Effects of Head Blight Caused by *Fusarium culmorum* on Toxin Content and Weight of Wheat Kernels

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ABSTRACT

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Ten winter wheat genotypes were inoculated with three Dutch strains of *Fusarium culmorum* (IPO 39-01, IPO 348-01, and IPO 436-01). Seed samples were analyzed for several trichothecene mycotoxins and zearalenone. Deoxynivalenol was detected in concentrations ranging from 0 to 48 mg/kg. The mycotoxins 3-acetyldeoxynivalenol, nivalenol, fusarenon-X, and zearalenone were not detected. Interactions between strains and genotypes were observed for head blight and kernel deoxynivalenol content. For each strain, high correlations were found between deoxynivalenol content and yield reduction. Path analysis suggested a relation between deoxynivalenol and kernel weight reduction. Infection by a highly pathogenic strain reduced yield in terms of kernel number. In the case of two moderately pathogenic strains, yield loss was ascribed to lower kernel weight. This is the first report on the relationships among head blight caused by *Fusarium culmorum*, kernel toxin content, and reduction of yield.

Additional keywords: Fusarium head blight, plant breeding, resistance, *Triticum aestivum*.

In the Netherlands, Fusarium head blight in wheat is caused by *Fusarium culmorum* (W. G. Smith) Sacc. and to a lesser extent by *F. graminearum* Schwabe. These two pathogens are closely related, and plant resistance to *F. culmorum* is correlated with resistance to *F. graminearum* (7,8). Various *Fusarium* species including *F. culmorum* and *F. graminearum* are capable of producing mycotoxins in crops (5). The most notorious mycotoxins of *F. culmorum* and *F. graminearum* in wheat are deoxynivalenol (DON) and 3-acetyldeoxynivalenol (3-ADON) (25). These toxins are harmful to human and animal health. Canada allows a maximum DON concentration in unground wheat for human consumption of 1–2 mg/kg; in the United States this tolerance level is advised (24).

Reports on the relationships among Fusarium head blight, toxin concentration, and yield reduction are scarce. Head blight-susceptible cultivars of wheat contained much higher concentrations of DON in the kernels than resistant cultivars after inoculation with a single strain of *F. graminearum* (mean 6.56 versus 0.83 mg of DON per kilogram) (12). From these data, a correlation of 0.85 (8 df) between DON and spike weight reduction can be derived. No correlation was found between DON and kernel weight reduction. In a comparable study, a correlation of 0.74 (12 df) was observed between Fusarium head blight and kernel DON content (26). From data on wheat heads enclosed in plastic bags after inoculation with *F. graminearum* and *F. culmorum* (6), a correlation between head blight and spike weight reduction can be derived of 0.88 (16 df), and a correlation between head blight and yield reduction can be derived of $r = 0.91$. In commercial wheat fields infected by *F. graminearum*, correlations between head blight (percentage of heads with bleached spikelets 10 days

after heading) and DON were found of 0.79 (20 df) (20) and 0.74 (23 df). No correlation was observed between blight and yield, and DON concentration and yield (21).

This study aims to establish the relationships among head blight caused by *F. culmorum*, kernel toxin content, and reduction of yield. It is the first of its kind involving *F. culmorum*.

MATERIALS AND METHODS

Source of wheat kernels. Sixty seed samples from 10 genotypes inoculated with three strains of *F. culmorum* were selected for mycotoxin analysis. The 10 genotypes were drawn from a set of winter wheat cultivars and SVP lines representing the whole range of Fusarium head blight susceptibility based on the SVP data available in 1985. Ten strains, taken from monospore cultures of wheat-derived isolates of *F. culmorum* collected in the Netherlands, were prescreened for pathogenicity in the greenhouse. Two nonpathogenic strains were discarded. From the remaining eight strains, three strains were drawn: IPO 39-01, IPO 348-01, and IPO 436-01, originating from isolations from a grain of seed, a head, and a leaf sheath, respectively. The lyophilized strains are deposited at the Research Institute for Plant Protection (IPO), Wageningen. Conidiospores for inoculation were produced in 1-L Erlenmeyer flasks containing 250 ml of sterilized wheat kernels of the cultivar Arminda. The cultures were incubated in darkness at 25 C for 2 wk, followed by 3 wk of incubation at 5 C. To prepare spore suspensions, conidia were washed from the kernels with water.

