Quantitative-Genetic Basis of Aggressiveness of 42 Isolates of *Fusarium culmorum* for Winter Rye Head Blight

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ABSTRACT

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Forty-two isolates of *Fusarium culmorum* obtained from diseased plant parts collected from fields in nine European countries and Australia were tested on a synthetic winter rye population. A spore suspension of each isolate was sprayed during midanthesis in five environments (location–year combinations) onto the heads. All isolates were pathogenic as judged by head blight rating scored on a 1 to 9 scale and grain weight relative to the noninoculated control. Isolates differed, however, in their ability to cause disease (aggressiveness). Quantitative genotypic variation for aggressiveness occurred, while isolate–environment interaction variance, although significant, accounted for only 14% of total variance averaged over both traits. Correlations for aggressiveness across environments ranged from 0.6 to 0.8 ($P = 0.01$). Estimates of heritability on an entry-mean basis were high ($h^2 = 0.9$) for both traits, indicating that a substantial proportion of the phenotypic variation was caused by genetic effects. It is concluded that aggressiveness of *F. culmorum* is inherited as a complex trait.

Additional keywords: genetic variance, population parameter, quantitative variation, resistance breeding, *Secale cereale*

Fusarium culmorum (W.G. Smith) Sacc. is a ubiquitous pathogen of winter rye (*Secale cereale* L.) and other cereal crops (3,6), infecting all plant organs and at all developmental stages. *F. culmorum* is a haploid, imperfect fungus with asexually formed macroconidia and no known teleomorph (23). Heterokaryosis is thought to be the most important source of asexual genetic variation (23). In culture, *F. culmorum* shows phenotypic variability, including altered morphological characters (3,19,22,30) and reduced aggressiveness on host plants (3,30). However, compared to other *Fusarium* species, *F. culmorum* is phenotypically rather stable. *F. culmorum* and *F. graminearum* were the *Fusarium* species with the highest pathogenicity and aggressiveness compared to five and 11 other *Fusarium* species, respectively, in artificial inoculation tests on wheat heads (29,33). According to Vanderplank (31), pathogenicity is the ability to cause disease, and aggressiveness designates the quantity of disease induced by a pathogenic isolate on a susceptible host. The term aggressiveness implies that the isolates do not interact differentially with host cultivars (2,31), i.e., that resistance is race nonspecific. This has been demonstrated for *F. culmorum*–incited head blight in wheat (28,32). In both studies, the relative ranking of isolates with host genotype was mostly the same, and no stable interaction patterns were found over environments. Among limited numbers of *F. culmorum* and *F. graminearum* isolates, phenotypic variation for aggressiveness in wheat was previously shown for seedling blight in the greenhouse (12–14), root rot infections (10,22), and head blight in the field (12,14,28). The economically most important disease caused by *F. culmorum* is head blight, which leads to severe yield loss, reduced kernel quality, and contamination with mycotoxins in wheat (12,26,27) and rye (4,16). Resistance of winter rye to *F. culmorum*–incited head blight was found to be quantitatively inherited, with no genotype being completely resistant (16,17).

Because little is known about the genetic basis of aggressiveness in *F. culmorum* populations, the objectives of this study were to estimate the relative importance of genotypic variance, genotype–environment interaction, and heritability for aggressiveness of 42 *F. culmorum* isolates inoculated on winter rye at anthesis in five environments.

