

SOYBEAN

Roundup Ready Soybean: Glyphosate Effects on *Fusarium solani* Root Colonization and Sudden Death Syndrome

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ABSTRACT

During 1997, the first year of widespread use of glyphosate (*N*-[phosphonomethyl]glycine) on Roundup Ready (RR) soybean [*Glycine max* (L.) Merr.] a severe sudden death syndrome (SDS) epidemic occurred and several RR cultivars were affected. Effects of glyphosate on colonization of soybean root by *Fusarium solani* (Mart.) Sacc f. sp. *glycines* (Fsg) and SDS were evaluated. Five RR cultivar pairs that contrasted for SDS resistance from maturity groups (MG) II to VI were evaluated with and without glyphosate application. The MG II and III cultivars were evaluated near Bloomington, Pontiac, and Mahomet in central Illinois and the MG IV, V, and VI cultivars were evaluated near Harrisburg, Ullin, and Valmeyer in southern Illinois. The Fsg root infection severity (IS), colony forming units per gram of root (CFU), SDS leaf scorch disease index (DX), and grain yield were determined. Across environments within each MG, there were no significant effects of glyphosate on IS, CFU, and DX. Significant differences were expected between cultivars but only observed in some MG. There was no significant effect of glyphosate on yield. Significant Glyphosate × Cultivar interactions occurred for yield in MG VI, in favor of the glyphosate sprayed subplots. In this study root colonization by Fsg and SDS leaf symptoms did not significantly increase following the application of glyphosate. Data from this study indicate that the development of SDS on RR soybean is influenced by genotype. Farmers planting RR soybean in Fsg infested fields are encouraged to select cultivars with resistance to SDS.

GLYPHOSATE (*N*-[phosphonomethyl]glycine) herbicide (Roundup Ultra, Monsanto, MO) controls a broad spectrum of grass and broadleaf weeds (Gonzini et al., 1999; Ateh and Harvey, 1999). It is a nonselective, nonresidual, low environmental impact herbicide used for total vegetation control in certain situations (Dyer, 1994). Glyphosate is used to burn-down weeds before no-till planting. It can be tank mixed with numerous preemergence herbicides to improve weed control (Landie et al., 1994). While the total vegetation control and low environmental impact of glyphosate provide it a niche in the market, the nonselective quality precluded its use as a postemergence treatment in field crops before 1996 (Delannay et al., 1995; Dyer, 1994).

Glyphosate-resistant soybean has been developed via genetic engineering (Padgett et al., 1995). Monsanto

markets and licenses glyphosate-resistant soybean under the trade name Roundup Ready (RR) soybean. During 1997, the first year of widespread use of RR soybean cultivars, environmental conditions were unusually wet and the southern Midwest had a severe epidemic of soybean sudden death syndrome (SDS) (Wrather et al., 2001). Soybean SDS is caused by the soil-borne fungus *Fusarium solani* (Mart.) Sacc f. sp. *glycines* (Fsg) (Roy et al., 1989; Rupe, 1989; Roy, 1997). Soybean SDS was identified more frequently on RR soybeans (Myers et al., 1999), suggesting that either RR soybeans were more susceptible to SDS, or that glyphosate application increased the severity of SDS.

Sudden death syndrome significantly reduces soybean yield in midwestern USA and South America (Wrather et al., 1997; Roy et al., 1997; Njiti et al., 1998b). Management of SDS is mainly from the use of SDS-resistant cultivars (Gibson et al., 1994; Njiti et al., 1998a). Although SDS is identified by foliar symptoms, the causal agent Fsg (Roy et al., 1989) infects only the roots and crowns. The leaf scorch or SDS leaf symptoms are thought to be a result of a toxin or toxins produced in the root by the fungus and transported to the leaves (Jin et al., 1996). Although very few cultivars are resistant to root infection (Njiti et al., 1997), several have good resistance to leaf scorch (Gibson et al., 1994). Root resistance is measured as infection severity (% of root segments with Fsg colonization) or colony forming units per gram of dry root tissue (Njiti et al., 1997; Luo et al., 2000) whereas leaf resistance is measured using a disease index (Gibson et al., 1994).

