A Common Genetic Basis for Cross-sensitivity to Mesotrione and Nicosulfuron in Sweet Corn Hybrid Cultivars and Inbreds Grown throughout North America

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ABSTRACT. Mutation of a cytochrome P450 (CYP) gene on the short arm of chromosome five, referred to as *nsfl* or *ben1*, conditions sensitivity to certain P450-metabolized herbicides in corn (*Zea mays* L.). Previous research has shown that the sweet corn inbred Cr1 is sensitive to nicosulfuron, mesotrione, and at least seven other P450-metabolized herbicides with five different modes of action. Although the *nsfl/ben1* CYP gene has not been sequenced from Cr1, a QTL that conditions cross-sensitivity to P450-metabolized herbicides was detected in a segregating population of Cr1 × Cr2 (herbicide tolerant) on the short arm chromosome five in tight linkage disequilibrium with the *nsfl/ben1* CYP locus. Sweet corn hybrid cultivars and inbreds that had been identified in previous research as being susceptible to injury from P450-metabolized herbicides were tested in this study to determine if they were allelic with Cr1 for cross-sensitivity to nicosulfuron and mesotrione. These cultivars and inbreds were developed by 12 independent commercial breeding programs. These cultivars include sugary, sugary enhancer, and shrunken-2 endosperm types that are grown for processing and fresh consumption in markets throughout North America and in other temperate climates throughout the world. Each hybrid cultivar, their F₂ progeny, and progeny from testcrosses of cultivars with Cr1 and Cr2 were evaluated for responses to mesotrione and nicosulfuron. Each inbred line, progeny from crosses of inbreds with Cr1 and Cr2, and F₂ progeny from crosses of inbreds with Cr1 were also tested. Based on segregation of progeny from testcrosses with Cr1 and Cr2 and the F₂ generation, 45 sweet corn hybrid cultivars and 29 sweet corn inbreds, including lines from each of the 12 breeding programs, appeared to be sensitive to nicosulfuron and mesotrione as the result of a gene that is the same as or very closely linked to the gene in Cr1. None of the cultivars or inbreds appeared to be sensitive to these herbicides as a result of other independent genes; however, additional genes that modify responses to these herbicides may be present in a few cases. The presence of a gene conditioning sensitivity to nicosulfuron and mesotrione, and probably to several other P450-metabolized herbicides, provides an explanation for varied levels of injury and inconsistent responses of sweet corn hybrid cultivars under differing environmental conditions. This information provides a basis from which an industry-wide concern with herbicide sensitivity in sweet corn can be addressed by various methods, including the elimination of an allele rendering germplasm sensitive.

Under certain environmental conditions in North America, some sweet corn hybrid cultivars and inbreds are injured by postemergence applications of cytochrome P450-metabolized

corn herbicides. Some of the sweet corn cultivars that are sensitive to specific herbicides have been identified in the past two decades in numerous field trials (Burton et al., 1994; Diebold et al., 2003, 2004; Grey et al., 2000; Monks et al., 1992; Morton and Harvey, 1992; O'Sullivan and Bouw, 1998; O'Sullivan and Sikkema, 2002; O'Sullivan et al., 1995, 2000, 2002; Robinson et al., 1993, 1994; Sikkema et al., 2008; Stall and Bewick, 1992; Williams and Pataky, 2008; Williams et al., 2005). Although useful, this information is limited because a relatively small percentage of nearly 600 sweet corn cultivars sold commercially in North America were tested in any one of these studies and the commercial availability of cultivars changes continually as new cultivars are introduced and older cultivars are discontinued. A better understanding of the genetic basis of herbicide sensitivity in sweet corn would help the sweet corn industry manage this problem more effectively and it may provide an explanation for varied responses of certain cultivars under differing conditions.

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In previous research, the sweet corn inbred Cr1 was found to be sensitive to multiple postemergence herbicides, including four acetolactate synthase (ALS)-inhibiting herbicides (foramsulfuron, nicosulfuron, primisulfuron, and rimsulfuron), two 4-hydroxyphenylpyruvate dioxygenase (HPPD)-inhibiting herbicides (mesotrione and tembotrione), a growth regulator herbicide combination (dicamba + diflufenzopyr), a protoporphyrinogen oxidase (PPO)-inhibiting herbicide (carfentrazone), and a photosystem II (PSII)-inhibiting herbicide (bentazon) (Nordby et al., 2008; Pataky et al., 2006; Williams and Pataky, 2008; Williams et al., 2005). Sensitivity to these herbicides appeared to be conditioned by a single gene or closely linked genes based on segregation of progeny in F₂, BC₁, and BC₂ generations of a cross of Cr1 with Cr2 (a herbicide-tolerant inbred) and cosegregation of phenotypic responses among BC₁S₁, BC₂S₁, F_{2:3}, F_{3:4}, and F_{3:5} families, (Nordby et al., 2008; Pataky et al., 2006; Williams and Pataky, 2008). Another independent gene from Cr2 also conditioned tolerance to bentazon.

These herbicides to which Cr1 is sensitive have different modes of action, but all are metabolized by cytochrome P450 monooxygenases. Barrett et al. (1994) previously proposed the existence of a "super P450" that metabolizes multiple herbicides. Although corn has many cytochrome P450 genes (CYP genes), the number of P450s involved in metabolic inactivation of herbicides, their expression levels, and their levels of herbicide metabolism are not clearly understood (Barrett, 1995, 2000; Frey et al., 1995, 1997; Persans et al., 2001).

A growing body of evidence suggests that sensitivity of corn to multiple P450-metabolized herbicides is regulated by a single CYP gene or a group of closely linked CYP genes on the short arm of chromosome 5. Kang (1993) associated nicosulfuron sensitivity in field corn inbred lines, including the inbred W703a, with a single, recessive gene designated as *nsf1*. Others also reported simple inheritance of tolerant and sensitive responses to nicosulfuron and similar ALS-inhibiting herbicides (Green, 1998; Green and Ulrich, 1993; Harms et al., 1990; Widstrom and Dowler, 1995). Green (1998) cited an unpublished report that mapped sensitivity to rimsulfuron in the field corn inbred F2 to the short arm of chromosome 5. Fleming et al. (1988), Bradshaw et al. (1994), and Barrett et al. (1997) associated bentazon and nicosulfuron sensitivity in field corn inbreds B90 and GA209 with a single, recessive gene designated as *ben1*. An independent, dominant gene, designated as *Ben2*, conditioned tolerance to bentazon in the inbred B73 (Barrett et al., 1997; Bradshaw et al., 1994). Williams et al. (2006) used a mapped-based cloning approach to locate the *Nsf1* gene on the short arm of chromosome 5 and to sequence the dominant, functional allele from a nicosulfuron-tolerant inbred, B73. The *Nsf1* gene was one of four closely linked genes with significant sequence similarity to CYP genes. The *Nsf1* gene also was very similar in sequence and function to a CYP gene in rice that conditions tolerant responses to multiple herbicides (Pan et al., 2006). Nicosulfuron-sensitive inbreds GA209 and W703a contained a 392-bp insertion in the *Nsf1* gene sequence relative to B73. Thus, it appears that the *nsf1* and *ben1* alleles identified from GA209 and W703a, respectively, are the same 392-bp insertion mutation of this CYP allele. The 392-bp insertion in this CYP gene sequence also occurs in several nicosulfuron-sensitive field corn inbreds (e.g., A180, B90, MS1334, NC22, and R4), whereas other nicosulfuron-sensitive field and sweet corn inbreds (e.g., B94 and Ia5125) do not contain this insertion. The *nsf1/ben1* CYP gene has not been

