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Factors Affecting Differential Sensitivity of Sweet Corn to HPPD-Inhibiting Herbicides

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Mutation of a cytochrome P450 (*CYP*) allele on the short arm of chromosome 5 affects sensitivity in sweet corn to mesotrione and to tembotrione plus isoxadifen applied POST. Hybrids that are homozygous for the functional allele (i.e., *CYPCYP*) are rarely injured at registered use rates, hybrids that are homozygous for mutant alleles (i.e., *cypcyp*) are frequently injured, and hybrids that are heterozygous for a functional and mutant allele (i.e., *CYPcyp*) have more variable responses over trials. The objectives of this work were (1) to conduct side-by-side comparisons of sweet corn hybrid responses to mesotrione, tembotrione plus isoxadifen, and topramezone under field conditions; and (2) to compare dose–response relationships among *CYPCYP*, *CYPcyp*, and *cypcyp* hybrids. Among 4-hydroxyphenylpyruvate dioxygenase (HPPD) inhibitors used POST in sweet corn, topramezone was safe on the 746 hybrids tested. When environmental conditions favored crop growth, mesotrione injured the largest number of hybrids, and these hybrids were almost exclusively *cypcyp* or *CYPcyp*. The safener isoxadifen added to the tembotrione product greatly reduced occurrence of injury to the *CYPcyp* genotypic class but not to the *cypcyp* hybrids. Despite a common genetic basis for herbicide metabolism, genotypic classes of sweet corn hybrids did not have identical field responses to mesotrione, tembotrione plus isoxadifen, and topramezone.

Nomenclature: Isoxadifen, ethyl-5, 5-diphenyl-2-isoxazoline-3-3-carboxylate; mesotrione; tembotrione; topramezone; sweet corn, *Zea mays* L.

Key words: Cross-sensitivity, cytochrome P450, dose–response, herbicide tolerance, *nsf1* gene.

In recent years, several new herbicides that inhibit 4-hydroxyphenylpyruvate dioxygenase (HPPD) have become available for POST broadleaf and grass weed control in sweet corn. Competitive inhibition of the HPPD enzyme, involved in carotenoid biosynthesis, ultimately leads to oxidative degradation of chlorophyll and photosynthetic membranes in growing shoot tissues. Chloroplast synthesis and function are compromised, and susceptible plants often have bleached leaves 1 wk after POST application. As of 2009, there were no reported biotypes of weed species resistant to HPPD-inhibiting herbicides (Heap 2009). In addition to contributing to the management of herbicide-resistant weed populations, the HPPD-inhibiting herbicides provide options for POST control of some grasses in sweet corn.

Three HPPD-inhibiting herbicides are currently registered for use in the United States for sweet corn production. Mesotrione, labeled in 2005, is a member of the benzoylcyclohexane-1,3-dione family of herbicides (Mitchell et al. 2001). Topramezone, labeled in 2006, is a member of the benzoyl pyrazole herbicide family (Grossman and Ehrhardt 2007). Tembotrione, labeled in 2008, is a member of the triketone herbicide family (USEPA 2007). The commercial formulation of tembotrione includes a safener, isoxadifen (ethyl-5, 5-diphenyl-2-isoxazoline-3-3-carboxylate), in a 2 : 1 ratio by weight. Differential tolerance among hybrids has been evaluated for HPPD-inhibiting herbicides individually (O'Sullivan et al. 2002; Soltani et al. 2007). Sweet corn hybrids have been tested for response to all three herbicides under common field conditions in only a single study (Bollman et al. 2008). Because only six to nine of the approximately 600 commercial sweet corn hybrids grown throughout North America were evaluated in any of those trials, a more complete assessment of the crop injury risks associated with use of these herbicides is warranted.

