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Genotype × strain interactions for resistance to *Fusarium* head blight caused by *Fusarium culmorum* in winter wheat

C. H. A. Snijders and F. A. Van Eeuwijk

Centre for Plant Breeding Research (CPO), P.O. Box 16, NL-6700 AA Wageningen, The Netherlands

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Summary. In 3 consecutive years, a set of 17 winter wheat genotypes, representing a wide range of *Fusarium* head blight resistance, was inoculated with four strains of *Fusarium culmorum*. *Fusarium* head blight ratings were analyzed. The interaction between genotypes, strains, and years was described using a Finlay-Wilkinson model and an Additive Main effects and Multiplicative Interaction effects (AMMI) model. The interaction consisted primarily of a divergence of genotypical responses with increasing disease pressure, modified by genotype-specific reactions in certain years. The divergence was mainly caused by one very pathogenic strain. The *Fusarium* head blight resistance in this study can be described as horizontal resistance in terms of Vanderplank, with the exception of three genotypes selected from one particular cross that showed a 'strain-year combination' dependent resistance which was ineffective in 1 year.

Key words: *Fusarium* head blight resistance – Plant breeding – *Fusarium culmorum* – Genotype × environment interaction – Wheat – AMMI model

Introduction

In The Netherlands, *Fusarium* head blight in wheat (*Triticum aestivum* L.) is predominantly caused by *Fusarium culmorum* (W. G. Smith) Sacc. and *Fusarium graminearum* Schwabe. Both species of *Fusarium* have a worldwide distribution as soil inhabitants and cause, in addition to head blight root, foot and stem rot. Both fungi are generalists infecting cereals and a large number of other hosts, including corn, peas, and alfalfa (Booth 1971). Both *Fusarium* spp. are nonobligate parasites and facultative saprophytes. Variation for *Fusarium* head blight resistance in wheat exists (Atanasoff 1924; Parry et al. 1984; Schroeder and Christensen 1963; Snijders 1990). The resistance found until now is of a moderate form. Complete resistance has not been demonstrated. It is not clear whether or not *Fusarium* head blight resistance can be described as horizontal resistance in terms of Vanderplank (1984), i.e., whether or not the variation in resistance in the population of the host is independent of the variation in the population of the pathogen.

In a 3-year study of *Fusarium* head blight resistance, Mesterhazy (1984) found significant genotype × isolate interaction each year between 11 isolates of *F. graminearum* and two wheat genotypes. Using two isolates of *F. graminearum* and two isolates of *F. culmorum* for artificial inoculation of 21 genotypes, Mesterhazy (1988) found significant interactions for genotype × *Fusarium* species and genotype × *Fusarium* isolate. However, interaction patterns were not stable over experiments, and genotype ranking was only slightly influenced by the isolates. No evidence has been found for the occurrences of races of *Fusarium culmorum* or *F. graminearum* adapted to different wheat genotypes. Also, in studies with *F. graminearum* in corn ear rot tests, significant but inconsistent isolate × genotype interaction patterns were found (Atlin et al. 1983; Mesterhazy 1982; Mesterhazy and Kovacs 1986). However, large genotype rank reversals did not occur. This phenomenon is not restricted to *Fusarium* of wheat and corn. Environmental lability of interactions between wheat cultivars and isolates were also reported for *Cercosporella herpotrichoides* (Scott and Hollins 1977).

In an initial study of *Fusarium culmorum* in wheat, a significant host genotype × pathogen strain interaction was observed (Snijders 1987). The experiments were continued to investigate the consistency of the interaction patterns, i.e., whether or not strain-specific resistance to *Fusarium culmorum* head blight in wheat exists.

