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INFLUENCE OF ANTIDOTES, MFO INHIBITORS, INSECTICIDES,
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INFLUENCE OF ANTIDOTES, MFO INHIBITORS, INSECTICIDES, AND CORN HYBRIDS ON ALS-INHIBITING HERBICIDE ACTIVITY

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ABSTRACT

INFLUENCE OF ANTIDOTES, MFO INHIBITORS, INSECTICIDES, AND CORN HYBRIDS ON SULFONYLUREA HERBICIDE ACTIVITY

by

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The effects of antidotes, mixed function oxidase (MFO) inhibitors, insecticides, and corn hybrids on acetolactate synthase (ALS) inhibiting herbicide activity were evaluated in greenhouse and field studies. Cross-resistance of chlorsulfuron [2-chloro-N-[[(4-methoxy-6-1,3,5-triazin-2-yl) amino] carbonyl] benzenesulfonamide]-resistant kochia (Kochia scoparia (L.) Schrad.) was evaluated in greenhouse.

Normal corn hybrids were sensitive to the interaction of sulfonylurea herbicides with terbufos [S-[[(1,1-dimethylethyl) thio] methyl] O,O-diethyl phosphorodithioate]. Pioneer 3377 IR and Ciba 4393 RSC corn hybrids showed excellent tolerance to the interaction of ALS-inhibiting herbicides with terbufos regardless of the presence of tank-mixed piperonyl butoxide (PBO) [α-(2-(2-butoxyethoxy) ethoxy)-4,5-methyl enedixoy-2-propyltoluene], a mixed function oxidase inhibitor. ICI 8532 IT showed cross-resistance to the interaction of thifensulfuron [3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl]

amino] sulfonyl]-2-thiophenecarboxylic acid] and imidazolinone herbicides with terbufos. The antidotes, CGA-154281 [4-(dichloro-acetyl)-3,4-dihydro-3-methyl-2H-1,4-benzoxazine] and NA [1,8-naphthalic anhydride], reduced corn injury from metolachlor [2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl) acetamide], nicosulfuron [2-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-N,N-dimethyl-3-pyridinecarboxamide] and primisulfuron [2-[[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl] amino] carbonyl] amino] sulfonyl] benzoic acid] with terbufos treatments, respectively. The combination treatment of primisulfuron and terbufos did not affect herbicidal activity to weed species.

PBO enhanced nicosulfuron activity on barnyardgrass (Echinochloa crus-galli (L.) Beauv.), velvetleaf (Abutilon theophrasti Medicus), and common lambsquarters (Chenopodium album L.), thifensulfuron activity on velvetleaf and common lambsquarters. Also, butylate hydroxyanisole (BHA) [2,[3]-tert-butyl-4-hydroxyanisole] and PBO enhanced nicosulfuron and primisulfuron activities on common lambsquarters and green foxtail (Setaria viridis (L.) Beauv.).

The enhancement of herbicide activity by 28% UAN (urea ammonium nitrate) was herbicide, adjuvant, and weed specific. Efficacy of the nonionic adjuvants was herbicide and weed specific.

The combination of thifensulfuron with PBO caused injury to Elgin '87 soybean (Glycine max (L.) Merr.), but the W20-STS soybean was tolerant to this combination treatment. Combination of imazethapyr with PBO or BHA had no effect on the growth of Elgin '87 soybean.

The chlorsulfuron resistant-kochia biotype was resistant to six herbicides: Triflusulfuron, 2-[[[[[4-(dimethylamino)-6-(2,2,2-trifluoroethoxy)-1,3,5-triazin-2-yl] amino] carbanyl]-amino] sulfonyl]-3-methylbenzoic acid, thifensulfuron, MON 12037, [methyl 3-chloro-5-(4,6-dimethoxypyrimidin-2-ylcarbamoylsulfamoyl)-1-methylpyrazole-4-carboxylate], imazamethabenz [(±)-2-[4,5-dihydro-4-methyl]-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-4(and 5)-methylbenzoic acid(3:2)], chlorsulfuron, and nicosulfuron. But, the resistant kochia biotype showed sensitivity similar to the susceptible biotype to three herbicides: metsulfuron [2-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] amino] sulfonyl] benzoic acid], imazethapyr [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylicacid], andimazaquin[2-[4,5-dihydro-4-methyl-4-(1-methylethyl-5-oxo-1H-imidazol-2-yl]-3-quinolinecarboxylic acid]. Addition of the PBO at 2 kg/ha to primisulfuron and thifensulfuron increased injury and reduced plant height of the chlorsulfuron resistant kochia biotype.

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INTRODUCTION

The sulfonylurea and imidazolinone herbicides have high specific activity and have been in commercial use since 1982. Sulfonylurea herbicides reduced the field application rates of herbicide to less than 50 g a.i./ha. Sulfonylurea and imidazolinone herbicides are used to control a broad spectrum of weed species in a variety of crops including corn (Zea mays L.), soybeans (Glycine max (L.) Merr.), and small grains. The selective action of these herbicides between crop and weed plants can be attributed to metabolism of the herbicides to inactive products in the various crop species. The mode of action of these compounds has been well established as the inhibition of acetolactate synthase (ALS), one of the enzymes important for the synthesis of branched amino acids. Sulfonylurea herbicides are initially metabolized by a cytochrome P450-dependent monooxygenase system located in the microsomal fraction. This enzyme system supports the hydroxylation of primisulfuron [2-[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl] amino] carbonyl] amino] sulfonyl] benzoic acid] and nicosulfuron [2-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-N,N-dimethyl-3-pyridinecarboxamide] at the phenyl ring and at the pyrimidine ring.

The recent introduction of the ALS inhibiting-herbicides have stimulated research on the interactions of herbicides with insecticides. Combinations of the organophosphate insecticides, especially terbufos, and primisulfuron or nicosulfuron were shown to interact synergistically resulting in corn foliar and root injury, plant stand reduction, and corn grain yield losses. The synergistic interaction was explained on the basis that the insecticide reduced metabolism and increased absorption of herbicides.

Several researchers have tried to find methods to protect crops from the interaction of ALS-inhibiting herbicides and corn insecticides. They found that antidotes or tank-mixing 2,4-D with sulfonylurea herbicides reduced corn injury.

Mixed function oxidase (MFO) inhibitors, or antioxidants, are possible herbicide synergist. Piperonyl butoxide [α-(2-(2-butoxyethoxy) ethoxy)-4,5-methyl enedioxy-2-propyltoluene] increased the activity of EPTC [S-ethyl dipropyl carbamothioate], atrazine [6-chloro-N-ethyl-N'(1-methylethyl)-1,3,5-triazine-2,4-diamine], bentazon [3-(1-methylethyl)-(1H)-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide] and oxadiazon [3-(2,4-dichloro-5-(1-methylethoxy) phenyl)-5-(1,1-dimethylethyl)-1,3,4-oxadiazol-2-(3H)-one] to corn. MFO inhibitors inhibit mixed-function oxidase systems which are possibly involved in the metabolism of ALS-inhibiting herbicides in corn.

The objectives of this study were 1) to identify interactions between corn herbicides and corn insecticides, 2) to evaluate the effect of MFO inhibitors on the activity of several sulfonylurea herbicides, 3) to identify effective adjuvants for the sulfonylurea herbicides, 4) to determine the effect of the MFO inhibitors on corn safety, 5) to evaluate the cross-resistance of a chlorsulfuron resistant kochia (Kochia scoparia (L.) Schrad.) biotype to various ALS-inhibiting herbicides, and 6) to evaluate potential synergistic effects of PBO with sulfonylurea herbicides to a chlorsulfuron-resistant kochia biotype.

CHAPTER 1

LITERATURE REVIEW

INTRODUCTION

The sulfonylurea herbicides are highly active and have been in commercial use since 1982. Among various classes of herbicides used today, sulfonylureas rank at the top in their specific activity. Sulfonylurea herbicides were discovered by Levitt in 1976 (1,2). Sulfonylurea herbicides reduced the field application rates from 0.5 kg a.i./ha or even greater to less than 50 g a.i./ha. Structure of a typical sulfonylurea is characterized by presence of a sulfonylurea "bridge" connecting two rings.

The second class of acetolactate synthase (ALS)-inhibiting herbicides are the imidazolinones. This class of chemistry is characterized by an imidazolinone ring bonded to an aromatic ring at the 2 position.

Sulfonylurea and imidazolinone herbicides are used to effectively control a broad spectrum of weed species in a variety of crops including corn (Zea mays L.), soybeans (Glycine max (L.) Merr.), and small grains. The selective action of these herbicides between crop and weed plants can be attributed to metabolism of the herbicides to inactive products in the various crop species.

Modes of Action of Sulfonylurea and Imidazolinone Herbicides

For the first time in the history of commercial herbicides, the mode of action of the new herbicide was known before the herbicides were widely commercialized. The new classes of herbicide chemistry, sulfonylureas and imidazolinones, were commercialized by separate companies.

Two lines of investigation came together to prove that acetolactate synthase (ALS) is the site of action of sulfonylurea herbicides. Experiments by LaRossa and Schloss (67) showed an inhibiting effect of sulfonylureas on growth of bacteria, concurrently, herbicide-resistant mutants of tobacco (Nicotiana tabacum L.) were selected in culture by Chaleff and Ray (19). Other researchers performed biochemical studies showing ALS inhibition by sulfonylureas in susceptible plants but not in selected resistant plants (18). Ultimate proof for this site of action came from studies on regenerated herbicide resistant tobacco, in which breeding experiments showed cosegregation of the herbicide resistance trait and herbicide insensitive enzyme (18,19). Anderson and Shaner (4,108) performed key experiments that lead to discovery of the mode of action of imidazolinones. Anderson and Hibberd (4) found a decline in levels of valine, leucine, and isoleucine in corn cell cultures treated with imazapyr [(±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-pyridinecarboxylic acid]. Supplementation of these amino acids reversed the growth inhibiting effects of imazapyr. Similar experiments were performed by Shaner (108) using corn root tips.

Recently, it has been discovered that ALS is the target of several structurally diverse herbicide compounds such as sulfonylureas, imidazolinones, and triazolpyrimidine sulfonanilides (63,67,76,94,108,113). ALS plays an important role in controlling carbon flow to the amino acids by feedback regulation (73). The mode of action of these compounds has been well established as the inhibition of acetolactate synthase (ALS), one of the enzymes important for the synthesis of branched amino acids. ALS (also known as acetohydroxy acid synthase, AHAS) is the first enzyme in the biosynthesis of Val, Leu, and Ile. This enzyme catalyzes the condensation of an acetoaldehyde moiety derived from pyruvate either with another molecular pyruvate to form 2-acetolactate or with 2-ketobutyrate to form 2-aceto-2-hydroxybutyrate. With microorganisms the enzyme and the inhibitory mode of action have been well studied. In Escherichia coli and Salmonella typhimurium, three isozymes (ALS I, II, and III), each of them encoded by a particular gene, differing in substrate preference and feedback regulation have been identified, purified and well characterized (32,104). Most of the enzymological and kinetic studies on inhibition of ALS by herbicides have been carried out with the ALS II from S. typhimurium, which resembles the plant enzyme with regard to its sensitivity to herbicides, but is different in subunit composition and feedback regulation (94,105). Duner et al. (30) reported that only partial agreement between plant ALS and bacterial ALS II is evident, while both the time-dependent inhibition and the slow disassociation of the enzyme-inhibitor complex are in accordance, there are decisive differences with regard to reversal of inhibition, i.e. recovery of enzyme activity. LaRossa and Smulski (66) using enzymological analysis found that ALS I activity derived from S. typhimurium and E. coli species was resistant to the herbicide. Their report demonstrated that the ALS I was insensitive to sulfometuron methyl [2-[[[[(4,6-dimethyl-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl] benzoic acid]. In contrast, activity of S. typhimurim ALS II and E. coli ALS III was inhibited by sulfometuron methyl (66). An interpretation is that sulfometuron methyl inhibits S tryhimurium ALS II but not ALS I (67). If this interpretation is correct, growth inhibition is only manifested when ALS I activity is blocked or absent. ALS I may simply represent an isozyme resistant to the herbicide.

Apparently, there are different physical and catalytic properties of plant and bacterial ALS, emphasizing the need for further studies on the plant enzyme (30). ALS is subject to feedback inhibition by the end products Val and Leu in both micro-organisms and in plants (39,74,118). Obviously, the branched-chain amino acid pools eventually decrease after addition of ALS inhibitor herbicides to plants (4). Each herbicide also rapidly increases the intracellular α-ketobutyrate concentration causing metabolic imbalances. LaRossa et al. (68) proposed that these α-ketobutylate-mediated imbalances contribute to the potency of herbicides interacting with ALS. Durner et al. (29) tried experiments to show whether active fractions of barley (Hordeum vulgare L.) ALS represented true isozymes or multiple polymeric forms of a basic ALS subunit. Their data showed that the two enzymically active forms were not isozymes but were different oligomeric species or aggregates of the basic subunit of ALS. These different ALS species exhibit little difference in feedback inhibition by valine, leucine, and isoleucine or inhibition by the sulfonylurea herbicide chlorsulfuron [2-chloro-N-[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] benzenesulfonamide]. The lack of cross-resistance in some of the variants has been used to support the hypothesis that there are two separable binding domains for sulfonylureas and imidazolinones on the ALS molecule (103).

Differences in sensitivity to nicosulfuron [2-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-N,N-dimethyl-3-pyridinecarboxamide] in two corn hybrids resistant to imazethapyr [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid] apparently resulted from the differential sensitivity of the ALS target enzyme. These results suggest that the sites of action of two different classes of herbicides, the imidazolinones and the sulfonylureas, may be overlapping but not identical (109). The sulfonylurea herbicide, chlorsulfuron, and the imidazolinone herbicide, imazaquin [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-quinolinecarboxylic acid], were shown to be noncompetitive and uncompetitive inhibitors, respectively, of purified ALS from barley with respect to pyruvate (30). Schloss et al. (105)

proposed that the herbicide-specific site of ALS is an evolutionary vestige of the quinone binding site of pyruvate oxidase. Consistent with this proposal, the ubiquinone homologues Q_0 and Q_1 (potent inhibitors of ALS), and Q_0 (an imidazolinone herbicide), and a sulphonanilide herbicide, each compete with a radiolabelled sulfonylurea herbicide for a common binding site on ALS. ALS from several of these variants has also been found to be altered in feedback sensitivity to Val, Leu, and Ile (38). The sulfonylurea herbicides have been found to act as slow-binding inhibitors of the enzyme (67).

Sulfonylurea herbicides have been shown to reduce pollen viability and influence other developmental processes that may account for decreased seed production by otherwise apparently healthy dyer's woad (Isatis tinctoria L.) plants treated with these materials. Pre-anthesis stage treatment with 3 g a.i./ha metsulfuron [2-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] amino] sulfonyl] benzoic acid] was enough to prevent dyer's woad fruit formation and seed production (5).