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MATERIALS AND METHODS

Isolates. Isolates of *F. culmorum* were obtained from naturally infected plant material or from soil collected in 10 different countries (Table 1). Eighteen isolates were collected by the authors, and the remaining 24 isolates were kindly provided by colleagues. The isolates FC10 and FC17 (32), FC20 (28), FC32 and FC39 (15,32), and FC40 (26,28,32) were previously used for inoculation on winter wheat heads. After isolation and identification of the isolates according to Nirenberg (21) in our lab, or receipt by mail, cultures were transferred to SNA (synthetic nutrient-poor mineral agar according to Nirenberg [21] containing the following composition per 1,000 ml of distilled water: 1.0 g of KH_2PO_4, 1.0 g of KNO_3, 0.5 g of $MgSO_4 \cdot 7H_2O$, 0.5 g of KCl, 0.2 g of glucose, 0.2 g of sucrose, and 15 g of agar, Oxoid L18, Detroit, MI) and incubated at 18°C in the dark until sporulation. Pure cultures from all isolates were derived by monoconidial transfers. Mycelial plugs from the monoconidial cultures were preserved in liquid nitrogen following the procedure described by Hoffmann (9). Isolates were cultured on SNA supplemented with glycerol at 5 ml liter^{-1}. Sterile polyvinylchloride "straws" with a diameter of about 1 mm and a length of 25 mm were used to cut plugs of mycelium and agar. Fifty straws per isolate were cryopreserved in liquid nitrogen in cryotubes, each containing up to 12 straws. To avoid accumulation of cultural variants during the experiments, conidia were always produced anew from isolates revived from cryopreservation. All cultures are deposited at the authors' laboratory.

Inoculum production and inoculation. Conidia were produced on wheat grain medium as previously reported by Miedaner et al. (17). Wheat grain was boiled with water for 15 min. Surplus liquid was removed, and 500 ml of grain was placed in 1,000-ml glass flasks. Flasks were sealed with aluminum foil and autoclaved twice, on successive days, at 121°C for 20 min at 1 atm. A starter culture was established from the cryopreserved isolates by placing one sterile straw on a SNA plate (60 mm diameter). The plates were incubated at 18°C in the dark for 5 to 7 days until sporulation. Spores were used for one cycle of subculture on SNA (60 mm diameter) to provide enough material for incubation on wheat. Subcultures were also placed at 18°C in the dark for 5 to 7 days until sporulation. Each flask of cooled wheat grain medium was mixed

with the spore suspension produced on one SNA plate and incubated at 13°C in the dark for 4 weeks. To prepare spores for inoculation of heads, colonized wheat grain was removed from the flasks and placed in a thin layer in plastic boxes (40 × 60 cm^2) covered with a punctured polyethylene foil about 40 cm below two black light tubes (Phillips TLO, 40 W/80, 24-h light) for 6 to 14 days until sporulation. During this period, the cultures were kept moist by spraying lightly with sterilized tap water under the foil when required. The colonized grain with the spores was air-dried for a further 2 to 3 days and stored in a refrigerator at 5°C until inoculation.

Shortly before inoculation, the wheat grain material was suspended in tap water for 10 min to produce spore suspensions, which were adjusted to a concentration of 3×10^5 spores ml^{-1}. Each plot was inoculated with a portable sprayer at a rate of about 140 ml of spore suspension per m^2 in the evening (5:30 to 10:00 P.M.). To maintain high humidity over the crop, experiments located at Hohenheim were sprinkled by a mist irrigation device for 2 min every 30 min in the morning after each inoculation date (6:00 to 12:00 A.M.).

Host. A synthetic winter rye population (SYN 8392) was used for all experiments. SYN 8392 had been established by crossing two self-fertile inbred lines (L283, L292) of the Carsten gene pool by hand-emasculation in the greenhouse. The resulting F_1 hybrid was propagated under open pollination in isolation cabins as commonly used for inbred line development in hybrid rye breeding. L283 is highly susceptible to *Fusarium* head blight; L292 is of medium susceptibility.

Table 1. Geographic origin, host or habitat, host organ, year of isolation, source, and means[a] of head blight rating and relative grain weight of 42 isolates of *Fusarium culmorum* (FC) inoculated at midanthesis on a synthetic rye population

