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ORIGINAL PAPER

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Inheritance of Fusarium head blight resistance in the soft red winter wheat Ernie

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Abstract Fusarium head blight (FHB), caused by *Fusarium graminearum* Schwabe [telomorph:*Gibberella zeae* Schw. (Petch)], is an increasingly important disease of wheat (*Triticum aestivum* L.). Host-plant resistance is considered to be the most economical means of control, but a lack of unique sources of resistance has hindered efforts to breed resistant varieties. The soft red winter wheat, Ernie, has moderately high FHB resistance and is widely used in U.S. breeding programs; however, the genetics of resistance have not been studied. The objectives of this study were to estimate the genetic effects, gene numbers, and heritability for traits related to FHB resistance in Ernie through generation means analyses and variance analyses of 243 F_3-derived F_8 and F_9 recombinant inbred lines (RILs). Replicated experiments were grown in the greenhouse, inoculated with *F. graminearum*, and evaluated for disease spread and the FHB index (FHBI). The latter was calculated as the percentage of diseased spikelets in inoculated spikes and is often referred to as type-II resistance. Gene action for both disease spread and FHBI was primarily additive with partial dominance for low disease. Broad-sense heritabilities for spread and FHBI were 78.2% and 78.3%, respectively, while the narrow-sense heritabilities were 51.3% and 55.4%, respectively. Line-mean heritabilities from analyses of variance of RILs were 0.70 and 0.87 for spread and FHBI, respectively. A minimum of four genes conditioned both disease spread and FHBI. These results suggest that breeders should be able to enhance FHB resistance by combining the resistance in Ernie with other complementary additive sources of resistance.

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Introduction

Fusarium head blight (FHB), caused by *Fusarium graminearum* Schwabe [telomorph: *Gibberella zeae* Schw. (Petch)], is a disease that affects wheat (*Triticum aestivum* L. and *T. durum* L.) and barley (*Hordeum vulgare* L.) in warm, humid areas of the world (Schroeder and Christensen 1963; Snijders 1990a). It is an increasingly serious problem in the north-central and eastern regions of the USA because of the emphasis on conservation tillage (Wilcoxson et al. 1988; Bai and Shaner 1994), rotations with corn (Windels and Kommedahl 1984), and the lack of effective cultural and/or fungicide control (McMullen et al. 1997). Wheat and barley losses caused by FHB epidemics in the USA during the 1990s were estimated to be close to $ US 3 billion (Windels 2000).

Host-plant resistance is considered to be the most practical and effective means of control (Schroeder and Christensen 1963); however, breeding has been hindered by a lack of effective resistance genes. As no source of complete resistance is known, and current sources provide only partial resistance, the identification and genetic analysis of different sources of resistance is critical to the continued improvement of FHB resistance in winter wheat.

Resistance to FHB is complex, consisting of several, apparently independent types of resistance (Mesterházy 1995). Resistant germplasm currently used in wheat breeding programs primarily provides type-II resistance, which has been defined by Mesterházy (1995) as resistance to pathogen spread within the head. The genetics of type-II resistance, however, are not well understood. Researchers have found FHB resistance to be quantitatively inherited (Liao and Yu 1985; Sheng and He

1989), oligogenic (Liu et al. 1989; Bai et al. 2000), or monogenic (Chen 1989). The contradiction in these results could be due to the source of resistance investigated, the contribution of genes from the susceptible parent, the disease screening methods used, and/or the isolate of the pathogen used for evaluating the phenotype. All of these factors can influence the number of genes detected in inheritance studies (Kolb et al. 2001).

Sumai 3 is widely used in U.S. wheat breeding programs as a source of type-II resistance. The genetics of this source of resistance have been studied in both Sumai 3 and its derivatives. Ban and Suenaga (2000) used doubled-haploid (DH) lines developed from two Sumai 3 crosses (Sumai 3/Emblem and Sumai 3/Gamenya) and recombinant inbred lines (RILs) of the cross Emblem/Saikai 165 to determine the inheritance of FHB resistance in Sumai 3 and its derivative Saikai 165 (Sumai 3/Aakaze-komugi). The results indicated that resistance in Sumai 3 was conditioned by two major genes with additive effects, while that in Saikai 165 was controlled by three genes, suggesting that one gene for resistance came from Aakaze-komugi. Van Ginkel et al. (1996) studied FHB resistance in the Sumai 3 derivative, Ning 7840 and the Brazilian spring wheat, Frontana. Their results indicated that Ning 7840 and Frontana each had two different dominant genes conditioning resistance.

