

Fusarium Head Blight Reaction of Langdon Durum-*Triticum dicoccoides* Chromosome Substitution Lines

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ABSTRACT

Fusarium head blight (FHB), caused by *Fusarium graminearum* Schwabe, is a serious disease problem on durum wheat (*Triticum turgidum* L. var. *durum*) in the USA. To date, the resistance to FHB available in hexaploid wheat (*T. aestivum* L.) sources has not been transferred successfully to tetraploid durum. In the 1980s, USDA geneticist L.R. Joppa produced a set of disomic lines [LDN(DIC)] derived from wild emmer (*T. turgidum* L. var. *dicoccoides*). Each line had a different pair of chromosomes from *T. dicoccoides* substituted for the corresponding 'Langdon' durum chromosomes. The purpose of this study was to determine if any of the LDN(DIC) lines showed useful levels of resistance to FHB. We tested these lines for FHB response by inoculation with *F. graminearum* in the greenhouse. Inoculation was accomplished via the single spikelet method, in which a droplet of conidiospore suspension is placed into one spikelet per spike followed by mist-high humidity for 3 d to establish infection. After 21 d, spikes were scored for extent of FHB symptoms and the average disease severity of the lines compared. A low disease severity score in this test indicated the presence of resistance to FHB in that line. Each test was replicated and the tests were done five times. In each test, some lines differed significantly. One line, LDN(DIC-3A), was consistently less susceptible and another line, LDN(DIC-2A), was consistently more susceptible than the parent Langdon durum, which itself showed an intermediate FHB reaction. Several other LDN(DIC) lines showed a trend either for increased or reduced susceptibility to FHB. Since each line differs by a single chromosome pair, the results suggest that genes affecting FHB resistance are present on several different *T. dicoccoides* or Langdon durum chromosomes.

FUSARIUM HEAD BLIGHT is a plant disease that adversely affects wheat and other small grains throughout the world. In North America, FHB is caused mainly by *Fusarium graminearum* Schwabe [sexual stage: *Gibberella zeae* (Schw.) Petch], with occasional involvement of other *Fusarium* species (McMullen et al., 1997; Stack and McMullen, 1985; Wong et al., 1992).

Outbreaks of FHB on wheat are unpredictable and highly dependent on weather conditions during and just following anthesis, when plants are at the most susceptible stage (Sutton, 1982). On spring wheat (*Triticum aestivum* L.) and durum (*T. turgidum* L. var. *durum*) in the northern Great Plains region of North America, the epidemics of 1993, 1994, and 1997 were very severe, with more localized losses occurring in other years between 1995 and 2000. Previous outbreaks, on a more limited scale, had occurred sporadically in the region

since the early 1980s (McMullen et al., 1997; Wilcoxson et al., 1988; Windels, 2000).

Management practices such as crop rotation have been ineffective in limiting the disease. Most researchers agree that incorporation of resistance is the best long-term answer to FHB (Meidaner, 1997). Sources of resistance to FHB are available in hexaploid wheat (Mesterhazy, 1995); however, transfer of that resistance from hexaploid wheat to durum has not been reported. Durum wheat cultivars differ in their response to FHB from moderately to highly susceptible (McMullen et al., 1994; Stack, 1988; Stack and Elias, 1995–2000, unpublished).

The greatest progress to date in identification of resistance to FHB has been with resistance expressed as limitation of the spread of infection within the spike (Wang and Miller, 1988). This phenotypic expression has been termed "Type II" resistance in the literature (Meidaner, 1997; Mesterhazy, 1995). Molecular markers for quantitative trait loci (QTL) associated with Type II resistance have been identified in hexaploid wheat (Anderson et al., 2001; Waldron et al., 1999).

