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Fusarium head blight and mycotoxin contamination of wheat, a review

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Summary

An infection of bread wheat by fusarium head blight contaminates the crop with mycotoxins, particularly deoxynivalenol (DON) and nivalenol (NIV). The toxicity and natural occurrence of these mycotoxins in wheat are reviewed. Based on 8 years data of fusarium head blight epidemics of wheat in the Netherlands, DON contamination of the grain was estimated. Fusarium head blight ratings averaged an infection of 1.7% of all spikelets; estimates for DON contamination averaged 0.9 mg kg^{-1}. Taking a guideline level for DON in uncleaned bread wheat of 2 mg kg^{-1}, in 1979 and 1982 a wheat crop was produced with estimated DON concentrations above the limit of tolerance. Human and animal exposure to mycotoxins in the Netherlands appears to be small but chronic. The information presented in this paper illustrates the need for an annual evaluation of the crop for fusarium head blight incidence and mycotoxin content, and the necessity of fusarium head blight resistant wheat cultivars.

Additional keywords: fusarium head blight, *Fusarium culmorum*, *F. graminearum*, wheat, mycotoxin, deoxynivalenol, nivalenol

Introduction

In wet summers, fusarium head blight can be an important problem in wheat. In the Netherlands, the species isolated from *Fusarium*-infected heads are mainly *F. culmorum* (W. G. Smith) Sacc. (teleomorph unknown) and *F. graminearum* Schwabe (teleomorph *Gibberella zeae* Schw.). Several forms of damage can be distinguished. An infection of the head by *Fusarium* can reduce kernel set and kernel weight, causing a yield reduction. Invasion of the kernel by *Fusarium* destroys the starch granules, storage proteins and cell walls, resulting in a poor quality product (Bechtel et al., 1985; Meyer et al., 1986). The embryo is usually not infected except in heavily invaded kernels. However, slightly infected kernels with apparently uninfected embryos exhibit reduced germination and vigour (Bechtel et al., 1985). As a result, a *Fusarium* epidemic can be a serious problem for seed production.

Various *Fusarium* species including *F. culmorum* and *F. graminearum* are capable of producing mycotoxins in crops. The most notorious mycotoxins of *F. culmorum* and *F. graminearum* in wheat are of the trichothecene class: deoxynivalenol (DON, vomitoxin), acetyldeoxynivalenol (the isomers 3-ADON and 15-ADON) and nivalenol (NIV) (Kurata and Ueno, 1984; Marasas et al., 1984), amongst which DON occurs most frequently. Quality loss because of mycotoxin content is an underestimated form of damage. Data on mycotoxin contents of West European wheat crops are scarce. The

objective of this study was to review the toxicity and occurrence of DON and NIV in wheat, and to estimate the DON contamination of wheat grown in the Netherlands based on 8 years data of fusarium head blight epidemics.

Toxicity of Fusarium toxins

Fusarium toxins are harmful to human and animal health. The toxicity of the isomers 3-ADON and 15-ADON is comparable, and is about twice that of DON (Mirocha et al., 1989). From the toxicity tests summarized by Joffe (1986), it can be concluded that NIV is about 10 times more toxic than DON.

Several papers report the effects on pigs fed on diets of DON-contaminated wheat (Bryden et al., 1987; Friend et al., 1986; Tobin, 1988). Swine diets of wheat naturally contaminated with *Fusarium* containing only 0.3 mg kg^{-1} DON resulted in decreased feed consumption and weight gain (Trenholm et al., 1981). Foster et al. (1986) concluded that 4.7 mg kg^{-1} DON added in pure form to the diet did not have any lasting effect on feed consumption and weight gain, while a diet of naturally contaminated wheat with the same concentration of DON had significantly lower feed intake and weight gain values. Apparently other toxic metabolites in *Fusarium* contaminated feed contributed to the poor performance. The reason for pigs refusing the feed is not known; there is little evidence that smell, taste, or a combination of the two is responsible (Friend and Trenholm, 1988). Toxin contaminated feed will lead to poor performance and health, and as a consequence economic losses.

Poultry can tolerate larger concentrations of DON in their diet (Bryden et al., 1987; Hamilton et al., 1985; Manley et al., 1988; Trenholm et al., 1981). Young chickens and turkey poults can tolerate diets that contain DON up to at least 5 mg kg^{-1} from wheat (Hamilton et al., 1985).

