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# Maize maturity and the development of gibberella ear rot symptoms and deoxynivalenol after inoculation

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**Abstract**

Development of gibberella ear rot disease symptoms and the accumulation of the mycotoxin deoxynivalenol (DON) in maize ears inoculated via the silk with *Fusarium graminearum* was determined at various times after inoculation. Ten hybrids ranging in maturity from early to late, were inoculated with a conidial suspension in 1993 and 1994 and harvested every 2 weeks for 14 weeks after inoculation. Disease symptom evaluations were conducted on all 10 hybrids; five of these hybrids were further analysed for DON concentrations. Disease symptoms reached a maximum and stabilized by 6 weeks after inoculation, approximately at physiological maturity (35% kernel moisture) for the early hybrids and the late dent stage of maturity for later hybrids. Deoxynivalenol accumulation was correlated with symptom development but did not stabilize at 6 weeks for all genotypes. Hybrid maturity did not influence symptom development or DON accumulation, but environment did. For the evaluation of hybrids, assessments of resistance to fungal invasion and mycotoxin accumulation based on symptom development could be made much earlier than the current 12-14 week harvest time commonly used in inoculated experiments.

## Introduction

The development of hybrid maize with improved resistance to gibberella ear rot (*Fusarium graminearum* Schwabe (sexual state: *Gibberella zeae* (Schw.) Petch) is a major goal of many breeding programes. This pathogen reduces maize yield and contaminates the grain with mycotoxins, such as deoxynivalenol (DON, vomitoxin), thus lowering grain quality. Deoxynivalenol induces emesis characterized by vomiting, feed refusal and decreased weight gain in swine (Prelusky et al., 1994; Vesonder et al., 1981), and is an immunosuppressant in livestock (Pestka and Bondy, 1990).

*Fusarium graminearum* enters into maize ears through two major routes: (1) by growth of mycelium, produced by germinated spores, down the silks to the kernels and cob (rachis); and (2), by entry through wounds created by insects and/or birds (Hesseltine and Bothast, 1977; Koehler, 1942; Sutton, 1982). Various inoculation techniques have been developed to screen maize genotypes for resistance to each mode of infection (Atlin et al, 1983; Chungu et al., 1996; Mesterhazy and Kovacs, 1986; Reid et al., 1996c; Schaafsma et al., 1993). Inoculation techniques enhance incubation and infection and overcome variability of infection during years when natural contamination is too low to identify genotypic differences. However, these techniques only measure resistance to an individual mode of infection.

Typically, resistance is assessed visually with the use of a rating scale of disease symptoms at normal grain harvest in late September or October (12–14 weeks after silking). Because these assessments are labour intensive, in large breeding nurseries or hybrid screening trials where many genotypes need to be assessed in a short period of time, it would be useful to know when disease symptoms have peaked and/or stabilized for optimal timing of assessments. As maize ears mature, the ability of *F. graminearum* to colonize decreases rapidly. Little infection will occur if silk and kernel inoculations are made later than 10 and 30 days after silk emergence, respectively (Reid et al., 1992; Reid and Hamilton, 1996a). The development

*Table 1.* Corn heat unit ratings (CHU) and relative days to silking of 13 hybrids in 1993 and/or 1994

| Hybrid | CHU[a] | Relative days to 50% silk | |
|---|---|---|---|
| | | 1993 | 1994 |
| Early | | | |
| Funk's G-4017 | 2400-2500 | 71 | 67 |
| Pioneer 3921 | 2500-2700 | 73 | 67 |
| Pioneer 3902 | 2500-2700 | 71 | 69 |
| Medium | | | |
| Pioneer 3790 | 2700-2900 | 76 | 73 |
| DeKalb DK403 | 2700-2900 | 79 | 72 |
| First Line H2343 | 2800-3000 | 76 | – |
| Funk's G-4153 | 2800-3000 | – | 74 |
| Late | | | |
| Cardinal MX320 | 3100-3400 | 77 | 76 |
| Dekalb DK535 | 3100-3400 | 83 | – |
| Hyland HL 2729 | 3300-3500 | 80 | – |
| Ferguson 8965 | 3300-3500 | 80 | 74 |
| Hyland HL2803 | 3300-3500 | – | 78 |
| Great Lakes GL582 | 3300-3500 | – | 78 |

[a] Corn heat unit rating from Ontario hybrid corn performance trials.

of disease symptoms in inoculated ears may also slow down as the ears mature. If so, assessment of disease symptoms could begin before traditional harvest times, especially for the earlier maturing genotypes.