Triticum turgidum L. var. *dicoccoides* (TDIC) possesses many interesting traits, including resistance to stem rust (*Puccinia graminis* Pers.) (Miller et al., 1998), resistance to stripe rust (*P. striiformis* Pers.) (Reinhold et al., 1983), resistance to FHB (Miller et al., 1998), and grain quality factors (Joppa et al., 1991). A set of disomic chromosome substitution lines from TDIC in the background of Langdon durum had been developed as described by Joppa and Williams (1988) using the TDIC line FA-15-3 ("Israel A"). A gene for high grain protein concentration in this population has been studied (Joppa and Cantrell, 1990; Steiger et al., 1996) and mapped (Joppa et al., 1997). These lines have also been evaluated for agronomic traits and grain quality (Cantrell and Joppa, 1991; Elias et al., 1996; Joppa et al., 1991).

In a search for potentially useful sources of resistance to FHB in durum, we tested FHB reactions of several hundred durum lines and accessions (Stack and Elias, 1994, unpublished). In one such screening test in 1994, we included several of the disomic chromosome substitution lines developed for high grain protein concentration (Joppa and Williams, 1988).

On the basis of results from those preliminary tests, we decided to examine the entire set of the LDN(DIC) disomic chromosome substitution lines for reaction to FHB. The purpose of this study was to determine if any of the LDN(DIC) lines showed useful levels of

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Abbreviations: FDK, Fusarium damaged kernels; FHB: Fusarium head blight; LDN(DIC), Langdon durum-*T. dicoccoides* substitution lines; QTL, quantitative trait loci; TDIC, *Triticum turgidum* var. *dicoccoides*.

resistance to FHB. This was done by inoculation with *F. graminearum* by a method and under conditions which minimized the effect of environmental and plant development factors. A preliminary report of a portion of the work presented in this paper has appeared (Stack et al., 1999).

MATERIALS AND METHODS

We tested the FHB response of the set of 13 LDN(DIC) chromosome substitution lines of Joppa and Williams (1988) plus the Langdon durum parent and multiple durum check lines of known FHB reaction. The substitution line LDN(DIC-2B) is unavailable, so we were not able to assess the effects of this chromosome.

The TDIC line FA-15-3 ("Israel A"), the source of the substituted chromosomes, was not included in the tests of the LDN(DIC) lines, but was tested for FHB reaction separately in replicated experiments evaluating TDIC accessions. Those experiments were done under conditions similar to the tests of the LDN(DIC) lines (Miller et al., 1998).

The set of 13 LDN-DIC lines were tested for FHB response in experiments across five environments. The five experiments were conducted from 1996 through 1998 in a controlled-environment greenhouse. Each experiment was arranged in a randomized complete block design with experimental units consisting of a single row of plants of a particular genotype. The LDN(DIC) lines and durum checks were randomly assigned to the rows of the replicate blocks.

Three experimental durum lines from the NDSU durum breeding program were used as checks. D91103 was included in all five tests as a moderately resistant entry. The NDSU breeding lines D87450 and D88541 were included as susceptible checks. D91103 has consistently shown one of the lowest FHB severity scores among numerous durum lines in many screening trials since 1995 (R.W. Stack, 1999, unpublished). D91103 was not released as it lacks acceptable grain quality (E.M. Elias, 1997, unpublished). Similarly, D87450 and D88541 have been used in numerous trials as susceptible check lines. All three lines also grow well in the greenhouse environment during the off-season (R.W. Stack, 1999, unpublished).

Plant Culture

In each of the five repeated experiments (Table 1), plants were grown in a temperature controlled greenhouse at $18 \pm 2^\circ\text{C}$ through the early stages of plant growth. From anthesis onward [after Feekes 10.0 (Simmons et al., 1985)], the green-

house controls were set to maintain 20 to 25°C where possible. Actual temperatures during the incubation period for each experiment are given in Table 1.

As described in Table 1, in Exp. #1 and #2, 6 seeds per 20-cm row were sown in raised ground beds, with rows spaced 15 cm apart. In Exp. #3 to #5, 5 or 8 seed per row were sown in large containers 23 cm by 32 cm by 40 cm high. In many previous tests under these conditions, these numbers of seed per row had been found to reliably produce 20 to 25 spikes per row, at least 10 of which could be expected to be at anthesis simultaneously.