Ill effects can occur also in humans because of the dietary intake of DON or its metabolites. The acute symptoms of trichothecene poisoning are characterized by: skin irritation, food refusal, vomiting, diarrhea, hemorrhages, neural disturbance, miscarriage and death (Joffe, 1986; Kuiper-Goodman, 1985). Human toxicoses due to ingestion of mycotoxin-contaminated food are well-documented (Bhat et al., 1989). Chronic ingestion of small amounts of trichothecenes may result in an important secondary effect: the predisposition to infectious diseases through suppression of the immune system (Kuiper-Goodman, 1985; Miller and Atkinson, 1987). Precautions should be taken to avoid inhalation of mycotoxin-containing spores and dust, and direct skin contact with infected kernels (Trenholm et al., 1989).

Carry over of DON into food for human consumption

Deoxynivalenol present in the rations of Leghorn chickens, laying hens and broiler chickens at dietary levels of 5 mg kg^{-1} was not detected in eggs or tissues at a detection limit of 10 μg kg^{-1} tissue (El Banna et al., 1983). None of the eggs collected from White Leghorn hens given a DON-contaminated wheat diet (18 mg kg^{-1}) contained detectable quantities (detection limit: 10 μg kg^{-1}) of DON (Kubena et al., 1987). Studies on the presence of DON-derived residues in milk of sheep indicated that only trace amounts (< 10 μg l^{-1}) were transmitted following either oral or intravenous administration of the toxin (Prelusky et al., 1987).

Milling did not remove DON from naturally contaminated wheat (Lee et al., 1987), but the toxin was distributed in the milling fractions (Scott et al., 1983). On average, baking and cooking reduces DON in wheat or wheat products by 40% (Abbas et al., 1988; Besling et al., 1983; Carvajal et al., 1987; Isohata et al., 1986; Young et al., 1984). Therefore, wheat products made from DON-contaminated wheat will still contain DON.

Current regulations

'Agriculture Canada' advises that animals should be fed diets containing less than 1 mg kg^{-1} DON and that clean grain should be fed to pregnant and lactating animals (J.D. Miller, Plant Research Centre Ottawa, personal communication). Sweden has a regulation advising a maximum of 0.5 mg kg^{-1} in the diets of swine and 2 mg kg^{-1} in cows (Pettersson and Wennberg, 1988).

For human nutrition, a tolerable daily DON intake of 3.0 μg kg^{-1} body weight for adults, and 1.5 μg kg^{-1} body weight for infants was established in Canada (Kuiper-Goodman, 1985). The guideline level for DON in uncleaned wheat and in the flour portion of finished foods made from bread wheat are therefore 2 mg kg^{-1} and 1.2 mg kg^{-1}, respectively. For the USA the tolerance levels advised by 'Food and Drug Administration' are 2 and 1 mg kg^{-1}, respectively (Van Egmond, 1989). In Europe only Rumania and the USSR have specified tolerance levels for DON: 0.005 mg kg^{-1} food and 0.5 mg kg^{-1} wheat, respectively (Van Egmond, 1989).

Fusarium head blight epidemics in the Netherlands

Fig. 1 illustrates the fusarium head blight occurrence in the Netherlands for the years 1979-1986, expressed in prevalence, i.e. the percentage of infected farmer fields, and the percentage infected spikelets of affected fields. The data were collected from the EPIPRE program (Anon. 1985b, 1987b; Daamen et al., 1990) and illustrate the irregular pattern of epidemics over the years. The period 1979-1982 had a higher prevalence and heavier spikelet infections than the subsequent four years. As the relative acreage of cultivars grown did not change in the period 1979-1986 (Fig. 2; Anon., 1987c), different resistance levels cannot explain the pattern of epidemics. Also, there was no qualitative change in the use of fungicides and seed coatings (Anon., 1979; 1981; 1985a; 1987a). Correlations coefficients for fusarium head blight prevalence and percentage infected spikelets of affected fields versus weather conditions of the year concerned and the preceding year, and versus fusarium head blight prevalence and percentage infected spikelets in the preceding year are given in Table 1. As in the Netherlands winter wheat (which covered in 1979-1986 92% of the total wheat acreage) flowers in the second half of June, data on temperature, relative humidity and precipitation were gathered for the period 11 June – 11 July (Anon., 1978-1986). The positive correlation between fusarium head blight prevalence and prevalence in the preceding year cannot be ascribed to the occurrence of soil-borne inoculum, as the crop rotation system prevents this. However, *Fusarium* winters on infected debris of cereals, corn or weeds, and wind and rain will further spread conidia and mycelium (Logrieco et al., 1988; Sutton, 1982). Another explanation for the correlation might be that a high head blight prevalence resulted in seed infections of sowing-seed for the following year. Seed infection therefore could also explain the correlation between head blight prevalence and relative humidity