Metabolism Study

Selectivity of ALS-inhibitor herbicides in cereal crops, corn, and soybean has been correlated with the ability of these plants to rapidly convert sulfonylureas to herbicidally inactive products. In contrast, susceptible weeds metabolize these herbicides much more slowly (13). Sulfonylurea tolerance of some weed species has been suggested to result from rapid herbicide inactivation (13).

Most tolerant plant species initially metabolize sulfonylurea herbicides by introduction of hydroxyl groups, frequently followed by carbohydrate conjugation (13). There have been several reports implicating cytochrome P-450 monooxygenase in oxidative herbicide metabolism in plants. Cytochrome P-450 is a ubiquitous family of hemoproteins, also referred to as mixed function oxidase; which catalyze a number of NAD(P)H-dependent monooxygenase reactions i.e., the incorporation of a single atom of oxygen from molecular O_2 into organic substances. P-450 are the terminal oxidases of a large number of biotransformations. The enzymic reactions include metabolism of steroids, fatty acids, prostaglandins, leukotrienes, biogenic amines, pheromones, drugs, plant metabolites, and numerous other substances, including mutagens (82).

The metabolic pathways in which these microsomal P-450 proteins participate are primarily involved in the biosynthesis and degradation of cellular components or the detoxification of xenobiotics. Some of the chemical reactions catalyzed by plant P-450 include aryl and alkyl hydroxylation and O- and N-dealkylation. Cytochrome P-450-mediated reactions are vital to the detoxification and selective phytoactivity of many herbicides (123).

There is evidence that cytochrome P-450 reactions are responsible for the detoxification of certain herbicides in wheat (Triticum aestivum L.) (42,91,108), corn (67,74,118), and other crop species (94) and that these reactions can enable certain crop species to tolerate a herbicide. The soil bacterium Streptomyces griseolus ATCC 11796 is capable of metabolizing several sulfonylureas. This metabolism is mediated by two inducible cytochrome P-450 monooxygenases (96). Each cytochrome P-450 appears to obtain reducing equivalents from NAD(P)H through a reductase and ferredoxin (85); the ferredoxins being the ultimate electron donors for the cytochrome P-450.

In plants, the induction of cytochrome P-450-dependent metabolism of xenobiotics has been reported (42,74,91,108,118), but there is no evidence that these reactions are catalyzed by enzymes exhibiting broad and overlapping substrate specificity. To the contrary, the approximately 30 cytochrome P-450 monooxygenase reactions already described in plants showed very narrow substrate specificity (39). Zimmerlin (124) has recently characterized a xenobiotic-inducible cytochrome P-450 from wheat microsomes that catalyzes the aryl hydroxylation of diclofop [($\pm$)-2-[4-(2,4-dichlorophenoxy) phenoxy]propanoic acid], a herbicide selective for wheat crop. Also, Zimmerlin (125) reported that wheat (cv Etoile de Choisy) microsomes catalyzed the cytochrome P-450-dependent oxidation of the herbicide diclofop to three hydroxy-diclofop isomers. Hydroxylation was predominant at carbon 4, with migration of chlorine to carbon 5 (67%) and carbon 3 (25%).

Fonne-Pfister et al. (34) found from microsomal corn seedlings that primisulfuron [2-[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl] amino] carbonyl] amino] sulfonyl] benzoic acid] is initially metabolized by a cytochrome P450-dependent monooxygenase system located in the microsomal fraction. This enzyme system supported the in vitro hydroxylation of primisulfuron at the phenyl ring and at the pyrimidine ring, respectively. Corbin et al. (23) suggested that primisulfuron metabolism in excised shoots and microsomal preparations of corn was mediated by cytochrome P-450 and that enhanced metabolism induced by an naphthalic anhydride (NA) seed-treatment can overcome the synergistic interaction imposed by terbufos [S-[(1,1-dimethylethyl) thio]methyl O,O-diethyl phosphorodithioate] on the metabolism of primisulfuron. Microsomes isolated from 3-day-old etiolated corn (Pioneer 3343 IR) shoots convert nicosulfuron to a single polar metabolite. This metabolite is hypothesized to be the 5-hydroxypyrimidinyl derivative of nicosulfuron found in intact corn plants (37). Formation of the nicosulfuron metabolite by the microsomes is NADPH dependent (9). Barrett (9) suggested that the nicosulfuron metabolism in the microsomal preparations is due to the activity of cytochrome P-450 mixed function oxidase.

Mechanisms For Herbicide Resistance

Among weeds, triazine resistance was first reported in 1970 (98). Subsequently, resistance to several other herbicides in addition to the triazines have occurred in weeds (8,40). A large number of herbicide-resistant mutants have been isolated and characterized from a wide range of organisms such as bacteria, yeast, and lower and higher plants (65).

There are four hypotheses to explain the herbicide resistant mechanisms.

1. Compartmentation of the herbicide preventing this herbicide from reaching the target site. Fuerst et al. (37) showed that paraquat [1,1'-dimethyl-4,4'-bipyridinium ion] resistance in conyza (Conyza bonariensis (L.) Cronq.) was related to compartmentation of the paraquat in the resistant biotype. They fed [^{14}C] paraquat through the petiole, and found that [^{14}C] paraquat distributed uniformly in the leaves of the susceptible biotype, but localized in the proximity of vascular tissue, and regions of the lower petiole, in the resistant biotype. From the autoradiograms, paraquat was compartmentalized at the cellular level and was excluded from the active site in the chloroplasts. They suggested that sprayed-paraquat would penetrate the cuticle of leaf but then become rapidly sequestered in leaf mesophyll tissue before reaching the active site in the chloroplast. Resistance to aryloxyphenoxypropionate (AOPP) and cyclohexanedione (CHD) herbicides in some populations of annual ryegrass (Lolium rigidum Gaudin) from Australia and wild oat (Avena fatua L.) from Canada is correlated with the ability of plants to sequester herbicide at the sub-cellular level (60). The various metabolites of most herbicides include conjugates of either the parent chemical or a phase I metabolite of parent chemical. Conjugation causes physical and chemical compartmentation of herbicides and may be of major importance for the sequestration of phytotoxic chemicals out of the cytoplasm (7).

2. Enhanced metabolism of the herbicide. Burnet et al. (15) reported that the resistant rigid ryegrass detoxified simazine [6-chloro-N,N'-diethyl-1,3,5-triazine-2,4-diamine], chlortoluron and metribuzin [4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one] faster than a control susceptible. The observation that resistance is reduced if metabolism is suppressed indicates that a major mechanism of resistance and cross-resistance to these herbicides is enhanced metabolism (15). Harms et al. (46) reported that the fate of ^{14}C-labeled primisulfuron in corn seedling tissues of inbred 4C0 and the hybrid, 4C0 x 4N5 indicated rapid metabolism with a half-life of approximately 3 h, but over 24 h in herbicide-sensitive inbred 4N5. This suggested that the observed primisulfuron tolerance of corn was probably due to a specific metabolic capacity rather than to herbicide-insensitive form of ALS (17). The differential tolerance of bahiagrass (Paspalum notatum Fluegge) and centipedegrass (Eremochloa ophiuroides (Munro) Hack.) to sulfometuron appears to be based on more rapid metabolism of the herbicide in centipedegrass than in bahiagrass (6). Herbicide selection pressure can lead to the mini-evolution of weed biotypes with enhanced MFO capacity to degrade herbicides, and therefore, resistant to herbicides such as chlortoluron, chlorsulfuron or diclofop-methyl (88).

3. Change the characteristics of cell membranes. The mechanism of paraquat resistance in wall barley (Hordeum glaucum Steud.) is related to an alteration in the membrane transport properties of paraquat in resistant plants (47).

Diclofop rapidly depolarized the cell membrane potential in peeled coleoptile sections, with no difference between the R and S biotypes of wild oat. However, when diclofop was removed from the treatment solution, the electrogenic potential remained collapsed in the susceptible cells, but recovered in the resistant cells. In whole plant experiments, acidification of the external medium continued in the presence of diclofop in the R biotype, but was slowly arrested in the S biotype. So, Hall et al. (45) suggested that the difference in the effect of diclofop on transmembrane proton flux in the R and S biotypes may be involved in conferring resistance to the R biotype.

4. Alterations in the target site of the herbicide. Thus far, weeds with alterations in the target site of the herbicide account for the majority of the herbicide-resistant weed problem, especially for sulfonylurea herbicide-resistant weeds. Westwood and Weller (121) supported a hypothesis that glyphosate [N-(phosphonomethyl) glycine] tolerance in field bindweed (Convolvulus arvensis L.) may be due in part to a greater level of activity in the shikimate pathway. This may lead to an increased ability of the tolerant biotype to respond to glyphosate challenge as reflected by EPSP synthase induction patterns. Peniuk et al. (86) reported that the two wild mustard (Sinapis arvensis L.) biotypes showed similar patterns of adsorption, translocation and exudation following application of ring ^{14}C-labelled 2,4-D [(2,4-dichlorophenoxy) acetic acid] and dicamba [3,6-dichloro-2-methoxybenzoic acid] to plants grown in a hydroponic system. Furthermore, reverse-phase HPLC indicated that there was no difference in the pattern or extent of metabolism of 2,4-D and dicamba between the resistant and susceptible biotypes. They suggest that sensitivity differences between the two biotypes of wild mustard to 2,4-D and dicamba may be the result of differential sensitivity at the target site(s) of action for auxinic herbicides. Hirshberg and McIntosh (57) found that the photosynthetic electron transport in triazine-resistant weeds was 1000-fold less sensitive to symmetrical triazines compared to wild-type chloroplasts. Specific knowledge about the alterations in kinetics of ALS resistant to herbicides is very limited. Rathinasabapathi et al. (92,93) reported that ALS from sulfonylurea-resistant and imidazolinone-resistant variants of Datura innoxia Mill. exhibited different degrees of cross-resistance to sulfonylureas and imidazolinones. The lack of cross-resistance in some of the variants has been used to support the hypothesis that there are two separate binding domains for sulfonylureas and imidazolinones on the ALS molecule (102). A number of ALS mutants have been described which are selectively resistant to sulfonylureas (54) and imidazolinones (55,103). To understand the molecular basis of imidazolinone resistance, Sathasivan et al. (100) isolated the ALS gene from an imazapyr-resistant mutant GH90 of Arabidopsis thaliana (L.) Heynh. DNA sequence analysis of the mutant ALS gene demonstrated a single-point mutation from G to A at nucleotide 1958 of the ALS-coding sequence. This would result in Ser to Asn substitution at residue 653 near the carboxyl terminal of the matured ALS.

They found that the mutant ALS gene from GH90 conferred imazapyr resistance in transgenic plants. This is the first report of the molecular basis of imidazolinone resistance in plants (100). Resistance to both the sulfonylureas and imidazolinones has been shown in haploid Datura innoxia Mill. lines (102). Since these organisms contain single loci for ALS, multiple resistance may result from independent mutations within the ALS enzyme, each responsible for selective resistance to a specific herbicide class, or from single mutations responsible for cross resistance. One possibility is that each herbicide class interacts with the ALS macromolecule at separate sites. In yeast, at least ten independent sites within highly conserved regions of ALS have been mutated to yield sulfonylurea resistance (70). Hattori et al. (50) demonstrated that multiple-resistance phenotypes can be achieved through combinations of separate mutations, each of which individually confers resistance to only one class of herbicides. ALS from several of these variants also had altered feedback sensitivity to Val, Leu, and Ile (50).

Rathinasabapathi et al. (91) suggested that the herbicide resistance mutation somehow altered pyruvate binding to the ALS molecule. This alteration could also have physiological implications, for example, by resulting in less efficient synthesis of branched-chain amino acids in vivo.

Nearly all plant mutants resistant to the imidazolinone, sulfonylurea, or triazolopyrimidine herbicides isolated to date have been shown to have numerous similarities: a single semidominant gene conferring resistance, herbicide-resistant ALS enzyme activity, and a direct correlation between the in vivo, whole plant cross-resistance spectrum, and the in vitro, ALS activity cross-resistance spectrum.

Herbicide Resistance Crops

In recent years considerable research in the private and public sectors has been directed toward introducing herbicide resistance into normally susceptible crop species. Potential benefits of developing herbicide resistant crops include, an increased margin of safety, reduced risk of crop damage from residual herbicides from rotational crops, and introduction of new herbicides for use on normally susceptible crops. The advantage from the use of crop plants with herbicide resistance is not only to increase crop yield but also to increase quality of that product.

Triazine resistant canola (Brassica napus L.) was the first herbicide resistant crop developed by backcrossing. This procedure used triazine resistant bird's rape (Brassica campestris L.) (12). Resistant cultivars were unaffected by atrazine [6-chloro-N-ethyl-N"-(1-methylethyl)-1,3,5-triazine-2,4-diamine] or cyanazine [2-[[4-chloro-6-(ethylamino)-1,3,5-triazine-2-yl] amino]-2-methylpropanenitrile], and metribuzin [4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one]. However, the crop yield was reduced 20% to 30% compared with reciprocal susceptible cultivars (11). Researchers at Monsanto have successfully transferred genes conferring a degree of resistance to glyphosate into tomato (Lycopersicon esculentum L.) (61), and other crops.

Newhouse et al. (83) studied three corn lines resistant to imidazolinone herbicides. For all three lines, resistance was inherited as a single semidominant allele for each of the three independent mutations. All resistant selections have herbicide-resistant forms of ALS. The herbicide-resistant phenotypes displayed at the whole plant level correlate directly with herbicide insensitivity of the ALS activities of the selections (83). Mukaida et al. (79) reported that the ALS enzyme inhibition parallels whole plant injury produced with imazethapyr [2-[4,5-dihydro-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid] and chlorimuron-ethyl [2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]benzoic acid] and suggests that the mechanism for resistance was attributable to differential sensitivity at the target site. Pioneer 3343 IR corn is cross-resistant to imazethapyr and chlorimuron-ethyl at the whole plant and enzyme levels, while ICI 8532 IT corn is tolerant only to imazethapyr. Also, Pioneer 3343 IR showed resistance to interactions of sulfonylurea herbicides with terbufos (109) with/without piperonyl butoxide (PBO) [5-[[2-(2-butoxyethoxy) ethoxy] methyl]-6-propyl-1,3-benzodioxole] in the field experiment, but ICI 8532 IT did not. Primisulfuron metabolism occurred more rapidly in Pioneer 3343 IR than in the other normal corn hybrids (23). ICI 8532 IT was resistant to imazethapyr regardless of rate, application method, or presence of terbufos at the

Illinois locations, but ICI 8532 showed some injury (10,22).

A sulfonylurea resistant soybean line (106) and imidazolinone resistant corn have been developed using a chemical mutagen agent, such as ethyl methane sulfonate (EMS). Resistance in both species was due to an altered ALS binding site. Also, Moseley et al. (78) reported that the resistance of 'W-20' soybeans to chlorimuron was due to an altered target site.