Plants were grown in a soil mix or a commercial soil-less medium as specified in Table 1. During preparation, both substrates received additions of ground dolomitic limestone (692 g m^{-3}), magnesium-ammonium-phosphate fertilizer (166 g m^{-3} 7-17-6 +12 Mg) ("MagAmp-K" W.R. Grace & Co., Columbia, MD). The pH was adjusted to 6.5 with $\text{Ca}(\text{OH})_2$ on the basis of a soil test.

Throughout each experiment, plants were watered as needed, usually twice weekly, and fertilized weekly with 3 g L^{-1} of a complete soluble fertilizer (15-30-15, "Miracle Gro", Scotts Miracle Gro Products, Port Washington, NY) applied to runoff. Supplemental lighting to increase daylength to 16 h was provided by lighting fixtures spaced 2.5 m apart each way in a grid located 3 m above the soil surface and fitted with 400-W wide spectrum high pressure sodium bulbs ("Lucalox LU400", General Electric Co., Cleveland, OH). Plants were grown to anthesis and inoculated. After inoculation, plants were maintained under the same greenhouse conditions through the incubation period and to maturity.

Inoculum Preparation

Preparation of inoculum and inoculation of plants generally followed published methods (Stack, 1989). Briefly, cultures of *F. graminearum* were grown in the laboratory on petri dishes of carnation leaf agar or half strength potato dextrose agar for 15 to 20 d at room temperature (22 – 24°C) under fluorescent lights. To prepare conidiospore inoculum, petri dish cultures were flooded with sterile distilled water and gently agitated; the resultant spore suspension was poured off and strained through several layers of sterile cheesecloth to remove hyphal fragments. Concentration was adjusted to $50\,000$ conidiospores mL^{-1} . Freshly prepared spore suspension was held on crushed ice and used within 4 h of preparation.

Three strains of *F. graminearum* were used in all experiments. Each strain was isolated originally from symptomatic plants and had been tested repeatedly for pathogenicity under

Table 1. Experimental conditions for testing Fusarium head blight (FHB) response of the Langdon durum (*T. dicoccoides*) chromosome substitution lines.

Experiment number	1	2	3	4	5
Environment	Spr96	Fall96	Fall97	Spr98	Fall98
Planting Site					
Raised Ground Beds	X	X			
Pots $23 \times 32 \times 40$ cm deep			X	X	X
Substrate†	soil mix	soil mix	soil-less mix	soil-less mix	soil-less mix
Row length, cm	20	20	20	20	20
Number seed per row	6	6	5	5	8
Planting Date	Feb. 15	Aug. 19	Aug. 27	Feb. 25	Sep. 09
Planting to Anthesis, d	53	47	55	49	67
Incubation time, d‡	21	21	22	23	23
Temperature during incubation	21 ± 4	20 ± 3	20 ± 3	25 ± 5	20 ± 3
Number replicates	3	5	6	3	6
Grain harvest	yes	yes	no	no	yes

† Ground beds were filled with a 1:1:1 mix of loam field soil, sphagnum peat, sand, and coarse vermiculite. Soilless mix was a coarse mixture of peat, sand, vermiculite, and ground bark (Metromix 550, Scotts-Sierra Horticultural Products, Marysville, OH).

‡ Time from inoculation at mid-anthesis to FHB disease scoring. The first 3 d were a mist/high humidity treatment.

a range of environments. Each strain was maintained, cultured, and prepared separately, then spore suspensions of equal concentration were mixed together immediately before use. All three cultures have maintained strong disease-causing ability by these methods (Mitchell Fetch et al., 1998; Stack et al., 1997; Waldron et al., 1999).

Inoculation

Inoculations were done by the single spikelet method, in which inoculum is placed into a single spikelet near the middle of each spike at anthesis (Stack, 1989). This method of inoculation selectively targets the kind of FHB physiological resistance expressed as a limitation of spread of infection within the spike, also called "Type II" resistance (Mesterhazy, 1995; Wang and Miller, 1988). Spikelet inoculation also excludes differences due to morphological and developmental factors—either genotypic or phenotypic—which can produce differences in FHB expression.