Using generation mean analyses, Bai et al. (2000) studied the inheritance of type-II resistance in six resistant cultivars from China, including: Fu 5114, Ning 7840, Ning 8306, Ning 8331, Sumai 3, and Sumai 49. Each was crossed to two susceptible cultivars, Clark and Morocco. While the additive genetic effects associated with higher levels of resistance were significant in all of the crosses studied, the choice of susceptible parent appeared to affect the extent of the dominance and epistatic effects involved. Dominance effects were only significant in Clark crosses involving the resistant parents Ning 8306, Ning 7840, and Sumai 49, while additive × additive and/or dominance × dominance were significant for the crosses Fu 5114/Clark and Sumai 3/Morocco. These results clearly indicate the impact that the choice of susceptible parent can have on the results of inheritance studies.

Although Asian sources of resistance frequently provide excellent levels of type-II resistance, the breeding of resistant winter wheat varieties using these sources is hindered by their lack of adaptation to winter wheat regions. Progeny are often late and tall and show a susceptibility to other economically important diseases in the U.S. winter wheat regions.

Routine screening of germplasm in U.S. wheat-breeding programs has enabled breeders to identify other incidental sources of resistance including those in Ernie (McKendry et al. 1995), Freedom (Gooding et al. 1997), and Roane (Griffey et al. 2001). These sources of resistance are important to U.S. breeding programs because they can produce segregating populations that are adapted, moderately resistant to FHB, have acceptable agronomic traits and have end-use quality suitable for the U.S. soft wheat market.

Ernie, a soft red winter wheat cultivar released by the University of Missouri Agricultural Experiment Station in 1994 (McKendry et al. 1995), has moderately-high type-II FHB resistance. Based on pedigree analysis, this resistance is not derived from Chinese or other known sources of resistance. Ernie is widely used in U.S. breeding programs as a complementary source of resistance to Sumai 3 and currently serves as an early-maturing resistant check variety in both the U.S. Northern and Southern Winter Wheat Scab Nurseries, however the genetics of resistance in Ernie are not well understood.

Hall and Van Sanford (2003) included Ernie in a diallel analysis of FHB resistance in soft red winter wheat and found significant general combining ability effects for reduced FHB severity in a field study of combining ability effects. While these data suggested that resistance in Ernie is conditioned by additive genetic effects, greenhouse data from a parallel study did not support this conclusion and, consequently, the genetics of resistance remain unclear. The purpose of the investigation reported here was to study the inheritance of type-II resistance in Ernie using generation means analyses and the analyses of variance of Ernie-derived RILs.

Materials and methods

Germplasm

Wheat (*Triticum aestivum* L.) cultivar Ernie originated from the cross Pike/MO 9965 made in 1980. Pike was derived from the cross Sava/Stoddard/3/Suwon 92/Burt//Stoddard. MO 9965 was developed from the cross Stoddard/Blueboy//Stoddard/D 1707. D 1707 is a two-gene semi-dwarf line from India derived from International Maize and Wheat Improvement Center (CIMMYT) germplasm. Based on pedigree analysis, the source of FHB resistance in Ernie differs from that in Sumai 3. Populations for genetic study were developed from a cross of Ernie with the highly FHB-susceptible Missouri genotype, MO 94-317, derived from the cross AgriproTraveller/Pioneer Variety 2555.

Five generations, including the F_1 (Ernie/MO 94-317), reciprocal F_1 (MO 94-317/Ernie), BC_1 (F_1/Ernie), BC_2 (F_1/MO 94-317), and the F_2 were developed in the greenhouse during the autumn and spring of 2000 and 2001. Two hundred and forty-three F_3-derived F_8 RILs were also developed from the cross Ernie/MO 94-317. Development of these lines was initiated in 1995. The F_2 and F_3 generations were advanced in the field as bulk populations without selection in 1997 and 1998. In 1998, a random set of 1,000 F_3 heads was taken and advanced to the F_7 by single-seed-descent in the greenhouse. A random sub-set of 243 F_8 and F_9 RILs was used for this study.