On 22 November 1985, seeds were sown at a standard density of 330 seeds/m^2 in rows 0.25 m apart. A split-plot design was established, with two blocks. Each plot, consisting of one genotype, was divided into subplots of 0.90×0.75 m across which the strains of *F. culmorum* and three controls were randomized so that the experimental subplots were separated from each other by border subplots of the same size.

Because wheat is most susceptible to Fusarium head blight at anthesis (14), experimental inoculations were made at flowering time on 19, 24, or 25 June 1986, using a spore suspension of 250,000 spores per milliliter, at 1 L/10 m^2. The spore suspension was sprayed from 0.25 m above the crop, for which a propane spray-gun was used. To ensure a high relative humidity during the nights after inoculation, the field was sprinkled in the evening for 1 hr each day over a period of 2 wk.

Incubation period, the period from inoculation to the appearance of first symptoms, was determined. These first symptoms consisted of light brown, water-soaked spots on the glumes. Soon, infected spikelets lost the water-soaked appearance, dried up, and assumed the color of the ripe heads. On 15 July, head blight values were determined as the product of the percentage of heads infected and the proportion of bleached spikelets per infected head. The third week of August, 25 leading tillers were randomly harvested per subplot. The heads were threshed by hand, and yield and kernel weight were assessed. Reduction of yield and 1,000 kernel weight was assessed by calculating the difference between a subplot and the mean of the three control subplots for each main plot. This difference was expressed as a percentage of the mean value of the control subplots. To examine control plots for *Fusarium* contamination, seeds were plated out on selective *Fusarium* agar (SFA) (3).

In vitro production of mycotoxins. The toxigenic potential of the strains was determined by analyzing the mycotoxins (DON, 3-ADON, and zearalenone) produced by in vitro cultures growing for 4 wk on wheat kernels (cultivar Grana) of 45% moisture content at a temperature of 20 C. The procedure for this toxin analysis has been described (2,25).

Chemicals and auxiliaries. All solvents were of analytical grade (Merck, Darmstadt, West Germany). Charcoal columns were prepared as follows. A small ball of glass wool was placed in a filtration column (SPE C18 3ml, Baker Chemicals, Deventer, The Netherlands), and acid-washed Celite (0.15 g) (#545, Serva, Heidelberg, West Germany) and a mixture containing 0.5 g of alumina (neutral activated, 70–230 mesh, Merck), 0.9 g of charcoal (SK-4, Serva), and 0.3 g of Celite were added. The column was connected to an assembly consisting of reservoir, filtration column, filter, and stopcock (Baker Chemicals, Deventer, The Netherlands). For better separation, high performance thin-layer chromatography plates (#5633, Merck) were activated for 1 hr at 110 C and then dipped in a solution of aluminum chloride (Sigma Chemical Co., St. Louis, MO)/water/methanol, 10:15:90 (w/v/v). The plates were dried for 12 hr at room temperature followed by 1 hr at 40 C. Plates were washed by development with chloroform/acetone/2-propanol (70:15:15), air dried for 2 hr, and further washed by development with benzene/acetone (60:35). The plates were dried as above. The trichothecene and zearalenone standards were obtained from Sigma, St. Louis, MO. The rotary evaporator was a product of Büchi, Switzerland. Glass capillary micropipets were obtained from Hirschmann Laborgeräte, West Germany. A Vitatron TLD 100 fluorodensitometer was used.