Anthesis is recognized as the time of peak susceptibility to FHB infection. To reduce the effect of differences in maturity, individual rows were inoculated as they flowered. Within each row of plants, 10 spikes at mid- to late anthesis (Feekes 10.52) were selected. Spikes to be inoculated were marked with colored paper tags so each could be identified later for disease scoring at the proper time. A 10- μ L droplet of *F. graminearum* conidial suspension was placed into one flowering spikelet near the middle of each selected spike with a repeating syringe dispenser (Nichiryō model #8100, Nichiryō, Ltd., Tokyo, Japan). At the conidiospore concentration used (50 000 mL⁻¹), each droplet contained approximately 500 conidiospores. This concentration was chosen to give maximum incidence while not obscuring expression of Type II resistance. Spikes in each replicate row of each line were inoculated only once, as that row flowered.

Following inoculation, plants were lightly misted with a hand fogging nozzle ("Fogg-it" Dramm Corp., Manitowoc, WI) attached to a watering hose. To maintain high humidity after misting, plants were covered with a plastic tent. Misting and covering were done on three successive nights after inoculation. The plastic tent was opened during the day to prevent overheating. Following the third night, no further misting or covering was done. In all experiments, numerous noninoculated spikes provided a check on both inoculation technique and for unintended secondary spread of infection.

FHB Evaluation

At 3 to 3.5 wk post-inoculation, plants were evaluated for FHB. Each inoculated spike was individually examined and

the extent of blight scored on a 0 to 100% scale (Stack and McMullen, 1995). Disease incidence was recorded as the percentage of inoculated spikes in the row showing any symptoms of FHB. A representative selection of the noninoculated spikes also was examined for symptoms in the same manner. Following scoring, plants were grown to maturity. In three of the five experiments, inoculated spikes were harvested at maturity and threshed. The proportion of Fusarium damaged kernels (FDK) in the harvested grain was visually determined (Clear and Patrick, 2001) as a second measure of disease severity (Ittu et al., 2000; Meidaner, 1997)

Data Analysis

For each experiment, analysis of variance was performed on the FHB scores and mean separation was determined by use of Fisher's protected least significant difference (LSD) at $\alpha = 0.05$. A combined analysis of variance was also done and the experiment \times genotype interaction variance was used to test for the presence of significant experiment to experiment variation. In addition, Bartlett's test for homogeneity of error variance was done. The correlation between the proportion of Fusarium damaged kernels in each line and the FHB severity scores of those lines was determined for each experiment where both measures were taken.

RESULTS

FHB disease scores of the five experiments are shown in Table 2. A combined analysis of variance and use of Bartlett's test for homogeneity of error variance indicated the presence of significant experiment to experiment variation. A combined analysis was not conducted and the results of individual experiments are presented (Table 3).

In each experiment, the lines displayed a wide range of FHB response, from moderately resistant to very susceptible, based on disease severity (Table 3). FHB incidence was very high in all lines and no further analysis was done with disease incidence data (Table 2). The reaction of the durum check lines in these experiments was as expected, based on numerous previous screening tests of durum lines (R.W. Stack and E. Elias, 1994–1999, data not shown). The lines D91103 (FHB severity = 30%) and D88541 (FHB severity = 70%) represent the extremes of FHB response of commercial durum cultivars under these experimental conditions. Exami-

Table 2. FHB disease scores in five experiments to test the Langdon durum (*T. dicoccoides*) chromosome substitution lines for Fusarium head blight (FHB) response.