Plants for all generations and eight plants per RIL were vernalized at 4°C for 2 months and transplanted into Promix (Premier Horticulture, Quakertown, Pa.) in D40 Deepots (Hummert International, Earth City, Mo.) in the greenhouse. For generation means analysis, plants were arranged in a completely random design (CRD), and the experiment was repeated twice in the winter of 2003, separated in time by approximately 2 months. Because the greenhouse environment differed significantly in terms of ambient light and temperature for these two experiments, each repetition was considered to be a separate environment.

RILs were arranged in a randomized complete block design with three replications. Within each replication, RILs were randomized by line rather than by individual plant to reduce the within-line variation and maximize the between-line variation. The experiment was repeated in 2002 and 2003 with F_8 and F_9 RILs, respectively.

Disease evaluation

This investigation was designed to evaluate the inheritance of type-II resistance in Ernie. The repeatability of this type of genetic analysis requires that disease levels be high, phenotypic evaluations be accurate, the effect of environmental variation and variation due to technique be minimized, and that the resulting data not be confounded by other types of resistance. Greenhouse evaluations provide the level of control of environment and technique necessary to measure type-II resistance with the level of precision necessary to ensure the repeatability of results. As such, these experiments were conducted in the greenhouse environment.

A Missouri isolate of *Fusarium graminearum* that had been earlier tested for aggressiveness was used for all inoculations. The isolate used was selected because it was the most aggressive isolate on the University of Missouri's most resistant genotypes (Anne McKendry, personal communication). Each plant was inoculated at first anthesis with 10 µl of a macroconidial suspension of *F. graminearum* concentrated to 50,000 macroconidia per millilter. The inoculum was placed in the basal floret of a central spikelet. Plants were incubated in a mist chamber at 100% relative humidity for 72 h post-inoculation to initiate disease and then returned to the greenhouse bench to enable the disease to develop. Ratings for type-II resistance were made at 21 days post-inoculation. The data collected included: (1) the total number of spikelets in the inoculated head; (2) the number of spikelets showing symptoms of FHB, including those spikelets that wilted due to disease (hereafter referred to as disease spread). The Fusarium head blight index (FHBI) was calculated as the number of diseased spikelets/total number of spikelets in the inoculated head and was expressed as a percentage.

Statistical analyses

Statistical analyses were performed using SAS systems for Microsoft Windows ver. 8.0 (SAS Institute, Cary, N.C.). Analyses of variance for all experiments were done using SAS PROC GLM. For generation means analyses, both environments and generations were considered fixed effects. Effects of generation, environments, and generation × environment interaction were tested using the pooled CRD error. Generation means analysis, as outlined by Mather and Jinks (1977), was used to estimate the type of gene action and the conformity of the genetic system governing the expression of each trait to a simple additive-dominance genetic model. Since the means of each generation were not determined with equal precision, generation means and their expectations were weighted by the reciprocal of the variance of the means. Lack of fit of the genetic model to the data, as indicated by a χ^2 test, implied the existence of epistatic effects. These were accommodated by increasing the complexity of the model to include the additive × additive, additive × dominance, and dominance × dominance non-allelic interaction components (*i*, *j* and *l*, respectively).

Analyses using second-degree statistics were performed for each trait on the variances of all generations. The variation of parental and F_1 generations was used to estimate the non-heritable variation, while the variances in the F_2, BC_1, and BC_2 generations were partitioned into additive (D), dominance (H), and environmental (E) components as outlined by Mather and Jinks (1977). The model was estimated using an unweighted-least-squares approach. Broad-sense heritabilities for the F_2 were calculated as the total genetic variance (1/2D + 1/4H) divided by the total phenotypic variance (1/2D + 1/4H + E), while narrow-sense heritabilities were calculated as the additive variance in the F_2 (1/2D) divided by the phenotypic variance.

Analysis of variance of RILs for both disease spread and FHBI was carried out individually by experiment. Data for both disease spread and FHBI approximated normal distributions. Bartlett's test indicated that error variances were homogeneous ($P = 0.05$). Pearson correlation coefficients between experiments in 2002 and 2003 were calculated with the SAS PROC CORR procedure. Broad-sense heritability estimates on an entry-mean basis were determined from the combined analysis of variance for both FHBI and disease spread as $h^2_{BS} = \sigma^2_G/(\sigma^2_G + \sigma^2_{G\times E}/E + \sigma^2_e/R\times E)$, where σ^2_G is the genetic variance among RILs, R is the number of replications, and E is the experiment or year. Ninety percent confidence intervals for heritability estimates were determined according to Knapp et al. (1985). Precise values from the F distribution were determined at $P = 0.95$ and $P = 0.05$ using the FINV function of Excel (Microsoft Excel 2003).