Cytochrome P450 (CYP) enzymes are a major family of monooxygenase enzymes responsible for metabolism of many herbicides, thus conferring selectivity in crops (Barrett 2000). Mutation of a *CYP* gene on the short arm of chromosome 5, referred to as the *nsf1* or *ben1* gene in previous research, conditions sensitivity to multiple P450-metabolized herbicides, including nicosulfuron (Williams et al. 2006), mesotrione (Nordby et al. 2008), and tembotrione plus isoxadifen (Williams and Pataky 2008). The functional allele at this CYP locus conditions responses to several other P450-metabolized herbicides with various modes of action (Nordby et al. 2008) and appears to be the "super P450" proposed by Barrett (Barrett et al. 1994). The mutant allele of this *CYP* gene (or closely linked *CYP* genes) was found in hybrids and inbreds from every major food processor and seed company in the sweet corn industry (Pataky et al. 2009), revealing an industry-wide occurrence of herbicide sensitivity in sweet corn.

Despite a common genetic basis of sensitivity to mesotrione and tembotrione plus isoxadifen, response to these two herbicides was not identical within genotypic classes of the *CYP* gene (Williams and Pataky 2008). Hybrids homozygous for functional alleles (i.e., *CYPCYP*) were uninjured or sustained minor injury from either herbicide. Hybrids homozygous for mutant alleles (i.e., *cypcyp*) were severely injured (> 50%) by both herbicides. Most hybrids heterozygous for a functional and a mutant allele (i.e., *CYPcyp*) were moderately (> 10 to 50%) injured by mesotrione but showed no injury from tembotrione plus isoxadifen. In other research, no hybrids were injured by topramezone, including *cypcyp* hybrids (i.e., 'DelMonte 20–38', 'Merit'; Bollman et al. 2008; Soltani et al. 2007). An understanding of the variable responses of hybrids with mutant *CYP* alleles to the HPPD-inhibiting herbicides and the effects of isoxadifen in relation to the genotypic class remain unresolved.

Advances in the knowledge of sweet corn sensitivity to herbicides have enabled sweet corn breeders to begin

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eliminating mutant *CYP* alleles from inbreds to avoid sensitivity among future hybrids; however, elimination of these alleles in commercial germplasm will require several generations of testing and selection. In the meantime, weeds remain a serious problem, and POST herbicides are used widely. Mesotrione, tembotrione plus isoxadifen, and topramezone are among the newest weed control tools available to sweet corn growers. Although weed control efficacy of these herbicides has received considerable attention, herbicide manufacturers, seed companies, extension personnel, and growers have little information upon which to base recommendations for hybrid tolerance to the above three herbicides.

Our goal was to use knowledge of the recently discovered genetic basis of sweet corn cross-sensitivity (Nordby et al. 2008; Pataky et al. 2008, 2009; Williams and Pataky 2008; Williams et al. 2008), in combination with a more comprehensive examination of sweet corn germplasm, to provide a greater understanding of the risks associated with using HPPD-inhibiting herbicides POST in sweet corn production. We wanted to test the hypothesis that differences in CYP genotypes is a factor in the degree of injury (i.e., leaf bleaching) to sweet corn hybrids treated with HPPD-inhibiting herbicides. Therefore, our objectives were (1) to conduct a side-by-side comparison of sweet corn hybrid response to mesotrione, tembotrione plus isoxadifen, and topramezone under field conditions; and (2) to quantify dose–response relationships among *CYPCYP*, *CYPcyp*, and *cypcyp* hybrids.

Materials and Methods

Plant Material. Every major seed company in North America with a sweet corn breeding program provided seed of hybrids[1] for this study. Germplasm included widely grown hybrids and precommercial hybrids in the final stages of development. All three major endosperm types (i.e., sugary, sugary enhancer, and shrunken-2), and hybrids grown for fresh consumption and processing markets, were included.

A total of 746 hybrids were evaluated in these field experiments, including 142 hybrids phenotypically characterized in previous studies for their genetic condition at the CYP locus associated with injury following applications of mesotrione and nicosulfuron. Based on responses of the F_2 progeny and/or progeny from testcrosses with Cr1, a mesotrione- and nicosulfuron-sensitive inbred, these 142 hybrids were classified as homozygous for a functional *CYP* allele, homozygous for a mutant *cyp* allele, or heterozygous (*CYPcyp*; Pataky et al. 2008, 2009). Not all 746 hybrids were available for both years of the trial.

