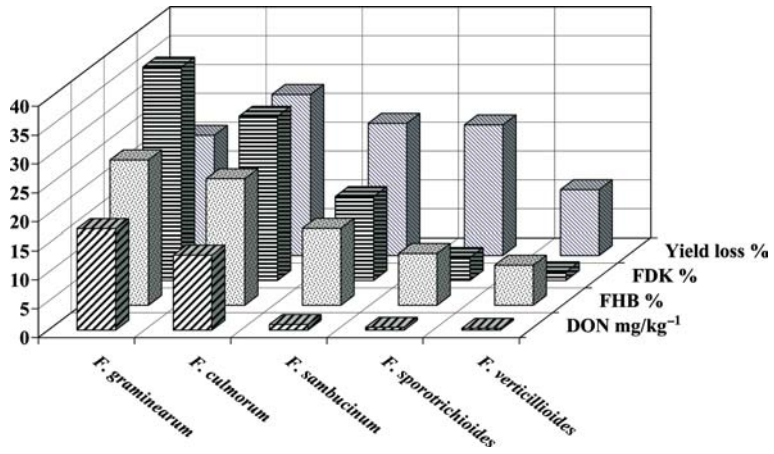

Figure 1. Aggressiveness of the *Fusarium* spp. according to FHB, FDK, yield loss values and DON contamination; means for 16 cultivars, 1998 and 1999.

variability, from traces up >400 mg kg^{-1}. The *F. graminearum* and *F. culmorum* strains produced large amounts of DON, whereas the other *Fusarium* spp. did not produce DON, or only traces originating from the natural background infection were detected. When only the DON-producers were considered, the susceptible Hungarian genotypes such as Zugoly had a mean DON content of 45 mg kg^{-1}, while the much more susceptible western European genotypes showed up to 234 mg kg^{-1} at nearly the same FDK values. The LSD value was higher than for the FHB or FDK values, because the three replicates were pooled and hence fewer data were available for the ANOVA.

ANOVAs (very similar to Table 3) indicated highly significant main effects and interactions. The main effects differed significantly from the interactions, meaning that the interaction modified only the effect of one agent, but the basic ranks did not vary much. The genotype/isolate and genotype/year interactions were smaller, but the isolate/year interaction was much higher in 2001 and 2002 than in 1998 and 1999. This means that the aggressiveness changed strongly, but this did not greatly affect the ranking of the cultivar response to the isolates used.

Table 8 lists the general means for the parameters across all isolates. Sumai 3 proved to be the best for all parameters. The ratio of DON/FDK is also given to illustrate any cultivar differences. For 1% FDK, 0.15–3.53 mg kg^{-1} DON was calculated for DON-producing isolates. Resistance components too were identified. A majority of the genotypes had one or more resistance components. The most resistant genotypes had such low values for every parameter that no additional resistance component could be identified. It is important that these components could be identified at higher susceptibility levels. The correlations between the parameters were close (r = 0.7718–0.9092, $P > 0.001$, $n = 26$).

The correlations between the pooled reactions to different *Fusarium* spp. according to the parameters (Table 9) showed the highest correlations for FHB and FDK: normally $r > 0.90$ between *F. graminearum*, *F. culmorum* and *F. sambucinum*. The correlations between these and the other two *Fusarium* spp. were lower because of the lower infection severities; the genotypes were less strongly differentiated, but in most cases significantly. DON is a special case since three of the *Fusarium* spp. were not DON-producers, and therefore a high correlation could not be demonstrated.

The background infection was tested in two ways. In 2003, 0–0.4 mg kg^{-1} DON was found for non-inoculated control materials. From healthy-looking inoculated materials, we checked 50 samples at 1% FDK or lower. Six contained 10 mg kg^{-1} or higher, five cases 5–10 mg kg^{-1} and the remainder < 5 mg kg^{-1} DON. For 22 samples, the DON content was below the detection limit of 0.2 mg kg^{-1}. For samples inoculated with DON non-producing strains, reisolations were made. In a low percentage, *F. graminearum* and *F. culmorum* infected grains were detected as in the non-inoculated check samples.