sequenced from the herbicide-sensitive sweet corn inbred Cr1; however, a QTL that conditions sensitivity to multiple P450-metabolized herbicides was detected in a segregating population of Cr1 × Cr2 on the short arm chromosome 5 in tight linkage disequilibrium with the *nsf1/ben1* CYP locus (Nordby et al., 2008).

The genetic condition at a single locus explained varied responses of 149 commercially adapted sweet corn hybrid cultivars to three P450-metabolized, postemergence herbicides (foramsulfuron, mesotrione, and nicosulfuron) in 12 field trials throughout the United States (Pataky et al., 2008). Seven cultivars classified as homozygous for alleles conditioning herbicide sensitivity were injured most severely and often were killed by the two ALS-inhibiting herbicides. Ninety-five cultivars classified as homozygous for an allele conditioning herbicide tolerance were uninjured or injured least. Forty-seven cultivars classified as heterozygous with one allele each conditioning herbicide tolerance and sensitivity displayed intermediate responses that were more similar to homozygous tolerant cultivars than homozygous sensitive cultivars. Injury to heterozygous and homozygous tolerant cultivars was minimal and often not significantly different in trials in dry, western climates (e.g., Caldwell, ID; Nampa, ID; and Olathe, CO), whereas injury to heterozygous cultivars was significantly greater than injury to homozygous tolerant cultivars in trials in eastern climates (e.g., Georgetown, DE and LeRoy, NY). Nevertheless, it was not evident that the seven homozygous sensitive cultivars and the 49 heterozygous cultivars carried the same allele conditioning sensitivity. The objective of this study was to determine if sensitivity to nicosulfuron and mesotrione among commercially adapted, sweet corn cultivars and inbreds is allelic with or closely linked to the recessive gene conditioning sensitivity in the sweet corn inbred Cr1.

Materials and Methods

PLANT MATERIALS AND CROSSES. Fifty-four sweet corn cultivars (i.e., hybrids) and 40 sweet corn inbreds were evaluated in this study. All of the sweet corn cultivars and most of the inbreds were obtained from 12 independent commercial breeding programs. Most of the cultivars were selected based on their potential for herbicide injury as indicated from results of previous public or proprietary trials, although some herbicide-tolerant controls also were included. Fifteen of the 54 cultivars in this study were among the 47 cultivars heterozygous for alleles conditioning herbicide tolerance and sensitivity in a previous study (Pataky et al., 2008). The group of cultivars in this study included those grown for processing and fresh market as well as white, yellow, or bicolored sweet corn with sugary, sugary enhancer, or shrunken-2 endosperm types. Thirty-seven proprietary sweet corn inbreds from nine commercial breeding programs were selected by their breeders as representative of herbicide-sensitive and -tolerant inbreds. The three major endosperm types and processing and fresh market types of sweet corn also were represented by this group of inbreds. Three nicosulfuron-sensitive, public sweet corn inbreds (Ia5125, Ia5125sh2, and IL14h) also were evaluated.

Hybrid cultivars and inbreds were planted in pollination nurseries in 2005 with pollinator rows of Cr1, Cr2, and three nicosulfuron-sensitive, field corn inbreds (B90, B94, and GA209). Cr1 is a nicosulfuron- and mesotrione-sensitive sweet corn inbred. Cr-2 is a nicosulfuron- and mesotrione-tolerant

sweet corn inbred. B90 and GA209 have the same 392-bp insertion in the *Nsf1/Ben1* CYP gene sequence as W703A, but B94 does not have this insertion nor does the sweet corn inbred Ia5125 (Williams et al., 2006). Each sweet corn cultivar was self pollinated to produce seed of F₂ progeny. Each cultivar also was crossed with Cr1, Cr2, and one or more of the three field corn inbreds to produce testcross progeny. Each sweet corn inbred also was crossed with Cr1, Cr2, and one or more of the three herbicide-sensitive field corn inbreds. F₁ progeny from crosses of sweet corn inbreds with Cr1 were self pollinated in a winter nursery in Chile to produce seed of F₂ progeny. The three field corn inbreds also were crossed with Cr1 and Cr2.

FIELD EVALUATIONS OF RESPONSES TO NICOSULFURON AND MESOTRIONE. Sweet corn cultivars, sweet corn inbreds, field corn inbreds, and progeny from crosses were evaluated for responses to nicosulfuron and mesotrione in field trials in 2006 and 2007. Four separate trials were planted each year for each combination of two groups of plant materials (i.e., cultivars and inbreds) and two herbicides. Each trial included two replicates of plant materials arranged in randomized blocks of a split-plot experimental design. Main plots were planted with all of the crosses derived from a cultivar or an inbred. For sweet corn cultivars, the number of rows of each subplot varied according to the generation being evaluated and availability of seed, and included one row of the cultivar, three rows of the F₂ generation, two rows of the Cr1-testcross, and one row each of the Cr2-testcross and testcrosses with field corn inbreds. For sweet corn inbreds, subplots were one row each of the inbred, the F₁ hybrids from crosses with Cr1, Cr2, and field corn inbreds, and the F₂ generation from the cross with Cr1. In all trials, each row was ≈ 3.1 m in length with 12 to 22 plants per row, depending on availability and germination of seed. Trials were planted 8 May 2006 and 3 May 2007. One row each of Cr1, Cr2, Cr1 $\times$ Cr2, and a sensitive sweet corn cultivar (Merit) were included in each replicate of each trial. One row each of B90, B94, and the F₁ hybrids: Cr1 $\times$ B90, Cr1 $\times$ B94, Cr2 $\times$ B90, Cr2 $\times$ B94, B90 $\times$ GA209, and B94 $\times$ GA209 also were included in each replicate of the cultivar trials.

Commercial formulations of herbicides were applied when plants had leaves with four to six visible collars. In both years, nicosulfuron was applied at 70 g·ha⁻¹ with 1% (v/v) crop oil concentrate (COC) and 3.6% (v/v) spray solution of 28% urea ammonium nitrate (UAN). This rate, which is twice the recommended usage rate in corn, was used to ensure distinct differences between sensitive and tolerant responses. In 2006, mesotrione was applied at 158 g·ha⁻¹ with 1% (v/v) COC and 2.5% (v/v) spray solution of 28% UAN. This rate, which is 1.5 times the recommended usage rate in corn, also was used to ensure distinct differences between sensitive and tolerant responses; however, intermediate responses to mesotrione occurred in 2006 that often were difficult to differentiate from sensitive responses. Therefore, in 2007, mesotrione was applied at the recommended usage rate of 105 g·ha⁻¹ with 1% (v/v) COC and 2.5% (v/v) spray solution of 28% UAN.