Field Comparison. Field trials of 565 and 387 hybrids were evaluated in 2008 and 2009, respectively, for response to mesotrione, tembotrione plus isoxadifen, and topramezone. Separate, side-by-side field experiments were conducted at the University of Illinois Crop Sciences Research and Education Center near Urbana, IL, on Flanagan silt loam (fine, smectitic, mesic Aquic Argiudoll) soils averaging 3.8% organic matter and a pH of 5.8. Each experiment consisted of two (2009) and five (2008) replicates of the hybrids arranged in a randomized complete-block design. An experimental unit was a single 3.5-m row with 12 to 18 plants. Fields were fertilized with 202 kg N ha^{-1} at 2 to 3 wk before planting. Preplant tillage included two passes each of a field cultivator and spring-tine harrow. Early season weeds were controlled with a PRE application of atrazine at 2.2 kg ai ha^{-1} plus *S*-metolachlor at 1.8 kg ai ha^{-1}. Seeds were planted on May 29 during both years. Each herbicide was applied at 2× registered use rates using a tractor-mounted, compressed-air sprayer equipped with nozzles delivering 187 L ha^{-1} of water at 276 kPa when sweet corn had four to five visible leaf collars. Rates of the commercial formulation of herbicides were mesotrione[2] at 210 g ai ha^{-1}, tembotrione[3] at 184 g ai ha^{-1} (with 96 g isoxadifen ha^{-1}), and topramezone[4] at 36 g ai ha^{-1}. Adjuvants included 1% (v/v) crop oil concentrate (COC) in 2008 and 1% (v/v) COC plus 2% (v/v) urea ammonium nitrate (UAN, with 28% nitrogen) in 2009. Plants were evaluated visually 1 and 3 wk after treatment (WAT) for percentage of leaf bleaching. Mean responses of all hybrids to mesotrione, tembotrione plus isoxadifen, and topramezone were summarized, including comparisons of the hybrids with their genotype characterized at the *CYP* locus. Because not all hybrids were available both years, data are presented separately by year.

Dose–Response. Responses of each genotypic class (i.e., *CYPCYP*, *CYPcyp*, and *cypcyp*) were evaluated in dose–response studies in the greenhouse using near-isogenic hybrids. Near-isogenic hybrids were made from two pairs of inbred lines selected in the S_6 generation for functional (*CYP*) or mutant (*cyp*) alleles. In addition to mesotrione, tembotrione plus isoxadifen, and topramezone, a formulation of tembotrione without the safener was included. Each herbicide was a separate trial, and all trials were repeated. Ten seeds of a hybrid of each genotype were planted into 30 by 30 by 7-cm flats that contained a sterilized 1 : 1 : 1 mixture of soil, peat, and perlite. Trials consisted of a factorial arrangement of three genotypes and eight herbicide doses in a randomized complete-block design with four replications. Herbicide dose, the main plot factor, was assigned to individual flats, whereas genotype was the subplot factor. Mesotrione doses were 0, 26, 53, 105, 210, 420, 840, and 17,00 g ha^{-1}. Tembotrione-containing treatments were applied at doses of 0, 46, 92, 180, 370, 740, 1,500, and 2,900 g ha^{-1}. Topramezone doses were 0, 72, 140, 290, 580, 1,150, 2,300, and 4,600 g ha^{-1}. Adjuvants included 1% (v/v) COC plus 2% (v/v) UAN (28% nitrogen). Natural sunlight was supplemented with metal halide lamps for an intensity of 1,000 μmol m^{-2} s^{-1} at the plant surface for 14 h. Plants were watered as needed for active growth. The greenhouse was maintained at 24 ± 4 C during a 14-h day.

Herbicides were applied when plants had one to two visible leaf collars. Flats of plants were treated in an herbicide spray chamber equipped with a flat-fan nozzle that delivered 187 L ha^{-1} of spray solution at 262 kPa. Plants were evaluated visually 1 WAT as described above.

