Further Evidence of a Genetic Basis for Varied Levels of Injury to Sweet Corn Hybrids from Cytochrome P450-metabolized Herbicides Applied Postemergence

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Abstract. **Over the last two decades, sweet corn injury from postemergence herbicides has resulted in routine screening of combinations of new and existing hybrids and herbicides. Sensitivity of sweet corn to several cytochrome P450-metabolized herbicides is simply inherited and has a common genetic basis, a single P450 locus that may account for a large amount of the variation in sweet corn injury commonly observed among screening trials. Using data from 13 hybrid-herbicide screening trials, the objective of this work was to determine the extent to which injury from P450-metabolized herbicides was associated with the genotypes of hybrids at a locus affecting herbicide sensitivity. Of the 703 hybrids evaluated in the University of Illinois sweet corn hybrid nurseries from 2002 to 2007, previous work showed that a total of 104, 70, and nine of the hybrids were known to be homozygous-tolerant, heterozygous, or homozygous-sensitive, respectively, for an allele affecting herbicide response. Nurseries from 2002 to 2007 included six trials with mesotrione, three trials with nicosulfuron, and one trial each with foramsulfuron, tembotrione, halosulfuron, and carfentrazone. When means of hybrids in genotypic classes were compared, homozygous-sensitive hybrids were consistently injured more severely than homozygous-tolerant and heterozygous hybrids. When environmental conditions favored crop injury, heterozygous hybrids had an intermediate response that was closer to homozygous-tolerant hybrids than homozygous-sensitive hybrids. These data are further evidence that the probability of injury from several P450-metabolized herbicides, including mesotrione, nicosulfuron, foramsulfuron, tembotrione, halosulfuron, and carfentrazone, is highest in homozygous-sensitive hybrids and least in homozygous-tolerant hybrids and that variability of responses among sweet corn hybrids to these P450-metabolized herbicides can be explained largely by the genotype of a hybrid at a single locus.**

Because certain sweet corn hybrids are sensitive to postemergence herbicides, new hybrids frequently are screened for responses to existing herbicides, and new herbicides are routinely tested for potential injury to existing hybrids (e.g., Diebold et al., 2003; Morton and Harvey, 1992; Stall and Bewick, 1992; Williams and Pataky, 2008). Although this type of screening identifies hybrids that may be injured from postemergence herbicides, a more comprehensive understanding of the genetic basis of herbicide sensitivity in sweet corn offers a mechanism by which this problem can be addressed and possibly solved by elimination of alleles that render sweet corn sensitive.

A sensitive or tolerant response of sweet corn to several cytochrome P450-metabolized herbicides is a simply inherited trait. Alleles at a single cytochrome P450 (CYP) locus (or a group of very closely linked loci) on the short arm of chromosome 5S condition response of corn to multiple, P450-metabolized herbicides, including: bentazon, carfentrazone, dicamba plus diflufenzopyr, foramsulfuron, imazethapyr, mesotrione, nicosulfuron, primisulfuron, rimsulfuron, and tembotrione (Barrett et al., 1997; Nordby et al., 2008; Pataky et al., 2006b; Williams and Pataky, 2008). A single gene conditioning sensitivity to nicosulfuron was identified from a field corn inbred W703a and designated as *nsf1* (Kang, 1993). A single gene conditioning cross-sensitivity to bentazon and nicosulfuron was identified in the field corn inbred GA209 and designated as *ben1* (Barrett et al., 1997; Bradshaw et al., 1994; Fleming et al., 1988). An allele at the Nsf1 locus on chromosome 5S from a nicosulfuron-tolerant field corn inbred, B73, contained the highly conserved heme-binding sequence FxxGxxCxG found in most cytochrome P450 genes (Williams et al., 2006). Alleles in the field corn inbreds, GA209 and W703a, contained identical 392 base pair insertions in this gene sequence relative to B73. Thus, it appears that this insertion mutation results in a nonfunctional P450 allele resulting in herbicide sensitivity and that the *nsf1* and *ben1* genes are the same mutant of a CYP gene.

Cross-sensitivity to multiple P450-metabolized herbicides also is simply inherited in the sweet corn inbred Cr1 (Nordby et al., 2008; Pataky et al., 2006b; Williams and Pataky, 2008). The allele (or closely linked alleles) in Cr1 that conditions the cross-sensitivity map to the same region of chromosome 5S as the Nsf1/Ben1 locus and progeny from crosses of Cr1 with GA209 and W703a have homogeneous responses to nicosulfuron and mesotrione (Nordby et al., 2008). Thus, cross-sensitivity in Cr1 appears to be the result of the same insertion mutation as *nsf1/ben1*, a similar mutation, or an allele at a very closely linked locus.