The objective of this study was to evaluate the effects of glyphosate on root colonization by Fsg and development of SDS on RR soybean. Results of this study will help soybean growers determine whether glyphosate application on RR soybean increases the risk of SDS problems in Fsg-infested fields.

MATERIALS AND METHODS

Ten RR soybean cultivars from maturity group (MG) II to VI were used for this study. The cultivars were provided by Monsanto without disclosing their identity. There were two cultivars per maturity group. Each cultivar was previously characterized for SDS resistance (by the companies) and was identified as either partially resistant (referred to as *resistant* in this paper) or susceptible to SDS. The cultivars in each

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Abbreviations: CFU, colony forming unit; DI, disease incidence; DS, disease severity; DX, disease index; IS, infection severity; Fsg, *Fusarium solani* f. sp. *glycines*; MG, maturity group; RR, Roundup Ready.

maturity group included one SDS-resistant and one SDS-susceptible cultivar.

Each maturity group was considered a separate experiment. In this study cultivar pairs within each experiment were evaluated at two locations per year for 2 yr. In 1998, MG II and III were evaluated in central Illinois in a field near Bloomington on Sable silty clay loam soil (fine-silty, mixed, superactive, mesic Typic Endoaquolls), and in a field near Pontiac on Andres loam soil (fine-loamy, mixed, superactive, mesic Aquic Argiudolls); MG IV, V, and VI were evaluated in southern Illinois in a field near Harrisburg on Reesville silt loam soil (fine-silty, mixed, superactive, mesic Aquic Hapludalfs), and in a field near Ullin on Bonnie silt loam soil (fine-silty, mixed, active, acid, mesic Typic Fluvaquents). In 1999 the MG II and III were evaluated in central Illinois in a field near Pontiac on Tama silt loam soil (fine-silty, mixed, superactive, mesic Typic Argiudolls), and in field near Mahomet on Drummer silty clay loam soil (fine-silty, mixed, superactive, mesic Typic Endoaquolls); MG IV, V, and VI were evaluated in southern Illinois in a field near Valmeyer on Ambraw silty clay soil (fine-loamy, mixed, superactive, mesic Fluvaquentic Endoaquolls), and in a field near Harrisburg on Patton silty clay loam soil (fine-silty, mixed, superactive, mesic Typic Endoaquolls). All fields were selected based on a history of uniform SDS leaf symptom expression. All plots were established using conventional tillage.

In 1998, experiments were planted during the period of 15 to 19 May in central Illinois and on 15 to 16 May in southern Illinois. In 1999, experiments were planted during the period of 11 to 15 May in central Illinois and on 10 and 11 May in southern Illinois. All experiments were planted in a split-plot design with cultivar as the main plot and glyphosate as the subplots. There were four replications per experiment. Subplots consisted of four rows 3 m long and spaced 0.75 m apart. The center two rows were evaluated for SDS leaf symptoms and grain yield and the outside two rows were evaluated for Fsg root IS and CFU.

All plots were treated with a preemergence herbicide combination of 0.84 kg a.i. ha⁻¹ trifluralin [2,6-dinitro-N,N-dipropyl-4-(trifluoromethyl)-benzamine] and 0.07 kg a.i. ha⁻¹ imaziquin (2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-quinolinecarboxylic). Treatments consisted of glyphosate-sprayed and a nonsprayed control. At the V3 growth stage (Fehr et al., 1971), the treated subplots were sprayed with 1.12 kg a.i. ha⁻¹ glyphosate. Sprays were applied on wind-free days with necessary precautions to prevent drift.

At the R6 growth stage, subplots were rated for SDS disease incidence (DI) and disease severity (DS). Disease incidence was the percent of plants in the subplot with visible SDS leaf symptoms. Disease severity (percentage leaf surface chlorotic/necrotic) was rated on a scale of 1 to 9 as described: (1 = 0–10%/1–5%, 2 = 10–20%/6–10%, 3 = 20–40%/10–20%, 4 = 40–60%/20–40%, 5 = >60%/>40%, 6 = up to 33% premature defoliation, 7 = up to 66% premature defoliation, 8 = >66% premature defoliation, and 9 = premature death of plant). Disease index (DX, expressed as %) was then calculated as (DI × DS)/9, with a possible range of 0 (no disease) to 100 (death of all plants).