Chemical analysis. In a first analysis, the 60 samples of infected wheat kernels were analyzed for the trichothecene content of DON, 3-ADON, nivalenol (NIV), fusarenon-X (FUS-X), and zearalenone (ZEA) by thin-layer chromatography (TLC), two-dimensional TLC, and quantitative TLC, described by Visconti et al (25). The detection limit of this method was 0.1–0.5 mg/kg, depending on the toxin. Because initially only DON was found, only DON was analyzed in further chemical analyses. Samples were extracted with aqueous acetonitrile and cleaned up using charcoal columns (4,19,22,23). Each wheat sample (10 g) was ground and placed in a 200-ml Erlenmeyer flask with 40 ml of acetonitrile/water (82:18). After being shaken vigorously for 15 min, the mixture was left for 12 hr and shaken again for 15 min. The samples were filtered under vacuum with a Büchner funnel on Whatman (No. 2) filter paper. The prepared columns were washed with 15 ml of acetonitrile/water (82:18) mixture. After the vacuum had been disconnected, a clean filter flask was inserted and 20 ml of collected extract was put on the column. After 5 min, vacuum was again applied, providing a flow rate through the column of 1 ml/min. The extract volume was filtered to reach the glass wool in the bottom of the column and 30 ml of acetonitrile/water (82:18) was added. The solution containing DON was transferred to a round-bottom flask. After the solvents had been evaporated under vacuum at 40 C, the residue was dissolved using two aliquots of 2 ml of ethyl acetate and 1 ml of chloroform/acetonitrile (4:1), and transferred to the vial. The solvent was evaporated under a stream of nitrogen. The dry residue was dissolved by sonification for 2 min in 200 μl of chloroform/acetonitrile (4:1). Extracts of 5 and 10 μl volume and the standard were applied to the prepared TLC plates by glass capillary micropipets. The plate was placed in an unsaturated tank containing chloroform/acetone/2-propanol (8:1:1). Each determination was carried out in five replicates. Blue fluorescent DON spots were quantified visually and via a fluorodensitometer using 20 μg/ml of DON as a working standard prepared in an ethyl-acetate/methanol (9:1) solution.

Recovery tests for DON ($n = 5$) carried out according to the above method gave the following yields: at 0.5 mg/kg 88.5% $\pm$ 2.6; at 0.1 mg/kg 84.0% $\pm$ 3.0; and at 0.05 mg/kg 86.6% $\pm$ 3.9. The limit of detection for DON was 0.05 mg/kg, and minimum detection equaled 0.02 mg per spot. A measure for the repeatability of the DON analysis is the intraclass correlation (18). Based on 10 genotypes (classes) and two observations per genotype, r_1 had a value of 0.99.

Statistical analysis. Both wheat genotypes and strains of *F. culmorum* were random samples from fixed sets. For the analysis of variance a split-plot model with random effects was used (18). For variance and regression analyses an angular transformation was applied for the ratings of Fusarium head blight, and the square root transformation for kernel DON content. From the mean squares of the variance analysis and the expected mean squares based on a random effect model (18), the variance components were estimated. Because the variance component due to genotypes requires more than two mean squares for its estimation, an approximate F test with quasi F ratio was used (27),

TABLE 1. Fusarium head blight and kernel deoxynivalenol (DON) content of 10 wheat genotypes inoculated with three strains of *Fusarium culmorum*

	F. culmorum strains							
	IPO 39-01		IPO 348-01		IPO 436-01		Mean	
Genotype	Head blight[a,b] (%)	DON[a] (mg/kg)	Head blight (%)	DON (mg/kg)	Head blight (%)	DON (mg/kg)	Head blight (%)	DON (mg/kg)
SVP 72017-17-5-10	2.0	4.6	3.0	0.8	1.5	0.6	2.2	2.0
SVP 77078-30	9.0	3.4	1.0	ND[c]	2.5	0.4	4.2	1.2
SVP 75059-28	11.0	3.4	3.0	ND	1.5	0.2	5.2	1.2
Saiga	4.5	4.2	4.5	0.6	9.0	2.1	6.0	2.3
SVP 72003-4-2-4	23.5	5.8	9.5	0.5	3.5	0.7	12.2	2.3
SVP 73030-8-1-1	60.0	33.2	7.5	1.9	9.0	6.8	25.5	13.9
SVP 75059-32	32.5	13.3	9.0	ND	25.0	1.4	28.0	4.9
SVP 73016-2-4	47.0	31.5	14.5	0.4	22.5	4.5	28.0	12.1
SVP 73012-1-2-3	67.5	34.0	17.5	1.5	17.0	8.4	34.0	14.6
SVP 72005-20-3-1	62.5	37.0	27.5	1.6	23.0	5.6	37.7	14.7
LSD ($P = 0.05$)	29.6	17.1	9.0	2.2	10.7	5.4		
Mean	32.0 ± 8.8[d]	17.0 ± 5.0	9.7 ± 2.8	0.7 ± 0.3	13.2 ± 3.0	3.1 ± 1.0	18.3	7.0

