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Heritabilities and Correlations of Fusarium Ear Rot Resistance and Fumonisin Contamination Resistance in Two Maize Populations

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ABSTRACT

Fusarium verticillioides **(Sacc.) Nirenberg (synonym *F. moniliforme* Sheldon) (teleomorph: *Gibberella moniliformis*) and *F. proliferatum* (Matsushima) Nirenberg (teleomorph: *G. intermedia*) are fungal pathogens of maize (*Zea mays* L.) that cause ear rot and contaminate grain with fumonisins, a family of mycotoxins that adversely affect animal and human health. The objective of this study was to estimate heritabilities of and genotypic and phenotypic correlations between fumonisin concentration, ear rot, and flowering time in two maize populations. In the (GE440 × FR1064) × FR1064 backcross population, the genotypic and phenotypic correlations between ear rot and fumonisin concentration were 0.96 and 0.40, respectively. Heritability estimated on an entry mean basis was 0.75 for fumonisin concentration and 0.47 for ear rot resistance. In the NC300 × B104 recombinant inbred line population, the genotypic and phenotypic correlations between ear rot and fumonisin concentration were 0.87 and 0.64, respectively. Heritability estimated on an entry mean basis was 0.86 for fumonisin concentration and 0.80 for ear rot resistance. Correlations between fumonisin concentration and silking date were not significant in either population, and correlations between ear rot resistance and silking date were small (less than 0.30) in both populations. Moderate to high heritabilities and strong genetic correlations between ear rot and fumonisin concentration suggest that selection for reduced ear rot should frequently identify lines with reduced fumonisin concentration. Ear rot can be screened visually and so is less costly and less time-consuming to evaluate than laboratory assays for fumonisin concentration.**

Fusarium verticillioides (Sacc.) Nirenberg (synonym *F. moniliforme* Sheldon) (teleomorph: *Gibberella moniliformis*) and *F. proliferatum* (Matsushima) Nirenberg (teleomorph: *G. intermedia*) cause Fusarium ear rot of maize and contaminate the grain with fumonisins, a family of mycotoxins that adversely affect animal and human health (Munkvold and Desjardins, 1997). These toxins are of increasing concern due to mounting evidence of their involvement in a number of human and animal diseases. These diseases include esophageal cancer (Gelderblom et al., 1988; Rheeder et al., 1992) and neural tube birth defects (Hendricks, 1999) in humans, and equine leukoencephalomalacia (Ross et al., 1992) and porcine pulmonary edema (Colvin and Harrison, 1992) in animals. The FDA has published "Guidance for Industry" that suggests limiting fumonisin concentrations to between 2 and 4 $\mu g\ g^{-1}$ for corn flour and other milled corn products used for human consumption (CFSAN, 2001a, 2001b).

Conditions such as high humidity, hot weather, and drought at or just before flowering (Shelby et al., 1994; White, 1999) are favorable on the coastal plain of North Carolina for high levels of ear rot and fumonisin contamination of maize. A study with 14 hybrids typically grown in North Carolina showed that by harvest, 11 of the 14 hybrids were naturally infected with *Fusarium* spp. and contained fumonisin at concentrations above 20 $\mu g\ g^{-1}$. Grain of the hybrid with the greatest amount of fumonisin had a concentration of 77 $\mu g\ g^{-1}$, while grain from the hybrid with the least amount of fumonisin had a concentration of 7 $\mu g\ g^{-1}$ (Heiniger and O'Neal, 2002). No effective control strategies are available to prevent fumonisin accumulation in grain.

In food processing for human consumption, nixtamalization (treating maize grain with lime) can eliminate a portion of the total amount of fumonisins. Palencia et al. (2003) found that nixtamalization, when performed appropriately with adequate amounts of fresh water, eliminated approximately 50% of the fumonisin in maize used to make tortillas. Grain processing methods can be inefficient and time consuming, however, and grain used for animal feed is not processed by nixtamalization. Therefore, researchers have proposed that breeding for resistance to fumonisin accumulation should be the most effective and sustainable approach, as economical management options are limited (Bush, 2001).

Genetic variation for resistance to Fusarium ear rot exists among inbred lines and hybrids of field corn (Smith and Madsen, 1949; Koehler, 1953; King and Scott, 1981; Clements et al., 2004) and sweet corn (Headrick and Pataky, 1989; Nankam and Pataky, 1996). There is no evidence of complete resistance to either ear rot or fumonisin contamination in maize. Pérez-Brito et al. (2001) demonstrated that resistance to Fusarium ear rot in two tropical maize populations is polygenic with relatively low heritability. Shelby et al. (1994) reported significant variation among commercial maize hybrids for fumonisin concentration, but no hybrid was found to be completely resistant. Clements et al. (2004) screened topcrosses of more than 1000 inbred lines to the susceptible inbred tester FR1064 and found significant genetic variation for both Fusarium ear rot and fumonisin

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Abbreviations: GEFR, (GE440 × FR1064) × FR1064; GEI, Genotype × environment interaction; NCB, NC300 × B104; RIL, Recombinant inbred lines.

concentration, but no complete resistance to either. Analysis of the most resistant inbreds, their F_1 hybrids with FR1064 (a more susceptible B73-type inbred), and their F_2 and backcross to FR1064 progeny demonstrated nearly complete dominance or overdominance of fumonisin concentration resistance alleles.

Fumonisin concentration is generally greater in symptomatic kernels than in asymptomatic kernels (Desjardins et al., 1998). However, maize is often colonized endophyticially by *Fusarium* spp. (Oren et al., 2003), and it has been argued that significant amounts of fumonisin can be produced in symptomless or only slightly rotten grain (Bacon and Hinton, 1996; Munkvold and Desjardins, 1997). Furthermore, the phenotypic correlation between Fusarium ear rot and fumonisin concentration has been reported to be moderate to low.

Clements et al. (2004) estimated the correlation between fumonisin concentration and ear rot in maize to be $r = 0.54$ ($P < 0.0001$) in Illinois and $r = 0.60$ ($P < 0.0001$) in North Carolina. Furthermore, Clements et al. (2003a) found that ear rot and fumonisin concentration were not significantly correlated in plants inoculated with *Fusarium* spp., but ear rot and fumonisin concentration were significantly correlated in plots that had not been inoculated, although this phenotypic correlation was low ($r = 0.38$, $P = 0.0002$). Clements et al. (2004) observed low ear rot (less than 5%) and high fumonisin concentrations (greater than 20 $\mu g\ g^{-1}$) for 6% of hybrids evaluated in Illinois and 3% of hybrids evaluated in North Carolina.