Materials and methods

Host and pathogen

A set of 17 winter wheat cultivars and SVP[1]-lines was composed, representing the whole range of *Fusarium* head blight susceptibility based on data available in 1985. In field trials in 3 consecutive years this set was tested for resistance to *Fusarium* head blight. Ten strains taken from monospore cultures of isolates of *Fusarium culmorum*, collected in The Netherlands, were prescreened for pathogenicity in the glasshouse. Two nonpathogenic strains were discarded, and from the remaining strains four were drawn: IPO 39-01, IPO 329-01, IPO 348-01, and IPO 436-01, originating from isolations from a grain of seed, culm, head, and leaf sheath, respectively. The lyophilized strains are deposited at the Research Institute for Plant Protection (IPO), Wageningen. Each year, conidiospores for inoculation were produced in 1-l Erlenmeyer flasks containing 250 ml sterilized cereal seeds: the 1st year, wheat seeds (cultivar Arminda), the next 2 years, a wheat (Arminda) and oat (bulk) seed mixture (3:1). A lyophilized strain was used as starting inoculum. The cultures were incubated in darkness at 25 °C for 2 weeks, followed by 3-week incubation at 5 °C. To prepare spore suspensions, conidia were washed from the kernels with water. Since wheat is most susceptible to *Fusarium* head blight at anthesis (Schroeder and Christensen 1963), experimental inoculations were made at that time. The spore suspensions were applied at 1 l/10 m^2. To ensure a high relative humidity during the nights after inoculation, the field was sprinkled in the evening for 1 h each day over a period of 2 weeks. Head blight ratings were determined as the product of the percentage of heads infected and the proportion of infected spikelets per infected head (Snijders and Perkowski 1990). In all experiments, interplot interference was prevented.

Field trial 1986

On November 22, 1985, seeds were sown in sandy soil in Wageningen 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 main plot, consisting of one genotype, was divided into subplots of 0.90×0.75 m, over which the strains of *F. culmorum* were randomized so that the experimental subplots were separated from each other by border subplots of the same size. Further details are described in Snijders and Perkowski (1990). *Fusarium* head blight was assessed 26 days after first inoculation.

Field trial 1987

On November 4, 1986, seeds were sown in Flevoland in clay soil at a standard seed density of 330 seeds/m^2 in rows 0.25 m apart. A split-plot design was established, with three blocks. The four strains of *Fusarium culmorum* were randomized over the main plots. A distance of at least 4 m between the main plots prevented interplot interference. The main plots were divided into subplots of 2.00×0.75 m, over which the wheat genotypes were randomized. On June 25, when 30% of the wheat genotypes flowered, all genotypes were inoculated. The spore concentrations varied from 25,000 to 250,000 spores per milliliter. At the time when 100% of the genotypes flowered, July 2, a second inoculation was done. For the inoculation a spraying machine was used, which sprayed from 0.3 m above the crop. Spore concentrations varied from 25,000 to 250,000 spores per milliliter. On July 21, 26 days after the first inoculation, head blight was assessed. Observations were made on culm length.

1 The Foundation for Agricultural Plant Breeding (SVP) is now part of the Centre for Plant Breeding Research (CPO)

Field trial 1988

On November 10, 1987, seeds were sown in Flevoland in clay soil. The same design was used as in field trial 1987. Each subplot consisted of a hill plot (Ø 0.25 m) seeded with 3 g seeds, at 0.5 m apart. Experimental inoculation was done on June 2, when 30% of the wheat genotypes flowered, and repeated on June 9 and June 16, by which time 100% of the genotypes flowered. For the inoculation a spraying machine was used. The spore suspensions had a concentration of 250,000 spores per milliliter. On June 30, 28 days after the first inoculation, head blight was assessed. Observations were made on time of anthesis and culm length.

Statistical analysis

For the analysis of variance of *Fusarium* head blight ratings in the three consecutive experiments, the split-plot model with fixed effects was used (Steel and Torrie 1981). For a description of the interactions, a Finlay-Wilkinson regression model (Finlay and Wilkinson 1963) and an Additive Main effects and Multiplicative Interaction effects (AMMI) model (Bradu and Gabriel 1978; Gauch 1988; Kempton 1984; Zobel et al. 1988) were used.

Results and discussion

As no head blight was observed in control and border plots, interplot interference was assumed to be absent. Inoculum concentration for individual inoculations and total amount of inoculum had no influence on the *Fusarium* head blight ratings. No significant correlations were found between *Fusarium* head blight and time of anthesis, and head blight and culm length. From preliminary analyses (data not shown), it was concluded that within the experiments of 1986 and 1987, there was a statistically significant interaction between wheat genotypes and *Fusarium* strains, which could not be removed by transformation of the data to an angular or logistic scale. In 1988 there was no significant interaction between genotypes and strains, nor was there a significant strain effect.