A logistic model was used to quantify plant response over a range of herbicide doses. Injury data were fitted to the logistic model:

$$y = C + \left\{ [D - C] \Big/ \left[1 + (x/I_{50})^{b} \right] \right\} \quad [1]$$

where y is the percentage of injury, x is the herbicide dose, C is the upper asymptote, D is the lower asymptote, I_{50} is the dose eliciting 50% injury, and b is the slope at the I_{50} dose.

Table 1. Response to mesotrione, tembotrione plus isoxadifen, and topramezone of sweet corn hybrids that differ in functional (*CYP*) and mutant (*cyp*) alleles that affect P450-metabolism of herbicides.[a]

Year	All hybrids and hybrids with known genotypic class[b]	n[c]	Mesotrione			Tembotrione + isoxadifen			Topramezone		
			≤ 5	> 5–30	> 30	≤ 5	> 5–30	> 30	≤ 5	> 5–30	> 30
			no. of hybrids								
2008	All hybrids	565	540	19	6	562	0	3	565	0	0
	cypcyp	5	0	1	4	3	0	2	5	0	0
	CYPcyp	65	59	6	0	65	0	0	65	0	0
	CYPCYP	72	72	0	0	72	0	0	72	0	0
2009	All hybrids	387	234	142	11	371	10	6	387	0	0
	cypcyp	5	0	0	5	0	1	4	5	0	0
	CYPcyp	42	8	34	0	41	1	0	42	0	0
	CYPCYP	32	27	5	0	32	0	0	32	0	0

[a] Response to 2× rates of mesotrione, tembotrione plus isoxadifen, and topramezone on the basis of mean plant response from injury ratings taken 1 wk after treatment in field trials in 2008 and 2009.

[b] Hybrids identified previously as homozygous for mutant alleles (*cypcyp*), heterozygous (*CYPcyp*), or homozygous (*CYPCYP*) for functional alleles that affect P450-metabolism of herbicides as based on segregation of F_2 progeny and testcross progeny after applications of mesotrione and nicosulfuron from Pataky et al. (2008, 2009).

[c] Total number of hybrids.

Nonlinear regression methods were used to fit injury response to herbicide dose for each genotype. Parameter estimates were determined using an iterative least-squares procedure (SYSTAT Software, Inc.[6]). The extra sum of squares principle for nonlinear regression analysis (Ratkowsky 1983) was employed to evaluate the similarity of parameter estimates among genotypes. Comparisons among genotypes were made by calculating a variance ratio of individual and pooled residual sums of squares and performing an *F* test described by Lindquist et al. (1996).

Results and Discussion

Field Comparison. At 1 WAT, the greatest number of hybrids were injured by mesotrione, and the fewest hybrids were injured by topramezone. In 2008, 25 hybrids had > 5% injury from mesotrione, compared with only 3 hybrids with > 5% injury from tembotrione plus isoxadifen (Table 1). In 2009, 153 hybrids had > 5% injury from mesotrione, compared with only 16 hybrids with > 5% injury from tembotrione plus isoxadifen. None of the 565 and 387 hybrids evaluated in 2008 and 2009, respectively, were injured by topramezone.

Relative to 2008, a greater number of hybrids injured in 2009 may be attributed largely to environmental conditions around the time of herbicide application. Rainfall in 2008 immediately after planting was excessive (e.g., 18.3 cm within 10 d of planting), followed by minimal rainfall events in the weeks before and after herbicide application (Figure 1). Plants had shallow roots and symptoms of moisture stress (e.g., leaf rolling, poor growth) were observed at the time of herbicide application in 2008. In contrast, the rainfall pattern was more evenly distributed in 2009 and no symptoms of moisture stress were observed at the time of the herbicide application. Low levels of injury from mesotrione in *cypcyp* and *CYPcyp* hybrids were observed previously in dry environmental conditions, whereas moderate levels of injury were otherwise common (Pataky et al. 2008; Williams et al. 2008). Development of epicuticular wax, believed to be the main barrier to absorption and subsequent translocation of foliar-applied herbicides, is influenced by several correlated factors, including temperature, light, humidity, and moisture stress (Hull et al. 1975). In giant foxtail (*Setaria faberi* Herr.), moisture stress increased epicuticular wax content and reduced absorption of fluazifop-P (Hatterman-Valenti et al. 2006). In the present study, the dry conditions and poor plant growth around the time of herbicide application in 2008 may have impeded cuticular penetration and subsequent translocation of the herbicides. In addition, use of UAN in 2009 may have enhanced crop response; UAN is reported to improve absorption and efficacy of some herbicides (Bunting et al. 2003; Young and Hart 1998) and to reduce crop tolerance to mesotrione (Anonymous 2003).