The correlations reported between ear rot and fumonisin concentration phenotypes in these studies were affected by covariances between genetic, genotype × environment, and experimental error effects on these two traits. Therefore, the correlations reported do not necessarily provide an accurate indication of the magnitude of response of resistance to fumonisin concentration that would occur when selecting for ear rot resistance. To more precisely predict correlated responses to selection, the phenotypic correlation should be partitioned into genotypic and nongenotypic effects (Falconer and Mackay, 1996). This has not yet been reported for ear rot and fumonisin concentration. Due to the limited understanding of the relationships between *Fusarium* spp. infection, symptom development, and fumonisin contamination, the objective of this study was to estimate the heritabilities of, and the genotypic and phenotypic correlations between, fumonisin concentration, ear rot, and flowering time in two populations of maize. This information was used to predict the relative efficiency of direct selection on ear rot for indirectly improving fumonisin concentration levels.

MATERIALS AND METHODS

Population Development

GE440 (Clements et al., 2001) and NC300 (Holland, unpublished data, 2002) were identified in preliminary studies as potential sources for low fumonisin concentration and resistance to Fusarium ear and kernel rot. Two segregating populations, derived from the crosses GE440 × FR1064 and NC300 × B104, were created. Resistant inbred GE440 was crossed and backcrossed once to susceptible inbred FR1064 and BC_1F_1 plants were self-pollinated to form 215 $BC_1F_{1:2}$ families. This population will be referred to as the GEFR population. A second population was formed by crossing inbred NC300 to inbred B104. Random samples of F_2 plants from this cross were self-pollinated to form $F_{2:3}$ families. $F_{2:3}$ families were grown ear-to-row, and a single self-fertilized ear was harvested from each row without conscious selection. This procedure was repeated until 143 F_6–derived recombinant inbred lines (RILs) were developed. F_7 or F_8 generation seeds were used in this study to represent the RILs. This population was developed by Dr. M.M. Goodman and will be referred to as the NCB population.

GEFR Population Evaluation

In 2002 the GEFR population was grown at two locations, Mt. Olive, NC, and Urbana, IL. The experiment was planted in a randomized complete block design. Plots were single-row individual $BC_1F_{1:2}$ families replicated twice at each location. In 2003 the GEFR population was grown at two locations, Clayton, NC, and Plymouth, NC, with single-row plots of each $BC_1F_{1:2}$ family replicated twice at each location. The treatment design in 2003 was a replication within sets design, wherein each $BC_1F_{1:2}$ family was randomly assigned to one of three sets. Each set also contained the two parent lines and seven check lines (B73, Mo17, K55, NC300, NC390, NC402, and Va35). The experimental design for each set was an 8 by 10 α-lattice with two replications at each location. Sets were planted in adjacent plots at each location. At all locations, experimental units were single row plots of 3.05 m in length with 0.91 m between plots, and were overplanted and thinned to approximately 20 plants.

RIL Population Evaluation

In 2002, the NCB population was grown at Clayton, NC, with single row plots of each RIL in a randomized complete block design with two replicates. Each replicate also contained the two parent lines and the check inbred B73. In 2003, the population was grown at two locations, Clayton, NC, and Plymouth, NC. The NCB population plus the two parent lines and the check inbred B73 were evaluated using a 12 by 12 lattice design with two replications at each location. At both locations, experimental units were single row plots of 3.05 m in length with 0.91 m between plots. Plots were overplanted and thinned to approximately 20 plants.

GEFR and NCB Inoculation Procedures

To represent the heterogeneity of pathogen species in the natural environment, ears were inoculated with three isolates each of *F. verticillioides* and *F. proliferatum* (Clements et al., 2003b). *Fusarium verticillioides* isolates ISU95082, ISU94445, and ISU94040 and *F. proliferatum* isolates 310, 37–2, and 19 were removed from glycerol storage at −80°C and cultured separately on potato dextrose agar (Difco, Sparks, MD). Inoculum was prepared by washing conidia from the cultures with 0.05% Triton X-100 (Fisher Biotech, Fairlawn, NJ) and diluting the resulting conidial suspension of the six isolates to a final concentration to approximately 1×10^6 conidia mL^{-1} in water.

A preliminary experiment conducted in one environment to determine the effect of self-pollination vs. open pollination on fumonisin concentration in the NCB population revealed no significant interaction between genotype and pollination method (Robertson, unpublished data, 2005). Therefore, all ears were allowed to open pollinate. At each inoculation, the primary ear of each plant was inoculated with 10 mL of the inoculum. To reduce the chance of escapes, two inoculations,

1 wk apart, were done on primary ears of all plants in each plot, except for the NCB population in 2002, where only 12 plants were inoculated per plot. Silk channel inoculation (injecting inoculum into the silk channel while the silks are fresh) was performed approximately 10 d after mean silking date for each experiment within each location, and direct ear inoculation (injecting inoculum directly through the husk onto the ear) was performed 7 d later (Clements et al., 2003b).

When all plants reached physiological maturity, primary ears were hand harvested and air dried to approximately 14% moisture concentration. After drying, ears were rated by visually identifying the percentage of kernels exhibiting Fusarium ear rot symptoms on each inoculated ear. Grain from all inoculated ears within a plot was bulked and ground to a fine particle size with a Romer mill (Series II Mill, Romer Labs Inc., Union, MO). Fumonisin concentration was determined from a 25-g sample of ground grain from each plot. Samples were evaluated in triplicate with an enzyme-linked immunosorbant assay (Clements et al., 2003b).

Silk date was also recorded on every plot of the NCB population in Clayton in 2002 and 2003 as the number of days between planting and silk emergence on 50% of the plants in a plot. Silk date was recorded on every plot of the GEFR population in the Clayton 2003 environment only. The GEFR population was also grown in an independent field trial at Clayton, 2002. Each family was represented once in a single row plot in a randomized complete block. Silk date was recorded on each plot.