Plants were rated visually for injury 13 to 15 d after application of nicosulfuron and 6 to 8 d after application of mesotrione. Phenotypic responses were easily differentiated 2 weeks after application of nicosulfuron, as plants were healthy and green (i.e., tolerant phenotype) or nearly dead (i.e., sensitive phenotype). An occasional plant that was severely wilted or dead because of root-feeding insects, root rots, or other causes may have been mistakenly classified as sensitive to

nicosulfuron although it was actually tolerant. Stunting or other quantitative measures of injury from nicosulfuron were not recorded. Thus, plants that were stunted but not killed by nicosulfuron were classified as tolerant. Phenotypic responses also were distinct 7 d after application of mesotrione, although many plants displayed an intermediate phenotype in 2006 when a 1.5 $\times$ rate of herbicide was applied. Tolerant plants were healthy and green, whereas leaves growing from leaf whorls of sensitive plants were chlorotic and bleached. In 2006, some plants had chlorotic tissues occurring in a 1- to 5-cm band on leaves about 3 to 7 cm above the whorl. The chlorotic area appeared to be the leaf tissue that formed the whorl on the day that mesotrione was applied. Plants with these intermediate phenotypes were grouped with tolerant responses, although in some instances the difference between intermediate and sensitive phenotypes was subjective; thus, some plants were undoubtedly misclassified. Therefore, segregation of responses to mesotrione was analyzed from data collected in 2007 when phenotypes were more definitive.

The chi-square goodness of fit test was used to determine if the number of tolerant and sensitive plants in segregating generations was different from the ratio expected if the response to these herbicides was conditioned by a single gene that was allelic with or closely linked to the gene in Cr1. Nicosulfuron data were combined from a total of four replicates from trials in 2006 and 2007. Mesotrione data were from two replicates from trials in 2007.

Results

Across all trials, 99% of inbred and hybrid control plants displayed expected responses to nicosulfuron and mesotrione. None of 308 plants of the tolerant inbred Cr2 were injured by nicosulfuron or mesotrione, whereas 280 of 284 plants of the sensitive inbred Cr1 were injured. Similarly, only 6 of 342 plants of the F₁ hybrid Cr1 $\times$ Cr2 were injured while 382 of 386 plants of the sensitive hybrid 'Merit' were injured.

SWEET CORN HYBRID CULTIVARS. Nine cultivars were classified as homozygous tolerant and eight cultivars were classified as homozygous sensitive to nicosulfuron and mesotrione (Tables 1 and 2). Thirty-seven cultivars were classified as heterozygous for alleles conditioning response to mesotrione and nicosulfuron (Tables 2 and 3).

None of 1518 plants of the nine homozygous tolerant cultivars were sensitive to nicosulfuron or mesotrione, and only 14 of 7597 progeny (0.2%) of these cultivars in the F₂ generation and in testcross generations with Cr1 and Cr2 were sensitive (Table 2). Based on 99.9% tolerant F₂ progeny, these cultivars were considered to be homozygous for an allele conditioning tolerance to nicosulfuron and mesotrione.

Among eight cultivars classified as homozygous sensitive, 98% of 1050 plants were sensitive to nicosulfuron or mesotrione, and 99% of 4631 progeny in the F₂ and the Cr1-testcross generations were sensitive (Table 2). Only 4 of 247 progeny from testcrosses of these cultivars with Cr2 were sensitive. Based on 98.3% sensitive F₂ progeny and 99.6% sensitive progeny from testcrosses with Cr1, these eight cultivars were considered to be homozygous for an allele that is the same as or very closely linked to an allele in Cr1 that conditions sensitivity to nicosulfuron and mesotrione.

Among the 37 seven hybrid cultivars classified as heterozygous, 25 of 4717 F₁ plants were sensitive to nicosulfuron or

Table 1. Sweet corn hybrid cultivars classified as homozygous for alleles conditioning a tolerant or sensitive response to nicosulfuron and mesotrione.

Cultivars with a tolerant response	Seed source ^z	Cultivars with a sensitive response	Seed source ^z
Conquest	Cr	177A	IFSI
Empire	Rog	CrshYP3-112	Cr
GSS 6314	Rog	DMC 20-38	DM
HMX 2374 BS	HM	EX 087 0 5770	Sem
HY1558	SnRv	Gallant	Cr
Intrique	Cr	HY 1734	SnRv
Rustler	HM	Kahuna	SnRv
Snow White	HM	Shogun	Cr
Twin Star	HM		

^zCr = Crookham (Caldwell, ID); DM = Del Monte USA (Rochelle, IL); HM = Harris Moran (Modesto, CA); IFSI = Illinois Foundation Seeds (Champaign, IL); Rog = Rogers Seeds/Syngenta (Boise, ID); Sem = Seminis Vegetable Seeds (Oxnard, CA); SnRv = Snowy River (Orbost, Australia).

mesotrione, and 4 of 2843 progeny from testcrosses with Cr2 were sensitive (Table 2). For the entire group of 37 heterozygous cultivars, 21.3% of 9120 F₂ progeny were sensitive and 48.4% of 8080 Cr1-testcross progeny were sensitive (Table 2).

Individually, for each of the 37 cultivars classified as heterozygous, F₂ progeny and Cr1-testcross progeny segregated for tolerant and sensitive plants in ratios that usually were not significantly different from 3:1 and 1:1, respectively, which would be expected if a single allele in the cultivar conditioned sensitivity and that allele was the same as or closely linked to the QTL conditioning sensitivity in Cr1 (Table 3). Segregation in the Cr1-testcross generation treated with nicosulfuron or mesotrione was not significantly different ($P > 0.05$) from a 1:1 ratio of tolerant:sensitive progeny for 36 of the 37 heterozygous cultivars (Table 3). Only GH 2298 segregated significantly different from 1:1 with a greater than expected number of progeny tolerant to nicosulfuron and mesotrione. Segregation in the F₂ generation treated with nicosulfuron or mesotrione was not significantly different ($P > 0.01$) from a 3:1 ratio of tolerant:sensitive progeny for 24 of the 37 cultivars classified as heterozygous (Table 3). In all 10 instances where segregation of

F₂ progeny did not fit a 3:1 ratio following application of nicosulfuron, the number of sensitive F₂ progeny was less than expected. This may have occurred because some sensitive progeny were stunted rather than killed 2 weeks after application of nicosulfuron and thus were misclassified as tolerant. In the four instances where segregation of F₂ progeny did not fit a 3:1 ratio following application of mesotrione, the number of sensitive F₂ progeny was greater than expected. This may have occurred because some heterozygous F₂ progeny with an intermediate response were classified as sensitive rather than tolerant.