Whereas visual symptoms are correlated to the concentration of DON in the kernels at harvest (Reid et al., 1996b; Reid et al., 1996c) when maize has been successfully infected with *F. graminearum*, it is not known if this relationship exists before harvest. Miller et al. (1983) measured fungal propagule counts, ergosterol levels and DON levels from 2 to 9 weeks after a toothpick kernel inoculation of a single maize hybrid. They reported that all factors measured increased rapidly up to 6 weeks, after which, propagule counts sharply decreased, ergosterol levelled off and DON decreased from 580 to 430 $\mu g^{-1}$. The decrease in DON was speculated, in part, to be due to metabolism of the toxin by host plant enzymes.

The objectives of this study were to determine: (1) the time at which visible symptoms of disease reach a maximum and stabilize; (2) the progress of DON accumulation in the kernels and its correlation to symptom development; and (3), the effect of genotype maturity on disease development and DON accumulation in susceptible maize ears inoculated with *F. graminearum* in the silk channel.

## Materials and methods

In 1993 and 1994 at the Central Experimental Farm in Ottawa, field experiments were designed as a 10 × 7 split plot with three replicates. Main plots consisted of 10 susceptible (based on inoculated trials) maize hybrids (Table 1) ranging in maturity from 2400 corn heat units (CHU) (FAO 200) to 3500 CHU (FAO 500). Seven of the hybrids were repeated across years with the exception of First Line H2343, Dekalb DK535, and Hyland HL 2729 which were not available in 1994 and were replaced with hybrids of similar maturity groupings (Funk's G-4153, Hyland HL2803 and Great Lakes GL582). Hybrid main plot units were divided into seven single-row sub-plot units consisting of seven harvest times (2, 4, 6, 8, 10, 12, and 14 weeks after inoculation). Each sub-plot unit consisted of 14 plants in a 3.8 m long row with 76 cm between rows. The primary ears of the centre 10 plants of each row were inoculated.

Inoculum was prepared as a macroconidial suspension of *F. graminearum*, prepared as previously described (Reid et al., 1992; Reid et al., 1996c) using a single isolate, DAOM180378, obtained from the Canadian Collection of Fungus Cultures, Agriculture and Agri-Food Canada, Ottawa, Ontario. Individual plants were inoculated once by injecting 2 ml of a $5 \times 10^5$ conidia/$ml^{-1}$ suspension into the silk channel of the primary ear approximately 6 days after silk emergence for each plot (Reid et al., 1992). After inoculation, humid conditions were maintained by overhead sprinkler irrigation with 2–5 mm water daily in the late afternoon for 4 weeks. Daily minimum and maximum temperatures and total rainfall were monitored by a weather station located at the research centre.

At each harvest date, 10 inoculated ears were hand-picked, husked, and the severity of ear rot symptoms was evaluated using a 7-class rating scale where 1= no infection, 2= 1–3%, 3= 4–10%, 4= 11–25%, 5= 26–50%, 6= 51–75%, and 7= >75% of the kernels exhibiting visible symptoms or signs of infection such as rot and pinkish coloured mycelial growth. For five of the ten hybrids (Funk's G-4017, Pioneer 3902, DeKalb DK403, Cardinal MX320, and Ferguson 8965), harvested ears were bulked within each row, hand-shelled and the cob (rachis) was discarded. A 100 g sub-sample was dried in an oven at 80 °C for 24 h to determine kernel moisture. All samples were frozen at −20 °C until DON analysis.