Statistical Analyses

For the GEFR population, fumonisin data were natural log transformed to remove heterogeneity of variance. Although sets and blocks were not originally used in the 2002 experiments, sets were added using the same set designation as in 2003, wherein each $BC_1F_{1:2}$ family had been randomly assigned to one of three sets, and in 2002, a single incomplete block was assigned to each replication. These modifications were made so that the environments could be analyzed together and the added information of set and block effects from the 2003 experiments would not be lost.

The model used in all of the GEFR analyses was

$$Y_{ijklm} = \mu + E_i + S_j + SE_{ij} + R(SE)_{ijk} + B(RSE)_{ijkl} + G(S)_{jm} + GE(S)_{ijm} + \varepsilon_{ijkm},$$

where μ = overall mean; E_i = effect of environment i; S_j = effect of set j; SE_{ij} = interaction of environment i and set j; $R(SE)_{ijk}$ = effect of replication k within environment i and set j; $B(RSE)_{ijkl}$ = effect of incomplete block l within environment i, set j, and replication k; $G(S)_{jm}$ = effect of genotype m within set j; $GE(S)_{ijm}$ = effect of interaction between environment i and genotype m within set j; and ε_{ijkm} = effect of experimental error on plot containing genotype m in replication k within set j and environment i. For ear rot, data was collected on individual plants, so the term ω_{ijkmn} was added to represent the effect of plant n of genotype m grown in plot k of set j and environment i.

A univariate analysis was conducted on each trait using PROC MIXED in SAS version 8.2 (Littell et al., 1996; SAS Institute, 1999). All effects in the model except the overall mean were considered random. The model including plants within plots for ear rot would not converge, so in that case we eliminated the incomplete block term to obtain convergence.

Genotypic correlations between the same trait measured in different environments were estimated for the GEFR population (Cooper and DeLacy, 1994). Heritabilities for fumonisin concentration and ear rot resistance in the GEFR population were estimated from the univariate mixed model analyses (Holland et al., 2003). Heritabilities were estimated on a family mean and per-plot basis for both traits. Heritability on a per-plant basis was estimated for ear rot only.

For the purposes of comparing the experimental entries to the parental lines and checks, a separate mixed model analysis was done, which included parental lines and considered genotypes to be fixed effects (Holland et al., 2003). Least square means of parents and $BC_1F_{1:2}$ families adjusted for set effects were obtained and the average standard error of a mean comparison was estimated for each trait.

We attempted to estimate genotypic and phenotypic correlations between fumonisin concentration, ear rot, and the silk dates taken in the Clayton 2003 environment using multivariate REML in PROC MIXED of SAS version 8.2 (Holland et al., 2001). However, the multivariate REML model would not converge, so a multivariate analysis of variance was performed for ear rot and fumonisin concentration using the MANOVA option of PROC GLM of SAS version 8.2. This permitted estimation of covariances between the traits due to each effect in the model specified above. In addition, linear correlations between mean fumonisin concentrations or ear rot scores averaged over the four disease evaluation environments with silk date measured in the independent environment were estimated using PROC CORR of SAS. These estimate genotypic correlations because only the genotypic effects have a covariance between plots grown in separate environments (Casler, 1982). The silk dates taken in the Clayton 2003 environment were used to determine the significance of the silk dates among GEFR families.

For the NCB population, fumonisin data were square root transformed to remove heterogeneity of variance. A univariate analysis was done on each trait with PROC MIXED in SAS version 8.2, considering all effects in the model except the overall mean to be random. The model used in all NCB analyses was

$$Y_{ijklm} = \mu + E_i + R(E)_{ij} + B(RE)_{ijk} + G_l + GE_{jl} + \varepsilon_{ijl},$$

where μ = overall mean; E_i = effect of environment i; R_{ij} = effect of replication j within environment i; $B(RE)_{ijk}$ = effect of incomplete block k within environment i and replication j; G_l = effect of genotype l; GE_{il} = effect of interaction between environment i and genotype l; and ε_{ijl} = effect of experimental error on plot containing genotype l in replication j and environment i.

Genotypic correlations between the same trait measured in different environments were estimated for each population (Cooper and DeLacy, 1994). Heritabilities for fumonisin concentration and ear rot resistance in the NCB population were estimated from the univariate mixed model analyses (Holland et al., 2003). Heritabilities were estimated on a family mean and per-plot basis for both traits. Heritability on a per-plant basis was estimated for ear rot only. Approximate standard errors for heritability estimates were estimated with the delta method as described by Holland et al. (2003).

Genotypic and phenotypic correlations between fumonisin concentration, ear rot, and maturity in the NCB population were estimated using multivariate REML in PROC MIXED of SAS (Holland et al., 2001). Standard errors of genotypic and phenotypic correlations obtained with multivariate analyses were also obtained with the delta method (Holland et al., 2003).

Relative efficiency of indirect selection in each population was obtained by calculating the ratio of correlated response of fumonisin concentration (CR_{fum}) when selection is based on ear rot severity to direct response when selection is based

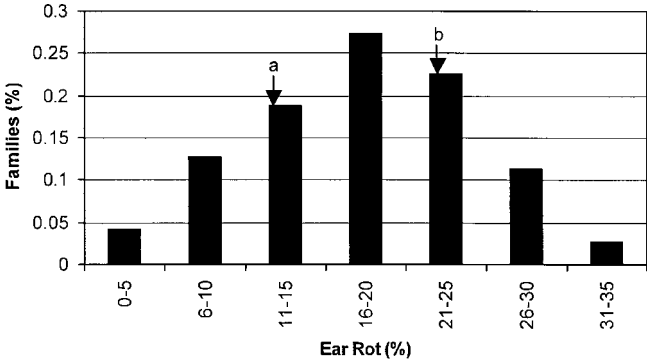

Fig. 1. Distribution of Fusarium ear rot severity among GEFR families. Values represent entry means across all environments. Parent line GE440 is represented by the letter a, and parent line FR1064 is represented by the letter b.