SWEET CORN INBREDS. Eleven sweet corn inbreds were classified as tolerant and 29 sweet corn inbreds were classified as sensitive to nicosulfuron and mesotrione (Table 4). Two of the 29 sensitive inbreds differed in response to nicosulfuron and mesotrione. One of the 29 sensitive inbreds appeared to be segregating for response to nicosulfuron and mesotrione.

Among the 11 inbreds classified as tolerant, only 2 of 619 inbred plants were sensitive to nicosulfuron or mesotrione, and only 4 of the 3144 progeny from testcrosses with Cr1, field corn inbreds, or Cr2 were sensitive to nicosulfuron or mesotrione (Table 4). Segregation of F₂ progeny from crosses of the 11 tolerant inbreds with Cr1 was not significantly different ($P > 0.05$) from a 3:1 ratio (tolerant:sensitive) for the entire group (i.e., 965:350) or for any of the 22 individual analyses of F₂ progeny treated with nicosulfuron or mesotrione (data not shown).

Among 26 inbreds with uniformly sensitive responses to nicosulfuron and mesotrione, 98.9% of 1710 inbred plants, 99.9% of 2647 progeny from crosses of the inbreds with Cr1, 99.8% of 2852 F₂ progeny from those crosses, and 99.6% of 2135 progeny from crosses of the inbreds with field corn inbreds B90, B94, and GA209 were sensitive to nicosulfuron and mesotrione (Table 4). Twelve of 2698 progeny from crosses of these 26 inbreds and Cr2 were classified as sensitive. Based on homogeneous sensitive responses of F₁ progeny from crosses with Cr1, B90, B94, and GA209, and F₂ progeny from crosses with Cr1, these 26 inbreds were considered to be homozygous for an allele that conditions sensitivity to nicosulfuron and mesotrione. This allele occurs at the same chromosomal location as the allele that conditions herbicide sensitivity in Cr1 and three field corn inbreds.

Table 2. Number of tolerant and sensitive progeny in F₁, F₂, and testcross^z generations for sweet corn hybrid cultivars classified as homozygous tolerant, homozygous sensitive, or heterozygous for alleles conditioning response to nicosulfuron and mesotrione.

Proposed genotype and herbicide	n ^y	Plants with tolerant or sensitive responses in each generation (no.)							
		F ₁ hybrid		Hybrid F ₂		Hybrid × Cr1 ^z		Hybrid × Cr2 ^z	
		Tolerant	Sensitive	Tolerant	Sensitive	Tolerant	Sensitive	Tolerant	Sensitive
Homozygous tolerant	9								
Nicosulfuron		772	0	1,867	4	1,502	3	479	1
Mesotrione		746	0	1,750	0	1,533	6	452	0
Homozygous sensitive	8								
Nicosulfuron		10	526	8	1,148	1	1,253	138	0
Mesotrione		7	507	30	1,026	8	1,157	105	4
Heterozygous	37								
Nicosulfuron		3,191	7	4,868	1,190	2,802	2,623	1,899	4
Mesotrione		1,501	18	2,313	749	1,367	1,288	940	0

^zTestcrosses of F₁ hybrid cultivars with Cr1 (a nicosulfuron- and mesotrione-sensitive inbred) and with Cr2 (a nicosulfuron- and mesotrione-tolerant inbred).

^yn = number of hybrid cultivars.

Table 3. Number of tolerant and sensitive testcross^z and F₂ progeny for sweet corn hybrid cultivars classified as heterozygous for alleles conditioning response to nicosulfuron and mesotrione.

Plants with tolerant or sensitive responses (no.) and chi-square probabilities													
Cultivar	Seed source ^y	Cultivar × Cr1 testcross ^z						F ₂ generation					
		Nicosulfuron			Mesotrione			Nicosulfuron			Mesotrione		
		T ^x	S ^x	P ^x	T	S	P	T	S	P	T	S	P
Segregation in all generations fit expected ratios													
178A	IFSI	78	79	0.94	25	29	0.59	93	22	0.15	42	22	0.08
Argent	Cr	79	66	0.28	24	27	0.67	104	39	0.53	56	24	0.30
Brocade	MM	71	75	0.74	32	39	0.41	86	20	0.14	35	9	0.4
Celestial	Cr	58	58	1.00	31	26	0.51	156	43	0.27	46	17	0.72
Coho	HM	97	87	0.46	35	38	0.73	155	74	0.01	73	37	0.04
CshBF3–122	Cr	76	78	0.87	38	37	0.91	85	25	0.58	64	21	0.95
CsuWP1–7	Cr	63	58	0.65	39	30	0.28	154	49	0.78	62	30	0.09
Double Gem	MM	72	75	0.80	39	44	0.58	78	38	0.05	60	13	0.16
Eliminator	Cr	84	89	0.70	33	33	1.00	173	36	0.01	67	24	0.76
GG Code 17	GG	72	68	0.74	36	39	0.73	164	44	0.20	71	17	0.22
GH 2669	Rog	91	88	0.82	49	48	0.92	205	63	0.57	115	27	0.10
Ivanhoe	MM	6	5	0.76	6	5	0.76	121	31	0.19	67	13	0.07
Jubilee	Rog	90	73	0.18	46	38	0.38	219	68	0.61	101	26	0.24
K3–312	Sak	64	56	0.47	37	30	0.39	108	25	0.10	46	18	0.56
Merkur	Sem	77	85	0.53	41	37	0.65	161	38	0.05	69	23	1.00
Mirai 002	Cent	89	87	0.88	35	34	0.90	41	6	0.05	17	4	0.53
Mystique	Cr	37	34	0.72	15	22	0.25	76	21	0.45	43	10	0.30
SCH 70064 RR	IFSI	93	78	0.25	40	43	0.74	175	57	0.88	90	29	0.87
Silver Queen	Rog	99	82	0.21	56	38	0.06	187	66	0.69	95	30	0.80
SS Jubilee	Rog	66	70	0.73	52	45	0.48	73	21	0.55	45	16	0.82
SS Jubilee Plus	Rog	55	50	0.63	30	37	0.39	109	36	0.96	46	19	0.43
UY 0657	SnRv	85	70	0.23	39	48	0.33	151	43	0.36	88	22	0.23
XTH 2477	IFSI	64	69	0.66	45	37	0.38	62	12	0.08	55	11	0.12
Segregation in the F ₂ generation treated with nicosulfuron did not fit the expected ratio													
277A	IFSI	57	64	0.52	32	29	0.70	79	8	0.001	65	13	0.09
Bold	Rog	81	87	0.64	41	32	0.29	175	30	0.001	79	22	0.46
El Toro	Sem	76	69	0.56	32	31	0.90	179	34	0.002	71	12	0.03
GH 0991	Rog	97	88	0.51	48	41	0.46	205	42	0.004	88	28	0.83
Hollywood	Sem	72	72	1.00	50	36	0.13	164	14	>0.001	81	23	0.50
How Sweet It Is	Cr	87	75	0.35	37	33	0.63	91	11	0.001	53	16	0.73
Mirai 003	Cent	66	86	0.10	21	25	0.56	95	7	>0.001	45	9	0.16
Polaris	HM	84	67	0.17	42	35	0.43	86	4	>0.001	36	9	0.44
XTH 1173	IFSI	69	61	0.48	37	43	0.50	159	17	>0.001	57	30	0.04
Segregation in the F ₂ generation treated with mesotrione did not fit the expected ratio													
170A	IFSI	75	77	0.87	33	39	0.48	66	15	0.18	29	22	0.003
Millenium	Sak	92	81	0.40	37	40	0.73	118	45	0.44	40	26	0.007
XTH 1178	IFSI	78	80	0.87	45	36	0.32	157	19	0.01	53	33	0.004
Segregation in the F ₂ generation treated with nicosulfuron and mesotrione did not fit the expected ratio													
Code 61	GG	83	80	0.81	30	38	0.33	149	22	>0.001	86	12	0.004
Segregation in the Cr1-testcross generation treated with nicosulfuron and mesotrione did not fit the expected ratio													
GH 2298	Rog	119	56	>0.001	65	31	0.001	209	46	0.01	87	22	0.25
Total		2,802	2,623		1,373	1,293		4,868	1,191		2,323	739	