The common genetic basis for cross-sensitivity to several P450-metabolized herbicides is prevalent in sweet corn grown in nearly every market in North America and throughout the world. Forty-five sweet corn hybrids and 29 sweet corn inbreds were either homozygous or heterozygous for an allele that is the same as or very closely linked to alleles in Cr1 that condition cross-sensitivity (Pataky, unpublished data). This group of hybrids and inbreds represents sweet corn germplasm from 12 different commercial

breeding programs and includes sugary, sugary enhancer, and shrunken-2 endosperm mutants. Some of these hybrids were among a group of 149 sweet corn hybrids evaluated for responses to foramsulfuron, mesotrione, and nicosulfuron in 12 trials in six states (Bollman et al., 2005). Those 149 hybrids differed genotypically for the presence or absence of alleles affecting herbicide metabolism and phenotypically for their response to the three P450-metabolized herbicides (Pataky et al., 2008). When trial mean levels of injury were above minimal levels (e.g., 1% to 6% depending on the herbicide), response of the three genotypic classes of hybrids followed a consistent pattern. Homozygous-sensitive hybrids were killed or severely injured. Heterozygous hybrids had intermediate responses that were more similar to homozygous-tolerant than homozygous-sensitive hybrids; however, injury to heterozygous hybrids was 1.5 to 2.3 times greater than injury to homozygous-tolerant hybrids. In another trial, homozygous-sensitive hybrids were killed by tembotrione plus the safener isoxadifen-ethyl applied at a standard use rate, whereas homozygous-tolerant and heterozygous hybrids were uninjured (Williams and Pataky, 2008).

Since 2002, 249 to 379 sweet corn hybrids were evaluated annually for responses to postemergence herbicides as part of the University of Illinois sweet corn hybrid nurseries (Pataky et al., 2002, 2003, 2004, 2005, 2006a, 2007). Hybrid responses to mesotrione were evaluated in each year. Other herbicides evaluated in at least 1 year included carfentrazone, foramsulfuron, halosulfuron, nicosulfuron, and tembotrione. Herbicide evaluations were added to the UI sweet corn hybrid nurseries at a time when little was known about the inheritance of herbicide sensitivity in sweet corn. Although these data have been used to report individual hybrid responses, further analysis would provide an independent test of the genetic basis for varied levels of injury to sweet corn from P450-metabolized herbicides. The objective was to determine the extent to which injury observed in these nurseries was associated with the genotypes of hybrids at a locus conditioning herbicide sensitivity.

Materials and Methods

Hybrid nurseries. Sweet corn hybrids were obtained from every major seed company with breeding programs in North America and included widely grown hybrids and precommercial hybrids in the final stages of development (Pataky et al., 2002, 2003, 2004, 2005, 2006a, 2007). Field experiments were conducted from 2002 to 2007 at the University of Illinois Crop Sciences Research and Education Center near Urbana, IL, on Flanagan silt loam (fine, smectitic, mesic Aquic Argiudoll) soils. Fields were cultivated and fertilized using practices typical to the region and planted from 13 May to 31 May. Early-season weeds were controlled with a preemergence application of 2.2 kg·ha^{-1} atrazine plus 1.8 kg·ha^{-1} *S*-metolachlor.

A total of 13 experiments evaluating P450-metabolized herbicides were conducted from 2002 to 2007 testing six herbicides representing three herbicide modes of action (Table 1). Each experiment consisted of two to 10 replicates of 249 to 379 hybrids arranged in a randomized complete block design. An experimental unit was a single row with 12 to 18 plants. Commercial formulations of mesotrione, nicosulfuron, foramsulfuron, tembotrione, halosulfuron, and carfentrazone were applied at 1× or 2× the recommended use rates with standard adjuvants (Table 1). Herbicides were applied when sweet corn had four to six visible leaf collars, ranging from 14 June to 21 June, using backpack or tractor-mounted compressed-air sprayers delivering 130 to 187 L·ha^{-1} of water. Injury from herbicide application was observed primarily as chlorosis and bleaching (mesotrione and tembotrione), whole-plant stunting and necrosis (halosulfuron, foramsulfuron, and nicosulfuron), and leaf necrosis (carfentrazone). Hybrids were rated visually for percentage of injury once or twice at 7 to 21 d after treatment (DAT).