[a] Values shown are the means over two blocks.
[b] Head blight ratings were determined as the product of the percentage of heads infected and the proportion of infected spikelets per infected head.
[c] No DON detected.
[d] Standard deviation of the mean.

TABLE 2. Estimates of variance components[a] of the data in Table 1, absolute and as percentage of the total variance, and narrow sense heritability for head blight and kernel deoxynivalenol (DON) content

	Head blight		DON	
	Absolute	In % of total variance		In % of total variance
$\hat{\sigma}^2_g$	97.18**[b]	38	0.79*[c]	18
$\hat{\sigma}^2_s$	68.20**	26	2.42**	56
$\hat{\sigma}^2_{g\times s}$	39.54*	15	0.55*	13
$\hat{\sigma}^2_b$	13.38**	5	0[d]	0[d]
$\hat{\sigma}^2_\tau$	0[d]	0[d]	0[d]	0[d]
$\hat{\sigma}^2_e$	40.28	16	0.57	13
$\hat{h}^2_n$	0.67		0.42	

[a] The indices g, s, b, τ, and e stand for genotype, strain, block, whole-plot error and subplot error, respectively.
[b] ** = significant at $P = 0.01$.
[c] * = significant at $P = 0.05$.
[d] Based on F statistics, computed from the split-plot analysis of variance, the estimate was not significantly different from 0.
[e] Heritability was estimated on a phenotypic mean basis averaged over blocks and strain.

TABLE 3. In vitro toxin production[a] of three strains of *Fusarium culmorum*

Strain	Zearalenone (mg/kg)	3-Acetyldeoxynivalenol (mg/kg)	Deoxynivalenol (mg/kg)
IPO 39-01	20	4.8	2.0
IPO 348-01	40	ND[b]	ND
IPO 436-01	1,728	1.5	0.5

[a] Strains were cultured for 4 wk on wheat grains (cultivar Grana) of 45% moisture content, at a temperature of 20 C.
[b] ND = nondetected.

with the approximate degrees of freedom calculated by the method of Satterthwaite (13). The broad sense heritabilities were estimated on a phenotypic mean basis averaged over blocks and strains as follows:

$$h^2 = \frac{\sigma^2_g}{\frac{\sigma^2_b}{r} + \sigma^2_g + \frac{\sigma^2_\tau}{r} + \sigma^2_s + \frac{\sigma^2_{g\times s}}{s} + \frac{\sigma^2_e}{rs}}$$

where σ^2_g stands for the variance component due to genotypes within populations, while σ^2_b, σ^2_s, and $\sigma^2_{g\times s}$ stand for the variance components due to blocks, strains, and genotype × strain, respectively. σ^2_e and σ^2_τ are variance components due to genotype × block (whole-plot error) and block × genotype × strain (subplot error), while r and s stand for number of blocks ($r = 2$) and strains ($s = 3$), respectively. As the SVP lines are considered to be homozygous, the broad sense heritability is equal to the heritability in narrow sense. Path analysis (17) was used to test a theoretical model for establishing possible causal relationships. A path coefficient is the standardized partial regression coefficient and estimates the strength of the relationship between cause X and effect Y. The indirect path coefficient between two variables is the sum of the product of the chain of path coefficients along all the indirect paths by which they are connected.