^zTestcross of F₁ hybrid cultivars with Cr1 (a nicosulfuron- and mesotrione-sensitive inbred).

^yCent = Centest (Harvard, IL); Cr = Crookham (Caldwell, ID); GG = Green Giant (LeSueur, MN); HM = Harris Moran (Modesto, CA); IFSI = Illinois Foundation Seeds (Champaign, IL); MM = Mesa Maize (Olathe, CO); Rog = Rogers/Syngenta (Boise, ID); Sak = Sakata Seed USA (Morgan Hill, CA); Sem = Seminis Vegetable Seeds (Oxnard, CA); SnRv = Snowy River (Orbost, Australia).

^xT = tolerant, S = sensitive, and P = probability associated with a chi-square goodness of fit test for 1:1 segregation of testcross progeny and 3:1 segregation of F₂ progeny for tolerant and sensitive responses.

Two inbreds classified as sensitive had different responses to nicosulfuron and mesotrione (Table 4). All 94 plants of these two inbreds were sensitive to mesotrione, but only 10 of 94 plants were sensitive to nicosulfuron. Nevertheless, all 275 F₁ progeny from crosses with Cr1 and 336 of 344 F₂ progeny from

those crosses were sensitive to nicosulfuron or mesotrione. All 53 progeny from crosses with sensitive field corn inbreds were sensitive to mesotrione, but only 42 of 61 progeny from crosses with sensitive field corn inbreds were sensitive to nicosulfuron. Two weeks after application of nicosulfuron, plants of these

Table 4. Number of plants tolerant and sensitive to nicosulfuron and mesotrione for 40 sweet corn inbreds and their F₁ progeny from crosses with Cr1-, Cr2-, and herbicide-sensitive field corn inbreds and the F₂ progeny from crosses with Cr1.

Proposed response and herbicide	n ^v	Plants with tolerant or sensitive responses to nicosulfuron or mesotrione in each generation (no.)									
		Inbred ^z		Inbred × Cr1 ^y		(Inbred × Cr1) F ₂		Inbred × field corn ^w		Inbred × Cr2 ^x	
		Tolerant	Sensitive	Tolerant	Sensitive	Tolerant	Sensitive	Tolerant	Sensitive	Tolerant	Sensitive
Tolerant	11										
Nicosulfuron		366	2	792	0	634	232	234	0	679	0
Mesotrione		251	0	452	3	331	118	259	1	724	0
Sensitive	26										
Nicosulfuron		6	988	2	1,634	4	1,716	9	998	1,690	12
Mesotrione		13	703	1	1,010	3	1,129	0	1,128	996	0
Sensitive ^a	2										
Nicosulfuron		84	10	0	129	8	156	19	42	153	0
Mesotrione		0	94	0	146	0	180	0	53	160	3
Sensitive ^t	1										
Nicosulfuron		7	2	27	29	12	58	30	29	51	0
Mesotrione		11	6	34	25	4	37	0	73	44	0

^zSweet corn inbreds include three sensitive public inbreds (Ia5125, Ia5125sh2, and IL14h) and 37 proprietary inbreds from nine commercial breeding programs, including Abbott & Cobb (Feasterville, PA) (3); Crookham (Caldwell, ID) (8); Del Monte USA (Rochelle, IL) (2); Green Giant (LeSueur, MN) (2); Harris Moran (Modesto, CA) (8); Illinois Foundation Seeds (Champaign, IL) (4); Mesa Maize (Olathe, CO) (1); Rogers Seeds/Syngenta (Boise, ID) (7); and Snowy River (Orbost, Australia) (2).

^yCr1 is a nicosulfuron- and mesotrione-sensitive sweet corn inbred from Crookham.

^xCr2 is a nicosulfuron- and mesotrione-tolerant sweet corn inbred from Crookham.

^wHerbicide-sensitive field corn inbreds: B90, B94, or GA209.

^vn = number of inbreds.

^tTwo sensitive inbreds with different responses to mesotrione (i.e., sensitive) and nicosulfuron (i.e., not killed) as inbreds per se.

^aOne sensitive inbred that appeared to segregate for tolerant and sensitive responses to nicosulfuron and mesotrione.

two inbreds were rated as tolerant because they were not dead or nearly dead; however, from 3 to 10 weeks after treatment, these plants were stunted. Although these two inbreds were not killed by nicosulfuron as were other sensitive inbreds, they appear to be homozygous for an allele that conditions sensitivity to nicosulfuron and mesotrione based on sensitive responses of F₁ and F₂ progeny from crosses with Cr1. These inbreds may possess another independent gene or several modifying loci with small effects that prevent nicosulfuron from killing them even though severe stunting occurs.

One inbred classified as sensitive appeared to be segregating for alleles conditioning herbicide responses. Eight of 26 inbred plants were sensitive to nicosulfuron or mesotrione. Among 115 F₁ progeny of this inbred crossed with Cr1, 47% were sensitive, and 30 of 59 F₁ progeny of this inbred crossed with field corn inbreds were sensitive to nicosulfuron.

FIELD CORN AND SWEET CORN INBRED CONTROLS. Among the control inbreds B90, B94, and Ia5125, 457 of 458 inbred plants, all 515 F₁ plants from crosses with Cr1, and 339 of 343 F₁ plants from crosses with GA209 were sensitive (Table 5). Only 2 of 292 F₁ plants from crosses with Cr2 were sensitive.