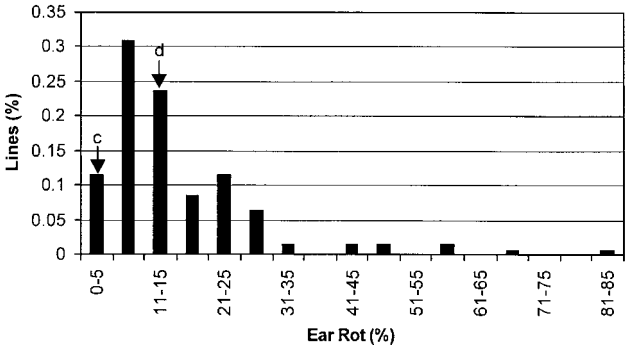

Fig. 2. Distribution of Fusarium ear rot severity among NCB recombinant inbred lines. Values represent entry means across all environments. Parent line NC300 is represented by the letter c, and parent line B104 is represented by the letter d.

on fumonisin concentration (R_{fum}). Equal selection intensities were assumed, so that $CR_{fum}/R_{fum} = \hat{r}_G\hat{h}_{rot}/\hat{h}_{fum}$, where $\hat{r}_G$ is the estimate of genotypic correlation between fumonisin and ear rot and $\hat{h}_{rot}$ and $\hat{h}_{fum}$ are the square roots of the family mean basis heritability estimates for rot and fumonisin, respectively (Falconer and Mackay, 1996).

RESULTS

In the GEFR and NCB populations, entries differed significantly ($P < 0.0001$) for ear rot, fumonisin concentration, and silk date. In the GEFR population, entry mean rot scores were distributed normally (Fig. 1). The entry mean rot scores in the NCB population were highly skewed toward lower rot scores (Fig. 2), probably because both parents have good resistance to Fusarium ear rot. In the GEFR population, entry mean fumonisin levels were skewed slightly toward lower fumonisin concentration (Fig. 3). In the NCB population, entry means for fumonisin concentration, like the rot scores, were highly skewed toward lower fumonisin concentration (Fig. 4).

In the GEFR population, GE440, the parent line that was selected on the basis of previous information indicating that it possessed some resistance to fumonisin concentration, differed significantly from the susceptible parent line, FR1064, for mean fumonisin concentration within and across environments (Table 1). In the

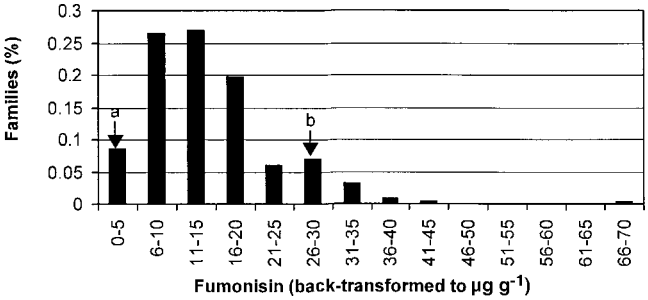

Fig. 3. Distribution of fumonisin concentration among GEFR families. Values represent back-transformed entry means across all environments. Parent line GE440 is represented by the letter a, and parent line FR1064 is represented by the letter b.

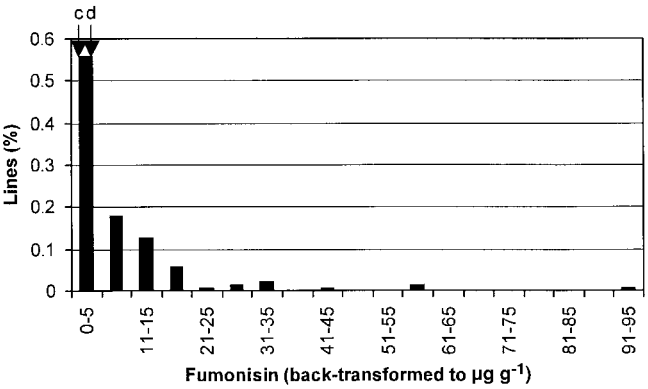

Fig. 4. Distribution of fumonisin concentration among NCB recombinant inbred lines. Values represent back-transformed entry means across all environments. Parent line NC300 is represented by the letter c, and parent line B104 is represented by the letter d.

NCB population, however, neither ear rot nor fumonisin concentration differed significantly between the two parents across environments, but both lines had lower mean fumonisin concentrations than the check inbred B73 (Table 2).

Evidence of transgressive segregation exists in both populations. One family in the GEFR population had significantly greater fumonisin concentration and one family had significantly greater ear rot than the susceptible parent, FR1064, across environments (Table 1). In the NCB population, ear rot and fumonisin concentration levels significantly greater than the B104 parent were observed in 18 and 26 lines across environments, respectively. In neither population was there a family or line that had a significantly lower fumonisin concentration or ear rot than the more resistant parent across environments. The GEFR population mean values within and across all locations for ear rot and fumonisin concentration were intermediate between the two parental lines, but closer to the FR1064 values, as expected because the population is derived from a backcross to FR1064 (Table 1). In contrast, the NCB population mean ear rot and fumonisin concentration exceeded both parental values within and across all locations (Table 2). Fumonisin concentration was significantly affected by genotype × environment interaction (GEI) in the GEFR ($P < 0.0001$) and in the NCB ($P = 0.0021$) populations. Ear rot was also significantly affected by GEI in both populations ($P < 0.0001$). Despite the significant GEI for both disease

Table 1. Mean natural log transformed fumonisin concentration and ear rot of BC_1S_1 families with greatest and least fumonisin concentrations, checks, and parents in the GEFR population, averaged across four environments and across replications within environments.†,‡