The means over the replicates of the genotypical assessments per strain within each of the 3 years are presented in Table 1. This table shows the high pathogenicity of strain IPO 39-01. The nonadditivity of the head blight ratings is striking. The head blight data of Table 1 were subjected to a Finlay-Wilkinson analysis, for which each strain-year combination was treated as a separate environment. The model may be written

$$Y_{ijk} = \mu + G_i + \beta_i E_j + I^*_{ij} + e_{ijk},$$

where μ is the mean value over all genotypes and environments, G_i is the effect of the i^{th} genotype, the regression coefficient β_i is a measure of the stability of the i^{th} genotype, E_j is the effect of the j^{th} environment, I^*_{ij} is the residual interaction after allowing for differences in stability between the genotypes, and e_{ijk} is the error for the k^{th} individual within the ij^{th} genotype-environment.

Table 1. *Fusarium* head blight incidence[a, b] of 17 wheat genotypes for four *F. culmorum* strains and 3 years. Genotypes are presented in ascending order of incidence averaged over strains and years

Wheat genotype	1986				1987				1988			
	IPO 39-01	329-01	348-01	436-01	39-01	329-01	348-01	436-01	39-01	329-01	348-01	436-01
SVP 72017-17-5-10[c]	2.0	1.5	3.0	1.5	7.3	0.8	0.5	2.1	5.3	2.7	2.0	2.7
SVP 77076-4	9.0	1.0	3.0	1.5	13.5	0.3	0.1	0.2	7.0	1.7	3.3	3.7
Arina	8.0	2.5	5.0	4.0	12.0	0.3	0.1	2.8	6.3	1.0	2.0	3.3
SVP 77076-38	18.0	1.0	4.5	7.0	8.9	0.2	0.1	0.7	2.0	2.3	1.3	1.7
SVP 77076-1	6.0	3.0	1.0	1.5	11.1	0.1	0.4	2.7	7.0	4.7	5.0	8.3
Saiga	4.5	7.5	4.5	9.0	15.5	1.1	0.3	1.7	9.3	9.7	6.0	11.7
SVP 77078-30	9.0	13.0	1.0	2.5	17.8	0.6	0.4	8.4	15.7	5.3	3.3	4.7
SVP 72003-4-2-4	23.5	4.0	9.5	3.5	16.3	0.7	0.4	1.4	5.0	6.3	7.3	13.7
SVP 77079-15	27.5	4.5	2.5	8.5	35.2	1.9	0.5	5.8	3.3	6.3	4.0	8.3
SVP 75059-28	11.0	3.5	3.0	1.5	54.0	1.2	1.2	19.3	4.0	2.7	1.3	6.3
SVP 73030-8-1-1	60.0	7.0	7.5	9.0	36.3	3.0	5.0	7.5	13.0	4.7	5.0	5.7
SVP 73016-2-4	47.0	18.0	14.5	22.5	44.1	5.4	3.5	11.6	6.7	12.5	3.3	4.0
SVP 73012-1-2-3	67.5	16.0	17.5	17.0	34.2	7.0	2.5	9.3	11.0	9.0	6.0	25.7
SVP 75059-46	25.5	5.0	5.0	10.5	69.3	5.0	1.7	13.2	30.0	22.3	20.0	20.3
Nautica	62.5	20.5	20.0	30.5	32.2	1.3	0.8	4.8	40.0	25.0	18.0	20.3
SVP 75059-32	32.5	5.0	9.0	42.5	57.3	5.2	4.3	30.5	37.7	14.7	38.3	31.0
SVP 72005-20-3-1	62.5	16.5	27.5	23.0	58.5	3.7	2.7	21.7	36.7	26.3	13.3	20.3
Mean	28.0	7.6	8.1	11.5	30.8	2.2	1.4	8.5	14.1	9.2	7.6	11.3

[a] Head blight ratings were determined as the product of the percentage of heads infected and the proportion of infected spikelets per infected head

[b] Values presented are means over blocks

[c] SVP-line code: the first two digits indicate the year of crossing, followed by three digits representing the crossing number. The number after each hyphen is a selection number

Table 2. Summary of the results from the Finlay-Wilkinson analysis

Term	*df*	SS	MS
Genotype	16	12,368	773**
Environment	11	15,201	1,382**
Genotype × environment	176	13,867 (100%)	79**
Regressions	16	5,662 (41%)	354**
Concurrence	1	4,586	4,586**
Deviations from concurrence	15	1,076	72*
Deviations from regressions	160	8,205 (59%)	52*
Error	>51		25