Discussion

Forty-five sweet corn hybrid cultivars and 29 sweet corn inbreds evaluated in this study and representing germplasm from 12 independent commercial breeding programs were sensitive to nicosulfuron and mesotrione as the result of a single recessive gene that occurs at the same chromosomal location as a gene conferring sensitivity in the sweet corn inbred Cr1. None of the cultivars or inbreds appeared to be sensitive to these herbicides as a result of another independent gene. A single gene or closely linked genes in the sweet corn inbred Cr1 condition sensitivity to nicosulfuron, mesotrione, and at least

seven other P450-metabolized herbicides and map to the same location on chromosome 5S as the *nsf1/ben1* gene, which is an insertion mutation of a CYP gene (Nordby et al., 2008; Pataky et al., 2006; Williams and Pataky, 2008; Williams et al., 2006). Nicosulfuron- and mesotrione-sensitive field corn inbreds GA209 and B90 have the same 392-bp insertion in the *Nsf1/Ben1* gene, whereas nicosulfuron- and mesotrione-sensitive inbreds Ia5125 and B94 do not have this insertion (Williams et al., 2006). Based on segregation of testcross progeny in this study, it appears that the gene(s) conditioning sensitivity in Cr1 and the other sweet corn inbreds and cultivars evaluated in this study may be the same insertion mutation as in GA209 and B90, a different mutation as occurs with Ia5125 and B94, or a different gene that is very closely linked to the *Nsf1/Ben1* locus.

In addition to the hybrid cultivars evaluated in this study, 49 hybrid cultivars were classified in a previous study as heterozygous or homozygous sensitive for responses to foramsulfuron, mesotrione, and nicosulfuron (Pataky et al., 2008). Of those cultivars, 16 were tested in the present study and were allelic with Cr1. Because every nicosulfuron- and mesotrione-sensitive sweet corn cultivar and inbred we have tested for allelism with Cr1 appears to have the same or closely linked genes conditioning sensitivity, it seems likely that many, if not all, of the other 33 cultivars from the previous study are allelic with Cr1. Thus, based on these two studies, it appears that at least 15% of nearly 500 sweet corn cultivars presently sold commercially in North America have a common genetic basis for sensitivity to P450-metabolized herbicides. These cultivars and inbreds include sugary, sugary enhancer, and shrunken-2 endosperm types from every major commercial breeding program. Cultivars in this group are grown commercially for processing and fresh consumption in nearly every market segment in North America and in many regions throughout the world.

Table 5. Number of plants tolerant and sensitive to nicosulfuron and mesotrione for corn inbreds B90, B94, and Ia5125 and F₁ hybrids from crosses of those inbreds with Cr1, GA209, and Cr2.

Inbred and herbicide	Plants with tolerant or sensitive responses in each generation (no.)							
	Inbred		Inbred × Cr1 ^z		Inbred × GA209 ^y		Inbred × Cr2 ^x	
	Tolerant	Sensitive	Tolerant	Sensitive	Tolerant	Sensitive	Tolerant	Sensitive
B90 ^w								
Nicosulfuron	0	89	0	85	0	75	34	0
Mesotrione	1	88	0	103	0	98	45	0
B94 ^v								
Nicosulfuron	0	54	0	62	0	58	13	0
Mesotrione	0	91	0	88	4	68	19	0
Ia5125 ^u								
Nicosulfuron	0	62	0	83	0	18	87	0
Mesotrione	0	73	0	94	0	22	92	2

^zCr1 is a nicosulfuron- and mesotrione-sensitive sweet corn inbred from Crookham (Caldwell, ID).

^yGA209 is a nicosulfuron- and mesotrione-sensitive field corn inbred from which the *ben1* gene (i.e., *nsfl* gene) was originally identified and which has a 392-bp insertion in the *Nsfl* gene sequence.

^xCr2 is a nicosulfuron- and mesotrione-tolerant sweet corn inbred from Crookham.

^wB90 is a nicosulfuron- and mesotrione-sensitive field corn inbred that has the same 392-bp insertion in the *Nsfl* gene sequence as GA209.

^vB94 is a nicosulfuron- and mesotrione-sensitive field corn inbred that does not have the same 392-bp insertion in the *Nsfl* gene sequence as GA209.

^uIa5125 is a nicosulfuron- and mesotrione-sensitive sweet corn inbred that does not have the same 392-bp insertion in the *Nsfl* gene sequence as GA209.

Despite the presence of a recessive allele(s) for herbicide sensitivity in prominent sweet corn germplasm, substantial injury may be relatively uncommon because many sweet corn cultivars are heterozygous for alleles conditioning herbicide response. Heterozygous cultivars are uninjured or suffer only minor injury under normal production practices. In this study and several previous studies investigating the inheritance of herbicide sensitivity (Bradshaw et al., 1994; Fleming et al., 1988; Green, 1998; Green and Ulrich, 1993; Kang, 1993; Pataky et al., 2006; Widstrom and Dowler, 1995), phenotypes were classified as tolerant or sensitive, and alleles conditioning tolerance were reported to be dominant to those conditioning sensitivity. In fact, alleles conditioning tolerance may be partially dominant, and under certain circumstances, plants heterozygous for alleles conditioning tolerance and sensitivity may have an intermediate response to these herbicides. In this study in 2006, intermediate responses occurred on heterozygous cultivars and on plants in segregating generations presumed to be heterozygous when a 1.5× rate of mesotrione was applied. In another study (Pataky et al., 2008), 47 sweet corn cultivars that were heterozygous for alleles conditioning herbicide response displayed intermediate responses to nicosulfuron, mesotrione, and foramsulfuron, and levels of injury appeared to be affected by environmental conditions as well as the genotype of the cultivar at the locus controlling herbicide response. Injury to heterozygous cultivars in the previous study was about 1.5 to 2.3 times greater than injury to homozygous tolerant cultivars, but was considerably less than injury to homozygous sensitive cultivars that often were severely injured or killed.

The results of Landi et al. (1989) and Volenberg et al. (2006) provide further evidence of intermediate responses of heterozygous hybrid cultivars. Landi et al. (1989) observed an intermediate response to chlorsulfuron, a different P450-metabolized sulfonylurea herbicide, in crosses of tolerant (T), Va85 and Mes44, by sensitive (S), B73 and B79, field corn inbreds compared with crosses of the two tolerant inbreds and

crosses of the two sensitive inbreds. Root length and shoot growth were affected by the genotype of the hybrid and dosage of chlorsulfuron applied. At low dosages (e.g., 0.25 ng·g⁻¹), root length of T × S and T × T hybrids was similar. At high dosages (e.g., 1 ng·g⁻¹), root length of T × S and S × S hybrids was similar. At intermediate dosages (e.g., 0.625 ng·g⁻¹), root length of T × S hybrids was intermediate to that of T × T and S × S hybrids. Although the chlorsulfuron-sensitive inbreds examined by Landi et al. (1989) were unaffected by nicosulfuron or mesotrione and did not appear to be allelic for the mutant CYP genes in GA209 or Cr1 (J.K. Pataky, unpublished data), we propose that a similar dose-response relationship may occur for hybrids that are homozygous tolerant, heterozygous, and homozygous sensitive to nicosulfuron and mesotrione due to the CYP gene(s) on chromosome 5S. In accordance with this hypothesis, results of a preliminary greenhouse study (Volenberg et al., 2006) demonstrate that following applications of mesotrione in dose-response studies similar to those of Landi et al. (1989), biomass of a sweet corn hybrid heterozygous for CYP alleles from Cr1 and Cr2 was intermediate to that of near-isogenic, homozygous tolerant, and homozygous sensitive hybrids.