	Across environments		MTO 2002		URB 2002		CLY 2003		PLY 2003	
Family	Fumonisin	Rot	Fumonisin	Rot	Fumonisin	Rot	Fumonisin	Rot	Fumonisin	Rot
	μg g^{-1}	%	μg g^{-1}	%	μg g^{-1}	%	μg g^{-1}	%	μg g^{-1}	%
Families with least fumonisin concentration averaged over all environments										
397–8	0.4 (1.5)	<1	0.9 (2.5)	14	0.8 (2.2)	2	0.1 (1.1)	3	−0.1 (0.9)	3
399–3	1.2 (3.3)	3	2.7 (14.9)	20	1.4 (4.1)	4	0.7 (2.0)	1	2.5 (12.2)	8
400–9	1.2 (3.3)	17	2.4 (11.0)	71	1.5 (4.5)	7	0.4 (1.5)	5	0.8 (2.2)	7
397–10	1.2 (3.3)	7	3.1 (22.2)	37	1.0 (2.7)	4	1.1 (3.0)	4	0.2 (1.2)	5
412–13	1.3 (3.7)	21	1.8 (6.0)	22	1.5 (4.5)	2	0.0 (1.0)	1	1.4 (4.1)	53
396–14	1.3 (3.7)	11	3.4 (30.0)	55	0.9 (2.5)	2	−0.3 (0.7)	3	1.4 (4.1)	4
402–3	1.4 (4.1)	8	2.3 (10.0)	20	0.3 (1.3)	1	1.2 (3.3)	1	2.1 (8.2)	5
399–13	1.4 (4.1)	2	2.2 (9.0)	17	2.5 (12.2)	6	1.8 (6.0)	2	−0.4 (0.7)	6
400–3	1.4 (4.1)	7	3.4 (30.0)	39	1.2 (3.3)	2	0.5 (1.6)	2	1.1 (3.0)	5
399–1	1.5 (4.5)	6	2.7 (14.9)	36	2.1 (8.2)	7	0.8 (2.2)	1	0.8 (2.2)	1
Mean of 10 families with least fumonisin	1.2 (3.3)	9	2.5 (12.2)	33	1.3 (3.7)	4	0.6 (1.8)	2	1.0 (2.7)	10
Families with greatest fumonisin concentration averaged over all environments										
402–10	3.4 (30.0)	25	3.7 (40.4)	67	3.6 (36.6)	9	2.8 (16.4)	3	3.7 (40.4)	13
405–1	3.5 (33.1)	23	4.8 (121.5)	56	3.3 (27.1)	8	3.3 (27.1)	12	2.9 (18.2)	8
402–11	3.5 (33.1)	30	4.1 (60.3)	53	2.8 (16.4)	10	3.2 (24.5)	9	4.1 (60.3)	42
401–6	3.5 (33.1)	27	4.3 (73.7)	67	3.8 (44.7)	14	3.3 (27.1)	21	3.2 (24.5)	29
397–13	3.5 (33.1)	22	4.3 (73.7)	57	3.8 (44.7)	14	4.0 (54.6)	30	2.4 (11.0)	12
403–11	3.5 (33.1)	19	4.2 (66.7)	39	3.5 (33.1)	7	3.7 (40.4)	19	3.4 (30.0)	10
399–7	3.6 (36.6)	23	4.3 (73.7)	51	3.5 (33.1)	16	3.5 (33.1)	15	3.4 (30.0)	30
410–4	3.6 (36.6)	36	4.3 (73.7)	94	3.0 (20.1)	11	2.6 (13.5)	6	3.9 (49.4)	16
413–5	3.7 (40.4)	26	4.2 (66.7)	50	3.4 (30.0)	12	3.2 (24.5)	7	3.7 (40.4)	21
413–7	4.2 (66.7)	32	3.9 (49.4)	46	4.8 (122.0)	20	3.5 (33.1)	19	4.1 (60.3)	28
Mean of 10 families with greatest fumonisin	3.6 (36.6)	26	4.2 (66.7)	58	3.6 (36.6)	12	3.3 (27.1)	14	3.5 (33.1)	21
Mean of GEFR population	2.5 (12.2)	18	3.6 (36.6)	45	2.3 (10.0)	7	2.0 (7.4)	7	2.2 (9.0)	14
Parental lines and checks										
GE440	0.8 (2.2)	11	–	–	–	–	0.5 (1.6)	2	0.1 (1.1)	4
FR1064	3.3 (27.1)	22	–	–	–	–	2.5 (12.2)	8	3.4 (30.0)	22
B73	4.2 (66.7)	41	–	–	–	–	3.8 (44.7)	37	3.7 (40.4)	31
K55	3.0 (20.1)	38	–	–	–	–	3.1 (22.2)	29	2.0 (7.4)	31
Mo17	2.0 (7.4)	31	–	–	–	–	0.5 (1.6)	9	2.7 (14.9)	38
NC300	1.2 (3.3)	16	–	–	–	–	0.5 (1.6)	5	1.1 (3.0)	11
NC390	2.1 (9.0)	20	–	–	–	–	1.5 (4.5)	12	1.8 (6.0)	13
NC402	3.3 (27.1)	36	–	–	–	–	3.1 (22.2)	28	2.6 (13.5)	29
Va35	2.4 (11.0)	27	–	–	–	–	1.4 (4.1)	8	2.6 (13.5)	31
LSD-1 (0.05)§	0.9 (NA¶)	13	1.1 (NA)	26	1.4 (NA)	7	1.6 (NA)	15	1.7 (NA)	26
LSD-2 (0.05)#	0.2 (NA¶)	3	0.3 (NA)	6	0.3 (NA)	2	0.4 (NA)	3	0.4 (NA)	6

† MTO, Mt. Olive, NC; URB, Urbana, IL; CLY, Clayton, NC; PLY, Plymouth, NC.
‡ Values in parentheses represent means back-transformed to approximate their original value of μg g^{-1}.
§ LSD-1 is appropriate to compare a pair of entry (family or check) means.
¶ Not applicable.
# LSD-2 is appropriate to compare the mean of 10 families to the overall population mean.

traits in both populations, the mean genetic correlations of traits measured in different environments were always equal to or greater than 0.70. In the GEFR population, the mean genetic correlations between a disease trait measured in different environments were high for both ear rot (r_G = 0.70) and fumonisin concentration (r_G = 0.79). The mean genetic correlations between environments were also high in the NCB population for both ear rot (r_G = 0.74) and fumonisin concentration (r_G = 0.87).

Further evidence of the stability of resistance is provided by the ear rot and fumonisin concentration values within each environment of the 10 lines with lowest and 10 lines with highest mean fumonisin concentrations across environments in each population (Tables 1 and 2). In both populations and within every environment, except for ear rot in the Plymouth 2003 environment for the GEFR population, the mean ear rot and fumonisin concentration of the 10 lines with lowest mean fumonisin concentrations was significantly lower than the overall population means. Similarly, the 10 lines with highest mean fumonisin concentrations had significantly higher mean fumonisin concentration and ear rot values than the overall population mean within each environment in both populations.