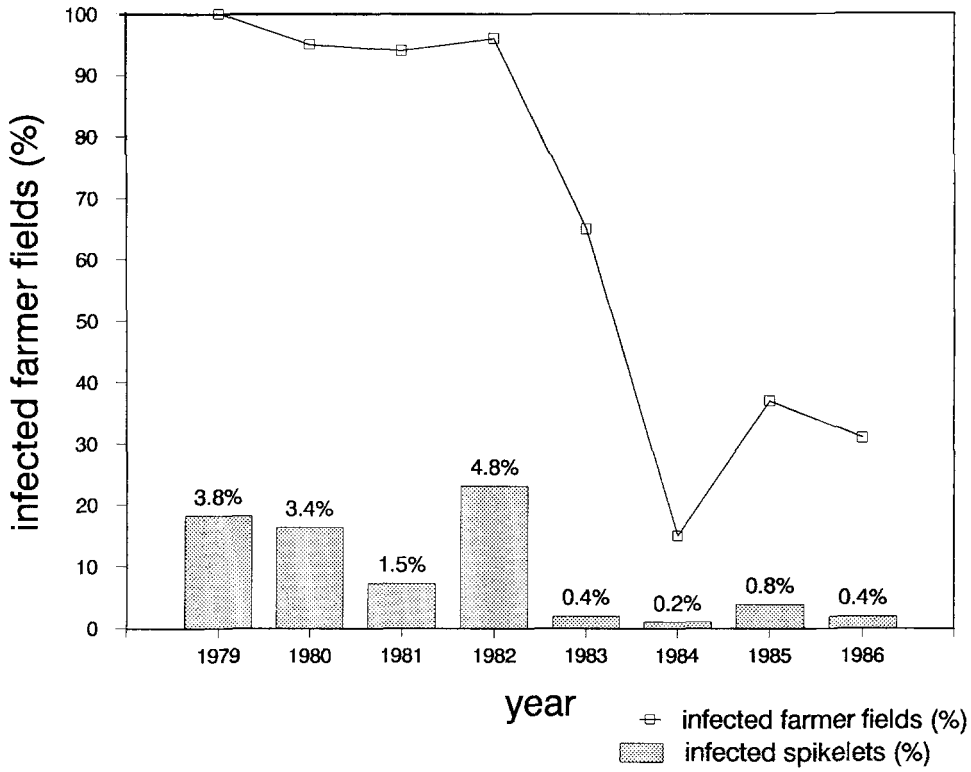

Fig. 1. Fusarium head blight prevalence (percentage infected farmer fields) and percentage infected spikelets of affected fields in the Netherlands for the years 1979-1986. Source: EPIPRE (Anon., 1985b; 1987b; Daamen et al., 1990)

of the period 11 June to 11 July in the preceding year (Table 1). The high correlation between percentage infected spikelets and precipitation in the period 11 June to 11 July is explained by the fact that *Fusarium culmorum* and *F. graminearum* are favoured by wet conditions (see also Daamen et al., 1990). Above all, they are typical splash-borne pathogens (Sutton, 1982).