Responses of *CYPCYP* hybrids were fairly consistent across herbicides. These hybrids had ≤ 5% injury from mesotrione, tembotrione plus isoxadifen, and topramezone at 1 WAT, with the exception of five hybrids ('Miracle', 'Prime Plus', 'Protégé', 'Sentinel', and 'Snow White') that had 6 to 10% injury from mesotrione trials in 2009 (Table 1).

Responses of *cypcyp* and *CYPcyp* hybrids varied with herbicide. Of the 65 and 42 *CYPcyp* hybrids tested in 2008

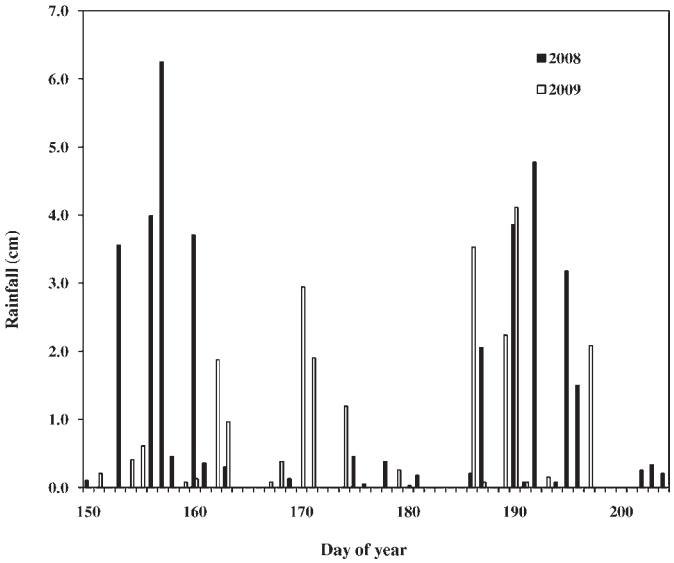

Figure 1. Daily rainfall at University of Illinois Crop Sciences Research and Education Center near Urbana, IL, in 2008 and 2009. Trials were planted on the day of year (DOY) 151 and 150 in 2008 and 2009, respectively. Trials were sprayed with mesotrione, tembotrione plus isoxadifen, or topramezone on DOY 179 and 182 in 2008 and 2009, respectively.