Heritabilities on an entry mean basis were moderately high to high for fumonisin concentration in both the GEFR (h^2 = 0.75, SE = 0.03) and NCB (h^2 = 0.86, SE = 0.02) populations. Heritability of ear rot on an entry mean basis was moderately high in the GEFR population (h^2 = 0.47, SE = 0.06) and high in the NCB population (h^2 = 0.80, SE = 0.03). Heritabilities on a plot basis were much lower than entry mean heritabilities for fumonisin concentration in both the GEFR (h^2 = 0.31, SE = 0.03) and NCB (h^2 = 0.54, SE = 0.04) populations. Heritabilities on a plot basis for ear rot were also lower in the GEFR (h^2 = 0.13, SE = 0.03) and NCB (h^2 = 0.47, SE = 0.04) populations. Heritabilities on an individual plant basis for ear rot were h^2 = 0.21 (SE = 0.03) in the NCB population and only h^2 = 0.03 (SE = 0.01) in the GEFR population. For both traits, corresponding heritability estimates were always higher in the NCB population than in the GEFR population.

Table 2. Mean square root transformed fumonisin concentration and ear rot of RILs with greatest and least fumonisin concentrations, checks, and parents in the NCB population, averaged across replications within environments.†,‡

	Across environments		CLY† 2002		CLY 2003		PLY 2003	
Line	Fumonisin	Rot	Fumonisin	Rot	Fumonisin	Rot	Fumonisin	Rot
	μg g^{-1}	%	μg g^{-1}	%	μg g^{-1}	%	μg g^{-1}	%
Lines with least fumonisin concentrations averaged over all environments								
RIL 41	0.5 (0.3)	3	0.9 (0.8)	5	0.4 (0.2)	2	0.3 (0.1)	1
RIL 135	0.8 (0.6)	5	0.6 (0.4)	3	0.5 (0.3)	3	1.3 (1.7)	8
RIL 73	0.9 (0.8)	4	0.5 (0.3)	4	1.0 (1.0)	2	1.4 (2.0)	7
RIL 42	1.0 (1.0)	11	–	–	1.3 (1.7)	4	1.2 (1.4)	16
RIL 137	1.0 (1.0)	8	0.9 (0.8)	5	0.7 (0.5)	3	1.3 (1.7)	17
RIL 120	1.0 (1.0)	8	0.5 (0.3)	5	1.1 (1.2)	5	1.5 (2.3)	14
RIL 68	1.1 (1.2)	3	1.0 (1.0)	5	0.8 (0.6)	2	1.5 (2.3)	2
RIL 118	1.1 (1.2)	8	0.4 (0.2)	5	1.6 (2.6)	6	1.4 (2.0)	11
RIL 1	1.2 (1.4)	5	1.0 (1.0)	5	0.7 (0.5)	2	1.8 (3.2)	8
RIL 5	1.2 (1.4)	6	0.8 (0.6)	5	1.7 (2.9)	6	1.0 (1.0)	6
Mean of 10 lines with least fumonisin	1.0 (1.0)	6	0.7 (0.5)	5	1.0 (1.0)	4	1.3 (1.7)	9
Lines with greatest fumonisin concentrations averaged over all environments								
RIL 91	4.8 (23.0)	26	6.8 (46.2)	26	2.7 (7.3)	6	4.4 (19.4)	45
RIL 126	5.2 (27.0)	26	6.2 (38.4)	31	2.8 (7.8)	9	6.6 (43.6)	36
RIL 65	5.4 (29.2)	31	6.7 (44.9)	30	3.9 (15.2)	15	5.6 (31.4)	49
RIL 113	5.5 (30.3)	67	8.0 (64.0)	92	3.2 (10.2)	32	6.6 (43.6)	85
RIL 139	5.8 (33.6)	49	6.9 (47.6)	75	5.4 (29.2)	36	5.2 (27.0)	37
RIL 117	5.9 (34.8)	24	5.2 (27.0)	35	4.8 (23.0)	19	7.9 (62.4)	18
RIL 80	6.6 (43.6)	24	9.0 (81.0)	37	6.5 (42.3)	20	4.3 (18.5)	15
RIL 57	7.6 (57.8)	30	7.2 (51.8)	31	–	–	–	–
RIL 105	7.7 (59.3)	59	8.4 (70.6)	82	8.0 (64.0)	60	6.9 (47.6)	36
RIL 64	9.7 (94.1)	82	–	–	10.1 (102.0)	76	9.8 (96.0)	85
Mean of 10 lines with greatest fumonisin	6.4 (41.0)	42	7.2 (51.8)	49	5.3 (28.1)	30	6.4 (41.0)	45
Mean of NCB population	2.6 (6.8)	16	2.1 (4.4)	17	2.2 (4.8)	12	3.3 (10.9)	19
Parental Lines and Checks								
NC300	0.9 (0.8)	5	1.3 (1.7)	6	1.0 (1.0)	6	1.5 (2.3)	5
B104	2.0 (4.0)	13	0.2 (0.0)	10	2.0 (4.0)	12	2.8 (7.8)	17
B73	3.8 (14.4)	21	–	–	3.5 (12.3)	7	4.6 (21.2)	33
LSD-1 (0.05)§	1.4 (NA¶)	13	2.2 (NA)	20	2.0 (NA)	17	2.3 (NA)	19
LSD-2 (0.05)#	0.5 (NA¶)	4	0.5 (NA)	5	0.5 (NA)	4	0.5 (NA)	4

† CLY, Clayton, NC; PLY, Plymouth, NC.
‡ Values in parentheses represent means back-transformed to approximate their original value of μg g^{-1}.
§ LSD-1 is appropriate to compare a pair of entry (line or check) means.
¶ Not applicable.
# LSD-2 is appropriate to compare the mean of 10 lines to the overall population mean.

Genotypic and phenotypic correlations between ear rot and fumonisin concentration were positive and significant in both populations. In the GEFR population, genotypic correlation was very high (r_G = 0.96, SE = 0.07), whereas the phenotypic correlation was only moderate (r_P = 0.40; SE = 0.03). Genotypic correlation between fumonisin concentration and silk date was not significant, and the correlation between ear rot and silk date was significant but low (r_G = 0.18, P = 0.0099).

In the NCB population, genotypic correlation between fumonisin concentration and ear rot was high (r_G = 0.87, SE = 0.04) and phenotypic correlation was moderate (r_P = 0.64, SE = 0.03). Genotypic and phenotypic correlations between fumonisin concentration and silk date did not exceed twice their standard errors, so were not considered significant (r_G = 0.20, SE = 0.11; r_P = 0.10, SE = 0.06). Genotypic and phenotypic correlations between ear rot and silk date were significant but low (r_G = 0.28, SE = 0.11; r_P = 0.15, SE = 0.06).