* Significant at $P \leq 0.01$
** Significant at $P \leq 0.001$

To fit the Finlay-Wilkinson model, first the genotypical and environmental main effects are estimated in the customary way for ANOVA. Subsequently, the individual genotypical responses are repressed on the estimated environmental main effects to find estimates for the parameters β_i. The heterogeneity between regression lines has to account for the genotype × environment interaction. This approach is quite usual for yield data, but may seem somewhat unorthodox for disease incidences. Problems with respect to inference may be expected from failure of the assumptions for analysis of variance, such as homogeneity of variance and normality. However, in this study the Finlay-Wilkinson model only served as a starting point for a more appropriate model, and no ultimate conclusions are derived from the model itself. The results of the Finlay-Wilkinson analysis are shown in Table 2. The heterogeneity between lines accounted for 41% of the total interaction and the description seems to be acceptable. The plot of the fitted regression lines, Fig. 1, approaches a special case of the Finlay-Wilkinson model, namely, the situation where all regression lines intersect at the same point. This model is equivalent to the concurrence model (Mandel 1969)

$$Y_{ijk} = \mu + G_i + E_j + cG_iE_j + I^*_{ij} + e_{ijk},$$

where μ, G_i, E_j, I^*_{ij}, and e_{ijk} have the same interpretation as in the Finlay-Wilkinson model, while c is the only extra parameter needed for a description of the interaction. With this model 81% of the interaction that was explained by the heterogeneity of the Finlay-Wilkinson fitted lines can be covered (Table 2). This means that the genotype × environment interaction as described by the Finlay-Wilkinson model consists mainly of a divergence of (centered) genotypical responses. This interpretation gains even more credibility from the strong associations existing between the evaluations of the genotypes over the set of environments as measured by Spearman rank

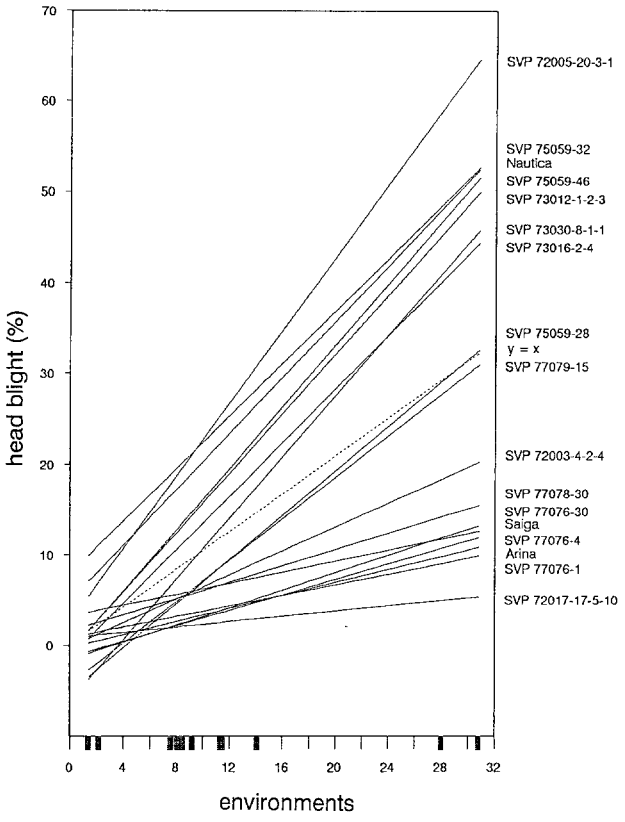

Fig. 1. Regression lines of individual head blight ratings on mean head blight ratings per environment (formed by strain-year combinations) for 17 individual genotypes. The hanging symbols on the abscissa represent the environments, which are presented in Table 1

correlations (data not shown). These were all positive; 58 out of 66 were significant at $P \leq 0.05$.

At first glance, this would seem an adequate explanation of the interaction. However, a first problem arises in the context of the deviations I_{ij}^* from the regressions. When tested against an error estimate of 25 with at least 51 *df* (Table 2), being the geometric mean of the error estimates for genotype-environment means over the 3 years, the deviations appear to be significant. This implies that in addition to the divergence of the regression lines, other factors are involved in the interaction. Obviously, the Finlay-Wilkinson model does not remove all pattern from the data. Furthermore, a plot of the residuals against the fitted values exhibited an increase of the variance with the mean.