Experiment number	1	2	3	4	5
Environment	Spr96	Fall96	Fall97	Spr98	Fall98
Number of replicates	3	5	6	3	6
Number inoculated spikes per rep	9	8	9	10	8
Number inoculated spikes per line	27	40	54	30	28
FHB INCIDENCE [†]	0.99	0.94	0.95	0.89	0.96
FHB SEVERITY (%) [‡]	51.9	42.1	45.9	71.2	42.5
FHB SEVERITY (%) - std. error	3.8	4.1	5.4	5.1	4.9
FHB SEVERITY (%) range	24–72	14–71	13–93	25–100	17–77
FHB SEVERITY (%) D91103 (resistant ck)	24.0	36.9	24.1	32.0	31.2
FHB SEVERITY (%)					
- susceptible ck D87450	68.4	38.6	–§	86.8	56.7
- susceptible ck D88541	–§	69.5	51.4	89.4	–§
FHB SEVERITY (%) Langdon	–§	43.9	49.8	76.0	33.4

[†] FHB incidence = proportion of spikes showing any degree of blight symptoms.

[‡] FHB severity = mean proportion of blighted spikelets per spike.

§ Not included in this test.

Table 3. Fusarium head blight (FHB) disease severity on Langdon durum (*T. dicoccoides*) chromosome substitution lines inoculated with *F. graminearum*.

Experiment number	FHB Severity					Mean
	1	2	3	4	5	
Environment	Spr96	Fall96	Fall97	Spr98	Fall98	
Line	%					
LDN(DIC-1A)	51.7	24.4	39.3	38.5	28.8	36.5
LDN(DIC-1B)	64.8	65.8	60.2	87.7	67.7	69.2
LDN(DIC-2A)	79.0	70.7	92.7	100.0	77.4	83.9
LDN(DIC-3A)	29.9	13.5	13.6	12.5	17.4	19.8
LDN(DIC-3B)	57.6	48.2	54.8	75.8	52.6	57.8
LDN(DIC-4A)	45.6	26.8	38.7	77.7	52.9	48.3
LDN(DIC-4B)	44.9	22.6	36.1	58.5	20.8	36.6
LDN(DIC-5A)	43.7	42.9	28.9	61.8	25.6	40.6
LDN(DIC-5B)	54.3	27.3	31.4	81.0	28.3	44.8
LDN(DIC-6A)	63.4	71.2	69.2	93.6	63.5	72.4
LDN(DIC-6B)	38.8	27.5	17.8	72.6	21.0	35.5
LDN(DIC-7A)	59.9	45.6	74.5	83.9	64.5	65.7
LDN(DIC-7B)	67.6	40.2	52.1	90.6	36.4	57.4
Langdon	–†	43.9	49.8	76.0	35.3	50.8
LDN(DIC) mean	53.9	40.5	46.9	69.6	42.8	(48.1)
Durum checks						
D91103 (resistant)	24.0	36.9	24.1	32.0	31.2	29.6
D87450 (susceptible)	68.4	38.6	–†	86.8	56.7	
D88541 (susceptible)	–†	69.5	51.4	82.0	–†	
FLSD (0.05)‡	29.1	21.5	15.1	25.1	21.6	
CV (%)	45.3	58.9	61.5	62.2	56.7	

† Not included in this trial

‡ Includes checks and Langdon.

nation of the noninoculated spikes revealed absence of secondary infections.

Langdon durum (FHB severity = 51%) showed an intermediate reaction relative to the durum check lines. The mean FHB severity score of all the LDN(DIC) lines for each experiment did not differ from Langdon (Table 3). In three separate experiments, TDIC FA-15-3 was tested for FHB along with many TDIC accessions. TDIC FA-15-3 showed a highly susceptible reaction in these tests. The FHB severity of FA-15-3 was 100% in a range of 10 to 100%, 82.4% in a range of 11 to 83%, and 88.4% in a range of 19 to 99% (R.W. Stack and J.D. Miller, 1997, 2000, unpublished).

Comparisons were made between the LDN(DIC) substitution lines and the Langdon durum parent. The LDN(DIC-3A) line showed the lowest FHB severity score in all experiments, significantly lower than Langdon in those experiments where both were present (Table 3). The FHB scores of three other lines, LDN(DIC-6B), LDN(DIC-4B), and LDN(DIC-1A) were greater than LDN(DIC-3A) but significantly less than Langdon in some individual experiments (Table 3).