Natural occurrence of Fusarium toxins

Table 2 illustrates the natural occurrence of DON in wheat samples of recent years. All the wheat samples were randomly collected from several growing areas, grain-elevators or graded lots. Average DON concentrations varied from 0.03 mg kg^{-1} to 1.78 mg kg^{-1}. Maximum DON concentrations varied from 0.09 mg kg^{-1} to 8.53 mg kg^{-1}. For the Netherlands data exist only for the crops of 1982, 1983 and 1984 (Besling et al., 1983; Besling, 1985; Tanaka et al., 1990). Samples from Austria, Hungary and Poland were also analyzed for 3-ADON, but none was detected (Ueno et al., 1985). Some of the authors cited analyzed the wheat samples also for the more toxic nivalenol (NIV). Sundheim et al. (1988) detected NIV in 53 out of 53 Norwegian samples with an average of 0.06 mg kg^{-1} and a maximum of 0.89 mg kg^{-1}. Ueno et al. (1985) detected NIV in

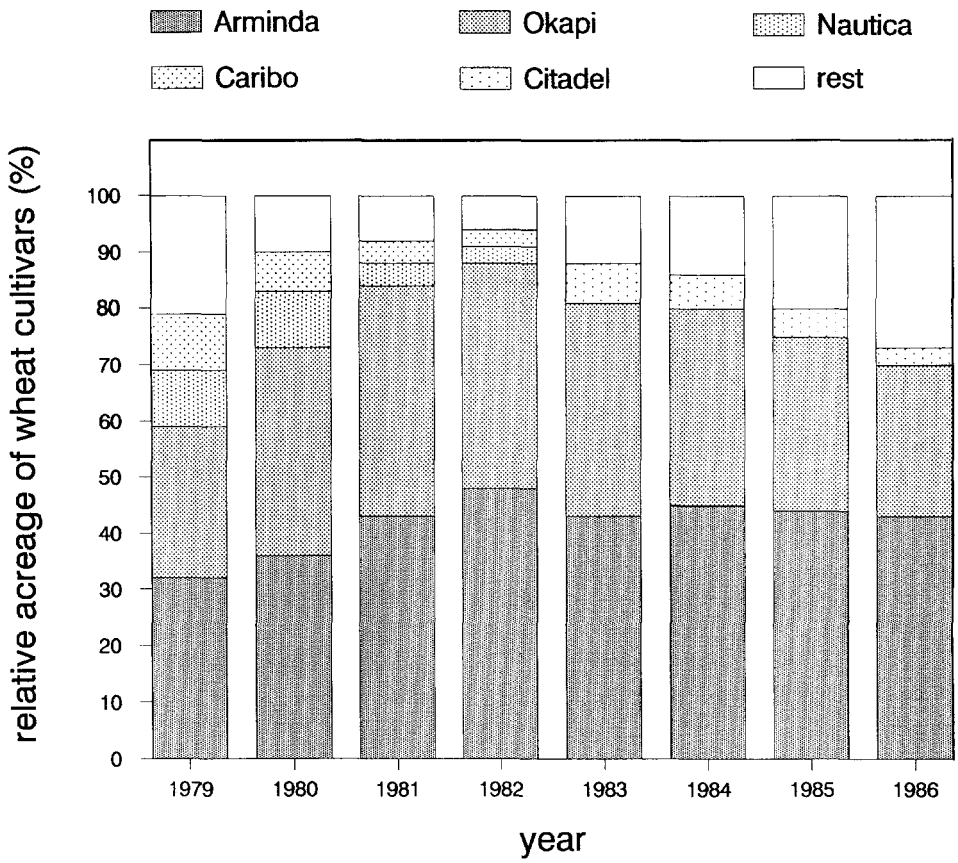

Fig. 2. Relative acreage of wheat cultivars in the Netherlands in the years 1979-1986.

4 out of 5 Austrian samples with an average of 0.02 mg kg^{-1} and a maximum of 0.04 mg kg^{-1}; in 37 out of 42 Polish samples an average of 0.05 mg kg^{-1} with a maximum of 0.35 mg kg^{-1} was detected. Tanaka et al. (1986) found NIV in 17 out of 31 UK samples with a mean of 0.10 and a maximum of 0.67 mg kg^{-1}. Tanaka et al. (1990) detected NIV in 12 out of 13 Dutch samples with a mean of 0.04 mg kg^{-1} and a maximum of 0.20 mg kg^{-1}. Osborne and Willis (1984) did not detect any NIV in UK samples at a detection limit of 0.05 mg kg^{-1}.

Lepschy-v. Gleissenthal et al. (1989) analyzed 'suspect' field samples of the 1987 Bavarian wheat crop for DON and NIV: 92 out of 106 DON-positive samples contained an average of 3.96 mg kg^{-1} with a maximum of 43.80 mg kg^{-1}. In samples with higher DON concentration NIV was detected with a concentration between 0.10 and 0.29 mg kg^{-1}.