In the GEFR population, when comparing indirect selection to direct selection, we predicted CR_{fum}/R_{fum} = 0.76. In the NCB population, we predicted CR_{fum}/R_{fum} = 0.84. In both populations, direct selection is expected to be more effective than indirect selection because $CR_{fum}/R_{fum} \leq 1$.

DISCUSSION

The component of the GEI that complicates selection in different environments is the change in rank of genotypes. By calculating the genetic correlation of a single trait measured in different environments (Cooper and DeLacy, 1994), we were able to estimate the importance of genotypic rank changes across environments for ear rot and fumonisin concentration. The high genetic correlations across environments in both populations indicated that the significant G × E effects were primarily due to heterogeneity of genotypic variance, rather than a lack of correlation of genotype performance, in the different environments. Therefore, genotype means across environments are presented, and significant differences among genotypic means across environments were observed (Tables 1 and 2).

The estimates of heritability on an entry mean basis provided further evidence of the greater importance of genetic effects in the estimation of family means across environments. Heritability estimated on an entry mean basis is relevant to predicting selection among families based on their mean phenotypes using the same experimental design as was used for heritability estimation. Although the heritability estimates on individual

plant bases (for ear rot) and on individual plot bases for both traits were very low to moderate, our results suggest that with adequate replication within and across environments, estimates of family means that are influenced primarily by genetic effects can be obtained. These heritability estimates suggest that ear rot and fumonisin contamination resistance should respond well to selection using family means.

Heritability estimated on an individual plant basis indicates the extent to which selection among individual plants will result in genetic gain. Heritability on an individual plant basis was almost zero in the GEFR population, but substantially greater (0.21) in the NCB population. This is likely because the NCB population is composed of inbred lines, whereas the GEFR population is composed of early generation segregating families. Therefore, plant-to-plant genetic and environmental variation within early generation families may be sufficiently large to make selection among individual plants unreliable. Even the heritabilities for ear rot and fumonisin on a plot basis were relatively low in the GEFR population, suggesting that replicated, multi-environment trials will be substantially more effective at identifying superior families than unreplicated progeny rows.

In the NCB population, genetic variance for ear rot resistance and fumonisin concentration is due to additive effects only, since families are composed of inbred lines. Therefore, the estimate of heritability from this population is appropriate for predicting gain from selection among RILs. The estimate of heritability from the GEFR population is also appropriate for predicting response to selection among $BC_1F_{1:2}$ families, in the absence of epistasis. This is surprising, because the families are not highly inbred. Dominance variance contributes to the genotypic variance, but it turns out that the deviations of $BC_1F_{1:2}$ families from the mean of their population, which is not a Hardy-Weinberg population, are directly proportional to deviations of their random-mated progeny from the mean of the resulting Hardy-Weinberg population (derivation not shown). Therefore, the ratio of the genotypic variance among $BC_1F_{1:2}$ families to the phenotypic variance of family means is an appropriate estimate of heritability for predicting response to selection (Holland et al., 2003).

Due to the silk dates being significantly different among genotypes in the GEFR and NCB populations and the small but significant positive correlation only between silk date and ear rot in the both populations, it seems that maturity plays a real but minor role in ear rot resistance. This suggests that the severity of ear rot symptoms depends, in part, on the developmental stage of plants when ears are infected with *Fusarium* spp. Alternatively, such a correlation also could arise due to linkage between resistance genes and flowering genes. Since the correlation between ear rot and silking date was positive, the later a plant flowered, the more likely the ear would display rot symptoms.

Genotypic correlation was greater than phenotypic correlation between ear rot and fumonisin concentration in both populations, indicating that genotypic effects on susceptibility to ear rot and fumonisin concentration are highly correlated, but that genotype $\times$ environment and error effects are not as highly correlated between the two traits. This suggests that the genetically controlled mechanisms of resistance to these two aspects of disease are largely the same, and that selection for ear rot should affect fumonisin concentration and vice versa. The GEFR population had a lower phenotypic correlation between ear rot and fumonisin concentration than the NCB population. This was probably a result of evaluating the GEFR population in more diverse environments (North Carolina and Illinois), and having greater within-plot variation due to genetic segregation within families. Although primary ears on all plants were injected with the same quantity of inoculum, there is a large environmental effect on *Fusarium* infection and symptom development. This can be illustrated by differences in mean rot scores and fumonisin concentrations between the North Carolina location (mean ear rot 45%; mean back-transformed fumonisin concentration 36.6 $\mu g\ g^{-1}$) and the Illinois location (mean ear rot 7%; mean back-transformed fumonisin concentration 10.0 $\mu g\ g^{-1}$) in the 2002 GEFR population study.

Genotypic and phenotypic relationships between fumonisin concentration and ear rot can be observed in scatter diagrams of the two traits measured on individual plots within each population (Fig. 5 and 6). In general, the trend of simultaneously increasing ear rot and fumonisin concentration is obvious, but outliers can be observed in the bottom right-hand corners of each plot. These outliers represent individual plots that had severe ear rot but low fumonisin concentrations. In some severely rotted ears, much of the kernel tissue was so badly destroyed that it did not contribute to the ground grain sample. Conversely, outliers also were observed in the upper left-hand corners of both diagrams. These outliers represent plots with low ear rot but high fumonisin concentrations. This supports the suggestion of Munkvold and Desjardins (1997) that fumonisin can accumulate in kernels with minimal ear rot.

High genotypic correlation between the two traits suggests that those genotypes that are most resistant to ear rot will, on average, across replications and environments, tend to be the same genotypes that are most resistant to fumonisin contamination. Thus, from a breeder's point of view, selecting against ear rot may be a useful strategy for selecting genotypes with less genetic susceptibility to high fumonisin concentration. In the NCB population (Fig. 5), if selection is made for lines with individual plot ear rot severity less than 20%, 95% of these plots would have represented the 10 lines with the lowest fumonisin concentrations. Or if 10 lines with least mean fumonisin concentrations were selected, only 5% of their individual plots would have fumonisin concentrations greater than 4 $\mu g\ g^{-1}$ (Fig. 5). Since families in the GEFR population are segregating, the families with the least ear rot or least mean fumonisin concentration could have high fumonisin concentrations in some individual plots (Fig. 6). Within-family segregation for resistance could have lowered the heritability of both traits, because data were collected on a plot basis and a few susceptible plants within a plot can make the entire plot more susceptible.