*Table 2.* Mean area under the disease progress curve (AUDPC) values (&-days) for disease severity and deoxynivalenol (DON) concentrations for maize hybrids silk channel inoculated with *Fusarium graminearum* in 1993 and 1994

| Hybrid | Disease severity[a] | | DON concentration[a] | |
|---|---|---|---|---|
| | 1993 | 1994 | 1993 | 1994 |
| Early | | | | |
| Funk's G-4017 | 349.9 e | 236.2 c | 6279.2 b | 379.9 a |
| Pioneer 3921 | 507.3 a | 302.3 abc | – | – |
| Pioneer 3902 | 487.9 ab | 344.5 ab | 27313.0 a | 923.4 a |
| Medium | | | | |
| Pioneer 3790 | 381.3 cde | 277.3 abc | – | – |
| DeKalb DK403 | 345.8 e | 315.4 abc | 6805.0 b | 2485.3 a |
| First Line H2343 | 373.9 de | – | – | – |
| Funk's G-4153 | – | 296.1 abc | – | – |
| Late | | | | |
| Cardinal MX320 | 346.7 e | 270.6 bc | 7862.4 b | 1014.8 a |
| Dekalb DK535 | 325.3 e | – | – | – |
| Hyland HL 2729 | 442.3 abc | – | – | – |
| Ferguson 8965 | 429.1 bcd | 357.0 ab | 12547.1 b | 2965.6 a |
| Hyland HL2803 | – | 282.4 abc | – | – |
| Great Lakes GL582 | – | 367.8 a | – | – |

[a] Means followed by the same letter for a given year and variable are not significantly different at the 0.05 probability level based on the Fisher (protected) least significant difference test. – = no DON analysis performed.

*Deoxynivalenol analysis*

For DON analysis, each bulked kernel sample from the 10 inoculated ears was mixed thoroughly to obtain a random distribution of the kernels and a 350 g subsample was freeze-dried for 5 days in a Pennwalt Stokes freeze-drier, after which a 50-g subsample was ground to a fine powder in a Retsch Ultra Centrifugal mill type ZM1 (Brinkman Instruments, Inc., Rexdale, Ontario) with a 0.75 mm wire mesh. From all samples, a 1– g subsample was used for DON analysis.

Deoxynivalenol was extracted in 5 ml of methanol/water (1:9) using end-over-end mixing for 1 h followed by centrifuging for 5 min at 2000 rpm in a table-top centrifuge, and filtering through a 0.22 Fm filter. The concentration of DON in the filtrates was determined by competitive direct-enzyme-linked immunosorbent assay (CD-ELISA) procedure using monoclonal antibodies (MABs) as described by Sinha et al., (1995) with slight modification.

*Statistical analyses*

For disease severity data, residual error terms were generated and tested for normal distribution using the Kolmogorov *D* statistic (SAS, 1985). Disease severity and DON concentrations were analysed separately by year, and error mean squares were tested for homogeneity to determine if data could be pooled over years. Univariate analysis of variance was conducted to assess the significance of block, hybrid, harvest time and interaction effects on mean disease ratings and DON concentrations. Pearson's correlation coefficients were calculated to determine the relationship between symptoms and DON concentration (SAS, 1985).

Area under the disease progress curve values (AUDPC) were calculated for each hybrid within each block and analysis of variance was used to test the significance of block and genotype effects for AUDPC values (Campbell and Madden, 1990).

## Results and discussion

In both years, residual error terms were found to be normally distributed ($P>0.05$). Error mean squares for both disease severity ratings and DON concentrations were heterogeneous; thus data were analysed separately for each year. In 1993, genotype effects were significant ($P<0.01$) for both disease severity and DON con-