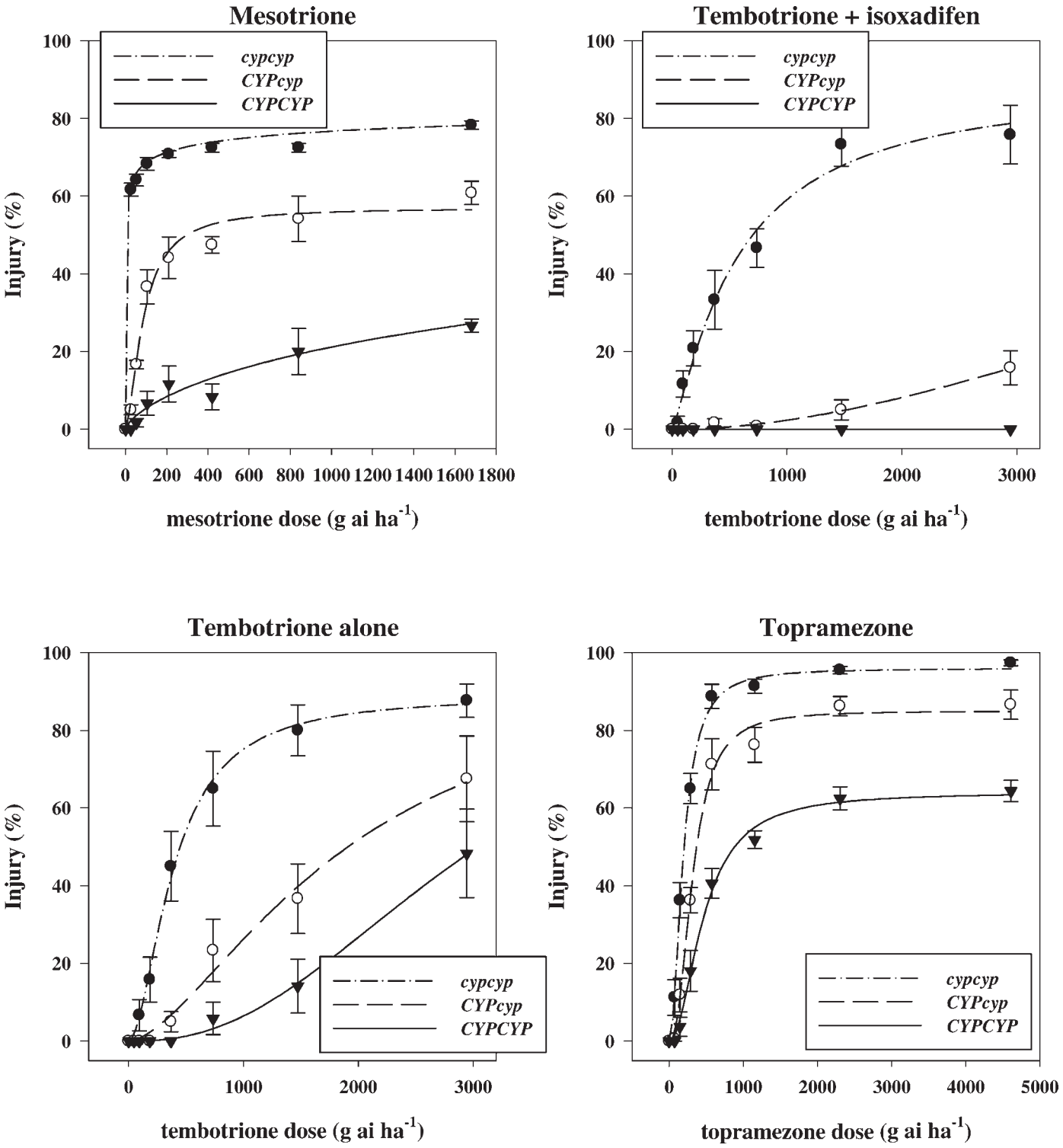

Figure 2. Dose–response relationships in greenhouse trials for hybrids that differ in functional (*CYP*) and mutant (*cyp*) alleles that condition tolerance to mesotrione and tembotrione one wk after treatment. Model predictions, mean plant responses, standard errors are shown. Parameter estimates and comparisons of model predictions are reported in Table 2.

and 2009, respectively, ≤ 8% injury was observed in response to tembotrione plus isoxadifen or to topramezone at 1 WAT. In contrast, as many as 81% of the *CYPcyp* hybrids had injury levels of > 5 to 30% in mesotrione trials (Table 1). Of the 5 *cypcyp* hybrids tested, most were injured 22% or more at 1 WAT with mesotrione or tembotrione plus isoxadifen. One exception was the tembotrione plus isoxadifen trials in 2008, where 3 *cypcyp* hybrids ('177A', 'HMX 6386 S', and 'XTH 3175') had ≤ 5% injury (Table 1). These three hybrids were moderately injured by tembotrione plus isoxadifen in earlier research (Williams and Pataky 2008), and the reason for a lack of injury in 2008 is unknown. In a subsequent greenhouse trial, plants grown from the same seed used in 2008 field trials were severely injured (≥ 38%) by the 2× rate of tembotrione plus isoxadifen (M. M. Williams and J. K. Pataky, unpublished data). Nevertheless, the highest levels of injury