TABLE 4. The effect of inoculation of wheat by *Fusarium culmorum* on incubation period[a] (IP)[b], 1,000 kernel weight reduction[a], and yield reduction[a] in 10 different genotypes

Genotype	IP (days)	Kernel weight reduction[c] (%)	Yield reduction[c] (%)
SVP 72017-17-5-10	15.0	5.5	6.7
SVP 77078-30	16.0	3.5	2.7
SVP 75059-28	19.5	4.8	4.5
Saiga	10.2	6.5	7.4
SVP 72003-4-2-4	11.3	5.9	7.4
SVP 73030-8-1-1	10.0	19.5	26.2
SVP 75059-32	14.8	2.8	2.7
SVP 73016-2-4	8.7	12.8	27.6
SVP 73012-1-2-3	8.5	13.5	35.0
SVP 72005-20-3-1	9.5	22.4	29.1
LSD ($P = 0.05$)	2.9	12.0	17.8
Mean	12.4	9.7	15.6

[a] Results presented are mean values over three *F. culmorum* strains and two blocks.
[b] IP is the number of days from inoculation time to the appearance of first symptoms.
[c] Yield and 1,000 kernel weight are based on 25 randomly harvested leading tillers. Reduction of yield and 1,000 kernel weight was assessed by calculating the difference between an inoculated subplot and the mean of the three control subplots for each main plot. This difference was expressed as a percentage of the mean value of the control subplots.

RESULTS AND DISCUSSION

Date of inoculation had no influence on head blight incidence or DON concentration. There was no visible head blight infection in the control plots. Seeds harvested from the control plots did not show any *Fusarium* infection. Therefore kernels harvested from the control plots were considered not to contain any DON

TABLE 5. Correlations[a] between incubation period (IP), head blight, kernel deoxynivalenol (DON) content, kernel weight reduction and yield reduction, of 10 wheat genotypes after inoculation by *Fusarium culmorum*, for three different strains

		IP	Head blight	Kernel DON content	Kernel weight reduction
Head blight	IPO 39-01	−0.64*[b]			
	IPO 348-01	−0.78**[c]			
	IPO 436-01	−0.64*			
Kernel DON content	IPO 39-01	−0.69*	0.96**		
	IPO 348-01	−0.77**	0.55		
	IPO 436-01	−0.82**	0.65*		
Kernel weight reduction	IPO 39-01	−0.40	0.74*	0.75**	
	IPO 348-01	−0.81**	0.73*	0.83**	
	IPO 436-01	−0.73*	0.50	0.86**	
Yield reduction	IPO 39-01	−0.69*	0.96**	0.97**	0.67*
	IPO 348-01	−0.69*	0.68*	0.73*	0.96**
	IPO 436-01	−0.83**	0.39	0.87**	0.91**

[a] Correlations are based on the means over two blocks. df = 8.
[b] * = significant at $P = 0.05$.
[c] ** = significant at $P = 0.01$.

(12). As the control plots were free of *Fusarium*, cross-contamination was considered to be absent.

Head blight incidence and kernel DON concentration are shown in Table 1. Estimates for variance components, their relative importance in the total variance, and estimates for heritabilities are given in Table 2. The Fusarium head blight ratings varied from 0% in several subplots to 85% in a subplot consisting of SVP 73012-1-2-3 inoculated with strain IPO 39-01. For Fusarium head blight significant interactions existed between plant genotypes and Fusarium strains. Strain IPO 39-01 is a highly pathogenic strain, while IPO 348-01 and IPO 436-01 are of moderate pathogenicity (Table 1). The estimated h_n^2 of 0.67 (Table 2) shows that the proportion of the total variability among wheat genotypes that was due to additive genetic causes was high.