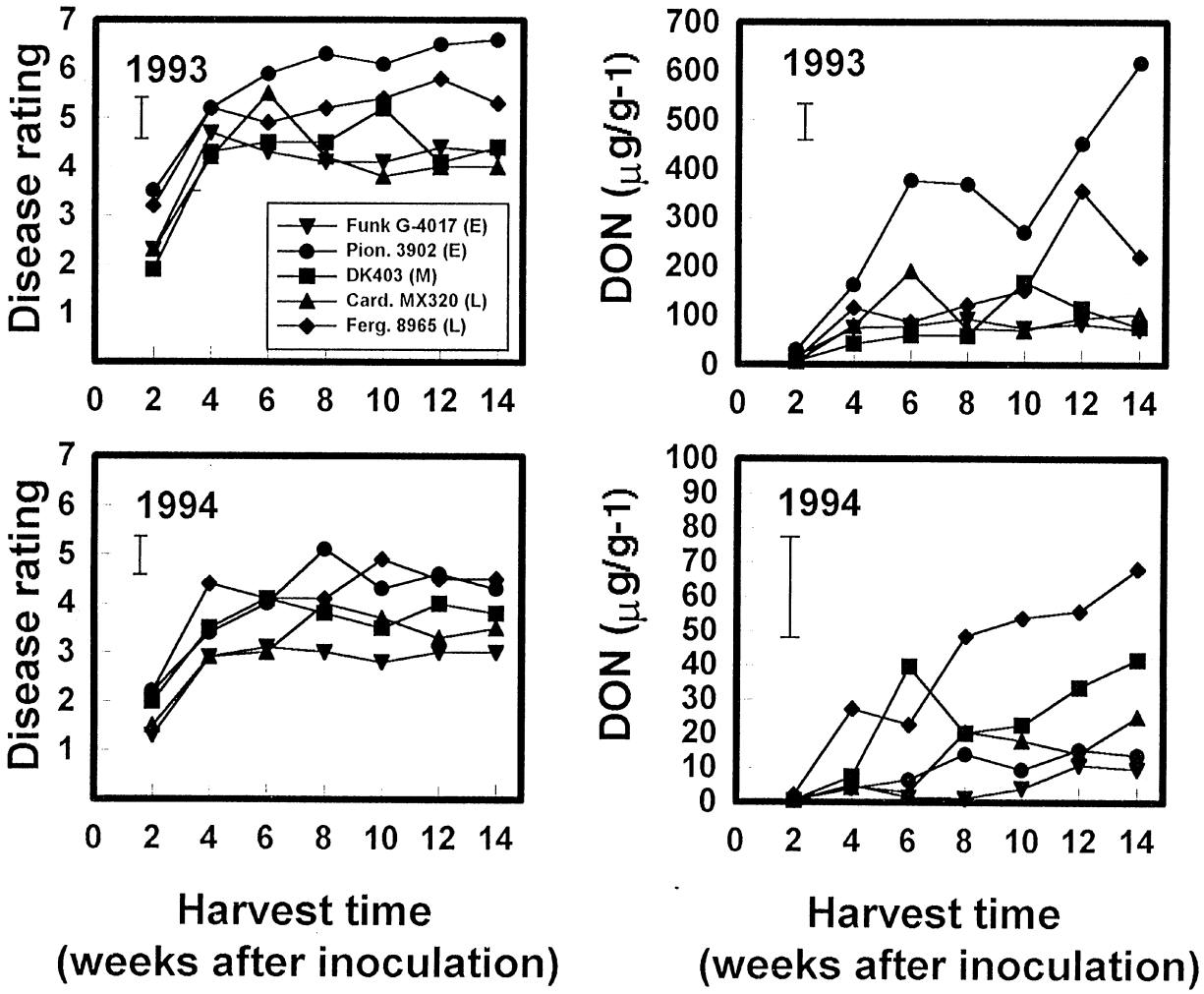


*Figure 1.* Disease progress curves for disease severity ratings and deoxynivalenol (DON) concentrations of five hybrids silk channel inoculated with *Fusarium graminearum* in 1993 and 1994. Disease severity ratings are on a 1-7 scale where 1= no infection and 7= >75% of the kernels visually moldy. Hybrid maturity groupings: E= early, M= medium and L= late. Bar indicates standard error of the difference = 0.88 (1993 disease severity), 74.3 (1993 DON), 0.78 (1994 disease severity) and 29.2 (1994 DON). Note: different y-axis scale for 1994 DON concentrations.

centration; however, there were no significant genotype effects for either variable in 1994. The effect of harvest time was significant ($P<0.01$) for both variables in both years. Hybrid X harvest time interaction effects were significant ($P<0.01$) only for DON concentrations in 1993. Analysis of the AUDPC values (Table 2) showed significant ($P<0.01$) hybrid effects for 1993 but not for 1994 for both disease severity and DON concentration.