Genotypes of hybrids. Several of the hybrids evaluated in the sweet corn nurseries were previously assigned to genotypic classes at a locus conditioning herbicide sensitivity (Pataky et al., 2008; Pataky, unpublished data). Hybrids were classified as homozygous-tolerant, heterozygous, or homozygous-sensitive based on responses

of F$_2$ progeny and/or responses of progeny from testcrosses with Cr1 to foramsulfuron, mesotrione, and nicosulfuron. Plants sensitive to nicosulfuron or foramsulfuron usually were dead, whereas the apical meristem of plants sensitive to mesotrione was severely chlorotic or bleached. The number of sensitive and tolerant progeny was counted. Hybrids with all F$_2$ and/or testcross progeny severely injured were classified as sensitive and homozygous for an allele conditioning herbicide sensitivity. Hybrids with uninjured progeny were classified as tolerant and homozygous for an allele conditioning herbicide tolerance. Hybrids with sensitive and tolerant progeny were tested by χ^2 analysis for goodness of fit for segregation of tolerant:sensitive F$_2$ and testcross progeny in ratios that would be expected if a single dominant gene conditioned a tolerant phenotypic response (e.g., the *Nsf1* allele). If segregation of progeny fit the expected ratios ($P > 0.01$), hybrids were classified as heterozygous for alleles conditioning tolerance and sensitivity.

Comparison of responses among genotypes. Means of injury of genotypic classes within each experiment were calculated from responses of hybrids with known genotypes. Mean levels of injury were compared among homozygous-sensitive, heterozygous, and homozygous-tolerant hybrids by multiple comparison tests (Fisher's LSD$_{0.05}$) and also by *t* tests among all three combinations of genotypic classes. When injury was rated at both 7 and 21 DAT within an experiment, data were analyzed separately by sampling time. Because mesotrione was evaluated in the nursery each year, distributions of hybrid response to mesotrione for each of the three genotypic classes were derived for data from all 6 years. Genotypic means of hybrid responses to mesotrione were tested as described.

Results

Based on injury responses to cytochrome P450-metabolized herbicides, a total of 104, 70, and nine hybrids were classified as homozygous-tolerant, heterozygous, and homozygous-sensitive, respectively (Table 2). In any single year, 26 to 72

Table 1. Details of herbicide applications in sweet corn hybrid nurseries, 2002 to 2007.

Herbicide	Mode of action[z]	Yr	Rates and adjuvants[y]
Mesotrione	HPPD	2002	105 g·ha^{-1} a.i. + 1% COC + 2.5% UAN
		2003	105 g·ha^{-1} a.i. + 1% COC
		2004	105 g·ha^{-1} a.i. + 1% COC
		2005	210 g·ha^{-1} a.i. + 1% COC + 2.5% UAN
		2006	210 g·ha^{-1} a.i. + 1% COC + 2.5% UAN
		2007	105 g·ha^{-1} a.i. + 1% COC + 3.6% UAN
Nicosulfuron	ALS	2003	35 g·ha^{-1} a.i. + 0.25% NIS + 2.5% UAN
		2004	35 g·ha^{-1} a.i. + 0.25% NIS + 2.5% UAN
		2006	70 g·ha^{-1} a.i. + 1% COC + 2.5% UAN
Foramsulfuron	ALS	2004	37 g·ha^{-1} a.i. + 37 g isoxadifen-ethyl ha^{-1} + 1% MSO + 2.5% UAN
Tembotrione	HPPD	2007	184 g·ha^{-1} a.i. + 92 g isoxadifen-ethyl ha^{-1} + 1% COC + 2% UAN
Halosulfuron	ALS	2002	36 g·ha^{-1} a.i. + 0.25% NIS + 2.5% UAN
Carfentrazone	PPO	2002	9 g·ha^{-1} a.i. + 0.25% NIS

[z]HPPD = 4-hydroxyphenylpyruvate-dioxygenase inhibitor; ALS = acetolactate synthase inhibitor; PPO = protoporphyrinogen oxidase inhibitor.
[y]COC = crop oil concentrate; MSO = methylated seed oil; NIS = nonionic surfactant; UAN = 28% urea ammonium nitrate.

homozygous-tolerant hybrids, 24 to 45 heterozygous hybrids, and two to four homozygous-sensitive hybrids were included in hybrid nurseries. Several hybrids were evaluated in more than 1 year such that the total number of observations of homozygous-tolerant, heterozygous, and homozygous-sensitive hybrids was 285, 199, and 20, respectively.