Minimum gene numbers were determined using Cockerham's modification (Cockerham 1983) of Wright's formula (Wright 1968) in which the F_8 and F_9

generations are considered to be homozygous. This modification, which factors broad-sense heritability into the equation, tends to eliminate the environmental variation in the expression of the trait. The minimum gene number (n) for each trait, therefore, was calculated as $n = (GR \times h^2_{BS})^2 / 4.27 \times \sigma^2_G$, where GR is the genetic range among RILs, h^2_{BS} is the broad-sense heritability, and σ^2_G is the genetic variance among RILs.

Results

Generation means analyses

For disease spread and FHBI, analyses of variance indicated that both generation and environmental effects were significant while the generation × environment interaction effect was not significant (Table 1). Although the experiment was conducted in the greenhouse, it was repeated in time and, consequently, ambient day length and temperature differed. Both of these environmental factors are known to impact disease, hence the large environmental component of the observed variation.

The means for disease spread in Ernie (1.7 spikelets) and MO 94-317 (11.8 spikelets) were significantly different (Table 2), while the means of the F_1 (3.7 spikelets) and reciprocal F_1 (4.1 spikelets) were not significantly different. These results suggested that resistance was predominantly under nuclear genetic control and, therefore, the data were analyzed as such. The mean for disease spread of the pooled F_1 was significantly less than that of the mid-parent, suggesting partial dominance for resistance. The mean of the F_2 was significantly less than that of the mid-parent using the minimum least significant difference (LSD) but not different from the mid-parent value when the maximum LSD was considered. The BC_1 generation mean was significantly less than the mid-parent, while the BC_2 generation mean was equal to that of the mid-parent. These results were consistent with additive or partial dominance gene action for low disease spread.

Table 1 Analyses of variance for disease spread and the Fusarium head blight index (FHBI) of seven generations of the soft red winter wheat cross Ernie/MO 94-317 following greenhouse inoculation with *Fusarium graminearum*

Source	df	Mean square	
		Disease spread	FHBI
Environments	1	1140.4***	54, 377.2***
Generations [a]	6	1107.5***	60, 504.6***
Generations × environment	6	7.6	1322.8
Pooled CRD error [b]	1174	16.2	897.2

*** Significant at the 0.001 probability level

[a] Generations include parents, F_1 (Ernie/MO 94-317), reciprocal F_1 (MO 94-317/Ernie), BC_1 (F_1/Ernie), BC_2 (F_1/MO 94-317), and the F_2

[b] Within experiments, plants were arranged in a completely randomized design (CRD)

For FHBI, similar trends were noted. The means for Ernie (14.4%) and MO 94-317 (89.4%) were significantly different. Consistent with data for spread, the mean FHBI in the F_1 (28.8%) and reciprocal F_1 (30.9%) were not significantly different, again indicating that FHBI was primarily conditioned by nuclear genes. The pooled F_1 mean was significantly less than the mid-parent value and significantly greater than the mean of the resistant parent, suggesting partial dominance for the resistant phenotype. The mean of the BC_1 was significantly less than that of the mid-parent, while the mean of BC_2 was not significantly different from the mid-parent value, again suggesting some dominance for low FHBI.

As expected, for both disease spread and FHBI, the variances of the means for non-segregating generations were lower than those for segregating generations (Table 2). For both traits, variances of the parental and F_1 generations were used to estimate the environmental component of the phenotypic variance in the F_2. Again, as expected, the variance of the F_2 generation was larger than that for backcross generations. It is of note that variances for susceptible generations were higher than those for resistant generations. This phenomenon has been frequently observed with FHB disease scores because disease spread in susceptible genotypes typically has a larger range than that in resistant genotypes. The variances of the generation means for both traits were heterogeneous, therefore, generation means were analyzed using weighted-least-squares analyses.