FC code	Location/country[b]	Host/habitat, host organ	Year of isolation	Original code/source[c]	Head blight rating (score 1-9)[d]	Rel. grain weight[e] (%)
01	Unknown/NL	Wheat, kernel	1952	251.52[6]	2.5	86.8
02	Puglia/I	Wheat, kernel	1979	ATCC 56088[8]	3.0	76.9
03	Unknown/NL	Wheat, leaf sheath	1982	IPO 433-01[3]	3.6	76.1
04	Unknown/G	Maize	Unknown	DSM 62184[5]	3.6	75.6
05	Unknown/G	Triticale, stem base	1988	289[4]	3.7	74.7
06	Crookwell/AUS	Maize, stalk	1975	F 4007[9]	3.8	72.4
07	Puglia/I	Wheat, kernel	1975	ATCC 56089[8]	3.9	73.6
08	Seymour/AUS	Soil	1991	F 10822[9]	4.0	71.7
09	Vaihingen/G	Emmer wheat, head	1989		4.1	68.4
10	Oldambt/NL	Wheat, head	1988	SVP 8901[3]	4.2	69.7
11	Söllingen/G	Wheat, kernel	1987		4.3	68.4
12	Hohenheim/G	Triticale, head	1988	257[4]	4.3	65.7
13	Svalöf/S	Rye, kernel	1991		4.5	61.5
14	Queensland/AUS	Soil	1976	F 4095[9]	4.5	62.9
15	Mainz/G	Wheat, head	1973	62191[1]	4.6	65.5
16	Svalöf/S	Rye, kernel	1991		4.6	62.9
17	Zelder/NL	Wheat, head	1988	SVP 8904[3]	4.6	60.5
18	Wageningen/NL	Wheat, head	1982	IPO 437-01[3]	4.7	62.2
19	Unknown/F	Barley, head	1982	64218[1]	4.8	63.5
20	Zelder/NL	Wheat, stem base	1981	IPO 329-01[3]	4.9	60.1
21	Bergen/G	Rye, stem base	1991		5.0	58.4
22	Hohenheim/G	Durum wheat, head	1989		5.0	58.9
23	Ellighausen/CH	Wheat, head	1989	SVP 8906[3]	5.0	58.5
24	Hohenheim/G	Durum wheat, head	1985	61[4]	5.1	61.5
25	Hohenheim/G	Durum wheat, kernel	1985	214[4]	5.2	58.1
26	Unknown/N	Soil	1982	64223[1]	5.4	58.9
27	Hohenheim/G	Rye, kernel	1990		5.3	54.4
28	Svalöf/S	Rye, kernel	1991		5.3	56.7
29	Hohenheim/G	Durum wheat, head	1987		5.4	57.7
30	Schönborn/G	Rye, stem	1989		5.4	54.8
31	Chewendowa/PL	Rye, stem	1990		5.4	55.8
32	Szeged/H	Wheat, stem base	1991[f]	12375[2]	5.4	57.1
33	Northeim/G	Wheat, leaf	1984	3.5[7]	5.5	55.8
34	Rottenburg/G	Spelt wheat, head	1988	274[4]	5.6	54.9
35	Svalöf/S	Rye, kernel	1991		5.6	52.5
36	Szeged/H	Wheat, head	1991		5.6	53.6
37	Grünbach/G	Wheat, stem	1984		5.6	55.0
38	Wetze/G	Wheat, head	1984		5.7	54.5
39	Szeged/H	Wheat, stem base	1991[f]	12551[2]	5.8	53.9
40	Flevoland/NL	Wheat, kernel	1966	IPO 39-01[3]	5.9	51.7
41	Bergen/G	Rye, stem base	1988		6.2	51.6
42	Chewendowa/PL	Rye, stem base	1990		6.2	48.2
Population mean					4.8	61.9
Least significant difference ($LSD_{5\%}$)					0.6	7.3

[a] Means are averaged over five environments.

[b] AUS = Australia, CH = Switzerland, G = Germany, F = Finland, H = Hungary, I = Italy, N = Norway, NL = The Netherlands, PL = Poland, S = Sweden.

[c] 1 = H. Nirenberg, Federal Biological Research Center for Agriculture and Forestry, Berlin, Germany; 2 = A. Mesterhazy, Cereal Research Institute, Szeged, Hungary; 3 = C. H. A. Snijders, CPRO-DLO, Wageningen, The Netherlands; 4 = H. Schmitz-El Sherif, State Plant Breeding Institute, University of Hohenheim, Stuttgart, Germany; 5 = DSM, Deutsche Sammlung für Mikroorganismen, Göttingen, Germany; 6 = CBS, Centraalbureau voor Schimmelcultures, Baarn, The Netherlands; 7 = J. Unger, Federal Biological Research Center for Agriculture and Forestry, Braunschweig, Germany; 8 = A. Logrieco, Istituto tossine e micotossine de parassiti vegetali, Bari, Italy; 9 = L. W. Burgess, University of Sydney, Sydney, Australia. Isolates without indication were isolated by the authors.