Among homozygous-sensitive hybrids, mean levels of injury after application of mesotrione ranged from 10% in 2003% to 86% in 2007 (Table 3). The lowest levels of injury within 1 week of treatment were in 2003 and 2005. These 2 years were characterized by both abnormally low rainfall preceding herbicide application and exceptionally cool (2003) or hot (2005) air temperatures the month herbicides were applied (Table 4). When heterozygous hybrids were injured 6% or greater, heterozygous hybrids were injured more severely than homozygous-tolerant hybrids, and homozygous-sensitive hybrids were injured more than homozygous-tolerant or heterozygous hybrids. When data were pooled across years, mean injury from mesotrione application was different ($P < 0.001$) for each genotypic class, averaging 47%, 11%, and 1% among homozygous-sensitive, heterozygous, and homozygous-tolerant hybrids, respectively (Fig. 1). Ninety-eight percent of 285 observations of homozygous-tolerant hybrids were 10% or less injured, whereas 17 of 20 observations of homozygous-sensitive hybrids were greater than 10% injured. Response of heterozygous hybrids was intermediate, with 52% of 199 observations having 10% or less injury and 48% having greater than 10% injury.

Injury from nicosulfuron application ranged from 0% to 90% (Table 5). When heterozygous hybrids were injured, injury was more severe than on homozygous-tolerant hybrids. Homozygous-sensitive hybrids were injured more severely than homozygous-tolerant and heterozygous hybrids in all cases. Foramsulfuron application in 2004 resulted in the highest level of injury to homozygous-tolerant hybrids (16%). By 21 DAT with foramsulfuron, homozygous-sensitive hybrids were dead or nearly dead, whereas minimal injury (1% or less) to heterozygous and homozygous-tolerant hybrids was not significantly different.

Heterozygous and homozygous-tolerant hybrids were not injured by tembotrione; however, homozygous-sensitive hybrids were substantially injured (76%) (Table 6). Injury from halosulfuron and carfentrazone was among the lowest observed in the hybrid nurseries, and only the homozygous-sensitive hybrids were significantly different among the three genotypic classes.

Mean levels of injury to heterozygous hybrids never exceeded injury to homozygous-sensitive hybrids nor did mean levels of injury to homozygous-tolerant hybrids exceed injury to heterozygous hybrids. Injury to homozygous-sensitive hybrids was significantly greater than injury to homozygous-tolerant hybrids in all trials, representing six herbicides and three modes of action.

Table 2. Total number of hybrids in sweet corn hybrid nurseries and number of hybrids known to be homozygous or heterozygous for alleles conditioning tolerance or sensitivity to cytochrome P450-metabolized herbicides.

Year	N	Number of hybrids with known herbicide genotype		
		Tolerant	Heterozygous	Sensitive
2002	274	26	24	4
2003	286	30	29	4
2004	378	57	42	3
2005	327	58	33	2
2006	379	72	45	3
2007	249	42	26	4
Total observations		285	199	20
Total hybrids		104	70	9

Table 3. Mean levels of injury on hybrids known to be homozygous or heterozygous for alleles conditioning tolerance or sensitivity after application of mesotrione in the sweet corn hybrid nurseries.

	2002		2003	2004		2005	2006	2007
	7 DAT[z]	21 DAT[z]	7 DAT[z]	7 DAT[z]	21 DAT[z]	7 DAT[z]	7 DAT[z]	7 DAT[z]
Genotypic class				——— % ———				
Sensitive hybrids	46 a[y]	25 a	10 a	46 a	13 a	14 a	73 a	86 a
Heterozygous hybrids	16 b	9 b	8 b	17 b	1 b	1 b	13 b	14 b
Tolerant hybrids	2 c	1 c	<1 c	3 c	0 b	<1 b	1 c	2 c

[z]Percent injury (chlorosis) rated visually 7 or 21 d after treatment (DAT) with mesotrione.
[y]Means followed by the same letter are not significantly different as separated by Fisher's least significant difference$_{0.05}$.

Table 4. Departure from 30-year average precipitation and mean air temperature for the month of May and June in Urbana, IL[z].