Eight taproots were harvested from each subplot at R6 growth stage. Four roots were used for IS and four for CFU determination. For IS determination, taproots were washed and surface sterilized in 0.5% (v/v) sodium hypochlorite. Each taproot was cut into several 1-cm segments. Ten root segments were randomly selected and placed on a Petri plate containing a selective medium composed of 20 g agar, 20 g sucrose, 1 g KH₂PO₄, 0.5 g MgSO₄·7H₂O, 1 g KNO₃, and 1 L distilled water; autoclaved at 110°C for 40 min and cooled to 42°C before

0.3 g streptomycin sulfate, 0.1 g neomycin, 0.1 g chlortetracycline, 0.05 g rifampicin, and 0.23 g pentachloronitrobenzene (PCNB) (Terrachlor, Uniroyal Chemical Co., Vaugntuk, CT) were added. Infection severity was determined as the percentage of root segments yielding slow-growing, blue fungus (Njiti et al., 1997). A total of 40 root segments from 4 taproots per subplot were used in IS determination.

For determination of CFU, the four taproots from each subplot were treated as one sample. The roots were washed, surface-sterilized in 0.5% (v/v) sodium hypochlorite, oven-dried at 28°C for 48 h, ground using a Wiley cutting mill (Thomas Scientific, Swedesboro, NJ) to pass through a 40-mesh screen, weighed, and suspended in 2 L of distilled water. One mL of the suspension was transferred onto each of five Petri plates containing the selective medium described above. Plates were inoculated and incubated at room temperature for 14 d. The number of slow-growing, appressed, and blue fungal colonies (Rupe et al., 1996) were counted as CFUs. The CFU per gram of root tissue was calculated (Luo et al., 2001).

At the R8 developmental stage, the middle two rows for each plot were harvested with a two-row combine. The seeds were cleaned without losing broken seeds. Yield was calculated for each plot at 13 g kg⁻¹ seed moisture content.

Data were collected for root IS in all test environments, CFU in 10 environments (2 per MG) in 1998, disease index in 4 environments for MG II and III; 3 environments for MG V and VI and 0 environment for MG IV, and grain yield in 4 environments in MG II, III, IV, and V; and 3 environments in MG VI.

In this study, environment was considered a random factor and cultivar and glyphosate were considered fixed factors. To avoid violation of the assumptions for analysis of variance, the CFU values were subjected to logarithmic transformation using the formula $\log_{10}(\text{CFU} + 1)$. The values of DX and IS were subjected to square root transformation using the formulae $\sqrt{(\text{DX} + 0.5)}$ and $\sqrt{(\text{IS} + 0.5)}$, respectively. Transformed data were subjected to analysis of variance (SAS Inst., 1985). Analysis of variance was conducted for combined environments to test possible interaction of treatments with environments.

RESULTS

There was significant ($P \leq 0.05$) variation among environments in MG III, IV, V, and VI for IS; MG II and VI for CFU; MG II, III, and VI for DX (Table 1). There was no significant Glyphosate × Environment interactions for any disease measure within any maturity groups (Table 1).

Across cultivars within each maturity group, there was no significant difference between glyphosate sprayed and nonsprayed subplots for all disease measures (Table 1). There was no Glyphosate × Cultivar interaction in any disease measure within any maturity group (Table 1).

For each disease measure, significant variation between cultivars was only observed in some maturity groups (Table 1). There was significant variation between cultivars for IS in MG IV and VI, for CFU in MG VI, and for DX in MG III and VI (Table 1). The SDS-resistant cultivar generally had less disease (as measured by IS, CFU, and DX) than the SDS-susceptible cultivar except for IS in MG III, V, and VI (Fig. 1 and 2).

Table 1. Mean square values from the analysis of variance of disease measures within maturity group.