All hybrids silked later in 1993 than in 1994 (Table 1). The months of May, July and September were warmer in 1994 than in 1993, resulting in higher accumulated CHU's for 1994 (3217 CHU) than 1993 (2924 CHU) compared to a 30-year average of 2980 CHU. Despite the early silking, cooler temperatures in August 1994 resulted in a delay in maturity for all hybrids. The time required to reach approximately 35% kernel moisture (physiological maturity) was approximately 2 weeks later in 1994 than in 1993.

Disease severity ratings and DON concentrations were higher in 1993 than in 1994 (Figures 1 and 2). There was a 7-to-10 fold decrease in DON levels from 1993 to 1994. Due to this difference, we repeated the DON analyses with additional sub-samples and found similar results.

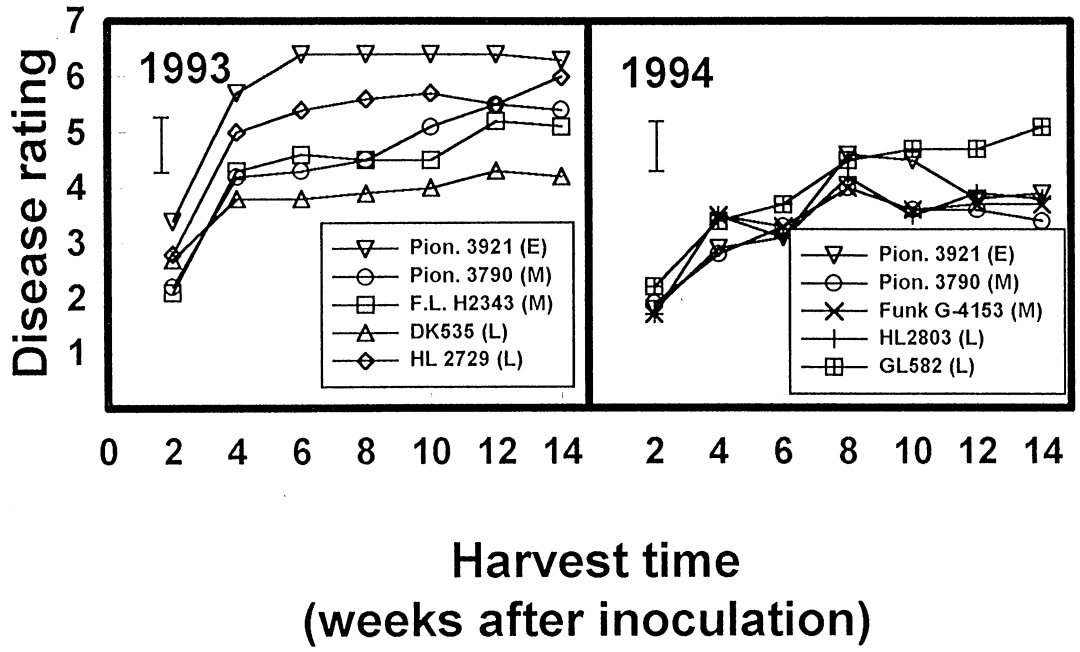


*Figure 2.* Disease progress curves for disease severity ratings of an additional eight hybrids silk channel inoculated with *Fusarium graminearum* in 1993 and/or 1994. Disease severity ratings are on a 1-7 scale where 1= no infection and 7= >75% of the kernels visually moldy. Hybrid maturity groupings: E= early, M= medium and L= late. Bar indicates standard error of the difference = 0.88 (1993 disease severity), and 0.78 (1994 disease severity).

Infections of *F. graminearum* require periods of warm temperatures(optimum 24–26 °C) and moderate rain especially during the months of July and August, i.e. during silking and early kernel development (Koehler, 1959; Sutton, 1982; Tuite et al., 1974). In this study, the earliest inoculations (on early maturing genotypes) were made by July 25; thus rainfall and temperatures in the month of August were more critical. Total rainfall in August 1993 (44.4 mm) was less than in 1994 (121.5 mm); however, the frequency of rainfall after inoculation was higher in 1993 (Figure 3). In both years, rainfall levels exceeded 60 mm over the 10–16 days after silk emergence previously reported by Lacey and Magan (1991) as optimal for *F. graminearum* growth. In our study, rainfall was not as important a parameter as temperature since the plots were irrigated.