A chlorsulfuron resistant sugarbeet (Beta vulgaris L.) line was selected from cell suspension cultures by Saunders et al. (101). Chlorsulfuron resistant sugarbeet was highly cross-resistant to other sulfonylurea herbicides including chlorimuron, thifensulfuron [3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] amino] sulfonyl]-2-thiophenecarboxylic acid], and primisulfuron applied at or exceeding field use rates (48). Hart et al. (49) showed that the sulfonylurea resistance in this sugarbeet was inherited in a semi-dominant fashion and that a 2- to 4-fold increase in resistance to primisulfuron and thifensulfuron was observed for homozygous resistant sugarbeet compared to heterozygous resistant sugarbeet. D'Halluin et al. (26) have developed an Agrobacterium mediated transformation procedure for sugarbeet. Sugarbeet was engineered for resistance to sulfonylurea compounds by introducing genes encoding mutant ALS. They confirmed that the transformants expressing a mutant ALS gene were resistant to field levels of sulfonylurea compounds.

Flax (Linum usitatissimum L.) and many other dicotyledonous species are sensitive to sulfonylurea residues in the soil, an interval of up to 4 years may be required before these crops can be sown in sulfonylurea-treated soil. Flax lines were transformed by Agrobacterium tumefaciens with a gene encoding a resistant ALS enzyme from Arabidopsis, generating 14 different, independently transformed lines (71). Resistance to chlorsulfuron was stablely inherited in all lines. McSheffrey et al. (72) suggested that the mutation in this particular ALS gene affects the binding of each sulfonylurea herbicide differently, and it is not possible to predict degrees of cross-resistance to other herbicides acting on the same target enzyme.

As part of a program to broaden the spectrum of herbicides useful in potato (Solanum tuberosum L.) production, a chimeric gene (bxn) for bromoxynil [3,5-dibromo-4-hydroxybenzonitrile] resistance has been introduced into potato cultivars. The bxn gene, which encodes a nitrilase specific for bromoxynil, was derived from Klebsiella ozanae, and was introduced into potatoes by Agrobacterium tumefaciens-mediated transformation. The transcription of the bxn gene was directed from a cauliflower (Brassica oleracea L. var. botrytis subvar. cultiflora DC.)-mosaic virus 35S promoter, and resulted in efficient expression in the potato plants (31).

Herbicide Resistant Weeds

The continued use of a single control agent is often a common feature in cases of resistance. The first herbicide resistant weed was reported from Belgium around 1950's. The reports said that dandelion (Taraxacum officinale Weber in Wiggers) and buttercup (Ranunculus) showed resistance to 2,4-D. Atrazine/simazine resistant common groundsel (Senecio vulgaris L.) was reported in Washington State in 1968 (89). Other triazine-resistant weed species, such as common lambsquarters (Chenopodium album L.), pigweed (Amaranthus spp.), witchgrass (Panicum capillare L.), kochia (Kochia scoparia (L.) Nees), annual bluegrass (Poa annua L.), downy brome (Bromus tectorum L.), barnyardgrass (Echinochloa crus-galli (L.) Beauv.), velvetleaf (Abutilon theophrasti Medicus), and giant foxtail (Setaria faberi Herrm.) have been reported worldwide and in the United States. Radosevich et al. (90) demonstrated that the resistant biotypes of common groundsel were resistant to all s-triazine herbicides due to a mutation in the chloroplast gene that encodes the herbicide binding protein of photosystem II where many photosynthetic inhibitors bind.

Rigid ryegrass (Lolium rigidum Gaudin) in Australia that was sulfonylurea-resistant, was first reported in 1986. This was followed by reports of sulfonylurea resistant prickly lettuce (Lactuca serriola L.) from Idaho, kochia, Russian thistle (Salsola iberica Sennen & Pau) from Montana, S. Dakota, N. Dakota, Colo., Kansas.

Wild mustard (Brassica kaber (DC.) L.C. Wheeler) populations resistant to auxin-type herbicides, including 2,4-D, MCPA [(4-chloro-2-methylphenoxy) acetic acid], dichlorprop [(±)-2-(2,4-dichlorophenoxy) propanoic acid] and dicamba [3,6-dichloro-2-methoxybenzoic acid] were identified in western Canada in 1990. Heap et al. (56) reported that plants from the resistant population appeared to be less competitive, had darker green foliage and were shorter than susceptible plants.

Burnet et al. (16) reported that rigid ryegrass biotype VLR 69 exhibited resistance to a number of ALS-inhibiting sulfonylurea and imidazolinone herbicides. On the basis that all VLR 69 plants exhibit resistance to chlorsulfuron but resistance to sulfometuron is restricted only to those that contain an insensitive ALS, it is suggested that more than one mechanism of sulfonylurea resistance is present in the biotype VLR 69.

Kochia biotypes that are sulfonylurea resistant have occurred through the continued use of chlorsulfuron in monoculture cereal-growing areas. Saari et al. (99) reported that ALS activity isolated from sulfonylurea-resistant kochia was less sensitive to inhibition by three classes of ALS-inhibiting herbicides, sulfonylureas, imidazolinones, and sulfonanilides. They also found that no differences were observed in the ALS-specific activities or the rates of [^{14}C] chlorsulfuron uptake, translocation, and metabolism between susceptible and resistant kochia biotypes. So, they concluded that the mechanism of sulfonylurea resistance in this kochia biotype is due solely to the less sulfonylurea-sensitive ALS enzyme.

Guttieri et al. (42) reported that most ALS resistance kochia (Kochia scoparia (L.) Schrad.) biotypes had mutation in the codon for the proline residue in Domain

A. Also, the nature of the amino acid substitution was highly variable. Four different amino acid substitutions, arginine, threonine, leucine, and alanine were observed in R biotypes. But, some R biotypes did not have an amino acid substitution in Domain A, although in vitro assays of ALS inhibition indicated resistance was due to an altered form of ALS. Therefore, other regions of the ALS gene may be involved in resistance to ALS inhibitors. They suggested that many resistance alleles are present in kochia.

The differences between sulfonylureas and imidazolinones with respect to the degree of ALS insensitivity are possibly due to slightly different binding domains in the common binding site of the protein.

Devine et al. (25) reported that the patterns of cross-resistance varied in two chlorsulfuron resistant biotypes of common chickweed (Stellaria media (L.) Viel) indicating that the alteration in ALS that confers chlorsulfuron resistance does not confer the same level of resistance to other sulfonylurea herbicides. The chlorsulfuron resistant common chickweed showed high levels of cross-resistance among sulfonylurea and triazolopyrimidine herbicides, but low levels of cross-resistance to imidazolinones. Whereas some sulfonylurea herbicides and the triazolopyrimidine herbicide are very effectively excluded from the altered binding niche, other sulfonylureas and the imidazolinone herbicides tested are restricted from the altered binding niche to a much lesser extent. They concluded that the differences in the patterns of cross-resistance observed reflect differences in the binding affinity of the herbicides for the altered ALS (25).

Tonks and Westra (117) showed that resistant and susceptible kochia responded similarly to the herbicides other than sulfonylureas. Christoffoleti (21) reported that the resistant biotype of kochia had no physiological disadvantage relative to the susceptible one in terms of biomass and seed production. This suggests that kochia resistant to sulfonylureas may not be less productive than susceptible ones. Thompson (116) found that the resistant kochia biotypes tended to have equal or greater leaf and stem dry weight, shoot height, and stem and shoot diameter than susceptible biotypes 13 weeks after establishment. The resistant and susceptible biotypes, on the average, produced 11,000 and 13,000 seeds per plant. Alcocer-Ruthling et al. (3) studied the seed biology of sulfonylurea-resistant and -susceptible biotypes of prickly lettuce. They found that seed longevity in soil, and fecundity or seed viability were not different between R and S biotypes. Also, they found seed from R biotype plants germinated as fast or faster than seed from S biotype plants (3).

Triallate [S-(2,3,3-trichloro-2-propenyl) bis(1-methylethyl) carbamothioate] resistant wild oat populations generally had higher seed germination rates than susceptible populations, but no difference in terms of shoot dry weight, shoot number, leaf number or leaf area (84).

Powles et al. (87) reported that multiple-resistance in Australian biotypes of rigid ryegrass, which were selected by heavy usage of diclofop-methyl, extends to chemicals in the cyclohexanedione, sulfonylurea, dinitroaniline, triazine, substituted urea, and triazole classes of herbicides. Rubin et al. (97) found that the sulfonylurea resistant redroot pigweed (Amaranthus retroflexus L.) from the forest which was annually treated for four years with a mixture of sulfometuron and simazine exhibited a 10 to 40 fold increase in cross-resistance to different sulfonylureas, depending on the method used. No cross-resistance was detected so far in the resistant population to imazapyr, another ALS inhibitor. Also, the resistant biotype shows a 2 to 5 fold increase in resistance to triazines. These data indicate that mixing herbicides of different modes of action may not be enough to prevent evolution of herbicide resistance especially when resistance is based on altered herbicide metabolism.

Interaction of ALS inhibiting Herbicides with Organophosphate Insecticides

Herbicides and insecticides may be required in the same growing season to control weeds and insects in corn. However, an interaction may occur between the pesticides that may be synergistic, antagonistic, or additive (52). In herbicide mixtures, chemicals can interact in the solution, at the plant surface, in the soil, within the tissues involved in absorption and translocation, as well as at the cellular site of action (69).

The first reports of herbicide interactions with insecticides came from studies with cotton (Gossypium hirsutum L.). Hacskaylo et al. (44) observed that severe injury or death occurred to seedling cotton when a monuron [N'-(4-chlorophenyl)-N,N-dimethylurea] or diuron [N'-3,4-dichlorophenyl)-N,N-dimethylurea] application was preceded with a systemic insecticide application of phorate [O,O-diethyl S-[(ethylthio) methyl] phosphorodithioate] or disulfoton [O,O-diethyl S-[2-(ethylthio) ethyl] phosphorodithioate]. Subsequent studies showed phosphate and other classes of insecticides to increase the crop phytotoxicity of diuron to corn (80).

Nicosulfuron, primisulfuron and imazethapyr have the potential to injure corn under unfavorable environmental conditions. Corn injury consisted primarily of height reduction and some malformation of plants. Further the injury may be accentuated by the addition of the insecticide terbufos. Combination of primisulfuron with terbufos in corn field resulted in foliar and root injury, plant height reductions, and yield losses (14). Corn injury with nicosulfuron, primisulfuron or imazethapyr was influenced by corn hybrid, terbufos placement, application time (81). Terbufos applied in soil can be absorbed by the corn seedling and moves systemically throughout the plant (107). In-furrow insecticide treatments in combination with any of the herbicides caused greater crop injury than band applied insecticides in combination with a herbicide (111). The 15% granular formulation of terbufos applied in-furrow, used in combination with any of the ALS inhibiting-herbicide treatments caused crop injury (chlorotic leaf spotting near the whorl, leaf crinkling, shortened internodes, and stunting of plant growth). Use of a controlled release formulation of terbufos in-furrow or in a band decreased visual injury symptoms when compared to 15% granular formulation (111).

Addition of the organophosphate insecticide terbufos to the germination medium prevented the metabolism of primisulfuron (23,111), and nicosulfuron (109,110,111) by shoots excised from unsafened seed, but not by shoots from NA-treated seed (23). HPLC analysis of extracted nicosulfuron and metabolites indicated greater parent herbicide longevity in plants treated with terbufos 15G. The rate of nicosulfuron metabolism decreased with increasing terbufos concentration (27,28). Herbicide uptake was not affected by terbufos form; however, all of the absorbed nicosulfuron was metabolized at 24 hr after treatment without an organophosphate insecticide pretreatment whereas 90%, 60%, and 35% was metabolized in the terbufos, terbufos-sulfoxide, and terbufos-sulfone treatments, respectively (28).

Terbufos form plays a major role in the level of nicosulfuron metabolism in corn (27,28). Terbufos 20 CR plus herbicide was safer than 15 G plus herbicide at 4 weeks after application and to a greater extent at 8 weeks after application (77,120). Corn injury was not increased with combinations of the sulfonylurea herbicides and carbofuran [2,3-dihydro-2,2-dimethyl-7-benzofuranol methylcarbamate], chlorpyrifos [O,O-diethyl-O-(3,5,6-trichloro-2-pyridinyl) phosphorothioate],tefluthrin[(2,3,5,6-tetrafluoro-4-methylphenyl)methyl-(1α,3α)-

(Z-(±)-3-(2-chloro-3,3,3,-trifluoro-1-propenyl)-2,2-dimethylcyclopropane-carboxylate] or chlorethoxyfos. Combination treatment of fonofos [O-ethyl-S-phenylethylphosphonodithioate] plus CGA-136872 resulted in 20% corn injury 1 week after treatment (120). Simulated rainfall increased the corn injury from primisulfuron-terbufos interaction (59).

Less injury occurred by delaying the application of nicosulfuron when terbufos was used (64). The difference in injury between times of postemergence application of nicosulfuron may be related to the more extensive root system beyond the insecticide-treated soil. So, corn plants probably were absorbing less insecticide when the herbicide was applied. Second, degradation of terbufos likely was occurring, resulting in less being available for uptake at the later corn stage. Furthermore, degradation of terbufos in the plants was occurring, resulting in less interacting or inhibiting the nicosulfuron metabolism in the plants.

Simpson et al. (110) found that tank-mixing 2,4-D with nicosulfuron decreased corn injury resulting from the nicosulfuron/terbufos interaction in field studies. Reduction of injury was observed when 2,4-D was applied at 0.28 kg ha^{-1} with and 1 day after nicosulfuron application. The decrease in nicosulfuron metabolism caused by terbufos was reversed when 2,4-D was applied. The 2,4-D did not affect uptake and translocation of nicosulfuron in the presence or absence of terbufos (110). Corbin et al. (23) suggested that primisulfuron metabolism in excised shoots and microsomal preparations of corn is mediated by cytochrome P-

450 and that enhanced metabolism induced by an NA seed-treatment can overcome the synergistic interaction imposed by terbufos on the metabolism of primisulfuron.

Metabolism of Terbufos

Soil-applied insecticides are widely used in corn at planting for wireworm (Melantus spp. and Conoderus spp.), rootworm (Diabrotica spp.), and nematode (Longidorus spp., Trichodorus spp., and Xiphinema spp.) control. A widely used soil insecticide in the United States is terbufos, an organophosphate insecticide. Terbufos is known to be effective against several soil insect pests that attack corn.