[d] 1 = no symptoms visible, 9 = 96 to 100% of spikelets per plot infected; see text for full scale.

[e] Grain weight of the inoculated relative to the noninoculated plots, determined from a sample of 60 main culm heads per plot harvested arbitrarily by hand.

[f] Reisolated from inoculated wheat heads in 1991.

Field trials. In 1992, the experiment was grown at Hohenheim near Stuttgart (400 m above sea level, 8.5°C mean annual temperature, 685 mm mean annual precipitation) and at Schönborn near Heidelberg (218 m above sea level, 10.2°C mean annual temperature, 730 mm mean annual precipitation). In 1993, the experiment was located at Hohenheim, Schönborn, and additionally, at the experimental station Lindenhof near Reutlingen (720 m above sea level, 6.6°C mean annual temperature, 952 mm mean annual precipitation). Plants were grown in three-row drilled microplots of 1.0-m length with approximate 0.21-m spacing between rows (0.625 m^2) in a noninoculated and an inoculated treatment block adjacent to each other. In the inoculated treatment block, isolates were randomized in a 7 × 6 lattice design with four replicates. The noninoculated treatment consisted of 10 plots of SYN 8392. To avoid infection with other pathogens, both treatment blocks were sprayed once against *Pseudocercosporella herpotrichoides* (Sportak Alpha [Prochloraz 450 g $liter^{-1}$ ha^{-1} + Carbendazim 120 g $liter^{-1}$ ha^{-1}], jointing stage), and *Erysiphe graminis* and *Puccinia recondita* (Simbo [Propiconazol 125 g $liter^{-1}$ ha^{-1} + Fenpropimorph 300 g $liter^{-1}$ ha^{-1}], shortly before heading) in both years. Application of a growth regulator (Terpal C [Ethephon 233 g $liter^{-1}$ ha^{-1} + Chlormequat-chlorid 458 g $liter^{-1}$ ha^{-1}], jointing stage) prevented lodging in both years.

Disease assessment. Two traits were assessed (16,17): head blight rating on a 1 to 9 scale and grain weight relative to the noninoculated control plots. Head blight was first rated with the onset of symptom development, i.e., 19 to 24 days after inoculation depending on the environment. To obtain a measure of disease progress, all plots were rated on successive dates (Table 2). We wanted a rating that would reflect the number of infected heads per plot and the number of infected spikelets per head. Therefore, the population was rated in terms of infected spikelets per plot with the following rating classes being assigned (18): 1 = no symptoms visible, 2 = <5% of all spikelets diseased, 3 = 5 to 15% of all spikelets diseased, 4 = 16 to 25% of all spikelets diseased, 5 = 26 to 45% of all spikelets diseased, 6 = 46 to 65% of all spikelets diseased, 7 = 66 to 85% of all spikelets diseased, 8 = 86 to 95% of all spikelets diseased, 9 = 96 to 100% of all spikelets diseased. Ratings were taken by the same person in each trial every 3 to 4 days according to the speed of symptom development (Table 2). Arithmetic means of the head blight ratings across all assessment dates were used for further analyses. Grain weight was determined with 60 main culm heads per plot harvested arbitrarily by hand. All heads from a plot were carefully threshed as a pooled sample.

Statistical analyses. All analyses were based on single plot data. Mean head blight rating of the noninoculated treatment never exceeded 1.4 across all environments, indicating virtually no natural infection. The values of the inoculated treatment were, therefore, used directly for analyses. For relative grain weight, trait values from the inoculated plots were calculated relative to the mean of the 10 noninoculated plots for each isolate, and all further analyses were performed with these percentage values. Lattice analyses of variance were performed on field data from each location. Entry means from individual environments followed a normal distribution for both traits, and error variances were homogeneous according to Bartlett's test (25). Adjusted entry means and effective error mean squares were used to compute the combined analysis across the five location–year combinations Hohenheim 1992 and 1993, Schönborn 1992 and 1993, and Lindenhof 1993 (= environments) according to Cochran and Cox (5, pp. 545-555), with the restriction that F tests are approximate. For each missing value, one degree of freedom is subtracted from the error variance. Estimates of variance components for σ^2_e (error variance), σ^2_{ge} (genotype–environment variance), and σ^2_g (genotypic variance) were calculated as described by Snedecor and Cochran (25, pp. 319-324) for factorial designs. Broad-sense heritabilities (h^2) on an entry-mean basis were estimated with the formula presented by Fehr (8, p. 98):