A second problem is that a considerable part of the environmental range is not represented by actual measurements, invalidating an interpretation of the regression coefficients as stability measures. The regressions mainly express a contrast between the high disease incidences in the environments formed by IPO 39-01 in 1986 and 1987, and the rest of the strain-year combinations. To a major extent the slopes were determined by the two high incidence environments (Fig. 1; Table 1). The influence of these two environments was investigated more closely by performing an analysis without them. The overall treatment sum of squares decreased dramatically from 41,436 to 13,579. However, the proportion of genotype × environment interaction remained more or less the same, 36% in the reduced set against 33% in the full set. Now a concurrence model gave an adequate description of the genotype × environment interaction, that is, deviations from the concurrence model were not significant anymore. However, the rank order of the slopes showed some clear reversals in comparison to the rank order derived from the Finlay-Wilkinson analysis for the full set of environments. This means that if circumstances had been such that only low disease pressures had occurred, an interaction analysis would have led to a concurrence model and the ranking of genotypes for stability would not have been predictive for situations with higher disease pressures.

It was evident that a description of the interaction in tems of a Finlay-Wilkinson model for the full set was not satisfactory. An alternative was a model with Additive Main effects and Multiplicative Interaction effects, an AMMI model. This model may be written

$$Y_{ijk} = \mu + G_i + E_j + \sum_{n=1}^{N} \lambda_n a_{ni}\, b_{nj} + I_{ij}^* + e_{ijk},$$

where μ, G_i, E_j, I_{ij}^*, and e_{ijk} have the same interpretation as above, while λ_n is the eigenvalue for axis n of the principal components analysis, and a_{ni} and b_{nj} are the corresponding genotypical and environmental scores. The a_{ni} may be interpreted as genotypical stabilities, while the b_{nj} may be seen as environment characterizations. N denotes the number of multiplicative terms necessary for an adequate description of the interaction. The model can be fitted by first calculating additive main effects for genotypes and environments, followed by a principal components analysis (singular value decomposition) of the matrix of the residuals (Gabriel 1978).

With respect to the assessment of N, two strategies are possible: (i) a strategy based on postdictive success, i.e., the ability of a model to fit its own data (e.g., traditional F-tests), and (ii) a strategy based on predictive success, the ability to predict validation data not used in constructing the model (Gauch 1988; Gauch and Zobel 1988). Because of the fact that in our experiment main- and subplot treatments changed over the years, assessment of predictive success was not straightforward. Therefore, model validation took place on postdictive grounds. Approximate F-tests were done after ascribing degrees of freedom to the eigenvalues following Mandel (1969) and calculating the corresponding mean squares. A summary of the ANOVA for the AMMI model is shown in Table 3. Three multiplicative terms seem necessary for an adequate description of the interaction. The

Table 3. Summary of the results from the AMMI analysis

Term	*df*	SS	MS
Genotype	16	12,368	773**
Environment	11	15,201	1,382**
Genotype × environment	176	13,867 (100%)	79**
Component 1	48[a]	6,199 (45%)	129**
Component 2	36	4,061 (29%)	113**
Component 3	27	1,756 (13%)	65*
Rest	65	1,851 (13%)	17
Error	>51		25

* Significant at $P \leq 0.01$
** Significant at $P \leq 0.001$
[a] The degrees of freedom for the components are calculated according to Mandel (1969)

Table 4. Genotypical scores ($\times 10^{-2}$) from the AMMI analysis, normalized at squared length 1

Genotype	Component		
	1	2	3
SVP 72017-17-5-10	−31	−13	1
SVP 77076-4	−21	−9	0
Arina	−23	−11	−1
SVP 77076-38	−12	−22	−3
SVP 77076-1	−27	−10	5
Saiga	−28	−7	12
SVP 77078-30	−23	−4	3
SVP 72003-4-2-4	−8	−18	−2
SVP 77079-15	4	6	−21
SVP 75059-28	−8	44[a]	−46[a]
SVP 73030-8-1-1	36	−13	−21
SVP 73016-2-4	23	−4	−32
SVP 73012-1-2-3	38	−27	−16
SVP 75059-46	5	57[a]	7
Nautica	32	−23	55[a]
SVP 75059-32	2	43[a]	49[a]
SVP 72005-20-3-1	38	12	11

[a] Genotypes with high scores used as a basis for component interpretation

Table 5. Environmental scores ($\times 10^{-2}$) from the AMMI analysis, normalized at squared length 1