For disease spread, neither simple additive nor additive-dominance genetic models adequately fit generation mean data. Model complexity was therefore

Table 2 Generation means and variances for disease spread and the FHBI of seven generations of the soft red winter wheat cross Ernie/MO 94-317 following greenhouse inoculation with *F. graminearum*

Generation	Number of plants	Mean		Variance	
		Spread	FHBI	Spread	FHBI
P_1 (Ernie)	91	1.7	14.4	1.14	86.78
P_2 (MO 94-317)	78	11.8	89.4	6.38	288.65
F_1 (P_1/P_2)	83	3.7	28.8	7.84	443.76
F_1 reciprocal (P_2/P_1)	70	4.1	30.9	8.54	501.04
F_2 (F_1 selfed)	404	5.4	40.7	23.45	1257.11
BC_1 (F_1/P_1)	215	3.9	29.6	14.93	823.33
BC_2 (F_1/P_2)	234	7.5	56.8	19.95	994.86
Mid-parent		6.7	51.9		
Minimum $LSD^a_{(0.01)}$		0.85	6.4		
Maximum $LSD^b_{(0.01)}$		1.71	12.7		

[a] Minimum $LSD_{(0.01)} = 2.58 \times \sqrt{[MSE \times (1/234 + 1/404)]}$, where MSE is the mean square error from the analysis of variance, 2.58 is the t value at $\alpha = 0.01$, and 234 and 404 are the two largest numbers of plants in the generations evaluated; therefore, the LSD is the minimum LSD

[b] Maximum $LSD_{(0.01)} = 2.58 \times \sqrt{[MSE \times (1/70 + 1/78)]}$, where MSE is the mean square error from the analysis of variance, 2.58 is the t value at $\alpha = 0.01$, and 70 and 78 are the two smallest numbers of plants in the generations evaluated; therefore, the LSD is the maximum LSD

increased to include the additive × additive (*i*), additive × dominance (*j*), and dominance × dominance (*l*) interaction components. The χ^2 test indicated that a model including additive, dominance, and epistatic (additive × dominant) gene action adequately fit the data (Table 3). Additive effects for reduced spread accounted for 95.7% of the variation. A smaller, yet significant ($P = 0.02$) dominance component was also evident that accounted for 3.4% of the variation. The additive × dominance effect accounted for 0.09% of the variation and was not significant at $P = 0.05$.

For FHBI, a genetic model containing additive, dominance, and additive × dominance epistatic effects also fit the observed data (Table 3). As was the case for disease spread, additive effects were the major effects conditioning resistance, explaining 95.2% of the observed variation. A small dominance effect, accounting for 4.2% of the variation, was significant at $P = 0.012$, while the additive × dominant epistatic effect, which accounted for 0.6% of the variation, was not significant at $P = 0.05$.

Generation variances (Table 2) provided estimates of the additive (D), dominance (H), and environmental (E) components of the F_2 phenotypic variance as outlined by Mather and Jinks (1977). These estimates were then used to determine F_2 heritabilities for disease spread and FHBI. Broad-sense heritabilities for disease spread and FHBI were 78.2% and 78.3%, respectively, while narrow-sense heritabilities were 51.3% and 55.4%, for disease spread and FHBI, respectively. These data are in agreement with the significant additive genetic variance indicated by the generation means analyses and suggest that both traits could be fixed in populations under selection for FHB resistance.

Analysis of phenotypic variation in recombinant inbred lines

Frequency distribution data of RILs for disease spread and FHBI approximated normal distributions in both 2002 and 2003 (Fig. 1). Mean disease spread in RILs ranged from 1.5 to 12.3 spikelets in 2002 and from 1.4 to 9.5 spikelets in 2003, reflecting the significant difference in disease resistance between the two parents. Mean disease spread was 2.6 and 2.3 spikelets for Ernie and 9.0 and 8.2 spikelets for MO 94-317, in 2002 and 2003, respectively. Although disease spread was somewhat lower in 2003, the correlation among mean values for RILs (0.57) was highly significant ($P = 0.001$).

Trends for FHBI data were similar to those for disease spread. Mean data for RILs ranged from 15% to 80% in 2002 and from 11% to 72% in 2003. These data again reflect the phenotypic range in disease resistance of the parents. Mean FHBI was 20.0% and 22.6% for Ernie and 76.3% and 65.3% for MO 94-317 in 2002 and 2003, respectively.

For both years of the experiment, variances associated with RILs (genetic variance) and replications were highly significant (Table 4). The significant replication effect reflects the fact that replications of both the 2002 and 2003 experiments were evaluated between January and March, during which time both natural photoperiod and temperature changed in the greenhouse environment. Although disease development was excellent in both years, FHB can be sensitive to these environmental factors.