Although the plant materials in this study were evaluated only for responses to mesotrione and nicosulfuron, the CYP gene(s) on chromosome 5S appear to affect metabolism of several other herbicides (Barrett et al., 1997; Green, 1998; Green and Ulrich, 1993; Nordby et al., 2008; Pataky et al., 2006; Williams et al., 2005; Williams and Pataky, 2008). Thus, sensitive inbreds and homozygous sensitive or heterozygous hybrid cultivars in this study may be affected by other P450-metabolized herbicides. Therefore, it seems pragmatic to determine more completely the range of herbicides metabolized by the CYP genes on chromosome 5S. Similarly, it may be useful to identify if other genes affect metabolism of nicosulfuron and mesotrione, such as genes in the two sweet corn inbreds that were stunted but not killed by nicosulfuron. In the past, we have observed different degrees of sensitive responses

to mesotrione (i.e., different levels of visual estimates of “bleaching” symptoms) among inbred lines selfed from the cross of Cr1 × Cr2, which indicates that other genes have modifying effects on responses conditioned by the CYP gene(s) on chromosome 5S (J.K. Pataky, unpublished data).

A number of different CYP genes occur in corn, of which only some are associated with herbicide metabolism (Frey et al., 1995, 1997; Persans et al., 2001). In addition to the cluster of four CYP genes identified by Williams et al. (2006) on chromosome 5S, another cluster of four CYP genes on chromosome 4S is related to production of DIMBOA (Frey et al., 1995, 1997). These genes, designated *Bx2* to *Bx5*, are closely related to the CYP71 family of CYP genes and encode four cytochrome P450 monooxygenases that are substrate specific. Persans et al. (2001) observed that CYP71C transcripts, encoding the third P450 in the DIMBOA biosynthetic pathway, were not induced in 2.5-d-old seedlings by the ALS-inhibiting, sulfonyleurea herbicide triasulfuron in combination with the herbicide safener naphthalic anhydride, but CYP71C3v2 transcripts, encoding the fourth P450 in DIMBOA synthesis, were additively induced in young seedlings by the combination of triasulfuron and naphthalic anhydride. Naphthalic anhydride and triasulfuron also induced expression of CYP genes on chromosomes 2 and 3. Thus, CYP genes affecting triasulfuron metabolism may be different from those affecting nicosulfuron and mesotrione metabolism. Also, the gene(s) affecting metabolism of chlorsulfuron appear to be different from the ones on chromosome 5S based on our observations of mesotrione and nicosulfuron tolerance among chlorsulfuron-sensitive field corn inbreds and their crosses with Cr1 and GA209 (J.K. Pataky, unpublished data). Additional research that associates groups of P450-metabolized herbicides with the CYP genes or other genes and detoxification enzymes governing their rates of metabolism will allow for more informed decisions concerning risks of crop injury from herbicides.

Injury or the potential for injury to sweet corn from postemergence applications of herbicides has been a concern of the sweet corn industry for nearly two decades. Using a group of sweet corn hybrid cultivars and inbreds that were subject to injury in previous trials, we observed a common genetic basis for cross-sensitivity to P450-metabolized herbicides that was present in germplasm used throughout the sweet corn industry. Confirmation of the presence of a gene(s) conditioning sensitivity to nicosulfuron and mesotrione, and probably to several other P450-metabolized herbicides, provides an explanation for varied levels of herbicide injury and inconsistent responses of sweet corn cultivars under differing environmental conditions. Hybrid cultivars heterozygous for CYP alleles in which rates of herbicide metabolism may be intermediate to that of homozygous tolerant and homozygous sensitive cultivars are more likely to display the greatest genotype × environment interactions with regard to herbicide responses. This information provides a rational basis from which herbicide sensitivity in sweet corn can be addressed. Alleles that render germplasm sensitive should be eliminated from breeding populations, inbreds, and commercial cultivars. Genotypes of cultivars at loci affecting herbicide metabolism also should be considered when establishing usage rates for new and existing herbicides. Alternatively, injury to heterozygous cultivars may be reduced if herbicides are formulated with safeners that enhance the detoxification activity of cytochrome P450 enzymes. Finally, applicators should inquire about the genetic

predisposition to injury of specific cultivars before using a P450-metabolized herbicide.