$$h^2 = \sigma^2_g/(\sigma^2_e/rt + \sigma^2_{ge}/t + \sigma^2_g)$$

where r = number of replicates and t = number of test environments. Exact 90% confidence intervals on heritability were computed according to Knapp and Bridges (11). Phenotypic correlations between both traits and between all pairs of environments were estimated on an entry-mean basis using standard procedures (20). All analyses of variance and coefficients of phenotypic correlation were calculated with the computer package PLABSTAT (H. F. Utz, Institute of Plant Breeding, Seed Science, and Population Genetics, University of Hohenheim, Stuttgart, Germany). The effects of isolates, environments, and replicates were assumed to be random variables.

RESULTS

Isolates of *F. culmorum* were collected from a broad range of environments (Table 1). They all caused head blight symptoms and reduced grain weight. Isolates differed significantly in their ability to cause head blight as indicated by both traits. Mean head blight ratings of the 42 *F. culmorum* isolates ranged from 2.5 to 6.2, and mean relative grain weight ranged from 48 to 87% across the five environments. Both traits showed continuous variation with no significant deviation (P = 0.05) from a normal distribution. Correlations between head blight rating and relative grain weight ranged between –0.88 and –0.97 (P = 0.01) across individual environments.

Disease progress in each environment was assessed by several head blight ratings at successive dates from 19 to 35 days after inoculation (Table 2). Disease progress was slightly different among environments. At 24 days after inoculation, for instance, Hohenheim 1992 and 1993, and Schönborn 1992 already showed medium head blight ratings; whereas at Schönborn 1993 and Lindenhof 1993, disease progress was delayed. All assessed mean head blight ratings showed genotypic differences at the 1% probability level and were highly intercorrelated ($r \geq 0.9$, P = 0.01). Mean disease severity was medium to high in each of the five environments (Table 3). Genotypic differentiation among the 42 *F. culmorum* isolates was significant at the 1% significance level in all instances. For relative grain weight, environments with highest reduction (Hohenheim 1992, Hohenheim 1993, Lindenhof 1993) tended to exhibit larger genotypic variances. Error variances relative to genotypic variances were lower for head blight rating than for relative grain weight.

Combined across environments, significant genotypic variation among isolates was found for head blight rating and relative grain weight (Table 4). Both environment variance and isolate–environment interaction variance were highly significant. Interaction variances were, however, about three times smaller than isolate variances. Heritability estimates were high throughout. Accordingly, phenotypic correlations between environments were significant for all pairwise comparisons

Table 2. Head blight rating on successive dates for a synthetic rye population inoculated with each of 42 isolates of *Fusarium culmorum* in five environments

	Days after inoculation					
Environment	**19**	**21**	**24**	**27**	**31**	**35**
Hohenheim 1992	4.7[a]	5.1	5.5	5.8	6.4	6.4
Schönborn 1992	4.3	ND[b]	5.0	5.3	5.5	ND
Hohenheim 1993	ND	4.0	4.9	5.5	6.4	ND
Schönborn 1993	3.3	ND	3.6	4.5	ND	ND
Lindenhof 1993	ND	ND	3.0	4.0	4.7	6.2

Significant (P = 0.01) genotypic variance at all assessment dates in each environment (F test).

[a] 1 = no symptoms visible, 9 = 96 to 100% of spikelets per plot infected; see text for full scale.
[b] Not determined.

ranging from 0.67 to 0.79 (P = 0.01) for head blight rating and from 0.62 to 0.77 (P = 0.01) for relative grain weight. The correlations between the years 1992 and 1993 at the locations Hohenheim and Schönborn were of the same magnitude as correlations among different location–year combinations.