From the fusarium head blight prevalence PRE (%) and the percentage infected spikelets of affected fields SPI, a fusarium head blight rating (%) can be calculated for each year as PRE × SPI/100. For the years 1979-1986 the fusarium head blight ratings averaged an infection of 1.7% of all spikelets. From the data reported by Snijders and Perkowski (1990) based on artificial inoculation of ten wheat genotypes with one pathogenic strain

Table 1. Correlation coefficients for fusarium head blight prevalence[a] and percentage infected spikelets of affected fields versus weather conditions[b] of the year concerned and the preceding year, and versus fusarium head blight prevalence and percentage infected spikelets in the preceding year.

	Fusarium head blight prevalence	Percentage infected spikelets
year concerned		
temperature	−0.16	−0.24
relative humidity	0.64	0.67
precipitation	0.70	0.77*
preceding year		
temperature	−0.48	−0.34
relative humidity	0.74*	0.29
precipitation	0.48	−0.03
prevalence	0.76*	0.52
percentage infected spikelets	0.67	0.16

[a] Based on the percentage infected farmer fields.
[b] Temperature (sum of the daily average over the 30 days), relative humidity (sum of the daily average over the 30 days) and total precipitation (mm) in the period 11 June to 11 July of the years 1978-1986. Presented values are means over 5 stations in the Netherlands, viz. De Kooy, Eelde, de Bilt, Vlissingen and Beek. Source: Anon., 1978-1986.
* Significant at $P \leq 0.05$.

of *F. culmorum* (IPO 39-01), a linear regression of DON concentration (mg kg^{-1}) on head blight rating (%) was estimated with a regression coefficient β of 0.54 (constant = 0; df = 9; R^2 = 93%). The yearly fusarium head blight ratings and the estimated DON levels in the kernels of the crops of the years 1979-1986 are given in Table 3. Estimates for DON contamination averaged 0.9 mg kg^{-1}. The estimates for kernel DON content in wheat grown in the Netherlands for the 1984 crop (Table 3) and the results from the toxin analysis (Table 2) both show low concentrations. Regarding the current regulations mentioned above, the estimated DON concentrations of the wheat crops of 1979 and 1982 were too high.

Estimates of DON-contaminated food intake in the Netherlands

During 1979-1986, the Netherlands produced annually an average of 946 276 tonnes of wheat, of which 23% was used for human consumption, 36% for feed and the rest for export and food aid (Anon., 1979-1987). Of the feed wheat, 55% was used for poultry and 39% for swine (Anon., 1988).

Indirect intake of DON by man through the consumption of animal tissues and produce from animals that were fed DON-contaminated feed is assumed to be essentially zero. Therefore, estimates of human exposure to DON are based on the direct intake by consumption of wheat products. Using a figure of 14.5 million people as the popula-

Table 2. Average and maximum deoxynivalenol contents (mg kg^{-1}) in positive samples from home-grown wheat.

Country	Crop year	Number of samples out of total	DON mean (mg kg^{-1})	DON max. (mg kg^{-1})	Reference
Australia	1983	20/25	0.14	1.7	Blaney et al., 1987
Austria	1984	3/4	0.36	0.91	Ueno et al., 1985
Canada	1980	36/77	1.06	8.53	Trenholm et al., 1983
	1986	8/8	0.27	0.45	Teich et al., 1987
FRG	1982-1983	3/5	0.11	0.22	Besling et al., 1983
	1984	5/9	0.06	0.09	Besling, 1985
France	1984	8/18	0.06	0.14	Besling, 1985
	1982-1983	22/25	0.09	0.27	Besling et al., 1983
Hungary	1984	2/2	0.70	1.29	Ueno et al., 1985
The Netherlands	1982-1983	12/18	0.05	0.16	Besling et al., 1983
	1984	8/20	0.06	0.14	Besling, 1985
	1984	13/13	0.12	0.51	Tanaka et al., 1990
Norway	1984	32/53	0.35	3.19	Sundheim et al., 1988
Poland	1984	13/42	0.09	0.31	Ueno et al., 1985
Sweden	1984	8/14	0.40	1.18	Pettersson et al., 1986
UK	1984	20/31	0.03	0.31	Tanaka et al., 1986
	1980-1982	32/200	0.20	0.40	Osborne & Willis, 1984
USA	1984	7/11	0.06	0.14	Besling, 1985
	1982-1983	11/13	0.22	0.50	Besling et al., 1983
	1982	31/33	1.78	5.50	Hagler et al., 1984

Table 3. Fusarium head blight rating (percentage infected farmer fields × percentage infected spikelets of affected fields/100) and estimated kernel DON content for the wheat crop of the years 1979-1986 in the Netherlands. DON concentration was estimated by a linear regression of DON mg kg^{-1} on head blight rating (%) with $\beta = 0.54$ and constant = 0.