	May		June	
	Temperature departure	Precipitation departure	Temperature departure	Precipitation departure
Year	------c ------	----- mm ----	------c ------	----- mm ----
2002	−2.2	+38	+1.2	−36
2003	−0.8	−31	−2.2	−30
2004	+1.6	−6	−1.0	−11
2005	−0.8	−97	+1.8	−45
2006	−0.4	−44	−0.2	−65
2007	+3.0	−81	+1.0	+37

[z]Based on data from a weather station within 1 km of experimental site (Illinois State Water Survey, Champaign, IL).
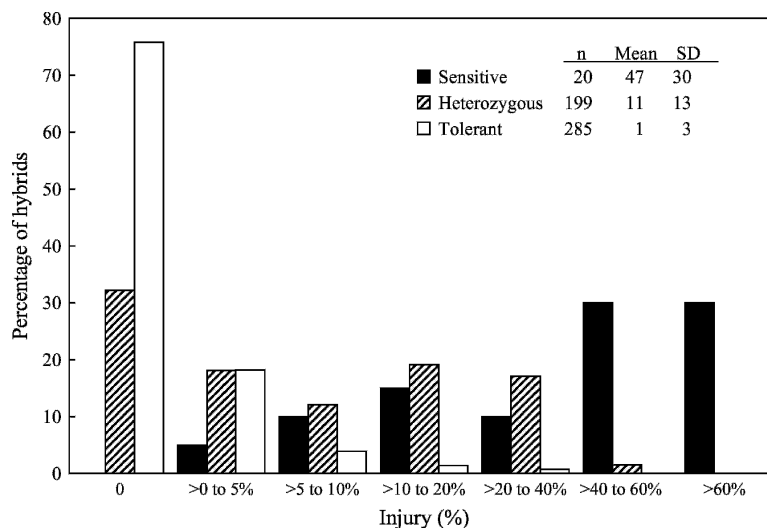

Fig. 1. Frequency distribution of levels of injury (chlorosis) from mesotrione observed in genotypes of hybrids at a locus conditioning herbicide sensitivity. Data from sweet corn hybrid nurseries from 2002 to 2007 and include 504 observations of 183 sweet corn hybrids.

Table 5. Mean levels of injury on hybrids known to be homozygous or heterozygous for alleles conditioning tolerance or sensitivity after application of nicosulfuron or foramsulfuron in sweet corn hybrid nurseries.

Genotypic class	Nicosulfuron					Foramsulfuron	
	2003		2004		2006	2004	
	7 DAT[z]	21 DAT[z]	7 DAT[z]	21 DAT[z]	14 DAT[z]	7 DAT[z]	21 DAT[z]
	--%---						
Sensitive hybrids	44 a	69 a	40 a	90 a	86 a	48 a	80 a
Heterozygous hybrids	10 b	10 b	21 b	6 b	0 b	24 b	1 b
Tolerant hybrids	3 c	<1 c	8 c	0 c	0 b	16 c	<1 b

[z]Percent injury (whole-plant stunting and necrosis) rated visually 7, 14, or 21 d after treatment (DAT) with herbicides.
[y]Means followed by the same letter are not significantly different as separated by Fisher's least significant difference$_{0.05}$.

Table 6. Mean levels of injury on hybrids known to be homozygous or heterozygous for alleles conditioning tolerance or sensitivity after application of tembotrione, carfentrazone, or halosulfuron in sweet corn hybrid nurseries.

Genotypic class	Tembotrione	Halosulfuron		Carfentrazone	
	2007	2002		2002	
	7 DAT[z]	7 DAT[z]	21 DAT[z]	7 DAT[z]	21 DAT[z]
	--%--				
Sensitive hybrids	76 a	4 a	2 a	4 a	2 a
Heterozygous hybrids	0 b	<1 b	<1 b	<1 b	<1 b
Tolerant hybrids	0 b	<1 b	<1 b	0 b	<1 b

[z]Percent injury (chlorosis, whole-plant stunting and necrosis, or leaf necrosis) rated visually 7 or 21 d after treatment (DAT) with herbicides.
[y]Means followed by the same letter are not significantly different as separated by Fisher's least significant difference$_{0.05}$.

Mean levels of injury to heterozygous hybrids was significantly greater than injury to homozygous-tolerant hybrids in 11 of 20 comparisons, the exception being when injury to heterozygous hybrids was 1% or less.