DISCUSSION

All *F. culmorum* isolates tested caused head blight symptoms and affected grain weight when inoculated to winter rye at anthesis. Isolates differed significantly in their aggressiveness for the synthetic rye population. According to Vanderplank (31), aggressiveness depends not only on the genotypes of pathogen and host but also on the environment in which it is assessed. However, when tested repeatedly with a reproducible procedure, aggressiveness is a stable, highly heritable trait for a given host genotype–isolate pair (2). This was demonstrated in our study. Genetic variance for aggressiveness was large in the tested *F. culmorum* population, and isolate–environment interaction was of minor importance only. Accordingly, phenotypic correlations among all pairs of environments were significant, indicating a high environmental stability of aggressiveness of *F. culmorum*. Means of head blight rating and relative grain weight both showed continuous variation. This type of variation might be caused by the simultaneous segregation of multiple genes that affect the trait (1,7). The number of genes involved cannot be estimated from the distribution, because environment may obscure genetic variation (1). However, in our study, isolate–environment interaction variance was only one-third of isolate variance for both traits (Table 3), and heritability estimates were high (h^2 = 0.9). Heritability can be overestimated by (i) sampling effects of genotypes when testing a small population, (ii) a narrow ecological range of environments, and/or (iii) low testing intensity (8). The isolates used, however, were randomly collected from a wide range of geographic areas, host species, and host organs. This results in a large genotypic variance. The test environments varied considerably according to their altitude, mean annual temperature, mean annual precipitation, and soil conditions. They belong to different ecological regions (Upper Rhine Valley, plains around Stuttgart, mountains) that represent most of the cereal growing areas in South Germany. Because we determined heritability on an entry-mean basis, as usual for plant breeding experiments, the estimate is influenced by the number of replicates and test environments (see formula in Materials and Methods). If the combined analysis of variance is calculated with a model estimating genotype–year–location instead of genotype–environment effects over a balanced set of two locations and two years (Hohenheim 1992 and 1993, Schönborn 1992 and 1993), the heritability estimate is not substantially different (h^2 = 0.85). Therefore, we conclude from our data that a high proportion of the total phenotypic variation was due to genetic effects. Quantitative differences in ability of *F. culmorum* and *F. graminearum* isolates to cause head blight in wheat in the field were reported earlier (12,14,28). However, these studies were restricted to two to three environments and four to 11 isolates without giving the relevant population parameters. Van Eeuwijk et al. (32) tested for *Fusarium* head blight at 18 European environments, but with only one isolate used at all environments.

In our study, head blight rating and relative grain weight were both used for measuring aggressiveness. Head blight rating estimates the proportion of prematurely bleached spikelets and could, therefore, only be assessed until the heads began to change color (yellow ripening). In contrast, pathogen-induced reduction of grain weight spans the whole infection period until harvest. Both traits were highly correlated and resulted in similar estimates of population parameters. Head blight rating, however, could be assessed more precisely than relative grain weight, as illustrated by the proportion of error variance relative to the genotypic variance.

Aggressiveness of the isolates we used did not depend on their geographic origin, year of isolation, host species' habitat, or host organ from which they were isolated. Frequently, it has been reported that older cultures lose their aggressiveness because of mutations during subculturing (3,30). The low aggressiveness of FC01 that was collected in 1952 seems to confirm this. However, FC40, which has been maintained in culture for 28 years, was highly aggressive. Among the 10 most aggressive isolates (FC33 to FC42), only three were derived from rye, and the remainder were from wheat. Eight of these isolates originated from environments with climatic conditions (North Germany, The Netherlands, Poland, Hungary, and Sweden) totally different from those tested in this study (South Germany). Moreover, isolates from Australia induced similar head blight ratings and relative grain weights to some German isolates. Thus, the data indicate that the *F. culmorum* isolates tested exhibited no particular ecological requirements for infection. This is also illustrated, on a smaller geographical scale, by the low importance of isolate–environment interaction reported here. These results corroborate earlier findings that show a low level of pathogenic specialization regarding host species and/or host organs (3,6) and a high climatic adaptiveness for *F. culmorum* (32). Nineteen isolates of the *F. culmorum* collection used in this study were additionally examined by using randomly amplified polymorphic DNA (RAPD) profiling as molecular genetic fingerprint markers. Based on fragment length data obtained with 25-decamer primers, cluster and principle coordinate analyses of similarity estimates for all pairwise comparisons of the isolates did not reveal any distinct associations among isolates with their geographic origin, host source, or age of culture (24). Expanding the PCR marker-based analysis on the