Literature Cited

- Barrett, M. 1995. Metabolism of herbicides by cytochrome P450 in corn. *Drug Metabol. Drug Interact.* 12:299–315.
- Barrett, M. 2000. The role of cytochrome P450 enzymes in herbicide metabolism, p. 25–37. In: A.H. Cobb and R.C. Kirkwoods (eds.). *Herbicides and their mechanisms of action*. CRC Press, Boca Raton, FL.
- Barrett, M., L.D. Bradshaw, N.D. Polge, R.J. Baerg, and C.G. Poneleit. 1994. Evidence for multiple herbicide metabolizing cytochrome P450 from maize. *Weed Sci. Soc. Amer. Abstr.* 34:60.
- Barrett, M., N. Polge, R. Baerg, R. Bradshaw, and C. Poneleit. 1997. Role of cytochrome P450 in herbicide metabolism and selectivity and multiple herbicide metabolizing cytochrome P450 activities in maize, p. 35–50. In: K. Hatzios (ed.). *Regulation of enzymatic systems detoxifying xenobiotics in plants*. Kluwer Academic Publishers, Dordrecht, The Netherlands.
- Bradshaw, L.D., M. Barrett, and C.G. Poneleit. 1994. Inheritance of bentazon susceptibility in a corn (*Zea mays*) line. *Weed Sci.* 42:641–647.
- Burton, J.D., E.P. Maness, D.W. Monks, and R.R. Robinson. 1994. Sulfonyleurea selectivity and safener activity in ‘Landmark’ and ‘Merit’ sweet corn. *Pestic. Biochem. Physiol.* 48:163–172.
- Diebold, S., D. Robinson, J. Zandstra, J. O’Sullivan, and P.H. Sikkema. 2003. Sweet corn (*Zea mays*) cultivar sensitivity to AE F130360. *Weed Technol.* 17:127–132.
- Diebold, S., D. Robinson, J. Zandstra, J. O’Sullivan, and P.H. Sikkema. 2004. Sweet corn sensitivity to bentazon. *Weed Technol.* 18:982–987.
- Fleming, A.A., P.A. Banks, and J.G. Legg. 1988. Differential responses of maize inbreds to bentazon and other herbicides. *Can. J. Plant Sci.* 68:501–507.
- Frey, M., P. Chomet, E. Glawischnig, C. Stettner, S. Grun, A. Winklmaier, W. Eisenreich, A. Bacher, R. Meeley, S.P. Briggs, K. Simcox, and A. Gierl. 1997. Analysis of a chemical plant defense mechanism in grasses. *Science* 277:696–699.
- Frey, M., R. Kliem, H. Saedler, and A. Gierl. 1995. Expression of a cytochrome P450 gene family in maize. *Mol. Gen. Genet.* 246:100–109.
- Green, J.M. 1998. Differential tolerance of corn (*Zea mays*) inbreds to four sulfonyleurea herbicides and bentazon. *Weed Technol.* 12:474–477.
- Green, J.M. and J.F. Ulrich. 1993. Response of corn (*Zea mays* L.) inbreds and hybrids to sulfonyleurea herbicides. *Weed Sci.* 41:508–516.
- Grey, T.L., D.C. Bridges, P. Raymer, D. Day, and D.S. NeSmith. 2000. Differential tolerance of fresh market sweet corn cultivars to the herbicides nicosulfuron and primisulfuron. *HortScience* 35:1070–1073.
- Harms, C.T., A.L. Montoya, L.S. Privalle, and R.W. Briggs. 1990. Genetic and biochemical characterization of corn inbred lines tolerant to the sulfonyleurea herbicide primisulfuron. *Theor. Appl. Genet.* 80:353–358.
- Kang, M.S. 1993. Inheritance of susceptibility to nicosulfuron herbicide in maize. *J. Hered.* 84:216–217.
- Landi, P., A. Vicari, and P. Catizone. 1989. Response of maize (*Zea mays* L.) inbred lines and hybrids to chlorsulfuron. *Weed Res.* 29:265–271.
- Monks, D.W., C.A. Mullins, and K.E. Johnson. 1992. Response of sweet corn (*Zea mays*) to nicosulfuron and primisulfuron. *Weed Technol.* 6:280–283.
- Morton, C.A. and R.G. Harvey. 1992. Sweet corn (*Zea mays*) hybrid tolerance to nicosulfuron. *Weed Technol.* 6:91–96.
- Nordby, J.N., M.M. Williams, J.K. Pataky, D.E. Riechers, and J.D. Lutz. 2008. A common genetic basis in sweet corn inbred Cr1 for cross sensitivity to multiple cytochrome P450-metabolized herbicides. *Weed Sci.* 56:376–382.

- O'Sullivan, J. and P.H. Sikkema. 2002. Sweet corn (*Zea mays*) cultivar tolerance to primisulfuron. *Can. J. Plant Sci.* 82:261–264.
- O'Sullivan, J. and W.J. Bouw. 1998. Sensitivity of processing sweet corn (*Zea mays*) cultivars to nicosulfuron/rimsulfuron. *Can. J. Plant Sci.* 78:151–154.
- O'Sullivan, J., J. Zandstra, and P. Sikkema. 2002. Sweet corn (*Zea mays*) cultivar sensitivity to mesotrione. *Weed Technol.* 16:421–425.
- O'Sullivan, J., P.H. Sikkema, and R.J. Thomas. 2000. Sweet corn (*Zea mays*) cultivar tolerance to nicosulfuron. *Can. J. Plant Sci.* 80:419–423.
- O'Sullivan, J., R.A. Brammall, and W.J. Bouw. 1995. Response of sweet corn (*Zea mays*) cultivars to nicosulfuron plus rimsulfuron. *Weed Technol.* 9:58–62.
- Pan, G., X. Zhang, K. Liu, J. Zhang, W. Wu, J. Zhu, and J. Tu. 2006. Map-based cloning of a novel rice cytochrome P450 gene CYP81A6 that confers resistance to two different classes of herbicides. *Plant Mol. Biol.* 61:933–943.
- Pataky, J.K., J.N. Nordby, M.M. Williams, II, and D.E. Riechers. 2006. Inheritance of cross-sensitivity in sweet corn to herbicides applied post emergence. *J. Amer. Soc. Hort. Sci.* 131:744–751.
- Pataky, J.K., M.E. Meyer, J.D. Bollman, C.M. Boerboom, and M.M. Williams, II. 2008. Genetic basis for varied levels of injury to sweet corn hybrids from three cytochrome P450-metabolized herbicides. *J. Amer. Soc. Hort. Sci.* 133:438–447.
- Persans, M.W., J. Wang, and M.A. Schuler. 2001. Characterization of maize cytochrome P450 monooxygenases induced in response to safeners and bacterial pathogens. *Plant Physiol.* 125:1126–1138.
- Robinson, D.K., D.W. Monks, and J.R. Schultheis. 1994. Effect of nicosulfuron applied postemergence and post-directed on sweet corn (*Zea mays*) tolerance. *Weed Technol.* 8:630–634.
- Robinson, D.K., D.W. Monks, J.R. Schultheis, and A.D. Worsham. 1993. Sweet corn (*Zea mays*) cultivar tolerance to application timing of nicosulfuron. *Weed Technol.* 7:840–843.
- Sikkema, S.R., N. Soltani, P.H. Sikkema, and D.R. Robinson. 2008. Response of sweet maize (*Zea mays* L.) hybrids to halosulfuron. *Crop Prot.* 27:695–699.
- Stall, W.M. and T.A. Bewick. 1992. Sweet corn cultivars respond differently to the herbicide nicosulfuron. *HortScience* 27:131–133.
- Volenberg, D.S., M.M. Williams, II, J.K. Pataky, and D.E. Riechers. 2006. Responses of tolerant and sensitive sweet corn inbreds and near isogenic hybrids to postemergence herbicides with different modes of action. *North Central Weed Sci. Soc. Abstr.* 61:213.
- Widstrom, N.W. and C.C. Dowler. 1995. Sensitivity of selected field corn inbreds (*Zea mays*) to nicosulfuron. *Weed Technol.* 9:779–782.
- Williams, M.M., II and J.K. Pataky. 2008. Genetic basis of sensitivity in sweet corn to tembotrione. *Weed Sci.* 56:364–370.
- Williams, M.M., II, J.K. Pataky, J.N. Nordby, D.E. Riechers, C.L. Sprague, and J.B. Masiunas. 2005. Cross-sensitivity in sweet corn to nicosulfuron and mesotrione applied postemergence. *HortScience* 40:1801–1805.
- Williams, M., S. Sowinski, T. Dam, and B.L. Li. 2006. Map-based cloning of the *nsf1* gene of maize. Program and Abstracts of the 48th Maize Genetics Conference, Pacific Grove, CA. (Abstr).