Discussion

Variation in levels of injury observed in sweet corn hybrid nurseries was associated with the genotypes of hybrids at a locus-conditioning herbicide sensitivity, presumably the Nsf1 locus. Homozygous-tolerant hybrids were injured the least. Homozygous-sensitive hybrids were injured the most. Injury to heterozygous hybrids was intermediate to the other two genotypic classes or similar to homozygous-tolerant hybrids. These results corroborate previous observations of variable responses of sweet corn hybrids to P450-metabolized herbicides. For example, Williams et al. (2005) observed an association between sweet corn responses to nicosulfuron and mesotrione now known to be the result of a common genetic basis (Nordby et al., 2008; Pataky et al., 2006b); however, the association was more evident among inbreds (homozygous for herbicide tolerance alleles) than hybrids (some of which were heterozygous). Williams et al. (2005) proposed that the lack of perfect association between hybrid responses to the two herbicides could have been the result of heterozygosity of genes affecting herbicide metabolism. In the present work, when heterozygous hybrids were injured (e.g., injury 6% or greater), the level of injury exceeded that of homozygous-tolerant hybrids. If genes for herbicide tolerance are not completely dominant, which appears to be the case (Volenberg et al., 2006; Williams and Pataky, unpublished data), then heterozygous hybrids could have a slower rate of metabolic inactivation compared with homozygous-tolerant hybrids, resulting in greater levels of injury and more variable responses under different conditions.

Severity of crop injury is the result of a combination of genotypic and other factors affecting herbicide action. For instance, variability of hybrid response to mesotrione among the six hybrid nurseries, particularly for homozygous-sensitive hybrids, was likely the result of an influence of environmental factors. In general, conditions favoring plant growth improve uptake and translocation of foliar-applied herbicides. Soil moisture deficit and air temperature before herbicide application were among the most influential environmental variables on graminicide efficacy in 59 field experiments (Medd et al., 2001). The lowest levels of injury from mesotrione application in the present study were observed in years with abnormally dry conditions and unusually warm or cool temperatures, suggesting the low level of injury may have been the result of poor cuticular penetration and/or translocation of the herbicide. Most evaluations of sweet corn tolerance to nicosulfuron have resulted in a continuous range of responses (Morton and Harvey, 1992; O'Sullivan and Bouw, 1998; Robinson et al., 1993). Williams et al. (2005)

hypothesized this range of responses could be the result of a combination of genetic and environmental factors affecting herbicide action. When homozygous-tolerant, heterozygous, and homozygous-sensitive sweet corn hybrids were evaluated for response to applications of nicosulfuron, mesotrione, and foramsulfuron in multiple locations, injury did not differ among genotypic classes when trial mean levels of injury were below 1% to 5% in trials located in dry climates, e.g., Idaho and Colorado (Pataky et al., 2008).

This study provides further evidence of a genetic basis for varied levels of injury to sweet corn hybrids from cytochrome P450-metabolized herbicides. Overlap in crop response exists among genotypic classes (Fig. 1), suggesting that other genes and/or environmental factors also influence crop injury. Nonetheless, the probability of injury from several P450-metabolized herbicides, including mesotrione, nicosulfuron, foramsulfuron, tembotrione, halosulfuron, and carfentrazone, is highest in homozygous-sensitive hybrids and least in homozygous-tolerant hybrids. Heterozygous hybrids appear to be most variable, having a response intermediate to the other two genotypic classes or similar to the homozygous-tolerant hybrids.

Enhancing cytochrome P450 activity may decrease the risk of crop injury in heterozygous hybrids. Two herbicides in these trials, foramsulfuron and tembotrione, were formulated with the safener isoxadifen-ethyl. By 21 DAT with foramsulfuron and 7 DAT with tembotrione, injury was 1% or less and similar between homozygous-tolerant and heterozygous hybrids (Tables 5 and 6). Williams and Pataky (2008) observed that four homozygous-sensitive hybrids were killed by application of tembotrione, but 22 heterozygous hybrids were not injured. They speculated that isoxadifen-ethyl reduced injury to the heterozygous hybrids by stimulating metabolism of the herbicide by cytochrome P450 enzymes, whereas the safener had little effect on homozygous-sensitive hybrids with two mutant CYP alleles. Bunting et al. (2004) found that isoxadifen-ethyl increased foramsulfuron absorption, translocation, metabolism, and crop tolerance of a hybrid injured by foramsulfuron alone. In dose–response studies, a heterozygous hybrid suffered up to 71% mean injury 7 DAT with tembotrione alone compared with only 9% mean injury when tembotrione was applied with isoxadifen-ethyl (Williams and Pataky, unpublished data). Thus, several new and emerging herbicide products formulated with P450-enhancing crop safeners such as isoxadifen-ethyl may reduce injury to heterozygous and homozygous-tolerant hybrids. Identifying sweet corn hybrids and inbred lines that carry mutant CYP alleles that condition herbicide sensitivity, and breeding with functional CYP genes into those hybrids and inbreds, also will reduce the frequency and severity of crop injury from several P450-metabolized herbicides.

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