Temperatures in August of 1994 were not optimum for *F. graminearum*. The minimum–maximum temperatures were 14.9–26.7 °C and 13.3–23.4 °C in 1993 and 1994, respectively (Figure 3). There were 24 days in August of 1993 with temperatures of 24 °C or greater compared to only 14 days in 1994. This may, in part, account for the lower disease ratings and DON concentrations in 1994, which was observed in other studies (Reid et al., 1996c; Reid et al., 1996d).

At the first sampling date, 2 weeks, all genotypes had some symptoms of infection (Figures 1 and 2), and some such as Funk's G-4017 and Pioneer 3902 had ratings greater than 3 (more than 10% of the ear had visual symptoms), suggesting that the fungus rapidly grew down the silk channel to infect the kernel. Disease severity increased rapidly from 2 to 4 weeks then began to stabilize and reached a maximum by 6 to 8 weeks for most genotypes in both years. Thus the first 4 weeks after inoculation are the most critical for determining the level of symptoms. Six weeks is the same time that Miller et al. (1983) observed a levelling off of ergosterol concentrations and a reduction in viable fungal propagule counts. Some genotypes appeared to have slight fluctuations in disease severity levels after 6 weeks, but most were not statistically significant. In 1994, the rate of disease progress from 2 to 4 weeks was much lower. This was probably due to the cooler temperatures in August of 1994 and could account for the failure of the fungus to reach high infection levels.

At 6 to 8 weeks, the early maturing hybrids had reached physiological maturity (35% moisture) while the later maturing hybrids were at the late dent stage.