Source of variation	df	<i>F</i> -test den†	Mean squares				
			MGII	MGIII	MGIV	MGV	MGVI
Infection severity (IS)							
1 Environment (Env)	3	2	3.694	6.686***	41.633**	30.393**	43.204***
2 Rep (Env)	12	11	1.417	0.445	5.426**	4.906	2.734
3 Cultivar (CV)	1	4	9.676	4.364	36.770*	4.259	5.960*
4 CV × Env	3	5	3.455**	3.766	2.199	3.916	0.233
5 CV × Rep (Env)	12	11	0.472	1.479	1.427	1.756	0.888
6 Glyphosate (Gly)	1	7	0.956	0.167	0.794	0.669	0.001
7 Gly × Env	3	8	0.732	0.644	0.350	1.210	0.821
8 Gly × Rep (Env)	12	11	0.578	0.817	1.206	1.909	0.700
9 Gly × CV	1	10	1.128	0.002	5.154	0.000	0.360
10 Gly × CV × Env	3	11	0.462	0.096	2.150	0.645	1.553
11 Error	12		1.056	1.227	0.919	4.266	1.380
Colony forming units (CFU)							
1 Environment (Env)	1	2	7.83*	4.61	0.01	0.02	5.03*
2 Rep (Env)	6	11	0.59	1.18*	1.22*	0.84	0.39
3 Cultivar (CV)	1	4	4.61	1.93	2.86	4.29	10.35*
4 CV × Env	1	5	0.73	0.60	4.53**	0.15	0.06
5 CV × Rep (Env)	6	11	0.57	0.54	0.26	0.42	0.47
6 Glyphosate (Gly)	1	7	0.20	0.51	0.40	0.29	0.92
7 Gly × Env	1	8	0.09	0.26	1.02	0.04	0.22
8 Gly × Rep (Env)	6	11	0.11	0.72	0.23	0.39	0.08
9 Gly × CV	1	10	0.07	1.91	0.34	0.08	0.09
10 Gly × CV × Env	1	11	0.18	0.25	0.38	0.04	0.00
11 Error	6		0.78	0.26	0.21	0.71	0.57
Disease index (DX)							
1 Environment (Env)	3	2	15.00***	9.160***	–	0.123§	7.821***‡
2 Rep (Env)	12	11	0.228	0.478*	–	0.254‡	0.402*§
3 Cultivar (CV)	1	4	4.207	42.365*	–	18.054	25.317*
4 CV × Env	3	5	0.703	7.050***	–	0.222‡	0.384‡
5 CV × Rep (Env)	12	11	0.324	0.232	–	0.282§	0.637**§
6 Glyphosate (Gly)	1	7	0.004	0.083	–	0.613	0.000
7 Gly × Env	3	8	0.245	0.012	–	0.413‡	0.102†
8 Gly × Rep (Env)	12	11	0.112	0.062	–	0.104§	0.064§
9 Gly × CV	1	10	0.091	0.000	–	0.439	0.035
10 Gly × CV × Env	3	11	0.152	0.068	–	0.583	0.087‡
11 Error	12		0.091	0.125	–	0.093§	0.074§
Grain yield							
1 Environment (Env)	3	2	4.047**	2.560**	20.791***	5.920***	12.430***
2 Rep (Env)	12	11	0.316*	0.165	0.396	0.503	0.133
3 Cultivar (CV)	1	4	7.931**	2.592	2.768	0.051	0.663
4 CV × Env	3	5	0.098	0.867*	0.647*	0.472	0.076
5 CV × Rep (Env)	12	11	0.078	0.224*	0.139	0.449	0.177
6 Glyphosate (Gly)	1	7	0.002	0.088	0.001	0.359	0.599
7 Gly × Env	3	8	0.028	0.525**	0.071	0.075	0.131
8 Gly × Rep (Env)	12	11	0.037	0.054	0.107	0.292	0.145
9 Gly × CV	1	10	0.110	0.014	0.035	0.110	0.193*
10 Gly × CV × Env	3	11	0.058	0.345*	0.045	0.355	0.012
11 Error	12		0.107	0.074	0.416	0.224	0.126

* Significant at the 0.05 level.

** Significant at the 0.01 level.

*** Significant at the 0.001 level.

† *F*-test denominator.

‡ Degrees of freedom (df) = 1.

§ Degrees of freedom = 6.

The application of glyphosate did not reduce yield in this study (Table 1). There was significant Glyphosate × Cultivar interaction for yield in MG VI (Table 1). In MG VI, the SDS-susceptible cultivar sprayed with glyphosate had significantly higher grain yield than the nonsprayed (Fig. 3).