Most of the terbufos was lost during the first month after application with corresponding increases in recoveries of terbufos-sulfoxide and terbufos-sulfone. Chapman et al. (20) found that terbufos residue persisted in organic soils because of a rapid conversion of terbufos to the less degradable terbufos sulfoxide. For this reason, terbufos has not been recommended as a soil insecticide on organic soil. But its use may be limited because it may be less persistent after a second consecutive year of applications on the same land because of enhanced microbial degradation (20). Szeto et al. (115) also reported that terbufos was oxidized to its sulfoxide and sulfone in soil. Felsot et al. (33) reported that terbufos sulfoxide was the principle oxidative metabolite which formed rapidly from terbufos and was followed by a slower oxidation to terbufos sulfone. Their findings are in general agreement with those reported by Chapman et al. (20). They found that terbufos translocated from soil into broccoli (Brassica oleracea L.). The plant residues consisted mostly of terbufos sulfoxide, terbufos oxon sulfoxide, and terbufos sulfone accounting for 90% or more of the total residues in broccoli, but the parent compound accounted for only 5% of the total. This suggests that terbufos and its sulfoxide and sulfone were translocated from soil into plants and they were further oxidized in the plants to terbufos oxon sulfone. Also they reported that the concentration of total resides was highest in young plants collected at the first thinning, i.e. 22 days after seeding. It decreased steadily as the plants matured. Sellers et al. (107) studied residues of terbufos in Iowa corn and soil. They reported that residues in field corn forage ranged from a high of 0.43 ppm 40 days posttreatment with 4.48 kg ha^{-1} furrow application to nondetectable residues 60 days posttreatment with 1.12 kg ha^{-1} band application. It is apparent that terbufos or its sulfoxide and sulfone were readily taken up by plants grown in treated soil, but residues in the plant tissues, including all toxic oxidative metabolites, degraded rapidly.

Interaction of Antioxidants with Herbicides

Piperonyl butoxide (PBO) a known insecticide synergist enhanced the herbicidal activity of atrazine and terbutryn [N-(1,1-dimethylethyl)-N'-ethyl-6-(methylthio)-1,3,5-triazine-2,4-diamine] applied postemergence to corn seedlings (119). PBO also reversed the chloroplastic triazine resistance in a resistant biotype of ryegrass, both at the whole plant and isolated chloroplasts levels (119). PBO applied in combination with either atrazine or terbutryn increased the foliar uptake of both herbicides in corn leaves, and lead to a light-dependent damage to membrane integrity. Rapid changes in the glutathione (GSH) levels were also observed following treatment with PBO alone and PBO with atrazine. PBO treated corn plants contained more terbutryn and its partially-dealkylated metabolites. Etiolated corn seedlings have shown that PBO binds to cytochrome P-450. In the microsomal fraction containing NADPH, terbutryn conversion to polar metabolites was inhibited by PBO by more than 50%. These results indicate that PBO inhibits terbutryn degradation mediated by cytochrome P-450. Varsano (119) suggest that PBO may act as a herbicide-synergist by several mechanisms simultaneously: increasing herbicide penetration, inducing membrane damage and inhibiting the metabolism of herbicides such as terbutryn.

Bentazon [3-(1-methylethyl)-(1H)-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide], tetcyclacis [5-(4-chlorophenyl)-3,4,5,9,10-pentaazatetrracyclo [5.4.$10^{2,6}$,$0^{8,11}$]-dodeca-3,9-dine] and PBO inhibited the metabolism of both primisulfuron and nicosulfuron in corn shoot tissue. But, tridiphane [2-(3,5-dichlorophenyl-2-(2,2,2,-trichloroethyl) oxirane] had no effect on metabolism (95). Two hydroxylation reactions in primisulfuron treated corn plants were inhibited in vitro by tetcyclacis (34). The catalytic reaction required NADPH and oxygen and was inhibited by the cytochrome P-450 monooxygenase inhibitors, PBO (23) and tetcyclacis(23,125) and carbon monoxide (125). Aminobenzotriazole, tetcyclasis and PBO inhibited nicosulfuron metabolism by corn microsomes (9).

Metabolism of simazine, chlortoluron, and metribuzin herbicides was inhibited by the mixed-function oxidase (MFO) inhibitor, 1-aminobenzotriazole (ABT). ABT in combination with each of the herbicides significantly reduced plant dry weight compared with either the ABT or the herbicide alone (15).

Action of Antidotes

Hoffmann (58) was the first researcher to report that a second chemical could selectively protect a whole plant from herbicide injury. He termed these chemicals 'herbicide antidotes'. The use of chemical antidotes or protectants or safeners has been widely studied. There are significant reasons for using herbicide antidotes including use of higher herbicide rates and therefore more effective weed control, use of herbicides under conditions where crop damage is liable to occur, such as susceptible varieties, carryover of herbicide residues from previous crop, or use of interacting insecticide. Antidotes also provide useful insights into herbicidal action and metabolism.

Chemical, biochemical, and competitive or physiological antagonisms of the activity of herbicides by the antidotes are potential mechanisms of protective action. Chemical antagonism occurs when an antidote reacts chemically or physically with herbicide to prevent herbicide absorption by crop. Biochemical antagonism occurs when a protectant reduces herbicide uptake and/or translocation or stimulates metabolism of herbicides. Competitive or physiological antagonisms occur when an antidote competes with a given herbicide for the same site of action in the cells of the protected plant.

Fuerst (35) proposed that two hypotheses for antidote mode of action seem plausible. Antidotes induce fast herbicide metabolism or protect the biochemical site of action of the herbicide. The other plausible hypothesis is that antidotes protect the biochemical site of herbicide action. Compounds with similar structures to thiocarbamate herbicides are often effective safeners (51,112). For example, dichlormid [2,2-dichloro-N,N-di-2-propenylacetamide] is structurally very similar to EPTC [S-ethyl dipropyl carbamothioate] and CDAA [2-chloro-N,N-di-2-propnylacetamide].

Safeners that protect corn from sulfonylurea herbicides induce both oxidation and glucosylation. Wheat safeners such as CGA-184967 and HOE-70542 induce oxidative metabolism of aryloxyphenoxypropionic acid herbicides. Several investigators have suggested that safeners may be inducing expression of genes that normally are induced by pathogens for the purpose of detoxifying phytotoxins (36). BAS 145138 and naphthalic anhydride (NA) treatments increased the metabolism of nicosulfuron and primisulfuron in corn shoots but did not increase in the roots (59,95). Also, metabolism was greatly enhanced by microsomes isolated from shoots of NA-treated corn seed (23).

Hatzios (53) reported that NA applied as a seed dressing offered good protection to the sensitive corn hybrids against injury caused by the PPI-applied thifensulfuron, but not against the highest rate of 96 g/ha. NA provided limited or no protection to any corn hybrid against injury caused by early postemergence-applied thifensulfuron. NA enhanced significantly the de-esterification of thifensulfuron-methyl causing a 1.5 - 2.0 fold increase in the formation of the parent acid, thifensulfuron. In addition to glutathione S-transferase (GST), MFO, and UDP-glucosyltransferase enzymes, hydrolytic enzymes are enhanced by herbicide safeners which confer tolerance to grass crops.

NA seed treatment (0.5% w/w) induced bentazon 6-hydroxylase activity 2.7 to 11.3 fold but did not induce cinnamic acid 4-hydroxylase activity (43). Seed treatment with the safener NA or treatment of seedlings with phenobarbital increased cytochrome P-450 content and lauric acid hydroxylase (LAH) activity, but decreased cinnamic acid hydroxylation of the wheat microsomes (125). This would suggest that in plants exposed to xenobiotics, some isoenzyme activities may be selectively stimulated, whereas others are unchanged or decreased. They concluded that cytochrome P-450 induction may be a general mode of action of those safeners that protect crops against herbicides undergoing oxidative metabolism (125). Sweetser (114) has shown that the rate of metabolism of the sulfonylurea herbicides, chlorsulfuron and metsulfuron methyl, was significantly increased in excised leaves from wheat and corn following treatment with crop safeners such as NA, and cyometrinil [(Z)-α-[(cyanomethoxy) imino] benzeneacetonitrile] (114). Treatment of corn seed with this safener CGA 154281 [4-(dichloro-acetyl)-3,4-dihydro-3-methyl-2H-1,4-benzoxazine], dramatically increased specific activities of cytochrome P-450 dependent primisulfuron hydroxylation. The total cytochrome P-450 content was also significantly increased in microsomes from safener-treated seedlings as compared to untreated controls (34). They suggested that the safener induces the biosynthesis of distinct cytochrome P-450 isozymes.

Zimmerlin et al. (124) reported that the content of cytochrome P-450 was enhanced in etiolated wheat shoot microsomes after treatment with either NA or phenobarbital (PB). A much greater stimulation of enzyme activity occurred when PB and NA were combined. For example, the oxidation of 2,4-D, which is too low to be detected in microsomes from untreated seedling, become measurable.

Benoxacor is a dichloroacetamide-safener used to protect corn against injury from metolachlor. Irzyk et al. (50) reported that corn (Pioneer 3906) treated at planting with 1 μM benoxacor contained elevated levels of total glutathione S-transferase (GST) activity (24,41). Miller (75) studied that the effects of benoxacor on protein synthesis and GST induction in corn cell suspension cultures. Treatment of cultures with 10 μM benoxacor for 24 hr resulted in a 2.5 fold increase in total GST activity using metolachlor [2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl)acetamide] as the substrate. ^{14}C-

Benoxacor metabolism studies, using TLC analysis, reveal that benoxacor is rapidly metabolized to at least seven metabolites more polar that the parent molecule.

Gronwald et al. (41) isolated the glutathione S-transferase (GST) isozymes from sorghum (DK 41Y) seedlings treated with the safener CGA-133205. Comparison of chromatogram with one for nonsafened sorghum showed that CGA-133205-treatment increased the GST activity of two peaks.

MON 13900 [3-(dichloroacetyl)-2,2-dimethyl-5-(2-furanyl) oxazolidine] is a new herbicide safener that can reduce corn injury from several classes of herbicides, especially effective for minimizing the deleterious effects of sulfonylurea herbicides on corn growth and development (122). Tank-mixtures of MON 13900 consistently reduced injury from MON 12000 [methyl 3-chloro-5-(4,6-dimethoxypyrimidin-2-arylcarbamoyl sulfamoyl)-1-methylpyrazde-4-carboxylate], a new sulfonylurea herbicide under development by Monsanto company (122). The mode of action of MON 13900 appears to be enhanced herbicide metabolism (76,122).

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CHAPTER 2

The Interaction of Insecticides with Herbicide Activity

ABSTRACT

Combination of certain herbicides with insecticides can negatively affect crop growth. Greenhouse studies were conducted to evaluate interaction effects of herbicides and insecticides with/without antidotes, on corn and weed species. Northrup King 9283 hybrid corn showed greater sensitivity to acetanilide herbicides than Cargill 7567. Both hybrids were sensitive to the interaction of chlorimuron, nicosulfuron and primisulfuron with terbufos. Cargill 7567 hybrid corn was more tolerant to the interactions of sulfonylurea herbicides with terbufos than Northrup King 9283. Imazaquin at 70 g/ha reduced corn height of Northrup King 9283 hybrid, but there was no interaction with terbufos. The antidotes, CGA-154281 and NA, reduced corn injury from metolachlor, and nicosulfuron and primisulfuron with terbufos treatments, respectively. Thus, these antidotes stimulate P-450 mixed function oxidase activity. Nicosulfuron and primisulfuron treatments combined with metolachlor showed less corn injury than metolachlor alone, but these herbicides increased corn injury combined with terbufos treatment.

The combination of primisulfuron and terbufos did not enhance herbicidal activity compared with primisulfuron alone to barnyardgrass, giant foxtail and velvetleaf control.

Nomenclature: Acetochlor, 2-chloro-N-(ethoxymethyl)-N-(2-ethyl-6-methylphenyl) acetamide;alachlor,2-chloro-N-(2,6-diethylphenyl)-N-(methoxymethyl)acetamide; chlorimuron, 2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl] benzoic acid; imazaquin, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-quinolinecarboxylic acid; metolachlor, 2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl) acetamide; nicosulfuron, 2,-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-N,N-dimethyl-3-pyridinecarboxamide;primisulfuron,2-[[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl] amino] carbonyl] amino] sulfonyl] benzoic acid; brace; terbufos, S-[[(1,1-dimethylethyl)thio]methyl] O,O-diethyl phosphorodithioate; CGA-154281, 4-(dichloro-acetyl)-3,4-dihydro-3-methyl-2H-1,4-benzoxazine;NA,1,8-naphthalic anhydride; barnyardgrass, Echinochloa crus-gali (L.). Beauv.; common lambsquarters, Chenopodium album L.; giant foxtail, Setaria faberi Herrm.; velvetleaf, Abutilon theophrasti Medic.; Corn, Zea mays 'Northrup King 9283', 'Cargill 7567';

Additional index words: Interaction, herbicide activity

INTRODUCTION

To produce corn (Zea mays L.), farmers need to control weeds, insects, and fungi which may reduce crop yield and quality. Thus, farmers may apply herbicides and insecticides to the same crop, during the same growing season. Interactions between pesticides may occur. The recent introductions of the sulfonylurea and imidazolinone herbicides have stimulated research on the interaction of herbicides with insecticides.

Nash (8) reported a synergistic interaction on oat (Avena sativa L.), corn, and cotton (Gossypium hirsutum L.) yields with monuron [N'-(4-chlorophenyl)-N,N-dimethylurea] or diuron [N'-(3,4-dichlorophenyl)-N,N-dimethylurea] plus phorate [O,O-diethyl S-[(ethylthio)methyl] phosphorodithioate] or disulfoton [O,O-diethyl S-[2-(ethylthio)ethyl] phosphorodithioate], herbicide-insecticide combinations. He stated that three possibilities of pesticide interaction may be visualized: at the site of absorption where one pesticide affects the absorption of the others; within plants in which one pesticide affects the primary and another pesticide affects a secondary pathway; or both jointly affecting a single pathway.

Rootworms (Diabrotica spp.) are a serious corn pest in the corn belt of the United States. Terbufos [S-[[(1,1-dimethylethyl) thio] methyl] O,O-diethyl phosphorodithioate] is the organophosphate insecticide widely used for rootworm control. The number of potential interactions between combinations of sulfonylurea herbicides and organophosphate insecticides are very high. In 1990, nicosulfuron [(2-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-N,N-dimethyl-3-pyridinecarboxamide] and primisulfuron [2-[[[[[4,6-bis (difluoromethoxy)-2-pyrimidinyl]amino] carbonyl] amino] sulfonyl] benzoic acid] became commercially available for the control of annual and perennial grasses, and annual and perennial broadleafs, respectively, in corn. In the field experiment, combinations of the organophosphate insecticides disulfoton, fonofos [O-ethyl S-phenylethylphosphonodithioate], isazophos [O-(5-chloro-1-[methylethyl]-1H 1,2,4-triazol-3-yl) O,O-diethyl phosphorotioate], or terbufos and primisulfuron were shown to interact synergistically resulting in foliar and root injury, plant height reduction, and corn grain yield losses (2,4). Morton et al. (7) observed that the application of terbufos increased corn injury from nicosulfuron to 'Pioneer 3751' and 'Jubilee' hybrids. Porpiglia et al. (9) suggests that the synergistic interaction occurs because the insecticide may reduce metabolism and increase the uptake of primisulfuron in plants.