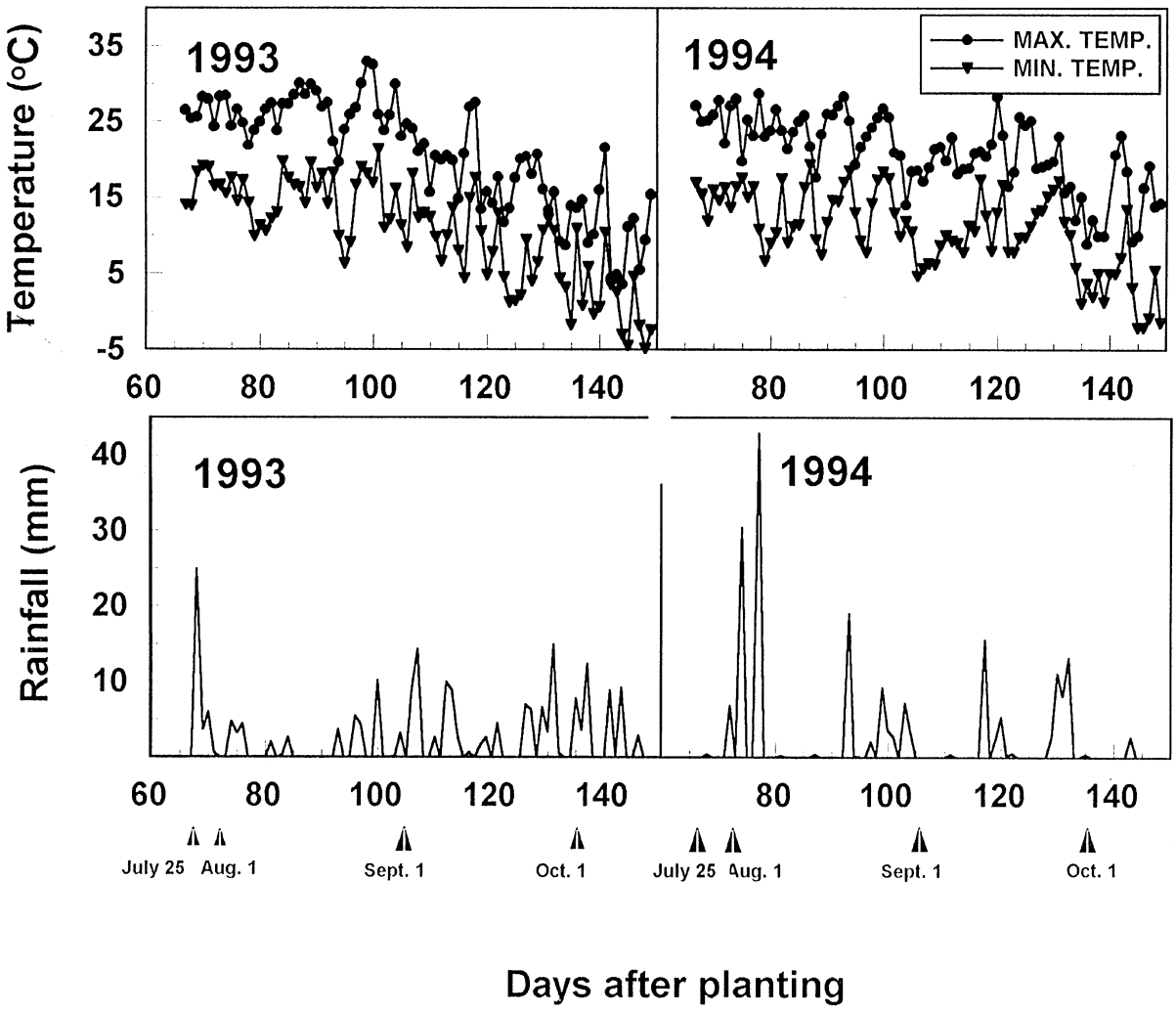


*Figure 3.* Minimum and maximum daily temperatures and total rainfall for 1993 and 1994 from July 25 (earliest inoculation) to October 15 (normal grain harvest).

The levelling off of the visual symptoms in this study and the results observed by Miller et al. (1983) would suggest that *F. graminearum* may not spread to additional kernels once drying and hardening of the kernels has started. With hardening, there would be an increase in the physical/morphological barriers to infection. This is the same observation we see when we try to inoculate maize ears by either the silk channel or through wounded kernels at later stages in ear development (Reid et al., 1992; Reid and Hamilton, 1996a). Miller et al. (1983) also proposed that the levelling off of fungal growth could be due to a lack of nutrients and/or moisture or end-product inhibition by DON.

Thus, for the purposes of screening maize for resistance based on visual symptoms from inoculation trials, harvesting can begin as soon as the plants reach physiological maturity or as early as 6 weeks after inoculation in some genotypes. This is a much earlier harvesting date than that currently used (12–14 weeks) in many screening programes.

Unlike disease symptoms, DON concentrations did not level off for all genotypes (Figure 1). In 1993, DON concentrations in Pioneer 3902 continued to increase with time and had a very high AUDPC value of 27313.0%-days. In contrast, in 1994, DON concentrations for this hybrid were low and constant. Ferguson 8965 had high DON levels in both years with consis-