DISCUSSION

Soybean SDS in Illinois was very severe in 1998 (Wrather et al., 2001). In 1998, the first year of this study, DX on susceptible checks averaged across maturity groups ranged from 14% in central Illinois to 38% in southern Illinois (data not shown). In 1999, the second year of this study, DX on susceptible checks averaged across maturity groups was significantly lower, and

ranged from 10% in central Illinois to 14% in southern Illinois (data not shown). Soil moisture and temperature in July (during soybean pod fill period) accounted for most of the year to year variation in DX. High soil moisture during this period results in severe SDS (Rupe et al., 1993). In July 1998, rainfall was 95.8 mm in southern Illinois and 106 mm in central Illinois, resulting in high soil moisture in the test regions. However, in July 1999, rainfall was lower in each test region, 45 and 97 mm for southern and central Illinois, respectively (Illinois State Water Survey, 2002).

The MG IV, the earliest of the cultivars evaluated in southern Illinois, escaped SDS symptoms (DX = 0). It has been observed that earlier cultivars may progress past the critical growth stages (<R6) for SDS leaf symptoms before favorable conditions for symptom occur-

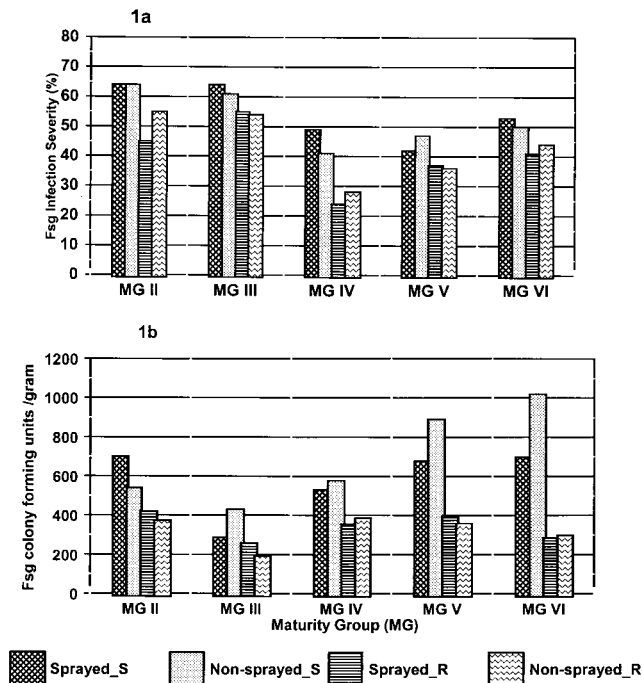


Fig. 1. Bar graphs of Glyphosate \times Cultivar interaction means for soybean root colonization by *Fusarium solani* f. sp. *glycines*. (a) Infection severity, clusters of bars represent averages over four environments; (b) colony forming units, clusters of bars represent averages over two environments.

rence become available (Rupe et al., 1993; Gibson et al., 1994; Hnetkovsky et al., 1996). Environmental dependence of leaf symptom expression highlights the importance of root infection as a measure of SDS resistance.

Development of SDS symptoms occurrence is influenced by both the genotype of the soybean cultivar and the environment. Significant differences in DS means among environments were consistent with previous studies (Gibson et al., 1994; Rupe et al., 1993). Disease development and severity in any given environment are functions of planting date, genotype, and soil factors including but not limited to soil moisture and temperature (Hershman et al., 1990; Rupe et al., 1993). As a result, some cultivars show inconsistent responses from one environment to another. However, there are a few cultivars (susceptible and resistant) that show low Genotype \times Environment interaction (Gibson et al., 1994; Njiti et al., 2002). Significant differences between cultivars were expected in this study since the cultivars were selected to contrast for disease resistance. However, in some environments (data not shown) and maturity groups, cultivar differences were not significant. This is an indication of Genotype \times Environment interaction ($G \times E$). Genotype \times Environment interaction is a large component of SDS resistance (Njiti et al., 1996) but the magnitude of $G \times E$ varies from cultivar to cultivar. The magnitude of $G \times E$ is influenced by the number of beneficial alleles for SDS resistance possessed by each cultivar (Iqbal et al., 2001) as well as the environmental factors.