Broad-sense heritability estimates for both disease spread and FHBI were determined from the combined analysis of variance. Heritability for disease spread was 0.70. Exact 90% ($1-\alpha = 0.90$) confidence limits for this heritability estimate were determined according to Knapp et al. (1985). The upper 90% confidence limit for the heritability estimate was 0.76, while the lower 90% confidence limit was 0.63. Heritability for FHBI was 0.87, with an upper confidence limit of 0.89 and a lower confidence limit of 0.84. These data suggested that the heritability of FHBI was estimated more precisely than that for disease spread.

The minimum number of effective genes conferring resistance to FHB was calculated from RIL variances for both disease spread and FHBI using the modified formula of Wright (1968). Estimates of the minimum number of genes conferring resistance were in close agreement in 2002 and 2003. Gene numbers conditioning disease spread were 4.3 and 4.2 for 2002 and 2003, respectively, while those conditioning FHBI were 3.8 in both years.

Table 3 Estimates (± standard error) of genetic effects for disease spread and the FHBI from six generations of the soft red winter wheat cross Ernie/MO 94-317 following greenhouse inoculation with *F. graminearum*

Parameter[a]	Disease spread	FHBI
m	6.78 ± 0.17	52.17 ± 1.00
[*d*]	5.13 ± 0.18	37.70 ± 1.01
[*h*]	−2.71 ± 0.39	−21.48 ± 2.41
[*j*]	−3.15 ± 1.02	−21.80 ± 6.52
χ^2	4.32	2.52
P	0.120	0.280

[a] *m*, Mid-parent value; [*d*], the additive genetic effect; [*h*], the dominance genetic effect; [*j*], the additive-by-dominance interaction effect; χ^2, chi-squared value testing the goodness-of-fit of the genetic model to the data; *P*, the probability associated with the χ^2 statistic

Discussion

Type-II resistance to FHB is frequently assessed as FHBI, which is given as the percentage of diseased spikelets in inoculated heads. However, more recently researchers have been using the simple number of diseased spikelets as a direct measure of type-II resistance to eliminate the confounding effects of total spikelet number on disease score (Buerstmayr et al. 2002). An understanding of the inheritance of both of these traits