LDN(DIC-2A) was the most susceptible to FHB in all experiments (Table 3). LDN(DIC-2A) also had a significantly greater FHB score than the most susceptible durum check line D88541. Three other lines, LDN(DIC-7A), LDN(DIC-1B), and LDN(DIC-6A), were significantly more susceptible than Langdon in some experiments (Table 3).

The other LDN(DIC) lines showed intermediate responses. Within that intermediate group, rankings varied from experiment to experiment but none was significantly different from Langdon. Since each LDN(DIC) line differs by a chromosome pair, the results suggest that the substitution lines with intermediate FHB scores

do not carry genes for FHB reaction that differ from those in Langdon.

Another measure of plant damage from FHB infection is proportion of Fusarium damaged kernels (FDK) observed in the grain harvested from infected spikes. In three of the trials, harvested grain was visually examined for FDK. In our tests, the proportion of such kernels in the grain closely reflected the FHB disease severity scores for all the lines. The correlations between percent FDK and FHB severity for the genotype means within the three experiments were: $r = 0.86$ ($P < 0.001$, $n = 15$) for Exp. 1; $r = 0.87$ ($P < 0.001$, $n = 16$) for Exp 2; and $r = 0.95$ ($P < 0.001$, $n = 16$) for Exp 5.

DISCUSSION

The FHB resistance exhibited by LDN(DIC-3A) has been confirmed in field nursery trials, and this line is currently being used as a source of FHB resistance in the NDSU durum breeding program. Experimental durum breeding lines derived from crosses with LDN(DIC-3A) show reduced levels of FHB similar to LDN(DIC-3A) (E.M. Elias and R.W. Stack, 2000, unpublished). Molecular markers for FHB resistance QTL in a population of LDN(DIC-3A)/Langdon recombinant chromosome lines have been identified (Otto et al., 2002).

The presence of progeny with a high level of resistance in a cross between a moderately susceptible (Langdon) and a highly susceptible (FA-15-3) line may be due to transgressive segregation. In hexaploid wheat, the widely used FHB resistance source 'Sumai-3' is itself derived from such a cross (Liu and Wang, 1990), and other examples have been reported in this pathosystem (Ittu et al., 2000).

The low phenotypic FHB score of LDN(DIC-3A)

may result from a gene or genes conditioning resistance located on chromosome 3A, in which case it was derived from the TDIC parent. Alternatively, the gene or genes conditioning resistance reside on one or more other chromosomes in Langdon, and the chromosome 3A of Langdon has some factor that is epistatic or inhibitory to the expression of that resistance. Substituting the TDIC 3A would then remove that factor, allowing the resistance to be expressed. Two lines of evidence support the former hypothesis. First, using another set of disomic substitution lines in which corresponding D genome chromosomes from a hexaploid 'Chinese Spring' wheat were substituted for Langdon A or B chromosomes (Joppa and Williams, 1988), we found that the LDN(3D-3A) line did not differ in FHB severity from Langdon (R.W. Stack, 2000, unpublished). Second, the marker studies of Otto et al. (2002), examining the segregation of resistance in a population derived from Langdon/LDN(DIC-3A), also support the hypothesis that a gene conditioning resistance to FHB is present on chromosome 3A of TDIC FA-15-3 (Otto et al., 2002).

TDIC FA-15-3 shows a highly susceptible phenotypic reaction despite the apparent presence of a strong gene for resistance to FHB. The substitution of TDIC 2A for Langdon 2A produces a line (LDN(DIC-2A)) in which the FHB score is significantly higher than Langdon itself. This chromosome may contain a factor that is epistatic to other factors that control or limit the expression of the FHB resistance on 3A, resulting in the susceptible phenotypic response observed in TDIC FA-15-3. Gilbert et al. (2000) found evidence of gene interaction for FHB response in hexaploid wheat. Alternatively, Langdon 2A may be the site of gene(s) conditioning the intermediate resistance of Langdon to FHB. Testing of the LDN(2D-2A) substitution line (Joppa and Williams, 1988) might help to clarify this.