Several isolates of other *Fusarium* spp. were also tested, but these data are omitted from the evaluations as we had results only for one year. One isolate each of *F. subglutinans, F. acuminatum* and *F. moniliforme*, two *F. poae* isolates from 2001 and nivalenol-producing *F. culmorum* strain 89.4 from France were additionally tested. Their reactions were similar to those listed, and we therefore presume resistance against these species is active.

Table 10 presents the data for the isolates as means for the genotypes tested, showing the level of aggressiveness according to the different parameters. As regards the head symptoms, *F. graminearum* was the most pathogenic, followed by *F. culmorum*, *F. sambucinum, F. sporotrichioides* and *F. avenaceum*. *Fusarium graminearum* and *F. sambucinum* gave identical FDK values significantly less than for *F. culmorum*. *Fusarium sporotrichioides* displayed similar values, for both FHB and FDK; *F. avenaceum* exhibited low FHB values, but five times higher FDK values. F*usarium culmorum* was the strongest DON-producer: at 1% FDK, it produced 60% more DON than did *F. graminearum*. At 1% FHB, *F. culmorum* produced three times more DON than did *F. graminearum*.

Discussion

Common resistance

The data for five years lend good support to the hypothesis that cultivar resistance to any *Fusarium*

spp. tested implies a similar level of resistance to other *Fusarium* spp. The eight *Fusarium* spp. involved in the study showed no exceptions. More species were examined here than in any previous study. In the first test (1998 and 1999), the genotype/isolate interaction did not prove significant; the later tests showed significant interactions, but they differed significantly from the main genotype and isolate effects. A non-significant genotype/isolate interaction is not a precondition for the demonstration of general resistance, but it must differ significantly from the main effects. The correlation coefficients are much higher than any published to date. We therefore consider this to be the most powerful support yet for the common resistance of wheat to *Fusarium* spp. Further, the large set of genotypes with highly differing genetical backgrounds support the conclusion that common resistance to different *Fusarium* spp. is a characteristic phenomenon in wheat. For this reason, the probability is low that wheat genotypes or *Fusarium* spp. not fitting into the system will be found.

It is important that this non-specificity could be demonstrated alike for kernel infection, yield loss and toxin contamination (except for non DON-producers) indicating that resistance to FHB to a large extent determines also the other parameters. Our data revealed that the correlation is strict for the FHB data, sometimes less strict for the FDK and yield loss values, while DON data are useful only for DON-producers. Accordingly, it is our opinion that the extent of the resistance is much broader than would be expected merely from the FHB values. When the disease-causing capacity is too low, differentiation of the genotypes is not possible. It is noteworthy that the most resistant genotypes were symptomless or exhibited only very moderate infection in response to all of the *Fusarium* isolates tested. There was no exception. This makes it possible to breed for general *Fusarium* resistance as wheat cannot differentiate between *Fusarium* spp. This justifies the relative simple breeding technology performed in many parts of the world. Even *M. nivale* seems to belong in this group, as indicated by van Eeuwijk et al. (1995) and Diamond and Cooke (1999). The fact that the cultivar resistance ranking to *M. nivale* seems to be very similar to that of the *Fusarium* spp. tested indicates that resistance can also be common to pathogens other than *Fusarium*. This promises to be an interesting research problem in the future.

Toxin production

We presumed that DON found in genotypes infected other than *F. graminearum* and *F. culmorum* is a consequence of the background infection. This is supported by the fact that the toxin contamination of the non-inoculated control grains contained normally 0–0.4 mg kg^{-1} DON. However, other reasons might also be responsible. Mirocha et al. (2003) cited Abramson et al. (1993) who stated that *F. poae* and *F. sporotrichioides* are able to produce DON. de Nijs et al. (1996) stated the same for *F. acuminatum* and *F. sambucinum*. We therefore conclude that besides background infection a small amount of DON can originate from *Fusarium* spp. that are generally assumed not to produce DON (or only several isolates produce it). This is in agreement with earlier findings (Mesterházy, 2002).

