# Reaction of Maize Hybrids to Ear Rot Caused by *Fusarium graminearum* Schwabe

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**Abstract**

Ear rot caused by *Fusarium graminearum* Schwabe (teleomorph stage: *Gibberella zeae* (Schwein.) Petch) is a destructive disease of maize. In our experiment we tested twenty maize hybrids. Two inoculation techniques differing in the way of application of a macro-conidial suspension, were evaluated for their effectiveness in assessing maize resistance to ear rot. Based on the results of one season, highly significant differences in sensitivity to Fusarium ear rot between genotypes for all variants under mist irrigation and without mist irrigation, were detected.

**Keywords**: fusariosis; maize; susceptibility; irrigation

## INTRODUCTION

Ear rot caused by *Fusarium graminearum* Schwabe is a destructive disease of maize (*Zea mays* L.) in many parts of the world, including maize-growing regions of Slovakia (ŠROBÁROVÁ *et al*. 2000). The fungus causes a pronounced reddish discoloration of the tip of the ear. The disease has economic implications and the infection may lead to contamination of grain with mycotoxins (NADUBINSKÁ *et al*. 2002). Development of resistant maize hybrids is the best way to control disease (REID & HAMILTON 1996). For this purpose artificial inoculation methods to test for *Fusarium* ears rot were developed (CHUNGU *et al*. 1996). Based on the results of one year we assessed genotypic differences in susceptibility to infection after silk and kernel inoculation.

## MATERIALS AND METHODS

We selected 20 genotypes of maize for testing maize ear rot (in alphabetic order): Ankora, AUS1, AUS2, AUS3, AV40299, Benicia, DK312, JASPE, Leona, Lucia, Monalisa, Norika, Pactol, Pavla1, Pavla2, TA10298, TA20188, TA30198, TA30299, Tina. These genotypes were coded.

The experiment was realised at the experimental field of the IFA-Tulln (Austria) in 2001. The experiment was grown in two blocks each with two replications, one block was with mist irrigation, the second block without it. For the experiment we used *Fusarium graminearum* (IFA66) which was isolated from maize. The inoculum was prepared according to the method described by MESTERHAZY and ROWAISHED (1977) on mung bean medium. Inoculum was stored in refrigerator. Artificial inoculation was realised in two blocks with different methods in two variants. In the first blocks without mist irrigation inoculation was done by wounding 4 kernels with simultaneous inoculation with *F. graminearum* $10^5$ macroconidia/ml (kernel resistance, var. 4). The effect of the variant was compared with natural infection (var. 3, treatment without artificial inoculation). All treatments were applied 14 days after 50% silking of each genotype. In

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the second block of the experiment mist irrigation was used to promote infection. The following treatments were used: inoculation with spraying *F. graminearum* ($5 \times 10^5$ macroc./ml, var. 2). The variant was compared with natural infection under mist irrigation (var. 1, treatment without artificial inoculation). These treatments were used 1 week after 50% silking of the individual maize lines. In the experiments under natural infection conditions and in the experiments designated to evaluate silk resistance, disease incidence (PDE, percentage of diseased ears) and disease severity of the diseased ears (PDSDE) were recorded. With these two parameters disease severity over all ears (PDSAE) was calculated as follows:

$$\text{PDSAE (in \%)} = \frac{\text{PDE (in \%)} \times \text{PDSDE (in \%)}}{100}$$

Disease severity over all ears equals the percentage of *Fusarium* damaged kernels in the harvest. Data were evaluated by analysis of variance (SAS Program ANOVA and GLM) (LEMMENS 1999).

## RESULTS AND DISCUSSION

Differences in reaction of the maize lines towards all variants was statistically highly significant in all cases. Silk resistance was tested under pure natural infection (Table 2 var. 3), under mist irrigation only (Table 1 var. 1) and after spraying the inoculum on the silks (Table 1 var. 2). Natural infection was already high (mean PDE = 28%). After application of mist irrigation PDE increased only marginally (mean PDE = 30%). The application of mist irrigation and *Fusarium* inoculum on the silks induced a further increase of PDE to 62%. The some conclusion was drawn for the PDSDE and the PDSAE. The genotype VG17 was the most resistant: it showed a low PDE- and a very low PDSDE-value. Kernel resistance was measured after wounding of the kernels (Table 2 var. 4). Most of the ears were diseased after this treatment which resulted in very heavy infections. The genotype VG15 was the most resistant (PDSAE = 20.8%) while VG10 reacted very susceptible (PDSAE = 90.6%).