Table 3. Means and estimates of genotypic (σ^2_t) and error (σ^2_e) variances of head blight rating and relative grain weight for a synthetic rye population inoculated with each of 42 isolates of *Fusarium culmorum* in five environments

	Head blight rating			Relative grain weight		
Environment	Mean (1-9)[a]	σ^2_t	σ^2_e	Mean (%)	σ^2_t	σ^2_e
Hohenheim 1992	5.7	1.33**[b]	0.18	52.1	146.4**	39.7
Schönborn 1992	5.0	1.06**	0.22	71.5	70.6**	27.1
Hohenheim 1993	5.2	0.69**	0.18	52.1	116.6**	29.5
Schönborn 1993	3.8	0.70**	0.28	70.9	60.8**	36.4
Lindenhof 1993	4.5	0.53**	0.14	63.1	110.3**	49.0

[a] 1 = no symptoms visible, 9 = 96 to 100% of spikelets per plot infected; see text for full scale.
[b] ** = significant P = 0.01 (F test).

Table 4. Variance component estimates and heritabilities of head blight rating and relative grain weight for a synthetic rye population inoculated with each of 42 *Fusarium culmorum* isolates combined across five environments

Source of variation	df	Head blight rating (×10²)	Relative grain weight
Isolate (I)	41	65.97 **[a]	66.13 **
Environment (E)	4	50.28 **	90.64 **
I × E	164	20.81 **	23.77 **
Pooled error	494	5.55	10.55
Heritability (h^2)		0.93	0.91
90% C.I. on h^2[b]		0.88 - 0.95	0.84 - 0.94

[a] ** = significant at P = 0.01 (F test).
[b] Confidence intervals (C.I.) on h^2 were calculated using the method of Knapp and Bridges (11).

remaining 23 *F. culmorum* isolates of this study confirmed this finding (A. G. Schilling, *personal communication*). In summary, great variation among *F. culmorum* isolates clearly exists for phenotypic as well as for molecular genetic characters, but no grouping of isolates according to their origin occurs. The four isolates from Svalöf/Sweden (FC13, FC16, FC28, FC35), for instance, were collected from the same field plot and revealed considerable differences in aggressiveness. Further conclusions for the population structure of *F. culmorum* are limited because other sampling locations are mostly represented by a single isolate only.

Inheritance of aggressiveness among *F. culmorum* isolates corresponds with the inheritance of host resistance in the same pathosystem (16,17). Pathogen aggressiveness and host resistance both show a continuous variation with no isolate being fully nonaggressive and no host genotype being fully resistant. However, host resistance has exhibited much larger genotype–environment interaction. For example, in a resistance test of 15 single-cross hybrids across 2 years, genotype–environment interaction variance for head blight rating amounted to 64% of the genotypic variance (16). For winter rye inbred lines, genotype–environment interaction of head blight rating in inoculation tests with *F. culmorum* is often as high or even higher than genotypic variance (16,17).

For resistance selection, any aggressive isolate can be used (27,32) without special regard to its origin or host source. However, aggressiveness should be tested in advance to ensure a disease severity optimal for genotypic differentiation. Single fungal strains may possess a low aggressiveness even when they are freshly collected from diseased plant tissue (Table 1).

Considering the quantitative variation of the assessed traits and their high heritabilities, we surmise that aggressiveness in *F. culmorum* is governed by several genes and/or an allelic series at one locus with the alleles having a different contribution to aggressiveness. Qualitative and quantitative differences in enzyme and mycotoxin production are possible causes for the great variation of aggressiveness (3,27). These fungal products, however, require complex biosynthetic pathways that are encoded by a large number of genes. The physiological basis of the continuously varying aggressiveness among *F. culmorum* isolates needs to be studied further.

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