Resistance components

The resistance of the cultivars varied from nearly symptomless to totally susceptible genotypes. The correlations for the parameters measured were very close, usually at $r \cong 0.90$; the high resistance measured based on head symptoms in most cases therefore implies similar values for the other parameters. Resistance components (Schroeder and Christensen, 1963; Miller et al., 1985; Mesterházy, 1995; Mesterházy et al., 1999) were considered in genotypes where significant differences were computed between FDK (type IV: resistance to kernel infection), yield loss (type V: tolerance) or DON contamination (type III: resistance to DON accumulation) for genotypes with FHB values without significant differences (Mesterházy, 1995). Our DON resistance component differs from that described by Miller et al. (1985), as we consider not only DON detoxication by the resistant cultivars, but also toxin production inhibition mechanisms. Genotypes were additionally found with significantly higher DON contamination than that supposed from the FHB rating. The extremely high DON levels in several genotypes pose important food and feed safety problems and may be responsible factors in western European epidemics. Of course, further research is needed for a better understanding of the components, but the phenomenon exists and is

of significance for practical breeding and selection methodology. QTLs responsible for the existence of these components should also be identified. These components have nothing in common with the components of the partial disease resistance described by Browne and Cooke (2004) as these relate to incubation period, latent period and lesion length which we did not measure. Partial disease resistance could also be investigated for FHB. It appears that more resistance components can exist than reported to date.

Pathogenicity of Fusarium *spp*

Although resistance against different *Fusarium* spp. is common, the *Fusarium* spp. differ in behaviour. As aggressiveness was measured as a mean for 16, 13 and 26 genotypes, the data are fairly informative. The *Fusarium* species and their isolates revealed large differences in disease-causing capacity in all the parameters tested. It is not possible to conclude that DON/FDK or FHB/FDK or other values are characteristic for the given *Fusarium* spp. or resistance classes: the two tests (1998–1999 and 2001–2002) did not give the same results, and a given isolate exhibited different profiles in different tests. One conclusion can be drawn: a given FHB value can lead to rather different levels of FDK, DON accumulation (for DON-producing isolates) and yield losses depending on the environmental conditions, even if the correlations between the parameters were generally close. An exact forecast of the DON or FDK level on the basis of field symptoms is hardly possible. It works reasonably well within one test, but not for samples differing in origin. DON is not necessarily a pathogenicity factor for all *Fusarium* spp. as DON non-producing species can also cause heavy infections. Furthermore, it seems that the fumonisins do not play any role in the pathogenesis (Proctor et al., 2002). This supports our conclusion that there is not a single toxin that is responsible for the pathogenicity of all *Fusarium* spp. For this reason, other common traits that could be the basis of the common resistance to different *Fusarium* spp. should be identified. However, for DON-producing isolates the amount of toxin produced is proportional to aggressiveness. Similar results were achieved by Gang et al. (1998).

Breeding aspects

The common resistance observed against the *Fusarium* spp. explains why highly-resistant genotypes resist infection in different parts of the world. Thus, selection against a highly pathogenic isolate of the given *Fusarium* spp. is sufficient and common resistance to the *Fusarium* spp. will be the result. As *Fusarium* resistance is not specific, the resistance of cultivars is durable. The main problem is now to combine *Fusarium* resistance with good agronomic parameters, quality, and resistance to other diseases. Another task is to detect resistance sources that differ from Sumai 3 and give similarly high resistance. Our present knowledge allows the breeding of highly resistant materials, even though many scientific problems have not yet been solved. The most important task is to construct a reliable selection system with artificial inoculation in all generations to allow for effective screening. Such systems are already functioning in several institutes and breeding companies, including Szeged.

The results clearly show that the FHB severity data alone are not enough to determine FHB resistance. This is even truer for screening tests where normally no replicates are made. As the agreement is not perfect, additional screening for FDK is necessary. There are several additional reasons for this: (1) We do not eat green heads, but grain, and therefore the infection severity and degree of toxin contamination of the grain are of the utmost important; (2) The FHB evaluation is completed about 26–30 days after inoculation, but at least one month then remains until harvesting and the infection can spread further in this period: in some cases 100% FDK was measured at relatively low FHB values. For 256 genotypes in 2003, the correlations were between them $r = 0.7579$; $P > 0.001$. In spite of this, FDK values fluctuated for genotypes with 20% FHB between 10 and 70% (unpublished data); (3) These tests revealed close correlations between FDK and DON contamination. This proves that the scabby or tombstone kernels are the most important carriers of the toxins. Mirocha et al. (1998) reached a similar conclusion. For this reason, we should select for low FDK at a reasonably high infection pressure. Additionally, we should select those lines or cultivars that have lower DON contamination than others with similar FDK values.

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