Table 1. Results of inoculation by spraying of a conidial suspension on the silks (var. 2) and natural infection (var. 1) in the block with mist irrigation (PDE, % of disease ears; PDSDE, % of disease severity of the diseased ears; PDSAE, % of disease severity over all ears)

| Genotype | PDE | | PDSDE | | PDSAE | |
|---|---|---|---|---|---|---|
| | var. 1 | var. 2 | var. 1 | var. 2 | var. 1 | var. 2 |
| VG1 | 21 | 64 | 1.9 | 1.9 | 0.2 | 1.2 |
| VG2 | 41 | 90 | 4.9 | 13.1 | 2.0 | 11.6 |
| VG3 | 32 | 81 | 6.0 | 10.9 | 1.5 | 8.8 |
| VG4 | 52 | 73 | 1.9 | 14.0 | 1.1 | 10.6 |
| VG5 | 15 | 64 | 3.4 | 12.3 | 0.6 | 7.5 |
| VG6 | 33 | 31 | 0.8 | 2.5 | 0.2 | 0.8 |
| VG7 | 20 | 76 | 5.3 | 18.8 | 1.0 | 14.3 |
| VG8 | 22 | 48 | 3.3 | 4.2 | 0.8 | 1.9 |
| VG9 | 23 | 49 | 2.2 | 5.3 | 0.6 | 2.6 |
| VG10 | 26 | 84 | 6.0 | 10.2 | 1.3 | 8.3 |
| VG11 | 63 | 89 | 4.9 | 7.5 | 3.1 | 6.5 |
| VG12 | – | 20 | – | 91.7 | – | 18.3 |
| VG13 | 44 | 68 | 6.8 | 37.7 | 2.9 | 24.7 |
| VG14 | 22 | 83 | 6.2 | 12.3 | 1.3 | 10.0 |
| VG15 | 26 | 36 | 4.6 | 7.2 | 1.1 | 2.7 |
| VG16 | 29 | 63 | 1.8 | 7.7 | 0.5 | 4.7 |
| VG17 | 21 | 23 | 0.4 | 1.2 | 0.1 | 0.3 |
| VG18 | 18 | 46 | 1.5 | 5.7 | 0.3 | 2.5 |
| VG19 | 29 | 62 | 0.5 | 8.4 | 0.1 | 5.1 |
| VG20 | 38 | 96 | 4.9 | 8.8 | 1.8 | 8.5 |
| Mean | 30 | 62 | 3.6 | 14.1 | 1.1 | 7.6 |

Table 2. Results of inoculation by wounding and application of a conidial suspension into the kernels (var. 4) and natural infection (var. 3) in the block without mist irrigation (PDE, % of disease ears; PDSDE, % of disease severity of the diseased ears; PDSAE, % of disease severity over all ears)

| Genotype | PDE | | PDSDE | | PDSAE | |
|---|---|---|---|---|---|---|
| | var. 3 | var. 4 | var. 3 | var. 4 | var. 3 | var. 4 |
| VG1 | 31 | 100 | 0.5 | 46.8 | 0.1 | 46.8 |
| VG2 | 41 | 96 | 3.2 | 48.9 | 1.4 | 46.3 |
| VG3 | 34 | 99 | 2.8 | 62.8 | 0.6 | 62.1 |
| VG4 | 36 | 98 | 0.8 | 59.3 | 0.3 | 57.9 |
| VG5 | 8 | 100 | 3.7 | 47.3 | 0.3 | 47.3 |
| VG6 | 21 | 99 | 0.8 | 37.5 | 0.2 | 37.0 |
| VG7 | 22 | 98 | 3.8 | 35.6 | 0.8 | 35.0 |
| VG8 | 15 | 99 | 1.1 | 52.3 | 0.2 | 51.6 |
| VG9 | 22 | 96 | 1.4 | 58.1 | 0.3 | 55.3 |
| VG10 | 19 | 100 | 0.8 | 90.6 | 0.2 | 90.6 |
| VG11 | 86 | 100 | 2.7 | 66.2 | 2.4 | 66.2 |
| VG12 | 2 | 100 | 20.0 | 79.6 | 0.4 | 79.6 |
| VG13 | 57 | 96 | 12.2 | 74.8 | 7.1 | 71.4 |
| VG14 | 34 | 100 | 2.7 | 80.5 | 0.9 | 80.5 |
| VG15 | 16 | 100 | 3.9 | 20.8 | 0.7 | 20.8 |
| VG16 | 29 | 100 | 2.8 | 55.3 | 0.8 | 55.3 |
| VG17 | 14 | 99 | 2.4 | 42.4 | 0.4 | 41.9 |
| VG18 | 22 | 100 | 0.4 | 76.7 | 0.1 | 76.7 |
| VG19 | 32 | 100 | 0.6 | 45.4 | 0.2 | 45.4 |
| VG20 | 26 | 100 | 1.3 | 71.9 | 0.4 | 71.9 |
| Mean | 28 | 99 | 3.4 | 57.7 | 0.9 | 56.9 |

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