Many researchers have reported that injury from nicosulfuron was greater when terbufos had been applied in-furrow at planting (15). Less injury occurred by delaying the application of nicosulfuron when terbufos was used (5). Kapusta et al. (5) suggested that the difference in injury between times of POST application of nicosulfuron may be due to the more extensive root system, and less absorption, and more degradation of terbufos at the later corn stage.

Several researchers have tried to find some methods to protect crops from the interaction of herbicides and insecticides. Simpson et al. (13), found that the tank-mixing 2,4-D [(2,4-dichlorophenoxy) acetic acid] with nicosulfuron decreased corn injury resulting from the nicosulfuron/terbufos interaction in the field studies. The decrease in nicosulfuron metabolism caused by terbufos was reversed when 2,4-D was applied. Corbin et al. (3), suggested that metabolism of primisulfuron of corn is mediated by cytochrome P-450 and that enhanced metabolism induced by an NA (1,8-naphthalic anhydride) seed-treatment can overcome the synergistic interaction imposed by terbufos on the metabolism of primisulfuron.

The objectives of this study were: a) to identify interactions between corn insecticides and the acetanilide herbicides or sulfonylurea herbicides, b) to determine whether the antidotes, CGA-154281 in CGA-180937, and NA seed-treatment, provided protection against the interaction of herbicides with the insecticide, terbufos, c) to determine whether sensitivity of corn to acetanilide herbicides was related to the other interactions, and d) to determine the interaction effect of the combination of primisulfuron with terbufos on herbicidal activity to several weed species.

MATERIALS AND METHODS

General greenhouse procedure

Weed seeds were planted in 945-ml plastic pots, which contained an air-dried Spinks sandy loam (mixed, mesic Psammentic Hapludalfs) soil consisting of 71.3% sand, 19.4% silt, and 9.4% clay with a pH of 6.2 or BACCTO soil. After emergence, the plants were thinned to one plant per pot for velvetleaf (Abutilon theophrasti Medic.) and common lambsquarters (Chenopodium album L.) and two plants per pot for barnyardgrass (Echinochloa crus-gali (L.). Beauv.) and giant foxtail (Setaria faberi Herrm.). Three corn seeds of selected hybrids were planted in 945-ml pots, containing air-dried Spinks sandy loam soil.

The plants were grown at 24 C $\pm$ 2 C with supplemental lighting from high pressure sodium lights to provide a midday light intensity of 1200 $\mu E\ m^{-2}s^{-1}$ for both supplemental and natural light. The day length was 18 h.

Soil insecticides, terbufos and brace, were incorporated into top 1.5 cm of soil. All herbicide treatments were applied with a flat-fan 8002E nozzle in a spray volume of 280 L/ha at 240 kPa using a chain link-belt compressed air sprayer. After PRE herbicide application, the pots were irrigated to activate herbicides.

Corn hybrid response

Two corn hybrids, which were previously identified as being tolerant (Cargill 7567) or sensitive (Northrup King 9283) to acetanilide herbicides were used in this study. Three corn seeds were planted 1.5 cm deep and covered with the soil which was treated with or without terbufos at 2.9 kg/ha. Metolachlor, alachlor and acetochlor at 1 and 3 times of field rates were applied preemergence with a chain link-belt compressed air sprayer. Antidote, CGA 154281, was used as a pre-mixed with metolachlor. Water was added to the soil surface for incorporation activation of the herbicide. Three factor (corn hybrid, insecticide, and herbicide) factorial, completely randomization design with four replications was used in this study.

The POST herbicides, nicosulfuron and primisulfuron, were applied when corn plants were in the three- to four-leaf stage. All postemergence treatments included 0.25% (v/v) of X-77[1].

Weed response

This study was conducted under the previously reported greenhouse conditions. To determine whether the interactions of primisulfuron with terbufos affected herbicidal activity, three weed species were planted in 945-ml pot and terbufos was

[1] X-77 Nonionic surfactant is a mixture of alkylarylpolyoxyethylene glycols, free fatty acids, and isopropanol marketed by Valent U.S.A. Corp., 1333 N. California Blvd., Walnut Creek, CA 94596.

applied preplant incorporated (PPI) at 2.9 kg/ha. Primisulfuron was applied at three rates of field rate when the weed species were at the three to five leaf stage or 5 to 8 cm of shoot height. After 14 days, plant height, visual injury, and fresh weight were taken. Three weed species, giant foxtail, barnyardgrass, and velvetleaf, were used in this study.

Data Presentation and Statistical Analysis

Shoot height, shoot fresh weight, and injury ratings were taken 3 weeks or 2 weeks, after PRE, and POST treatments, respectively. Plant injury rating was on a scale of 0 (no effect) to 100 (completely dead). The mean of two or three plants in each pot was considered one observation. The data presented are the means of two experiments. The data was analyzed for variance, and means were separated with LSD values at the 5% level of significance.

RESULTS AND DISCUSSION

Interaction of preemergence herbicides with insecticides.

There is a significant difference of plant height and visual injury of corn between corn hybrids and herbicide treatments (Tables 1 and 2). Cargill 7567 and Northrup King 9283 hybrids showed greater shoot reduction with increasing preemergence herbicides rates. Northrup King 9283 hybrid showed greater sensitivity to acetanilide herbicides than Cargill 7567 hybrid in shoot height reduction and visual injury. No interaction of preemergence herbicides with terbufos or brace was evident except that acetochlor at 6.7 kg/ha + terbufos combination enhanced recovery from high rate of acetochlor in Northrup King 9283 hybrid. The application of terbufos increased the Northrup King 9283 height from 13.7 to 21.8 cm at the acetochlor 6.7 kg/ha treatment. The Northrup King 9283 hybrid showed some visual injury even at the 2.2 kg/ha rate of acetochlor alone, or with insecticide treatment. Rowe et al. (12) reported that 'Cargill 7567' was more tolerant than 'Northrup King 9283' to metolachlor. These results conformed their report and also extended herbicide spectrum to alachlor and acetochlor.

Interaction of sulfonylurea herbicides with insecticides.

The application of sulfonylurea herbicides and imazaquin reduced the corn shoot heights of Cargill 7567 and Northrup King 9283 hybrids (Table 3). Terbufos combination with postemergence herbicides, chlorimuron, primisulfuron, and nicosulfuron further reduced the shoot height of both corn hybrids. Northrup King 9283 showed more sensitivity than Cargill 7567 to the interaction of terbufos and sulfonylurea herbicides. There was no interaction effect of imazaquin with terbufos. Morton et al. (7) reported that 'Pioneer 3751' field corn had shown more tolerance than 'Jubilee' sweet corn to the interaction of DPX-V9360 with terbufos. Also, the application of terbufos increased injury from DPX-V9360 to both of these hybrids (7). Northrup 9283 hybrid which was sensitive to acetanilide herbicides, showed more shoot reduction due to the interaction of sulfonylurea herbicides with terbufos than Cargill 7567.

Antidote effect on the interaction of herbicides with insecticides.

The application of a high rate of metolachlor induced considerable corn injury, but addition of terbufos reduced the corn injury from 69% to 28% (Table 4). The antidote CGA-154281 (premix with metolachlor) reduced the corn injury from metolachlor treatment. Chlorimuron, nicosulfuron and primisulfuron reduced corn injury from high rate of metolachlor, but increased corn injury was observed from these herbicides if terbufos was also applied. Antidote CGA-154281 reduced the corn injury from high rate of metolachlor even if applied with chlorimuron, nicosulfuron, primisulfuron and imazaquin with or without terbufos. The application of terbufos reduced the crop injury from the high rate of metolachlor treatment. The combination of metolachlor with primisulfuron and nicosulfuron reduced crop injury compare to metolachlor alone (Table 4). Rowe et al. (11) also reported CGA-154281 decreased the corn injury from high rate of metolachlor. They concluded that it was due to an enhanced rate of metabolism of metolachlor in the sensitive corn hybrids.

The seed treatment of NA reduced corn injury from the combination of terbufos with primisulfuron and nicosulfuron, but the antidote effect of NA was not enough to eliminate all injury (Table 5). The interaction of primisulfuron with terbufos caused greater injury to both corn hybrids than the nicosulfuron terbufos interaction. Seed treatment with NA decreased the visual injury to corn from the interaction of primisulfuron and nicosulfuron with terbufos (Table 6). Rehab et al. (10) reported that NA increased the metabolism of nicosulfuron and primisulfuron in corn shoots up to two-fold. Also, Corbin et al. (3) found that the shoots from NA-treated corn seed did not show the decreased metabolism of primisulfuron from adding the terbufos treatment.

Interaction effect of primisulfuron and terbufos on the herbicidal activity on weed species.

With increasing primisulfuron rate, the plant height and fresh weight of barnyardgrass, giant foxtail, and velvetleaf decreased (Table 7). The application of terbufos did not effect the herbicidal activity of primisulfuron. Arle (1) had found that the interaction of trifluralin with phorate or disulfoton had no effect on the herbicidal activity of trifluralin in annual grasses.

The synergistic effect of terbufos was evident with sulfonylurea herbicides, but not with acetanilide herbicides. The sulfonylurea and acetanilide herbicides should be metabolized by a different enzyme system. The enzyme system which metabolizes acetanilide herbicides may not sensitive to terbufos. The degree of sensitivity to the interactions of sulfonylurea herbicides and terbufos was different by hybrid. Terbufos inhibited the metabolism of sulfonylurea herbicides in corn plants. Corn injury might depend on the amount of inhibition of herbicide metabolism. Antidotes, CGA-154281 and NA, stimulated P-450 mixed function oxidase activity to increase the metabolism in corn plants. Thus, treatment of antidotes reduced the corn injury from the interaction of sulfonylurea herbicides with terbufos.

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Table 1. The combination effect of acetanilide herbicides with insecticides on the shoot height of two corn hybrids in the greenhouse 3 WAT.

Hybrids	Herbicides	Rate	Insecticides		
			Control	Terbufos[a]	Brace[b]
		(kg/ha)	-----------(cm/plant)-----------		
Cargill 7567	Control	0	48.4	48.0	46.2
	Metolachlor	2.2	45.9	45.1	47.0
		6.7	36.7	39.4	40.1
	Alachlor	2.2	47.1	47.6	48.0
		6.7	41.5	42.2	44.8
	Acetochlor	2.2	41.3	41.3	41.7
		6.7	34.1	33.8	33.4
NK 9283	Control	0	52.9	52.9	51.0
	Metolachlor	2.2	42.2	46.3	46.4
		6.7	28.5	34.0	34.7
	Alachlor	2.2	46.3	48.1	47.6
		6.7	35.7	36.0	33.2
	Acetochlor	2.2	33.0	31.1	33.7
		6.7	13.7	21.8	6.7
		LSD at 0.05		7.3	

[a] Terbufos 11.2 g/100 m row
[b] Brace 1.5 kg/ha

Table 2. The combination effect of acetanilide herbicides with insecticides on the visual injury of two corn hybrids in the greenhouse 3 WAT.

Hybrids	Herbicides	Rate	Insecticides		
			Control	Terbufos[a]	Brace[b]
		(kg/ha)	-----------(% injury)-----------		
Cargill 7567	Control	0	0	0	0
	Metolachlor	2.2	3	1	0
		6.7	15	26	14
	Alachlor	2.2	0	0	0
		6.7	7	2	3
	Acetochlor	2.2	3	5	6
		6.7	29	27	24
NK 9283	Control	0	0	0	0
	Metolachlor	2.2	9	7	5
		6.7	36	34	33
	Alachlor	2.2	3	2	2
		6.7	21	16	36
	Acetochlor	2.2	33	39	41
		6.7	84	67	85
		LSD at 0.05		15	

[a] Terbufos 11.2 g/100 m row
[b] Brace 1.5 kg/ha

Table 3. The interaction of corn hybrids, terbufos insecticide, and several POST herbicides on corn shoot height in the greenhouse 2 WAT.

		Cargill 7567		Northrup King 9283	
Herbicide	Rate	Control	Terbufos[a]	Control	Terbufos
	(g/ha)	-------------------------	(cm/plant)	-------------------------	
Control	-	39.8	39.7	37.7	35.0
Chlorimuron	12	26.4	22.6	32.3	14.0
Nicosulfuron	70	34.7	24.3	33.3	20.3
Primisulfuron	70	31.5	23.6	29.1	13.8
Imazaquin	70	24.2	25.2	14.5	13.8
LSD at 0.05			2.5		

[a] Terbufos 11.2 g/100 m row

Table 4. The interaction of terbufos insecticide with several POST herbicides on visual injury to the metolachlor sensitive Northrup King 9283 hybrid in the greenhouse 2 WAT.

		Control		Metolachlor[a]		CGA-180937[b]	
				Terbufos[c]			
Herbicide	Rate	-	+	-	+	-	+
	(g/ha)	-------------------------(% injury)-------------------------					
Control	-	0	0	69	28	8	6
Chlorimuron	12	31	85	65	75	20	60
Nicosulfuron	70	6	44	43	70	10	55
Primisulfuron	70	11	56	54	66	45	50
Imazaquin	70	65	58	70	70	53	50
LSD at O.O5				4			

[a] Metolachlor 6.7 kg/ha applied as PRE and supplied water to 12% moisture level.
[b] CGA-180937 (metolachlor + CGA 154281) 6.7 kg/ha applied as PRE and supplied water to 12% moisture level.
[c] Terbufos 11.2 g/100 m row.

Table 5. The effect of NA on the interaction between herbicides and terbufos on corn plant height in the greenhouse 2 WAT.

Hybrid	Herbicide	Rate	Control		NA[a]	
			Control	Terbufos[b]	Control	Terbufos
		(g/ha)	------------(cm/plant)-----------			
Cargill 7567	Control	-	59.4	54.3	59.9	53.6
	Nicosulfuron	52.5	59.9	46.9	57.0	53.2
	Primisulfuron	52.5	51.6	28.2	55.6	41.3
NK 9283	Control	-	63.7	56.5	60.3	57.9
	Nicosulfuron	52.5	64.8	35.8	61.9	42.3
	Primisulfuron	52.5	50.4	28.5	61.9	42.3
LSD at 0.05				**4.2**		

[a]NA seed dressing 1%(w/w)
[b]Terbufos 11.2 g/100 m row.

Table 6. The effect of NA on the interaction of sulfonylurea herbicides with terbufos on corn injury in the greenhouse 2 WAT.