DON was found in 53 of the 60 samples, with a maximum of 48 mg/kg in seeds of SVP 72005-20-3-1 inoculated with strain IPO 39-01. Strain IPO 39-01 had a very high DON producing capacity (Table 1). For statistical analysis, nondetection (ND) was regarded as a concentration with a value of 0. For DON content, variance analysis revealed interactions between plant genotypes and *Fusarium* strains. Table 2 shows that $\hat{\sigma}^2_{g\times s}$ is of about the same level as $\hat{\sigma}^2_g$. The largest part of the variance is determined by $\hat{\sigma}^2_s$. Except for DON, no 3-ADON or any other trichothecene looked for was found. As DON can be formed by hydrolysis of 3-ADON or 15-ADON (10,28), there may have been 3-ADON in the samples in trace amounts below the detection limit. No ZEA was detected. Usually there has been little, if any, ZEA in *Fusarium*-infected wheat (11,12,25). In the in vitro experiment, however, besides DON 3-ADON and ZEA were also produced (Table 3). As was the case in the field, IPO 39-01 had the highest toxigenic potential in vitro.

For incubation period (IP), reduction in 1,000 kernel weight and yield, no significant interactions were found. The mean values for IP, 1,000 kernel weight reduction, and yield reduction are given in Table 4. For IP only the genotype effect accounted for the variation observed, the strain effect was not significant.

Because of the significant interactions between genotype and strains for head blight and for DON concentration, correlations between head blight, kernel DON content, kernel weight, and yield reduction were calculated for each strain separately (Table 5). The significant negative correlations between head blight and incubation period show that the more resistant the genotype, the longer is the incubation period. This relation, which does not depend on the pathogenicity of the strain, makes the incubation period a potentially useful resistance component for selection. The correlations between DON concentration and kernel weight reduction and yield reduction, respectively, were high.

In Figure 1, a path diagram illustrates possible causal relationships for each strain separately. In comparison with the direct path (p_1) between head blight and yield reduction, for the pathogenic strain IPO 39-01 the calculated indirect path ($p_{1,\text{indirect}}$), via

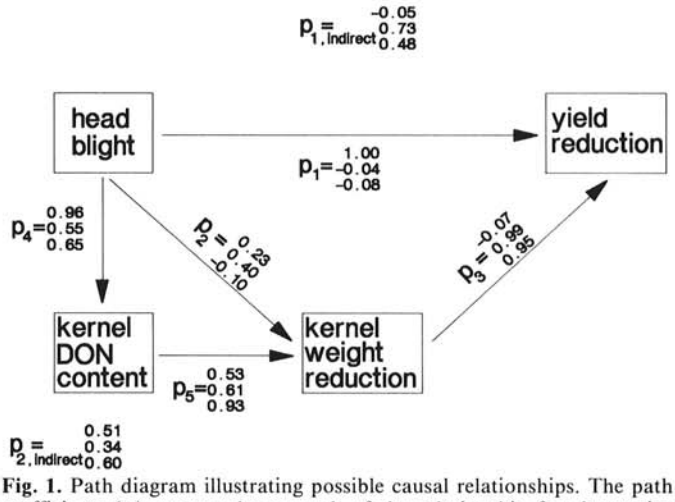

Fig. 1. Path diagram illustrating possible causal relationships. The path coefficients (p_i) express the strength of the relationship for the strains IPO 39-01 (top figure in each column of three), 348-01 (middle), and IPO 436-01 (bottom). The indirect path coefficient ($p_{i,\text{indirect}}$) between two variables expresses the strength of the relationship along all the indirect paths by which they are connected.