Recently, chromosomal locations of QTL responsible for FHB resistance in hexaploid wheat have been proposed (Anderson et al., 2001; Bai et al., 1999; Waldron et al., 1999). Chromosome 3A was not among those identified by those authors, but was reported by Buerstmayr et al. (1997) in one of two genotypes. The resistance found in LDN(DIC-3A) may or may not be at the same locus. If not, it may have value for additional gene pyramiding for FHB resistance in hexaploid wheat as well as durum. That possibility is further strengthened since the resistance is expressed in the same manner (Type II) as the major resistance on other chromosomes in hexaploid wheat.

Further study is needed to confirm if any of the other three lines [LDN(DIC-6B), LDN(DIC-4B), and LDN(DIC-1A)], which tend toward lower FHB severity also possess heritable resistance. The factor on chromosome 2A that appears to confer increased susceptibility should also be studied. If it were found to be widespread in durum lines, it might help to explain why so many durums are susceptible to FHB.

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Yield and Molecular Diversity of Soybean Lines Derived from Crosses of Northern and Southern Elite Parents

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ABSTRACT

Genetic diversity is low in southern United States elite soybean [*Glycine max* (L.) Merr.] cultivars. Multiple sources of diversity will be required to effectively diversify this gene pool. The objective in this study was to evaluate the genetic diversity and yield of families derived from crosses between northern elite (NE) by southern elite (SE) parents. Lines were derived from 10 crosses of NE × SE parents. Molecular markers were used to estimate genetic distance between each line and its SE parent. Yield and agronomic traits were measured in field trials from 1997 to 1999 in six of the crosses. The association of diversity with line yield, expressed relative to yield of the SE parent was determined with regression. On average, the use of NE parents reduced yield, relative to using other SE parents. Some crosses and NE parents were better than others and produced families with yield that exceeded that of their SE parent, indicating that some genes from the NE parents were superior to the genes in the SE parent. At least one line with yield either superior or similar to their SE parent was found in each cross. The finding of positive transgressive segregants in some crosses and the results of the regression analyses indicate that most of the NE parents possess some yield genes that are likely to be superior to those of the SE parents. Our approach to selecting for diversity and yield may be applicable to large introgression programs where diversity from many sources is desired.

SUCCESSFUL CROP IMPROVEMENT depends on genetic variability that arises from genetic diversity. The predominant use of selected elite parents in soybean cultivar breeding has led to a narrow genetic base of commercial soybean in the United States. Recent elite

public and proprietary U.S. soybean cultivars derived 90% of their parentage from only 11 ancestors (Sneller, 1994), while only 22 ancestors account for 90% of the parentage of the 258 public U.S. cultivars released from 1947 to 1988 (Gizlice et al., 1994). Gizlice et al. (1993) estimated that cultivars released in the public domain since 1983 have 50% more genes in common than public cultivars released prior to 1954.

There are strong patterns of diversity within the commercial U.S. gene pool. Elite cultivars adapted to the northern United States are quite distinct from southern cultivars (Delanney et al., 1983; Gizlice et al. 1993; Sneller, 1994). Sneller (1994) reported that the average coefficient of parentage was 0.23 among northern elite (NE) U.S. families 0.26 among southern elite (SE) U.S. cultivars, but only 0.10 between NE and SE cultivars. There appears to be less diversity among SE cultivars than among NE families. Nearly 47% of the SE parentage derives from two ancestors, CNS and S100 (Sneller, 1994; Gizlice et al., 1993).

Molecular analyses of diversity in US soybean support the conclusions of the pedigree analyses. RFLP (Keim et al., 1992; Sneller et al., 1997; Kisha et al., 1998) and SSR (Nelson et al., 1998) analyses have shown the clear separation of northern and southern cultivars and the limited diversity in the southern gene pool. In addition, RFLP analyses indicate that our current elite pool is less diverse than the ancestral pool (Kisha et al., 1998).

A lack of genetic diversity may limit breeding progress, though genetic gain in yield is still being made in U.S. soybean. Ininda et al. (1996) found the greatest mean yield and gain from selection in a population de-

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