			Control		NA[a]	
Hybrid	Herbicide	Rate	Control	Terbufos[b]	Control	Terbufos
		(g /ha)	---------------(% injury)--------------			
Cargill 7567	Control		0	1	0	2
	Nicosulfuron	52.5	0	14	0	3
	Primisulfuron	52.5	6	59	0	27
NK 9283	Control		0	2	0	0
	Nicosulfuron	52.5	0	38	1	4
	Primisulfuron	52.5	8	68	0	28
LSD at 0.05				**6**		

[a]NA seed dressing 1%(w/w)
[b]Terbufos 11.2 g/100 m row.

Table 7. The interaction effect on the primisulfuron herbicidal activity to three weed species in the greenhouse at 2 WAT.

Treatment	Rate	ECHCG[a] Plant ht	ECHCG[a] Fresh wt	SETFA[b] Plant ht	SETFA[b] Visual injury	SETFA[b] Fresh wt	ABUTH[c] Plant ht	ABUTH[c] Visual injury	ABUTH[c] Fresh wt
	(g/ha)	(cm/plant)	(g/plant)	(cm/plant)	(%)	(g/plant)	(cm/plant)	(%)	(g/plant)
Control	-	61.7	11.2	42.1	0	5.8	16.9	0	3.5
Terbufos	2858	64.3	11.6	42.6	0	5.2	16.7	1	3.1
Primisulfuron	2.8	61.3	10.2	20.6	43	2.7	13.7	13	2.1
+ Terbufos		59.7	11.6	18.2	45	4.4	13.2	15	1.9
Primisulfuron	14.2	44.6	7.9	16.5	57	0.8	10.5	52	1.7
+ Terbufos		48.6	8.2	16.2	55	0.8	10.3	53	1.6
Primisulfuron	28.3	-	-	14.6	67	0.6	7.8	74	0.7
+ Terbufos		-	-	16.0	68	0.7	8.1	71	0.9
LSD at 0.05		5.0	2.5	3.7	13	2.7	2.3	12	0.8

[a] ECHCG: Barnyardgrass
[b] SETFA: Giant foxtail
[c] ABUTH: Velvetleaf

CHAPTER 3

The Effect of Mixed Function Oxidase Inhibitors on Sulfonylurea Herbicide Activity

ABSTRACT

Greenhouse studies were conducted to evaluate the effect of mixed function oxidase (MFO) inhibitors or antioxidants on the efficacy of sulfonylurea herbicides, and to identify effective adjuvants for the sulfonylurea herbicides with 28% UAN (Urea Ammonium Nitrate) or piperonyl butoxide or both. Tank-mixed PBO enhanced nicosulfuron and thifensulfuron activity on barnyardgrass, velvetleaf and common lambsquarters, respectively. Also, BHA and PBO enhanced nicosulfuron and primisulfuron activities on common lambsquarters and green foxtail. The optimal rates of PBO ranged from 1 to 6 kg/ha dependent on weed species and herbicide. All three factors, PBO, nonionic adjuvants, and 28% UAN, enhanced activity of nicosulfuron on common lambsquarters, velvetleaf, barnyardgrass and giant foxtail, primisulfuron on giant foxtail and velvetleaf, and thifensulfuron on common lambsquarters and velvetleaf. But 28% UAN did not increase activity of primisulfuron on barnyardgrass, or common lambsquarters. Effective adjuvants with nicosulfuron were K-3000 on common lambsquarters,

and SYLGARD 309 on velvetleaf, K-2000, K-3000 and SCOIL on barnyardgrass. Effective adjuvants with primisulfuron were K-2000, SCOIL, and SYLGARD 309 on giant foxtail, X-77, K-2000, K-3000, SCOIL, and SYLGARD 309 on velvetleaf, K-3000 and SYLGARD 309 on common lambsquarters. Effective adjuvants for thifensulfuron were SCOIL on common lambsquarters, and SCOIL and SYLGARD 309 on velvetleaf.

Nomenclature: nicosulfuron, 2-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-N,N-dimethyl-3-pyridinecarboxamide; primisulfuron, 2-[[[[[4,6-bis(difluoromethoxy)-2-pyrimidinyl] amino] carbonyl] amino] sulfonyl] benzoic acid; thifensulfuron, 3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino] carbonyl] amino] sulfonyl]-2-thiophenecarboxylic acid; PBO, α-(2-(2-butoxyethoxy) ethoxy)-4,5-methyl enedioxy-2-propyltoluene; barnyardgrass, Echinochloa crus-galli (L.) Beauv.; common lambsquarters, Chenopodium album L.; giant foxtail, Setaria faberi Herrm.; green foxtail, Setaria viridis (L.) Beauv.; velvetleaf, Abutilon theophrasti Medicus.

Additional index words: 28% UAN, CHEMPRO, K-2000, K-3000, SCOIL, SYLGARD 309, X-77.

INTRODUCTION

Higher plants may metabolize xenobiotics that they absorb through their roots and leaves. The monooxygenases are enzymes involved in oxidative transformations forming primary metabolites. Mixed function oxidase (MFO) inhibitors, or antioxidants, are compounds used to prevent oxidative reactions. For these reasons, MFO inhibitors have been widely used to protect foods and other products from discoloration and spoilage.

O'Brien introduced piperonyl butoxide (PBO) and sesamex (a component of sesame oil) as insecticide synergists (6). PBO, the most effective and widely used insecticide synergist, enhances activity of many organophosphate, carbamate, and pyrethroid insecticides (6). Insecticide synergists are potentially important pest management compounds because they may increase insecticidal activity against resistant insects (1), enhance cost effectiveness, and natural enemy survival (7), and decrease environmental impact by using lower rate of toxic insecticides. PBO significantly enhances the toxicity of certain insecticides because it inhibits microsomal detoxification enzymes (3).

Komives et al. (5) and Rubin et al. (9) reported that PBO also acted as a herbicide synergist. PBO increased the phytotoxicity of EPTC [S - ethyl dipropyl carbamothioate], atrazine [6-chloro-N-ethyl-N' (1-methylethyl)-1,3,5-triazine-2,4-

diamine], bentazon [3-(1-methylethyl)-(1H)-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide], and oxadiazon [3-(2,4-dichloro-5-(1-methylethoxy)phenyl)-5-(1,1-dimethylethyl)-1,3,4-oxadiazol -2-(3H)-one] to corn (Zea mays L.)(5,9) and of atrazine and bentazon to soybean (Glycine max (L.) Merr.) (9). They proposed that antioxidants inhibited mixed-function oxidase (MFO) systems which were possibly involved in the metabolism of these herbicides in corn or soybean. Also, Hatzios (4) reported a combination effect of PBO and metolachlor [2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl) acetamide] on the growth of sorghum (Sorghum bicolor (L.) Moench 'Funk G522DR'] seedlings and called this a synergistic effect. Varsano et al. (10) observed that PBO reversed the chloroplastic triazine resistance in a resistant biotype of ryegrass (Lolium rigidum GAUD.), both at the whole plant and isolated chloroplasts levels.

Rehab et al. (8) reported that bentazon, tetcyclacis [5-(4-chlorophenyl)-3,4,5,9,10-pentaazatetracyclo [5.4.$10^{2,6}$,$0^{8,11}$]-dodeca-3,9-diene], and PBO inhibited the metabolism of both primisulfuron and nicosulfuron in corn shoot tissue, but not tridiphane [2-(3,5-dichlorophenyl-2-(2,2,2-trichloroethyl) oxirane].

The objectives of this study were a) to evaluate the effect of antioxidants on the activity of several sulfonylurea herbicides on several weed species, b) to determine the optimal rate of PBO, and c) to identify effective adjuvants for the sulfonylurea herbicides applied with or without PBO.

MATERIALS AND METHODS

Plant Materials

Five weed species, barnyardgrass, green foxtail, giant foxtail, common lambsquarters, and velvetleaf, were used in this study. Weed seeds were planted in 945-ml pots, which contained BACCTO media. After emergence, the plants were thinned to two plants per pot for barnyardgrass, green foxtail, and giant foxtail, and one plant per pot for common lambsquarters and velvetleaf. The POST herbicides, nicosulfuron, primisulfuron, and thifensulfuron, were applied when the weed species were at the three to five leaf stage or 5 to 8 cm shoot height. All postemergence treatments were applied with a flat-fan 8002E nozzle in a spray volume of 280 L/ha at 240 kPa using a chain link-belt compressed air sprayer. All postemergence treatments included 0.25% (v/v) of X-77[1]

MFO Inhibitor

Two MFO inhibitors, PBO, and butylated hydroxyanisole [2,[3]-tert-butyl-4-hydroxy-anisole : BHA], were evaluated for effect on the herbicidal activities of the sulfonylurea herbicides, nicosulfuron, primisulfuron, and thifensulfuron.

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[1]X-77 nonionic surfactant is a mixture of alkylarylpolyoxyethylene glycols, free fatty acids, and isopropanol marketed by Valent U.S.A. Crop., 1333 N. California Blvd., Walnut Creek, CA 94596.

INCITE[2] (insecticide synergist) was used as the PBO source and contained 92 % technical PBO. PBO and BHA at 4 kg/ha were applied with nicosulfuron and primisulfuron to common lambsquarters and green foxtail. PBO at 2 kg/ha was applied with thifensulfuron. To determine the optimal rate of PBO, rates of 1, 2, 4, and 6 kg/ha were applied in combination with nicosulfuron and primisulfuron.

Adjuvant studies

Four weed species, common lambsquarters, velvetleaf, giant foxtail, and barnyardgrass, were used to identify the most effective adjuvants for the sulfonylurea herbicides, nicosulfuron, primisulfuron and thifensulfuron, with PBO and 28% UAN (containing urea and ammonium nitrate).

Five adjuvants were tested with nicosulfuron. X-77 was added as a standard adjuvant for primisulfuron and thifensulfuron. The concentration of adjuvants was 0.25%, 1.25%, 1%, 1%, 1%, and 0.5% (v/v) for X-77, CHEMPRO[3], K-2000[4], K-3000[5], SCOIL[6], and SYLGARD 309[7], respectively. Four rates, 0, 0.1, 0.5,

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[2]INCITE sold by Loveland Industries Inc. Greeley, Colorado 80632.

[3]CHEMPRO: Chemorse LTD., Des Moines, Iowa 50322.

[4]K-2000: Central Soya. Fort Wayne, IN 46801.

[5]K-3000: Central Soya. Fort Wayne, IN 46801.

[6]SCOIL: Methylated seed oil from AGSCO Inc., Grand Forks, ND.

[7]SYLGARD 309: Dow Corning Corp, Midland, MI 48686.

and 1.0 kg/ha of PBO were applied with nicosulfuron, and three rates, 0, 0.5, and 1.0 kg/ha, of PBO were applied with primisulfuron and thifensulfuron. 4% (v/v) of 28% UAN was used.

The treatments were evaluated 2 weeks after treatments. Plant height and visual injury were recorded. Plant injury rating was on a scale of 0 (no injury) to 100 (completely dead). The mean of two plants in each pot was considered one observation for corn hybrids, barnyardgrass, green foxtail, and giant foxtail.

Statistical Analysis

All experiments were conducted separately on each species. A three-factor completely randomized design was used in PBO rates and adjuvant experiments. Each treatment was replicated four times and the data presented are the means of two experiments. The data were analyzed by ANOVA and means separated with LSD values at the 5% level of significance.

RESULTS AND DISCUSSION

Effects of MFO inhibitors on sulfonylurea herbicide activity.

Nicosulfuron was more effective for control of barnyardgrass than primisulfuron (Table 1). PBO tank-mixed with nicosulfuron increased the herbicidal activity to barnyardgrass, but not with primisulfuron. Nicosulfuron tank-mixed with PBO decreased plant height and fresh weight, and increased visual injury of barnyardgrass more than nicosulfuron alone.

Tank-mixture of PBO and BHA with nicosulfuron and primisulfuron increased the herbicidal activities of both herbicides on common lambsquarters (Table 2). PBO and BHA tank-mixed with primisulfuron enhanced control of common lambsquarters. Nicosulfuron at 7 g ai/ha plus PBO was more effective than nicosulfuron 35 g ai/ha alone. The synergistic action of PBO and BHA with nicosulfuron was greater than with primisulfuron for control of common lambsquarters.

PBO increased the phytotoxicity of primisulfuron and nicosulfuron on green foxtail, but BHA did not (Table 3). Addition of PBO to primisulfuron increased visual injury and decreased fresh weight, but not plant height of green foxtail. Tank-mixed PBO increased nicosulfuron phytotoxicity on green foxtail as measured by the effects on plant height, fresh weight, and visual injury.

Nicosulfuron at 7 g ai/ha applied with PBO showed the same effective green foxtail control as nicosulfuron at 35 g ai/ha alone. Nicosulfuron provided greater control of green foxtail than primisulfuron.

PBO tank-mixed with thifensulfuron increased visual injury and decreased plant height of velvetleaf and common lambsquarters (Table 4). The application of 1.1 g ai/ha of thifensulfuron with PBO decreased plant height from 17.1 to 12.5 cm, and increased visual injury from 24 to 52% compared to thifensulfuron alone. The lowest rate, 0.6 g ai/ha of thifensulfuron, tank-mixed with PBO increased the herbicidal activity to common lambsquarters up to the 2.2 g ai/ha rate of thifensulfuron alone.

PBO applied with various rates of thifensulfuron to barnyardgrass and giant foxtail did not significantly increase thifensulfuron activity, except for thifensulfuron at 4.5 g ai/ha plus PBO applied for giant foxtail control (Table 5).

Tank-mixing of PBO with thifensulfuron was more effective on broadleaf weed species than grass weed species in increasing weed control (Tables 4 and 5). Mixed function oxidases, PBO and BHA, inhibit oxidative reactions in plants. PBO may inhibit the metabolism of sulfonylurea herbicides (8). Due to the inhibition of sulfonylurea herbicide metabolism in the weed species, tank-mixed PBO may be useful in enhancing activity of nicosulfuron, primisulfuron, and thifensulfuron. The synergistic effect of PBO was greater on broad leaf than grass weed species.

Effects of PBO rates on sulfonylurea herbicide activity.

Two factors, herbicides and PBO rates, affected plant height and visual injury of common lambsquarters in the greenhouse (Table 6). The synergist action of PBO was evident with both herbicides on the growth of common lambsquarters. PBO alone had no effect on the growth of common lambsquarters or green foxtail (Tables 6 and 7). As PBO rates increased from 1 to 6 kg ai/ha, nicosulfuron activity on common lambsquarters and green foxtail increased (Tables 6 and 7). An increase in primisulfuron activity with increasing rates of PBO was not as evident, although PBO significantly increased primisulfuron activity to both common lambsquarters and green foxtail.

Effects of nonionic adjuvants, PBO rates and 28% UAN on the phytotoxicity of sulfonylurea herbicides.