kernel weight reduction, is of minor importance. The yield reduction caused by strain IPO 39-01 must result from a lower kernel number. In contrast, for the two less pathogenic strains IPO 348-01 and IPO 436-01, the indirect path is more important; yield reduction is almost exclusively caused by a lower kernel weight. Comparison of the direct path (p_2) between head blight and kernel weight reduction with the indirect path ($p_{2,\text{indirect}}$), i.e., via DON, shows that for the strains IPO 39-01 and IPO 436-01 the path via DON ($p_{4\text{-}5}$) is more important than the direct path, while for IPO 348-01 p_2 and $p_{2,\text{indirect}}$ are about the same. For these three strains there appears to be a relation between DON and kernel weight reduction. If the measurement of DON is expressed on a per kernel basis, for p_2 the coefficients change into 0.98, 0.46, and 0.04 for IPO 39-01, IPO 348-01, and IPO 436-01, respectively. The coefficients $p_{2,\text{indirect}}$ change into −0.24, 0.27, and 0.46. In case of measuring DON on a per kernel basis, the indirect path between head blight and kernel weight reduction via DON is only important for IPO 436-01.

Whether there is a causal, phytotoxic effect of DON on kernel weight reduction cannot be proved in this study, but is possible. There have been few reports concerning the phytotoxic action of DON. A DON concentration of 2×10^{-5} M (6 mg/L) strongly inhibited the growth of tomato seedlings (1). Coleoptile tissue segments from some wheat cultivars would not grow at a DON concentration of 10^{-6} M (0.3 mg/L) (26). A retarded germination and growth of wheat was observed at 1.2×10^{-5} M DON (4

TABLE 6. Head blight to deoxynivalenol (DON) ratios[a] of 10 wheat genotypes inoculated with three strains of *Fusarium culmorum*

Genotype	*F. culmorum* strains		
	IPO 39-01	IPO 348-01	IPO 436-01
SVP 72017-17-5-10	0.4	3.8	2.5
SVP 77078-30	2.6	...[b]	6.3
SVP 75059-28	3.2	...	7.5
Saiga	1.1	7.5	4.3
SVP 72003-4-2-4	4.1	19.0	5.0
SVP 73030-8-1-1	1.8	3.9	1.3
SVP 75059-32	2.4	...	17.9
SVP 73016-2-4	1.5	36.3	5.0
SVP 73012-1-2-3	2.0	11.7	2.0
SVP 72005-20-3-1	1.7	17.2	4.1

[a] The values presented are based on means over two blocks.
[b] As no DON was detected, no ratio was calculable.

mg/L), while 4.8×10^{-5} M DON (14 mg/L) inhibited seedling growth completely (16). The phytotoxic effect of DON can be explained by the fact that DON is a very potent inhibitor of eukaryotic protein synthesis (26). Comparing the above data on phytotoxicity with the high concentrations of DON found for strain IPO 39-01 in the wheat kernels of the susceptible wheat genotypes, a phytotoxic effect of DON during development of the seed is imaginable.

From the data in Table 1, the head blight to DON ratio was calculated for each genotype-strain combination (Table 6). Within each strain, the extremes for head blight to DON ratios varied by a factor of 10. This corresponds to previous studies of ergosterol/DON ratios, where ergosterol was an index for estimating fungal biomass (12). The head blight to DON ratios did not show significant correlations with any of the variates in Table 5. The correlations between head blight and DON in Table 5, and the variation in head blight to DON ratios in Table 6 confirm that besides the resistance mechanisms that determine the head blight severity (14), there may be a second type of mechanism that influences kernel toxin content (12). Several papers have reported mechanisms that may influence kernel DON content. The *Fusarium* resistant wheat cultivar Frontana degraded 18% of ^{14}C deoxynivalenol added to fragmented embryo callus cultures after 72 hr of incubation, while the susceptible wheat cultivar Casavant converted only 5% of the added DON (9). Declines of DON in vivo have also been observed. In wheat spikes a maximum of 9.5 mg/kg of DON was found 6 wk after inoculation with one strain of *F. graminearum*, after which time the concentration decreased to 2.5 mg/kg by week 9 (11). In naturally infected wheat a decline in kernel DON content was observed from 1.56 mg/kg to 0.11 mg/kg in only 11 days' time (15).

Infection by *F. culmorum* must be minimized in order to prevent yield reduction and to produce a crop sufficiently mycotoxin free for human and animal consumption. Selection must focus both on resistance to infection and on mechanisms that either block synthesis or promote degradation of DON.

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