Table 2. Logistic model parameter estimates (standard errors in parentheses) for the effects of herbicide dose on percentage of injury for sweet corn hybrids that differ in functional (*CYP*) and mutant (*cyp*) alleles that affect P450-metabolism of herbicides.[a]

Herbicide	Genotypic class[b]	*D*	*C*	I_{50}	*b*	Corrected R^2	ESSP[c]
		(SE)		g ai ha^{-1} (SE)			
Mesotrione	*cypcyp*	0.0 (1.2)	96 (29)	1 (2)	0.2 (0.2)	0.98	a
	CYPcyp	0.0 (3.4)	57 (3)	87 (14)	1.6 (0.3)	0.87	b
	CYPCYP	0.0 (2.9)	77 (205)	> 1,700	0.7 (0.6)	0.58	c
Tembotrione + isoxadifen	*cypcyp*	0.0 (4.4)	90 (14)	580 (200)	1.2 (0.3)	0.85	a
	CYPcyp	0.0 (0.9)	100 (1505)	> 2,900	1.9 (2.8)	0.60	b
	CYPCYP[d]	—	—	—	—	—	—
Tembotrione	*cypcyp*	0.0 (4.3)	89 (7)	390 (60)	1.8 (0.5)	0.87	a
	CYPcyp	0.0 (3.5)	100 (59)	1,900 (1,600)	1.6 (0.8)	0.75	b
	CYPCYP	0.0 (2.3)	100 (268)	> 2,900	2.4 (2.8)	0.67	c
Topramezone	*cypcyp*	0.0 (2.8)	96 (2)	190 (10)	2.0 (0.2)	0.96	a
	CYPcyp	0.0 (2.9)	85 (3)	320 (20)	2.4 (0.4)	0.92	b
	CYPCYP	0.0 (2.2)	64 (3)	470 (40)	2.0 (0.3)	0.91	c

[a] *C* is the upper asymptote, *D* is the lower asymptote, I_{50} is the dose eliciting 50% injury, and *b* is the slope at the I_{50} dose.
[b] Hybrids identified previously as homozygous for mutant alleles (*cypcyp*), heterozygous (*CYPcyp*), or homozygous (*CYPCYP*) for functional alleles that affect P450-metabolism of herbicides as based on segregation of F_2 progeny and testcross progeny after applications of mesotrione and nicosulfuron from Pataky et al. (2008, 2009).
[c] The extra sum of squares principal for nonlinear regression (ESSP) reports comparisons of dose-response curves among genotypic classes. Within each herbicide trial, response of genotypic classes differ ($P < 0.05$) when followed by a unique letter.
[d] There was no effect of tembotrione plus isoxadifen on *CYPCYP*.

among genotypic classes were observed in *cypcyp* hybrids treated with mesotrione and tembotrione plus isoxadifen, yet those hybrids were not injured by topramezone.

The *cypcyp* hybrids were slow to recover from tembotrione plus isoxadifen injury. Severity of injury generally decreased by 3 WAT in all other combinations of genotypic classes of hybrids and herbicide treatments (data not shown).

Dose–Response. Dose–response of sweet corn to mesotrione, tembotrione plus isoxadifen, tembotrione, and topramezone was influenced by genotypic class. In all trials, the *cypcyp* hybrid was injured the most across a range of herbicide doses (Figure 2). The hybrid that was injured the least had the *CYPCYP* genotype. No injury was observed on the *CYPCYP* hybrid in tembotrione plus isoxadifen trials at doses up to 2,900 g ai ha^{-1}.

The heterozygous hybrid exhibited levels of injury that were intermediate to responses of *cypcyp* and *CYPCYP* hybrids (Figure 2). At low doses, the *CYPcyp* hybrid was generally not injured and had a phenotype similar to the *CYPCYP* hybrid, thus suggesting dominant gene action. At higher doses, the *CYPcyp* hybrid had an intermediate phenotype that might be interpreted as a partial dominant or codominant response. These results suggest herbicide metabolism in *CYPcyp* hybrids may be more rapid than in *cypcyp* hybrids but slower than in *CYPCYP* hybrids. This work is consistent with Pataky et al. (2008), where mesotrione was reported to be two times more injurious to *CYPcyp* hybrids than to *CYPCYP* hybrids in 12 trials conducted in six states throughout the continental United States.