Nicosulfuron: All three factors, PBO, nonionic adjuvants, and 28% UAN, increased nicosulfuron activity on common lambsquarters in the greenhouse study. Application of 35 g ai/ha of nicosulfuron alone had no effect on the growth of common lambsquarters (Table 8). PBO tank-mixed with nicosulfuron reduced plant height and increased visual injury. Addition of 28% UAN to nicosulfuron enhanced common lambsquarters control. All adjuvants treatments increased nicosulfuron activity. Among adjuvants, K-3000 appeared most effective on common lambsquarters. Tank-mixing 1 kg/ha of PBO with nicosulfuron enhanced the visual injury from 76 to 95%, and from 69 to 80%, with SCOIL and SYLGARD 309, respectively. The addition of 28% UAN to CHEMPRO, or K-2000 did not affect phytotoxicity. The addition of 28% UAN to K-2000 plus PBO increased the visual injury up to 0.5 kg/ha rate of PBO.

All three factors, nonionic adjuvants, PBO, and 28% UAN, increased nicosulfuron activity on velvetleaf. Tank-mixed PBO increased nicosulfuron activity to velvetleaf in the absence of an adjuvant or with 28% UAN, CHEMPRO $\pm$ 28% UAN, and SCOIL (Table 9). Addition of 28% UAN alone enhanced velvetleaf control, and in combined with CHEMPRO, K-2000 and SCOIL. Combination of PBO and 28% UAN increased nicosulfuron activity in the absence of an adjuvant, or with CHEMPRO, K-2000, and SCOIL. All adjuvant treatments increased nicosulfuron activity to velvetleaf. Addition of 28% UAN with SCOIL and K-2000 increased velvetleaf control at all PBO rates. SYLGARD 309 adjuvant was more effective than the other nonionic adjuvants for velvetleaf control with nicosulfuron. All treatments of SYLGARD 309 adjuvant showed above 88% of visual injury and below 39% of plant height. From the results, SYLGARD 309 plus 28% UAN appeared to be the most effective adjuvant for velvetleaf control with nicosulfuron (Table 9).

All three factors, PBO, nonionic adjuvants, and 28% UAN, increased nicosulfuron activity on barnyardgrass. Nicosulfuron at 2.8 g ai/ha alone or with 28% UAN did not affect the growth of barnyardgrass (Table 10). Tank-mixing of 1 kg/ha of PBO with nicosulfuron increased barnyardgrass control without nonionic adjuvant. The 28% UAN alone did not increase nicosulfuron activity to barnyardgrass. All nonionic adjuvants increased barnyardgrass control with nicosulfuron. K-2000 appeared to be the most effect of the nonionic adjuvants and SYLGARD 309 the least effective with nicosulfuron for barnyardgrass control (Table 10). Also, combination of 28% UAN and SYLGARD 309 with nicosulfuron increased barnyardgrass control about 20 to 25%. From the results, K-2000, K-3000 and SCOIL adjuvants were considered good adjuvants to control barnyardgrass with nicosulfuron. With SYLGARD 309, the additions of 28% UAN and PBO were strongly recommended.

All three factors, PBO, nonionic adjuvants, and 28% UAN, enhanced giant foxtail control by nicosulfuron 6 g ai/ha. Nicosulfuron alone or plus 28% UAN did not affect the growth of giant foxtail (Table 11). Tank-mixed PBO with nicosulfuron increased activity to giant foxtail. In the absence of any nonionic adjuvant, the addition of 1 kg/ha PBO ± 28% UAN increased visual injury to giant foxtail by 48%. All adjuvants increased giant foxtail control with nicosulfuron. With all of the weed species, it appeared that as weed control increased with the addition of nonionic adjuvant and 28% UAN, the effect of PBO became less evident.

Primisulfuron:

Two factors, PBO and nonionic adjuvants, gave a significant increase to primisulfuron activity on plant height of common lambsquarters, but, the addition of 28% UAN did not (Table 12). From the visual injury data, it appeared that only nonionic adjuvants were effective in increasing primisulfuron activity. Application of 21 g ai/ha of primisulfuron alone reduced the growth of common lambsquarters, and tank-mixed PBO provided greater reduction of plant height. Addition of any of the adjuvants to primisulfuron decreased plant height of common lambsquarters.

All three factors, PBO, nonionic adjuvants and 28% UAN, increased primisulfuron activity on velvetleaf. The 7 g ai/ha rate of primisulfuron alone did not affect the growth of velvetleaf, but addition of PBO and/or 28% UAN increased visual injury up to 89% and reduced plant height up to 42% of control without any adjuvant (Table 13). In the absence of 28% UAN and PBO, the adjuvant K-3000 and SYLGARD 309 provided exceptionally good enhancement of primisulfuron activity on velvetleaf. With 28% UAN and 1 kg/ha PBO, all nonionic adjuvant provided excellent enhancement of primisulfuron activity.

Two factors, PBO and nonionic adjuvants, increased primisulfuron activity on barnyardgrass, but addition of 28% UAN did not change primisulfuron activity. Primisulfuron at 35 g ai/ha was not an effective treatment for barnyardgrass control with any adjuvant (Table 14). Only K-2000 enhanced primisulfuron activity. If 1 kg/ha PBO was also applied than SCOIL + 28% UAN and

SYLGARD 309 also enhanced primisulfuron activity.

All three factors, PBO, nonionic adjuvants, and 28% UAN, increased primisulfuron activity on giant foxtail (Table 15). In the absence of 28% UAN and PBO the nonionic adjuvant, CHEMPRO, K-2000, K-3000, and SCOIL were most effective in increasing primisulfuron activity on giant foxtail. If both 28% UAN and 1 kg/ha PBO were present all the nonionic adjuvants were equally good.

Thifensulfuron:

All three factors, PBO, nonionic adjuvants, and 28% UAN, increased thifensulfuron activity on common lambsquarters in the greenhouse study (Table 16). Treatment of 0.6 g ai/ha of thifensulfuron alone had no effect on the growth of common lambsquarters. Tank-mixed PBO increased thifensulfuron activity, but 28% UAN did not in the absence of nonionic adjuvants. All adjuvants increased thifensulfuron activity on common lambsquarters. Addition of 28% UAN with SYLGARD 309 significantly enhanced thifensulfuron activity on visual injury of common lambsquarters. Fielding et al. (2) found that addition of 28% UAN significantly increased velvetleaf control, but, it did not enhance common lambsquarters control. The effect of 28% UAN may be dependent on weed species, adjuvant, and herbicides.

All three factors, PBO, nonionic adjuvants, and 28% UAN, increased thifensulfuron activity on velvetleaf. Treatment of 1.5 g ai/ha of thifensulfuron alone did not effect on the growth of velvetleaf (Table 17). Tank-mixed PBO and/or 28% UAN increased thifensulfuron activities on velvetleaf. Addition of 28% UAN plus PBO to thifensulfuron provided 81% visual injury without any adjuvant. Tank-mixed PBO and 28% UAN enhanced velvetleaf control with K-2000, SCOIL adjuvants, but if both were added, there was no PBO effect. SYLGARD 309 adjuvant efficacy was greatly increased by the addition of 28% UAN.

The results of this study are consistent with the hypothesis that at sublethal rates of the herbicide, the activity of these herbicide is a function of the level of the free active herbicide at the site of action. This level is a function of the rate and amount of herbicide absorption and the rate of herbicide metabolism. Since the sulfonylurea herbicides appear to be metabolized at least to a limited degree in the weed species studies, blocking this metabolism with PBO and increasing herbicide absorption with effective adjuvant or 28% UAN can raise the herbicide activity to its maximum potential. Similarly if the absorption rate is enhanced or accelerated, metabolism is overwhelmed and maximum potential herbicide activity is observed. This hypothesis explains why a certain maximum level of herbicide activity is observed and why in the presence of effective adjuvants, no effect of PBO is observed.

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Table 1. The effect of PBO on primisulfuron and nicosulfuron activity to barnyardgrass in the greenhouse 2 WAT.

Treatment	Rate	Plant height	Visual injury	Fresh weight
	(g ai/ha)	(cm/plant)	(% injury)	(g/plant)
Control	-	55.4	0	21.0
PBO	4000	51.1	0	21.3
Primisulfuron	28	36.4	22	11.4
+ PBO	28 + 4000	34.8	23	10.3
Nicosulfuron	4	23.1	61	4.5
+ PBO	4 + 4000	17.5	86	1.2
LSD at 0.05		3.2	7	2.1

Table 2. The effect of PBO and BHA on primisulfuron and nicosulfuron activity to common lambsquarters in the greenhouse 2 WAT.

Treatment	Rate	Plant height	Visual injury	Fresh weight
	(g ai/ha)	(cm/plant)	(% injury)	(g/plant)
Control	-	21.5	0	7.1
PBO	4000	20.4	0	6.6
BHA	4000	20.2	0	6.5
Primisulfuron	35	8.9	69	1.3
+ PBO	35 + 4000	4.7	86	0.5
+ BHA	35 + 4000	6.6	86	0.4
Nicosulfuron	7	17.6	4	6.4
+ PBO	7 + 4000	7.5	72	1.1
Nicosulfuron	35	11.1	40	3.9
+ PBO	35 + 4000	5.5	92	0.4
+ BHA	35 + 4000	5.9	89	0.3
LSD at 0.05		2.0	8	0.9

Table 3. The effect of PBO and BHA on primisulfuron and nicosulfuron activity to green foxtail in the greenhouse 2 WAT.

Treatment	Rate	Plant height	Visual injury	Fresh weight
	(g ai/ha)	(cm/plant)	(% injury)	(g/plant)
Control	-	38.1	0	12.6
PBO	4000	35.1	0	10.6
BHA	4000	37.5	0	12.6
Primisulfuron	35	14.5	53	1.9
+ PBO	35 + 4000	12.6	72	0.7
+ BHA	35 + 4000	14.7	48	2.2
Nicosulfuron	7	15.5	51	1.6
+ PBO	7 + 4000	8.9	91	0.3
Nicosulfuron	35	9.5	86	0.3
+ PBO	35 + 4000	1.4	98	0.2
+ BHA	35 + 4000	9.2	85	0.4
LSD at 0.05		2.8	5	0.9

Table 4. The effect of PBO on the herbicidal activity of thifensulfuron on the growth of velvetleaf and common lambsquarters in the greenhouse 2 WAT.

		Velvetleaf		Common lambsquarters	
Treatment	Rate	Plant ht	Injury	Plant ht	Injury
	(g ai/ha)	(cm/plant)	(%)	(cm/plant)	(%)
Control	-	27.5	0	28.5	0
PBO	2000	27.6	0	28.5	0
Thifensulfuron	0.6	21.1	17	13.5	51
+ PBO	O.6 + 2000	18.3	24	7.0	74
Thifensulfuron	1.1	17.1	24	6.6	69
+ PBO	1.1 + 2000	12.5	52	6.8	84
Thifensulfuron	2.2	10.4	66	6.7	78
+ PBO	2.2 + 2000	10.1	76	6.7	91
LSD at 0.05		**2.9**	**8**	**2.0**	**8**

Table 5. The effect of PBO on the herbicidal activity of thifensulfuron on the plant height of barnyardgrass and giant foxtail in the greenhouse 2 WAT.

Treatment	Rate	Barnyardgrass	Giant foxtail
	(g ai/ha)	(cm/plant)	(cm/plant)
Control	-	64.3	68.4
PBO	2000	65.1	67.7
Thifensulfuron	1.1	64.7	68.8
+ PBO	1.1 + 2000	64.1	61.1
Thifensulfuron	2.2	63.9	67.1
+ PBO	2.2 + 2000	63.7	63.1
Thifensulfuron	4.5	63.8	67.3
+ PBO	4.5 + 2000	63.0	53.1
LSD at 0.05		NS	8.6

Table 6. The effect of various PBO rates on nicosulfuron and primisulfuron activity on common lambsquarters in the greenhouse 2 WAT.

Treatment	Rate	Plant height	Fresh weight	Visual injury
	(g ai/ha)	(cm/plant)	(g/plant)	(% injury)
Control	-	13.9	3.8	0
PBO	1000	13.3	4.4	0
PBO	2000	13.5	4.6	0
PBO	4000	13.0	4.1	0
PBO	6000	13.1	4.0	0
Nicosulfuron	35	8.7	2.9	49
+ PBO	35 + 1000	6.9	1.9	54
+ PBO	35 + 2000	5.7	1.1	73
+ PBO	35 + 4000	5.4	0.9	76
+ PBO	35 + 6000	5.4	0.7	81
Primisulfuron	21	6.4	1.4	57
+ PBO	21 + 1000	5.6	0.8	73
+ PBO	21 + 2000	5.5	0.7	81
+ PBO	21 + 4000	5.2	0.7	80
+ PBO	21 + 6000	5.6	1.5	68
LSD at 0.05		1.7	0.9	9

Table 7. The effect of various PBO rates on nicosulfuron and primisulfuron activity on green foxtail in the greenhouse 2 WAT.

Treatment	Rate	Plant height	Fresh weight	Visual injury
	(g ai/ha)	(cm/plant)	(g/plant)	(% injury)
Control	-	32.6	7.7	0
PBO	1000	31.7	8.6	0
PBO	2000	32.9	9.0	0
PBO	4000	32.1	7.6	0
PBO	6000	29.6	6.9	0
Nicosulfuron	6	22.2	3.1	51
+ PBO	6 + 1000	20.6	1.6	55
+ PBO	6 + 2000	20.8	1.5	59
+ PBO	6 + 4000	19.7	1.2	76
+ PBO	6 + 6000	19.8	1.1	84
Primisulfuron	21	21.7	3.3	44
+ PBO	21 + 6000	19.7	1.9	57
+ PBO	21 + 4000	19.6	1.9	53
+ PBO	21 + 2000	19.4	1.7	56
+ PBO	21 + 1000	19.2	1.6	60
LSD at 0.05		3.3	1.1	7

Table 8. The effect of various PBO rates and adjuvants on nicosulfuron activity on the growth of common lambsquarters in the greenhouse 2 WAT.

Nicosulfuron (35 g ai/ha) + Adjuvant	Rate	Plant height 0	Plant height 0.1	Plant height 0.5	Plant height 1.0	Visual injury 0	Visual injury 0.1	Visual injury 0.5	Visual injury 1.0
		PBO rate (kg/ha)							
	(% (v/v))	(% of control)				(% injury)			
None		102	91	75	65	0	5	9	17
+ 28% UAN	4	102	79	66	48	0	9	27	46
CHEMPRO	1.25	24	29	26	30	79	73	84	80
+ 28% UAN	1.25 + 4	27	27	25	31	82	76	84	81
K-2000	1	26	28	30	29	76	77	76	79
+ 28% UAN	1 + 4	27	27	27	30	84	89	85	77
K-3000	1	27	28	26	29	94	89	88	87
+ 28% UAN	1 + 4	25	30	29	28	94	86	85	87
SCOIL	1	28	28	29	30	76	83	83	95
+ 28% UAN	1 + 4	26	27	29	31	89	91	85	83
SYLGARD 309	0.5	32	28	33	29	69	77	68	80
+ 28% UAN	0.5 + 4	29	27	30	30	79	77	79	83
LSD at 0.05		7				10			

Table 9. The effect of various PBO rates and adjuvants on nicosulfuron activity on the growth of velvetleaf in the greenhouse 2 WAT.