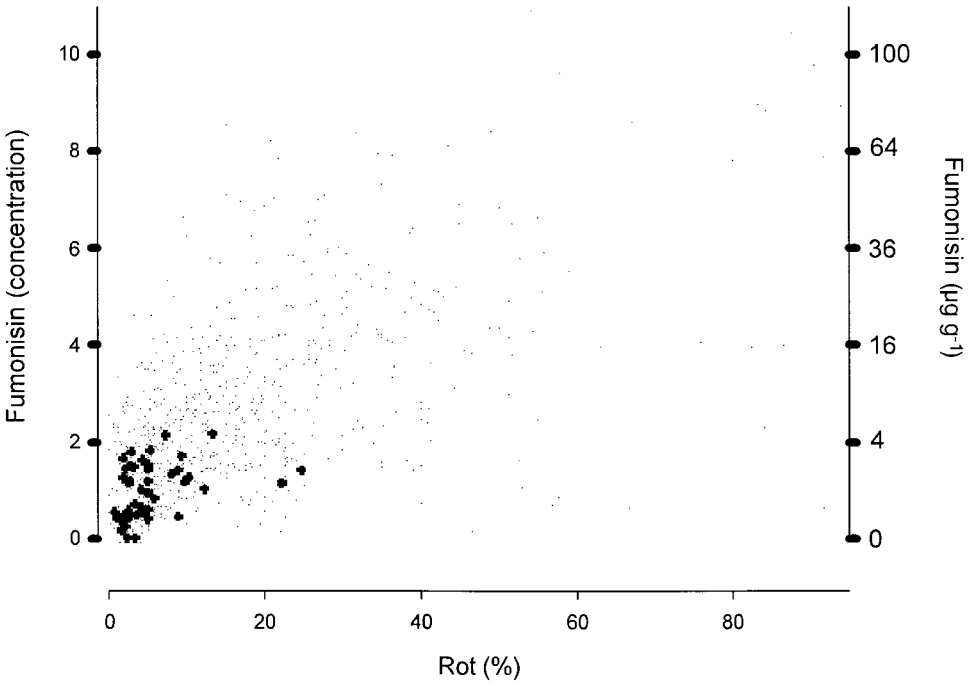

Fig. 5. Scatter diagram of individual plot values of Fusarium ear rot severity and transformed fumonisin concentration in the NCB population in 2002 and 2003. Darker points indicate 10 recombinant inbred lines with the lowest entry mean fumonisin concentrations across all environments. Fumonisin (concentration) represents square root transformed data and fumonisin (μg g^{-1}) represents back-transformed data.

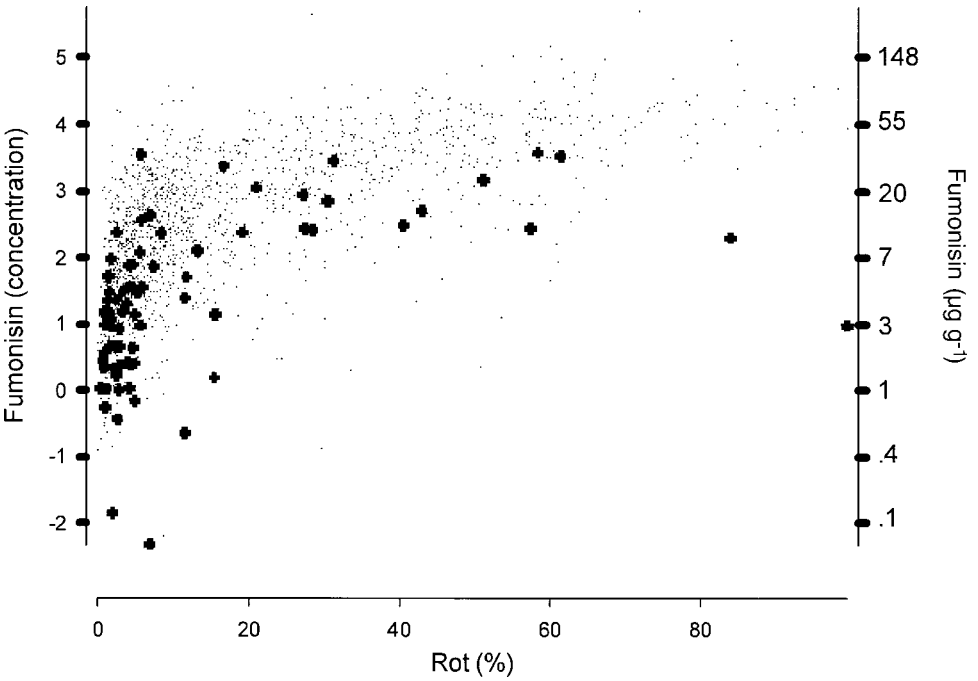

Fig. 6. Scatter diagram of individual plot values of Fusarium ear rot severity and transformed fumonisin concentration in the GEFR population in 2002 and 2003. Darker points indicate 10 families with the lowest entry mean fumonisin concentration across all environments. Fumonisin (concentration) represents natural log transformed data and fumonisin (μg g^{-1}) represents back-transformed data.

In both populations predicted correlated response on fumonisin concentration when selection is based on low ear rot severity was lower than the predicted response to direct selection for low fumonisin concentration. However, this calculation does not represent relative economic efficiency of indirect selection. Fumonisin assays are expensive, time consuming, and destructive, and it is not practical or cost efficient to perform evaluation on individual ears. Although heritability for ear rot was lower than heritability for fumonisin concentration estimated with a bioassay, a trained researcher can easily and rapidly visually select families and individual plants with low ear

rot. Since phenotyping ear rot is less costly and time consuming than performing laboratory analysis of fumonisin concentration, greater numbers of genotypes and greater numbers of replications of experiments can be screened. This should lead to greater selection intensity and perhaps higher entry mean basis heritability for ear rot for a fixed investment of resources. Nevertheless, later generation lines selected for reduced ear rot should be evaluated for low fumonisin concentration directly to verify that resistance has been identified. In conclusion, moderate to high heritabilities and strong genetic correlations suggest that selection for resistance to ear rot should result in lines with greater resistance to fumonisin contamination in grain.

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