Isoxadifen improved crop tolerance to tembotrione in *CYPcyp* and *CYPCYP* hybrids but not in the *cypcyp* hybrids. When the formulation of tembotrione included isoxadifen, the *CYPCYP* hybrid was not injured at doses well beyond the registered dose, and the I_{50} dose for the *CYPcyp* hybrid was > 2,900 g ai ha^{-1} (Table 2). In contrast, when tembotrione was applied alone, the *CYPcyp* hybrid had greater injury (e.g., I_{50} = 1,900 g ai ha^{-1}), and the phenotype of the *CYPcyp* hybrid was intermediate to *cypcyp* and *CYPCYP* hybrids (Figure 2). Previously, addition of isoxadifen increased the rate of tembotrione metabolism in two corn cultivars (Schulte and Köcher 2009); however, the genotypic class of *CYP* alleles of these cultivars was not reported. Our results suggest *CYPcyp* hybrids are less likely to be injured from tembotrione plus isoxadifen than by mesotrione because isoxadifen stimulates herbicide metabolism. However, tembotrione plus isoxadifen appears to provide little or no benefit to *cypcyp* hybrids with respect to herbicide tolerance, perhaps because plants with two mutant *cyp* alleles are unable to proceed with normal P450 metabolism of the herbicide.

Relative to *CYPCYP* hybrids in general, *CYPcyp* hybrids are more predisposed to injury from these HPPD-inhibiting herbicides applied POST. Regarding commercial field situations, our work shows risk of injury is greater from mesotrione than from tembotrione plus isoxadifen because isoxadifen enhances tolerance of *CYPcyp* and *CYPCYP* hybrids to tembotrione. However, *cypcyp* hybrids, which are most sensitive to mesotrione, also can be severely sensitive to tembotrione plus isoxadifen.

In field trials, topramezone appeared safe across all genotypic classes (and all 746 hybrids tested). At higher rates of topramezone used in the dose–response studies, *CYPCYP*, *CYPcyp*, and *cypcyp* hybrids displayed the same order of response to topramezone as mesotrione and tembotrione, suggesting that, in spite of field tolerance of hybrids to topramezone, the same *CYP* locus may be responsible for metabolism of this P450-metabolized herbicide.

When environmental conditions limited active crop growth (thereby reducing herbicide uptake and activity), few *CYPcyp* hybrids were injured. The same hybrids were injured to a greater extent when conditions for plant growth were favorable. This observation is consistent with responses of *CYPcyp* hybrids to mesotrione and other P450-metabolized herbicides in multistate and multiyear trials (Pataky et al. 2008, Williams et al. 2008). Over time, as plant breeders replace mutant *cyp* alleles with functional alleles in commercial hybrids, the occurrence of crop injury will decline. In the meantime, knowledge of the genetic predisposition of a hybrid to injury should be considered, particularly when mesotrione and tembotrione are applied POST.

Sources of Materials

[1] Abbot & Cobb, Inc., Feasterville, PA; Centest Inc., Harvard, IL; Crookham Company, Caldwell, ID; Del Monte USA, Rochelle, IL; General Mills Green Giant Agricultural Research, LeSueur, MN; Harris Moran Seed Company, Modesto, CA; Illinois Foundation Seeds Inc., Champaign, IL; Mesa Maize, Olathe, CO; Syngenta Seeds Inc./Rogers Brands, Boise, ID; Sakata Seed USA, Morgan Hills, CA; Seminis Inc., Oxnard, CA; and Snowy River Seeds, Orbost, Australia.

[2] Callisto™ herbicide, Syngenta, Greensboro, NC.

[3] Laudis™ herbicide, Bayer CropScience, Research Triangle Park, NC.

[4] Impact™ herbicide, AMVAC, Los Angeles, CA.

[5] Tembotrione herbicide, Bayer CropScience, Research Triangle Park, NC.

[6] SYSTAT Software, Inc. SYSTAT 11.0. Richmond, CA.

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