Environment Strain	Year	Component		
		1	2	3
IPO 39-01	1986	−83[a]	−37	−8
IPO 329-01	1986	−10	−26	−19
IPO 348-01	1986	−2	−25	−11
IPO 436-01	1986	4	−2	35
IPO 39-01	1987	30[a]	79[a]	−31
IPO 329-01	1987	−25	−14	−31
IPO 348-01	1987	−28	−16	−31
IPO 436-01	1987	−15	21	−20
IPO 39-01	1988	0	13	63[a]
IPO 329-01	1988	−8	−2	12
IPO 348-01	1988	−19	6	24
IPO 436-01	1988	−10	1	18

[a] Environments with high scores used as a basis for component interpretation

rest of the terms is not significant when tested against the error estimate introduced above. The plot of residuals showed no gross failures of the assumptions. The interpretation of the components is as follows:

1. The first component provides genotypical scores, 'stabilities' (Table 4), and environmental scores (Table 5) that are closely correlated with the stabilities and scores from the Finlay-Wilkinson model. From the environmental scores in Table 5 it can be seen that the first component is the contrast between IPO 39-01 in 1986 and 1987, on the one hand, and the rest of the strain-year combinations, on the other hand. The proportion of variance explained by this component is 45% (for comparison, 41% in the Finlay-Wilkinson model). It can be concluded that the first multiplicative term is more or less equivalent to the Finlay-Wilkinson regressions.
2. The second component arises from nonadditivity of the genotypes SVP 75059-28, SVP 75059-32, and SVP 75059-46 inoculated with strain IPO 39-01 in 1987 (Tables 4 and 5). These three selections from the same cross had a far higher *Fusarium* head blight incidence in 1987 after inoculation with IPO 39-01 than may be expected from the genotypical and environmental main effects plus the Finlay-Wilkinson coefficients.
3. The third component results from genotype SVP 75059-28, with a far lower than expected incidence, and genotypes SVP 75059-32 and Nautica, with a higher than expected incidence. Again, this component is mainly due to an IPO 39-01 reaction, this time in 1988. The interpretation of this component is not easy. It probably represents merely noise but, as a consequence of a postdictive validation strategy, prone to lead to overfitting (Gauch 1988), is not identified as such. An estimate for the amount of noise in the overall treatment sum of squares is the product of the treatment degrees of freedom with the error estimate: $203 \times 25 = 5{,}075$. Acknowledging the fact that the noise will predominantly turn up in the higher axes, a strong argument for an interpretation of axis three in terms of noise is given.

The AMMI model thus provides a good description of the data, including the genotype × environment interaction, and uncovers some features we were not able to disclose before. Altogether the interaction may be said to consist primarily of a divergence of the incidences at higher disease pressures, modified by genotype-specific reactions in certain years. However, the modifications are on the whole not such that they heavily disrupt the rankings of the genotypes over the environments, al-

though incidental changes occur. The divergence is mainly caused by the highly pathogenic strain IPO 39-01.

A last point concerns the scale of the measurement. A percentage scale was used, as experience has shown that this is a convenient scale for resistance breeding research. For the purpose of genetic analyses the scale should preferably be one on which the analysis is as simple as possible, which means one on which interactions are small or absent. Various empirical transformations were tried. The most successful was the complementary log log transformation, which removed the genotype × strain × year interaction completely. However, genotype × year interaction and, to a lesser extent, genotype × strain interaction remained significant. The conclusions with respect to the status of resistance type, horizontal, did not change. The complementary log log transformation confers extra weight to the lower percentages. This seems unjustifiable in the light of the size of the measurement error. Therefore, the original percentage scale was retained. For nonremovable interactions, Mather (1971) remarked that "we must always be prepared to bring interaction explicitly into an analysis."

Conclusions

The three environments with the highest disease pressure were the combinations of one particular strain (IPO 39-01) with the 3 years. No evidence was found for strain-specific resistance. The *Fusarium* head blight resistance in this study can be described as horizontal resistance in terms of Vanderplank (1984), with the exception of the lines selected from cross SVP 75059, which showed a 'strain-year combination' dependent resistance, ineffective in 1987. For large-scale screening for resistance to *Fusarium* head blight using experimental inoculation, highly pathogenic strains should be used. The use of an AMMI model for the description of genotype × strain interaction over years allows conclusions not obtainable by the additive models used in the studies reported in the introduction. It provides a means to check whether the environmental 'lability' of interaction in the aforementioned studies was really part of the pattern in the data and hence merits agricultural interpretation, or whether it was merely noise.

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