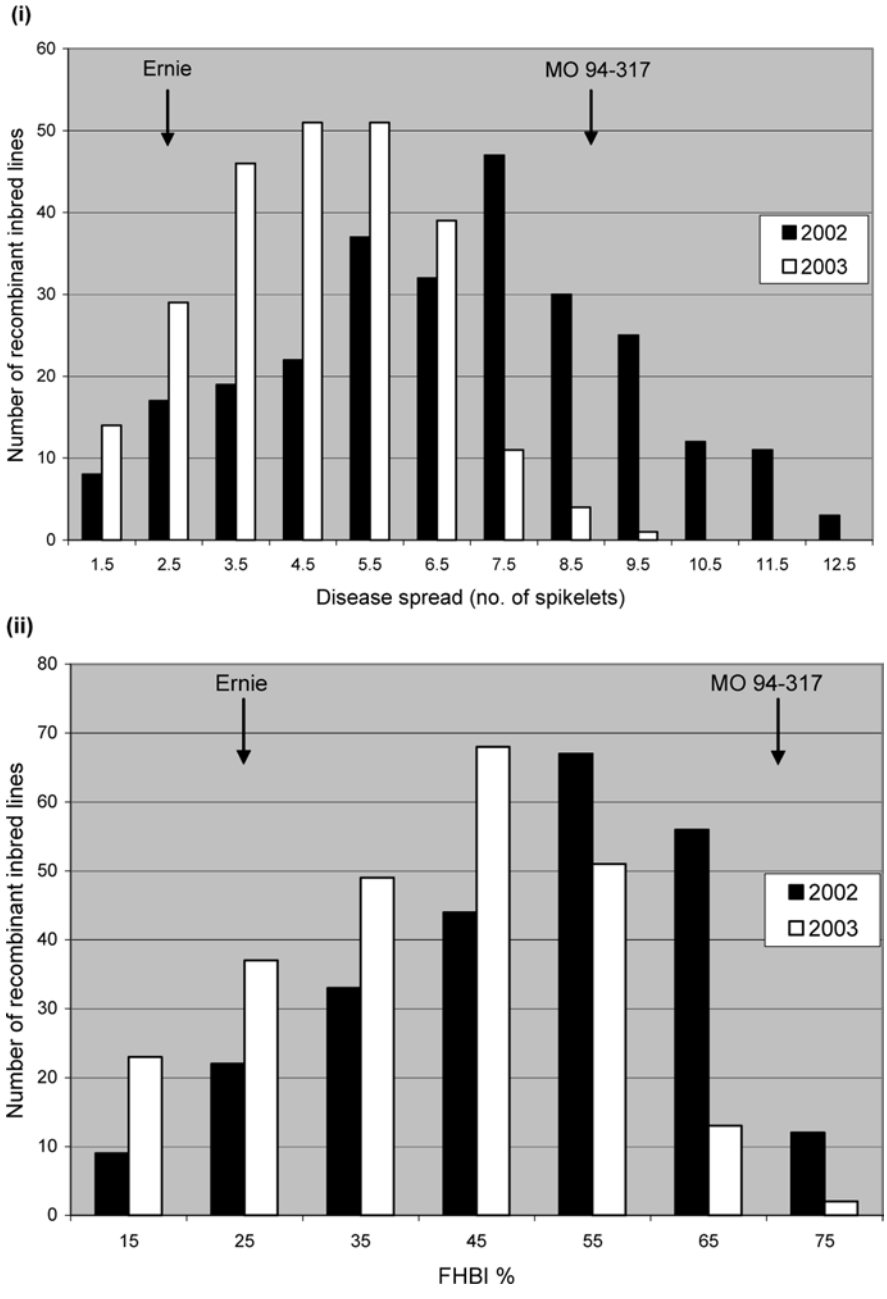

Fig. 1 Frequency distributions for disease spread (**a**) and Fusarium head blight index (FHBI) (**b**) of 243 recombinant inbred lines of the soft red winter wheat cross Ernie/MO 94-317 inoculated in 2002 and 2003. Disease spread was determined as the number of diseased spikelets, while FHBI was determined as the percentage of diseased spikelets in heads inoculated with *Fusarium graminearum*. Data for each year reflect mean data for three replications (eight plants per replication)

in a given source of resistance is critical to their efficient and effective exploitation in breeding programs.

Ernie, a soft red winter wheat developed and released by the University of Missouri, provides breeders with moderately high levels of type-II FHB resistance. This source of resistance differs from that in Sumai 3 and other known sources of resistance based on pedigree analysis and therefore, could provide wheat breeders with a source of resistance that may complement those sources that are widely used. The advantage of using Ernie as a source of resistance rather than Asian sources of resistance is that the former provides a highly functional level of resistance in an agronomic background suitable to winter wheat production regions of the USA.

Generation means analyses of type-II resistance in Ernie indicated that this source of resistance is primarily conditioned by additive genes when assessed either as disease spread or as FHBI. A smaller, yet significant, dominance component was also evident. These results agree with field data from Hall and Van Sanford (2003) who reported a high general combining ability effect when Ernie was used as a parent. Other researchers have also reported primarily additive genetic effects controlling FHB resistance in Sumai 3 (Waldron et al. 1999) and Frontana (Singh et al. 1995). Bai et al. (2000) reported that an additive-dominance model adequately explained resistance in a majority of crosses involving six Chinese sources of resistance.

Table 4 Analyses of variance for disease spread and the FHBI of 243 recombinant inbred lines (RILs) of the soft red winter wheat cross Ernie/MO 94-317 following inoculation with *F. graminearum*. Experiments were grown in the greenhouse at the University of Missouri in 2002 and 2003

Source of variation	*df*	Disease spread	FHBI
2002			
Replications	2	15.14***	2411.20***
RILs	242	17.00***	692.48***
Error	482	1.83	85.01
2003			
Replications	2	26.27***	432.23*
RILs	242	7.48***	570.45***
Error	482	1.44	100.58
Combined			
Replications (year)	4	20.71***	1421.71***
Year	1	1208.31***	29, 040.50***
RILS	242	18.78***	1115.15***
Year × RILs	242	5.69***	147.79***
Error	968	1.63	92.79

*, *** Significant at 0.05 and 0.001, respectively

For disease spread and FHBI, the estimated F_2 broad-sense heritabilities were 78.2% and 78.3%, respectively, while narrow-sense heritabilities were 51.3% and 55.4%, respectively. Line-mean broad-sense heritability estimates based on an analysis of variance of RILs for both disease spread (0.70) and FHBI (0.87) agreed reasonably well with those estimated from generation means analyses. The agreement of these heritability estimates coupled with the precision with which they were estimated are evidence that good progress can be made with selecting for either disease spread or FHBI. The high narrow-sense heritability estimates for both traits provide evidence of the magnitude of the additive effects controlling these traits and reinforce the conclusion that response to selection based on either trait will be good.