Crop year	Fusarium head blight rating (%)	DON content (mg kg^{-1})
1979	3.80	2.05
1980	3.23	1.74
1981	1.41	0.76
1982	4.61	2.49
1983	0.26	0.14
1984	0.03	0.02
1985	0.30	0.16
1986	0.12	0.07

Table 4. Percentage of imported wheat that was imported from EC countries (EC%), and estimated daily DON intake per person in the Netherlands for the years following the wheat crops of 1979-1986.

Crop year	EC%	Intake per person per day (μg)
1979	63	129.5
1980	67	114.2
1981	67	49.9
1982	76	176.3
1983	73	9.7
1984	83	1.2
1985	89	12.6
1986	95	5.5

tion of the Netherlands, it can be calculated that the human consumption of wheat grown in the Netherlands is ± 16 kg per person. The total consumption of wheat flour per person in 1986 was 53 kg (Anon., 1988). Averaged over the years following the crops of 1979-1986, the sources of imported wheat used in the Netherlands were: 77% imported from EC countries (Table 4) (of which 69% from France), 20% from the USA and 3% from Canada (Anon. 1979-1987). Assuming (*i*) 53 kg total consumption of wheat flour per person, of which 70% imported, (*ii*) all EC-grown wheat had the estimated DON concentration of the Dutch home-grown wheat, (*iii*) the American and Canadian wheat contained no DON, and (*iv*) cooking or baking reduced DON by 40%, the daily intake of DON per person can be estimated (Table 4). Exposure to DON was small, but chronic. In the year following the crop of 1982 as much as 176.3 μg DON/person/day was consumed. Given the tolerable daily DON intake of 3 μg kg^{-1} body weight and a mean human body weight of 60 kg, the estimated daily intake in 1982 was about equal to the limit of tolerance advised in Canada and the USA.

Conclusions

This analysis indicates the need for more information on the problem of *Fusarium* mycotoxins in wheat grown in Western Europe. More epidemiological data should be collected to develop a better model of the relation between climatic factors, fusarium head blight infection and mycotoxin content. The irregular pattern of the fusarium epidemics has led to an underestimation of the potential danger of a toxin contaminated wheat crop. In consideration of human and animal health, inspection of the crop and harvest is necessary. Breeding for resistance to fusarium head blight is of the utmost importance.

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Samenvatting

Het van nature voorkomen van deoxynivalenol in tarwe in relatie tot aaraantasting door Fusarium-soorten

Aaraantasting van tarwe door *Fusarium culmorum* en *Fusarium graminearum* leidt tot vorming van mycotoxinen in het graan, waarvan deoxynivalenol (DON) en nivalenol (NIV) de belangrijkste toxinen zijn. In dit artikel wordt een overzicht gegeven van de toxicologische aspecten, en het voorkomen van deze toxinen in tarwe. Informatie over DON en NIV in tarwe in West-Europa is schaars. Gebaseerd op gegevens van *Fusarium* epidemieën in de jaren 1979-1986 wordt een schatting gegeven van de concentratie DON in Nederlandse tarwe. Rekening houdend met de herkomst en verwerking van tarwe, blijken zowel in dierlijk als menselijk voedsel lage concentraties DON chronisch voor te komen. Op basis van een maximaal toelaatbare dagelijkse dosis DON van 3 $\mu g\ kg^{-1}$ lichaamsgewicht is de schatting van de dagelijkse opname van DON in het jaar volgend op de oogst van 1982 net op de grens. Zowel een jaarlijkse inventarisatie van *Fusarium* aantasting en DON besmetting van het graan, als de ontwikkeling van *Fusarium*-resistente rassen zijn noodzakelijk.

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