In this study, the application of glyphosate to RR

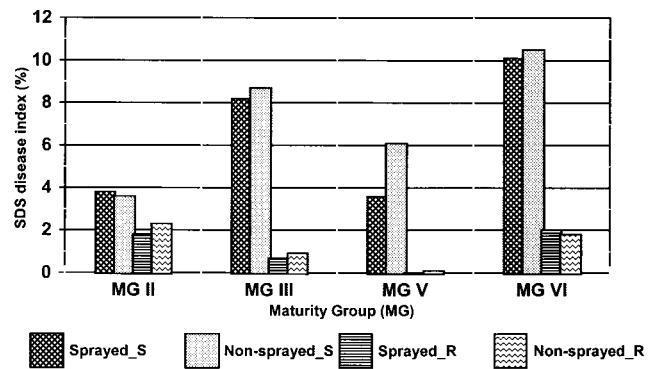


Fig. 2. Bar graphs of Glyphosate \times Cultivar interaction means for SDS disease index. Clusters of bars for MG II and III are averaged over four environments and MG V and VI are averaged over three environments; MG IV was not included in the analysis due to missing data.

soybean did not increase susceptibility to soybean root infection by Fsg or soybean SDS leaf symptoms caused by toxin produced in response to root infection. It has been suggested that there might be a relationship between the application of glyphosate to RR soybean and increased fungal diseases (Kremer, unpublished, 2001). This study found no relationship between the application of glyphosate to RR soybean and SDS. No increase in fungal colonization was noted. It has been reported that RR soybean plants inoculated with Fsg in the greenhouse had more root colonization by the fungus and more severe leaf symptoms when sprayed with glyphosate (Sanogo et al., 2000). However, the response was not limited to glyphosate herbicide. The difference between the greenhouse and field response may be related to the stage of evaluation and inoculum concentration (Njiti et al., 2001). Some of the cultivars, including Resnik (McBlain et al., 1990a), Flyer (McBlain et al., 1990b), and A5403 used as parents in the development of RR soybean cultivars have been shown to be very susceptible to SDS (Gibson et al., 1994; Prabhu et al., 1999). Alleles contributed by these parents may explain why some RR cultivars are very susceptible to SDS.

In most MG, the SDS-resistant cultivars had higher grain yield than the SDS-susceptible cultivars. It is not clear whether this was the effect of yield genes or SDS resistance genes.

CONCLUSION

In this study root colonization by Fsg and SDS leaf symptoms did not significantly increase following the application of glyphosate. Data from this study indicate that the development of SDS on RR soybean is influenced by the genotype of the cultivar. Farmers planting RR soybean in Fsg-infested fields are encouraged to select cultivars that have been shown to be resistant to SDS.

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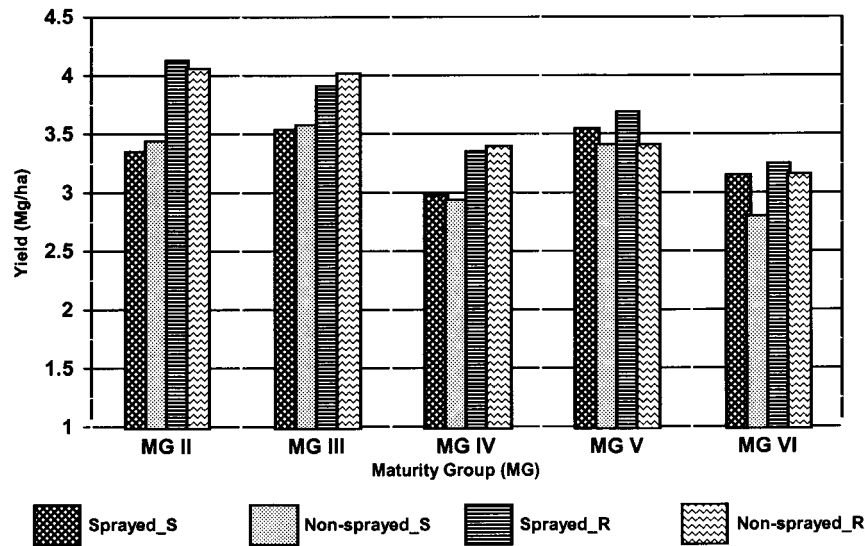


Fig. 3. Bar graphs of Glyphosate \times Cultivar interaction means for yield. Clusters of bars for MG II to V are averaged over four environments and MG VI is averaged over three environments.

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