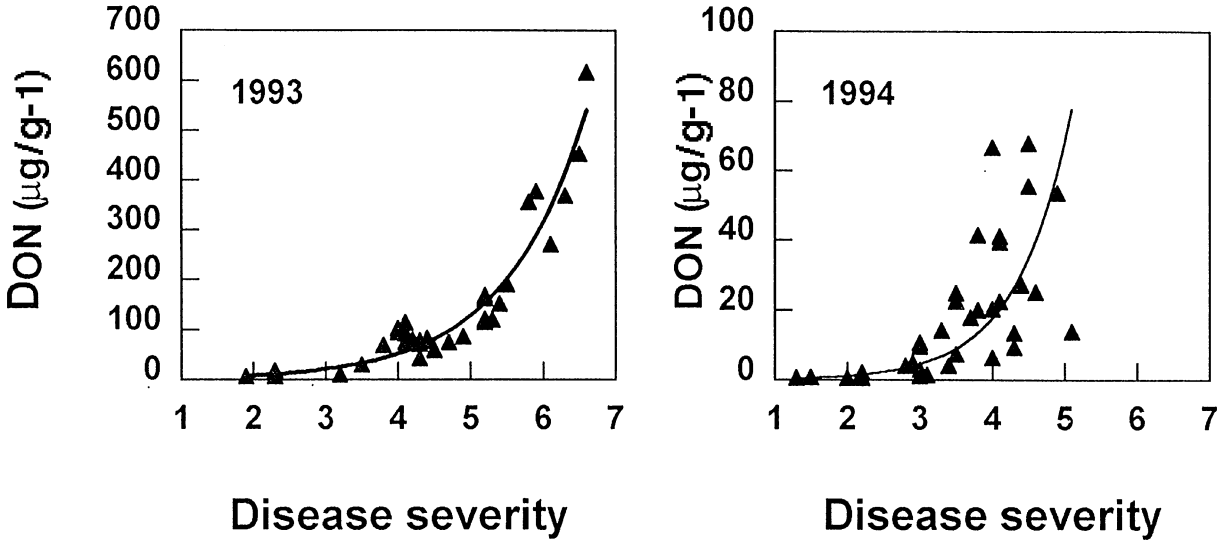


*Figure 4.* Correlation between disease severity and deoxynivalenol (DON) concentration. Disease severity ratings are on a 1-7 scale where 1= no infection and 7= >75% of the kernels visually moldy. r= 0.758 (1993), 0.682 (1994), $p<0.01$.

tently increasing levels in 1994 but a statistically significant decrease in levels at 14 weeks in 1993. Miller et al. (1983) observed a decrease in DON after 6 weeks, but only conducted measurements until 9 weeks. In our study, we observed the same trend for Pioneer 3902 and Cardinal MX320 in 1993 and for DeKalb DK403 in 1994. In Cardinal MX320, DON levels remained lower than the peak observed at 6 weeks; however, for the other two hybrids, DON levels increased again after 10 weeks to levels either the same as that observed at 6 weeks for DeKalb DK403 or significantly higher than at 6 weeks for Pioneer 3902. On average, DON concentrations either levelled off with time as did the disease symptoms or continued to increase with time.

Deoxynivalenol concentrations were significantly ($P<0.010$) correlated to visual disease symptoms for all genotypes (with r values ranging from 0.702 to 0.838), except Pioneer 3902 in 1994. For a given genotype, an increase in disease severity at a given harvest time was associated with an increase in DON concentration (Figure 4). As in our previous studies (Reid et al., 1996d), this relationship was exponential. Once infection rates were higher than 4 (25% of the ear showing symptoms), very high concentrations of DON could occur.

There was no trend for certain maturity groups (early, medium or late) to be more or less susceptible than others. The only maturity group in which AUDPC values for disease severity were not significantly different within the group was that of medium maturity (Table 2). The time period from inoculation to physiological maturity was longer for the later maturing hybrids (Table 3). In theory, this would imply that the fungus would have more time to infect and develop symptoms, and DON levels would be higher. However, this was not observed in our study. This was probably due to a gradual decrease in optimum fungal growth temperatures during August. For example, although Ferguson 8965 took almost twice as long to go from silking to 35% moisture as Funk's G-4017 (57 vs. 29

*Table 3.* Number of days from silking to physiological maturity (35% kernel moisture) and the number of those days with optimum fungal growth temperatures (greater than 24 °C) for five hybrids in 1993 and 1994

| Hybrid | Days from inoculation to maturity | | Number of days ≥ 24 °C | |
|---|---|---|---|---|
| | 1993 | 1994 | 1993 | 1994 |
| Early | | | | |
| Funk's G-4017 | 29 | 63 | 23 | 20 |
| Pioneer 3902 | 43 | 57 | 29 | 21 |
| Medium | | | | |
| DeKalb DK403 | 57 | 74 | 24 | 19 |
| Late | | | | |
| Cardinal MX320 | 59 | 73 | 24 | 17 |
| Ferguson 8965 | 57 | 75 | 24 | 18 |

days) in 1993, the number of days with temperatures of 24°C or greater were about the same for both hybrids (Table 3). These results show that hybrid maturity did not influence disease development. This result agrees with our other studies (Reid et al. 1992; Reid et al., 1996d).

## Conclusions

The results of this study indicate that assessment of maize for resistance to *F. graminearum* after inoculation of the silk channel can begin as early as 6 weeks after inoculation or as soon as the plants approach physiological maturity. By this time, symptom development reached a maximum and stabilized. Depending on genotype, DON accumulation may or may not have stabilized by 6 weeks; however, DON concentrations were correlated to visual symptoms. Genotype maturity does not have an effect on how soon symptoms stabilize or the severity of symptoms or accumulation of DON, so earlier maturing genotypes can be harvested before later maturing genotypes.

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