The heritability estimates obtained from this investigation are in agreement with those published from similar genetic analyses. Snijders (1990b) reported broad-sense heritabilities in the F_2 generation of a series of crosses involving nine SVP lines from the Netherlands that differed widely in FHB reaction: heritability estimates averaged 39% and ranged from 5% to 89%. Closer examination of these data indicated that, as expected, heritability was generally higher when the difference between parents— and therefore the genetic variance—was maximized. Our results, based on a wide difference between parents, agreed with the higher values reported by Snijders.

Our results based on analyses of variance of RILs involving Ernie are also in reasonable agreement with those reported by both Buerstmayr et al. (2000) and Bai et al. (2000) based on European and Chinese sources of resistance. Buerstmayr et al. (2000) reported line-mean heritability estimates from RILs of UNG-266 (potentially carrying the Sumai 3 source of resistance) that ranged from 0.77 to 0.80 and from RILs of SVP-72017 that ranged from 0.71 to 0.75. Confidence intervals for these heritability estimates were similar to those we report here for Ernie. Similarly, Bai et al. (2000) reported broad-sense heritability estimates from an analysis of the variance in several generations of RILs for the cross Ning 7840/Clark that ranged from 0.80 to 0.91. Each of these estimates, including those for Ernie, may be overestimated because each was estimated in a limited number of environments.

For both disease spread and FHBI, approximately four genes conditioned resistance. Ban and Suenaga (2000) found two genes conditioning resistance in Sumai 3 and three genes associated with resistance in Saikai 165, a Sumai 3 derivative. Van Ginkel et al. (1996) reported two dominant genes in the Sumai 3 derivative Ning 7840 and two different dominant genes in the Brazilian spring wheat, Frontana. Finally, Bai et al. (2000) reported from one to three genes conditioning resistance in six Asian varieties including Sumai 3 and Ning 7840. Our results suggest that the source of resistance in Ernie differs from other widely used sources of resistance.

It is important to note that this research was based on greenhouse evaluation of FHB resistance. Hall and Van Sanford (2003) reported differences in the results of genetic analyses based on field and greenhouse evaluations and raised concern for results based solely on greenhouse evaluations. However, as they pointed out in their study, despite attempts to have precisely similar inoculation protocols in the field and greenhouse environments, disease severity and the manifestation of symptoms differed in the two environments. In the field environment, heads are typically covered with glassine bags to prevent contamination with natural sources of inoculum. This frequently leads to the development of excessive amounts of mycelium in the injected spikelet that can colonize adjacent spikelets and compromise precise type-II measurements. Hall and Van Sanford noted that this mycelium development occurred in their study and may have contributed to the higher disease levels reported between the greenhouse and field. Although most researchers recognize that field data are critical to assessing performance under natural conditions, most also recognize that data generated in the field can be confounded by other components of FHB resistance. To offset the confounding effects that can occur in field environments, many of the quantitative genetic analyses of type-II resistance reported in the literature have been conducted under greenhouse conditions where inoculation and the subsequent exposure to humidity to promote disease development can be precisely controlled. In most cases this has led to results that are highly repeatable.

In summary, transgressive segregates have been shown to be important in breeding for FHB resistance. Both Sumai 3 and Ernie were derived as transgressive segregates from crosses involving two moderately susceptible parents. The results of our investigation agree with those reported elsewhere, indicating the importance of additive effects in the inheritance of FHB resistance.

Furthermore, the results from our study of inheritance indicate that type-II resistance in Ernie is heritable with a moderately high narrow-sense heritability so breeding progress should be expected when Ernie is used as a source of resistance. The similarity between the inheritance of disease spread and FHBI suggests that either trait can be effectively used as a selection criterion in breeding programs aimed at enhancing FHB resistance. More importantly, because the type-II resistance in Ernie is primarily conditioned by additive genes, it should be possible to develop varieties with high levels of FHB resistance by selecting for transgressive segregates in crosses involving different and complementary sources of resistance.

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