Nicosulfuron (35 g ai/ha)		Plant height				Visual injury			
		PBO rate (kg/ha)							
+ Adjuvant	Rate	0	0.1	0.5	1.0	0	0.1	0.5	1.0
	(% (v/v))	(% of control)				(% injury)			
None		93	84	84	79	3	17	19	21
+ 28% UAN	4	88	71	73	69	13	24	37	41
CHEMPRO	1.25	66	59	50	53	44	43	64	69
+ 28% UAN	1.25 + 4	56	52	43	46	58	59	79	71
K-2000	1	67	62	61	60	38	46	48	58
+ 28% UAN	1 + 4	48	50	49	45	66	56	68	71
K-3000	1	45	49	54	49	66	68	64	66
+ 28% UAN	1 + 4	43	45	46	40	69	72	75	81
SCOIL	1	62	57	53	49	49	54	57	66
+ 28% UAN	1 + 4	43	41	42	43	81	82	76	81
SYLGARD 309	0.5	38	33	39	38	88	95	88	94
+ 28% UAN	0.5 + 4	31	32	36	34	98	96	94	95
LSD at 0.05		--------- 10 ---------				--------- 14 ---------			

Table 10. The effect of various PBO rates and adjuvants on nicosulfuron activity on the growth of barnyardgrass in the greenhouse 2 WAT.

Nicosulfuron (2.8 g ai/ha) + Adjuvant	Rate	Plant height 0	Plant height 0.1	Plant height 0.5	Plant height 1.0	Visual injury 0	Visual injury 0.1	Visual injury 0.5	Visual injury 1.0
		PBO rate (kg/ha)							
	(% (v/v))	(% of control)				(% injury)			
None		100	96	97	74	0	3	8	19
+ 28% UAN	4	101	95	89	73	0	2	6	19
CHEMPRO	1.25	49	47	53	49	69	66	64	70
+ 28% UAN	1.25 + 4	46	49	50	46	74	68	69	73
K-2000	1	46	45	49	53	79	76	74	73
+ 28% UAN	1 + 4	47	48	47	48	81	79	76	74
K-3000	1	50	48	48	48	73	73	73	74
+ 28% UAN	1 + 4	49	50	46	47	79	77	74	78
SCOIL	1	50	51	49	50	71	70	71	68
+ 28% UAN	1 + 4	48	48	47	50	80	76	74	77
SYLGARD 309	0.5	80	76	70	67	17	21	45	50
+ 28% UAN	0.5 + 4	62	55	53	48	42	46	65	71
LSD at 0.05		11				6			

Table 11. The effect of various PBO rates and adjuvants on nicosulfuron activity on the growth of giant foxtail in the greenhouse 2 WAT.

Nicosulfuron (6 g ai/ha)		Plant height				Visual injury			
		PBO rate (kg/ha)							
+ Adjuvant	Rate	0	0.1	0.5	1.0	0	0.1	0.5	1.0
	(% (v/v))	(% of control)				(% injury)			
None		100	90	78	52	0	4	28	49
+ 28% UAN	4	94	79	66	53	3	11	34	52
CHEMPRO	1.25	44	45	43	43	67	70	68	70
+ 28% UAN	1.25 + 4	42	42	45	41	73	74	70	73
K-2000	1	38	41	42	42	76	74	79	73
+ 28% UAN	1 + 4	39	41	42	40	76	76	75	77
K-3000	1	40	44	46	44	72	72	74	74
+ 28% UAN	1 + 4	39	41	42	40	79	76	74	81
SCOIL	1	40	42	45	41	72	71	73	76
+ 28% UAN	1 + 4	40	41	39	43	75	71	78	78
SYLGARD 309	0.5	43	43	44	41	62	68	65	69
+ 28% UAN	0.5 + 4	40	45	43	41	73	69	73	72
LSD at 0.05		6				8			

Table 12. The effect of various PBO rates and adjuvants on primisulfuron activity on the growth of common lambsquarters in the greenhouse 2 WAT.

		Plant height			Visual injury		
Primisulfuron (21 g ai/ha)		PBO rate (kg/ha)					
+ Adjuvant	Rate	0	0.5	1.0	0	0.5	1.0
	(% (v/v))	(% of control)			(% injury)		
None	-	59	29	29	59	66	70
+ 28% UAN	4	61	27	30	59	69	68
X-77	0.5	21	22	31	88	81	73
+ 28% UAN	0.5 + 4	24	27	24	79	81	91
CHEMPRO	1.25	23	20	26	80	81	71
+ 28% UAN	1.25 + 4	24	20	29	78	78	66
K-2000	1	27	22	32	74	80	66
+ 28% UAN	1 + 4	22	21	35	83	80	60
K-3000	1	21	23	23	93	74	84
+ 28% UAN	1 + 4	21	28	24	86	66	79
SCOIL	1	19	27	28	84	68	75
+ 28% UAN	1 + 4	26	25	26	75	75	78
SYLGARD 309	0.5	24	25	31	74	70	63
+ 28% UAN	0.5 + 4	24	25	28	95	66	78
LSD at 0.05		------- 7 -------			------- 19 -------		

Table 13. The effect of various PBO rates and adjuvants on primisulfuron activity on the growth of velvetleaf in the greenhouse 2 WAT.

		Plant height			Visual injury		
Primisulfuron (7 g ai/ha)		PBO rate (kg/ha)					
+ Adjuvant	Rate	0	0.5	1.0	0	0.5	1.0
	(% (v/v))	(% of control)			(% injury)		
None	-	102	76	59	0	14	53
+ 28% UAN	4	62	49	42	34	85	89
X-77	0.5	89	73	41	7	17	91
+ 28% UAN	0.5 + 4	43	35	43	86	91	92
CHEMPRO	1.25	56	47	49	48	71	72
+ 28% UAN	1.25 + 4	43	38	39	89	93	91
K-2000	1	66	51	43	31	66	83
+ 28% UAN	1 + 4	42	39	40	81	89	96
K-3000	1	40	39	42	91	91	95
+ 28% UAN	1 + 4	36	33	39	97	99	96
SCOIL	1	48	43	44	65	83	81
+ 28% UAN	1 + 4	38	36	38	98	97	97
SYLGARD 309	0.5	37	44	47	93	86	84
+ 28% UAN	0.5 + 4	35	40	43	99	96	94
LSD at 0.05		------- 10 --------			------- 12 --------		

Table 14. The effect of various PBO rates and adjuvants on primisulfuron activity on the plant height of barnyardgrass in the greenhouse 2 WAT.

Primisulfuron (35 g ai/ha)		PBO (kg/ha)		
+ Adjuvant	Rate	0	0.5	1.0
	(% (v/v))	(% of control)		
None	-	101	101	100
+ 28% UAN	4	104	100	100
X-77	0.5	103	99	97
+ 28% UAN	0.5 + 4	104	102	101
CHEMPRO	1.25	101	101	95
+ 28% UAN	1.25 + 4	102	101	100
K-2000	1	83	87	83
+ 28% UAN	1 + 4	79	92	89
K-3000	1	102	100	100
+ 28% UAN	1 + 4	98	102	98
SCOIL	1	95	100	93
+ 28% UAN	1 + 4	93	98	90
SYLGARD 309	0.5	101	95	85
+ 28% UAN	0.5 + 4	101	100	90
LSD at 0.05		———— 8 ————		

Table 15. The effect of various PBO rates and adjuvants on primisulfuron activity on the growth of giant foxtail in the greenhouse 2 WAT.

		Plant height			Visual injury		
Primisulfuron (21 g ai/ha)		PBO rate (kg/ha)					
+ Adjuvant	Rate	0	0.5	1.0	0	0.5	1.0
	(% (v/v))	(% of control)				(% injury)	
None	-	81	71	61	10	40	53
+ 28% UAN	4	90	70	56	10	45	56
X-77	0.5	69	58	48	31	56	64
+ 28% UAN	0.5 + 4	63	51	45	39	65	71
CHEMPRO	1.25	58	45	45	61	74	71
+ 28% UAN	1.25 + 4	56	48	46	55	70	74
K-2000	1	53	47	52	56	67	62
+ 28% UAN	1 + 4	50	38	46	62	82	77
K-3000	1	55	47	51	63	66	61
+ 28% UAN	1 + 4	49	42	44	70	73	73
SCOIL	1	50	42	39	66	74	77
+ 28% UAN	1 + 4	48	41	38	77	76	84
SYLGARD 309	0.5	69	57	42	40	58	74
+ 28% UAN	0.5 + 4	57	44	40	64	84	79
LSD at 0.05		------- 16 -------			------- 13 -------		

Table 16. The effect of various PBO rates and adjuvants on thifensulfuron activity on the growth of common lambsquarters in the greenhouse 2 WAT.

		Plant height			Visual injury		
Thifensulfuron (0.6 g ai/ha)		PBO rate (kg/ha)					
+ Adjuvant	Rate	0	0.5	1.0	0	0.5	1.0
	(% (v/v))	(% of control)			(% injury)		
None	-	97	37	35	1	60	74
+ 28% UAN	4	89	35	35	3	63	76
X-77	0.5	40	39	36	51	57	73
+ 28% UAN	0.5 + 4	36	36	38	61	76	76
CHEMPRO	1.25	34	34	33	73	78	80
+ 28% UAN	1.25 + 4	37	36	33	77	77	78
K-2000	1	39	37	40	72	78	77
+ 28% UAN	1 + 4	33	32	36	74	82	74
K-3000	1	34	32	34	75	81	79
+ 28% UAN	1 + 4	33	33	34	76	79	76
SCOIL	1	35	31	37	73	83	81
+ 28% UAN	1 + 4	33	35	35	77	82	82
SYLGARD 309	0.5	38	39	36	53	68	73
+ 28% UAN	0.5 + 4	34	35	35	79	76	77
LSD at 0.05		------- 9 -------			------- 8 -------		

Table 17. The effect of various PBO rates and adjuvants on thifensulfuron activity on the growth of velvetleaf in the greenhouse 2 WAT.

		Plant height			Visual injury		
Thifensulfuron (1.5 g ai/ha)		PBO rate (kg/ha)					
+ Adjuvant	Rate	0	0.5	1.0	0	0.5	1.0
	(% (v/v))	(% of control)				(% injury)	
None	-	96	90	72	1	14	36
+ 28% UAN	4	61	52	50	55	81	81
X-77	0.5	93	77	71	3	26	41
+ 28% UAN	0.5 + 4	57	51	49	61	81	88
CHEMPRO	1.25	61	66	56	48	42	56
+ 28% UAN	1.25 + 4	46	51	48	81	81	89
K-2000	1	74	66	56	36	45	58
+ 28% UAN	1 + 4	44	47	46	88	89	87
K-3000	1	55	51	50	66	74	76
+ 28% UAN	1 + 4	47	47	46	84	90	88
SCOIL	1	63	58	50	52	53	74
+ 28% UAN	1 + 4	48	43	48	92	89	93
SYLGARD 309	0.5	52	69	57	67	38	58
+ 28% UAN	0.5 + 4	44	47	45	87	91	92
LSD at 0.05		------- 8 --------			------- 11 --------		

Chapter 4

The Effect of Mixed Function Oxidase Inhibitors on Crop Safety to ALS Inhibiting Herbicides

ABSTRACT

Greenhouse studies were conducted to determine the response of six corn hybrids and two soybean varieties to acetolactate synthase (ALS) inhibitor herbicides applied with terbufos and/or mixed function oxidase (MFO) inhibitors. Field experiments were also conducted to determine the response of six corn hybrids to the combination treatments, terbufos plus ALS inhibitor herbicides and/or pipernoyl butoxide (PBO) and/or antidote. PBO at 0.33 kg/ha tank-mixed with nicosulfuron and primisulfuron caused injury to the Northrup King 9283 corn hybrid. Great Lakes 584 corn showed less sensitivity than Northrup King 9283 to these combination treatments. Pioneer 3377 IR corn hybrid was resistant to the combination of nicosulfuron, primisulfuron plus PBO 2 kg/ha, and also to the combination treatments of imazethapyr herbicide plus PBO or butylated hydroxyanisole (BHA) even though terbufos was previously applied. ICI 8532 IT,

ICI 8532 and Pioneer 3377 hybrids showed injury to the combination of nicosulfuron, primisulfuron herbicides and/or terbufos insecticide and/or PBO 2 kg/ha. ICI 8532 IT corn hybrid showed resistance to the combination treatment of imazethapyr or thifensulfuron with terbufos.

In the field study, injury 2 WAT to ICI 8532 IT, ICI 8532, and Pioneer 3377 hybrids from sulfonylurea herbicides plus terbufos was more evident than 6 WAT. Injury to ICI 8532 IT and Pioneer 3377 hybrids by imazethapyr herbicide plus terbufos remained similar to that observed at the early stage (2 WAT). Pioneer 3377 IR and Ciba 4393 RSC hybrids showed cross-resistance to sulfonylurea and imidazolinone herbicides even applied with PBO regardless of the presence of terbufos. ICI 8532 IT was cross-resistant to thifensulfuron and imidazolinone herbicides plus terbufos. All treatments of chlorimuron plus terbufos caused considerable injury to ICI 8532 IT, ICI 8532, Pioneer 3377, and Ciba 4393, but not Pioneer 3377 IR and Ciba 4393 RSC.

The combination of thifensulfuron with PBO caused injury to Elgin '87 soybean, but the W20-STS soybean was tolerant to this combination treatment. Combination of imazethapyr with PBO or BHA did not effect the growth of Elgin '87 soybean hybrid.

Nomenclature: Chlorimuron, 2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl] benzoic acid; imazethapyr, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid;

nicosulfuron, 2-[[[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-*N*,*N*-dimethyl-3-pyridinecarboxamide; primisulfuron, 2-[[[[[4,6-bis(difluromethoxy)-2-pyrimidinyl] amino] carbonyl] amino] sulfonyl] benzoic acid; thifensulfuron, 3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] amino] sulfonyl]-2-thiophenecarboxylic acid; terbufos, S-[[(1,1-dimethylethyl) thio] methyl] O,O-diethyl phosphorodithioate; butylated hydroxyanisole (BHA), 2,[3]-tert-butyl-4-hydroxyanisole; piperonyl butoxide (PBO), 5-[[2-(2-butoxyethoxy) ethoxy] methyl]-6-propyl-1,3-benzodioxole; corn, *Zea mays* L. 'Great lakes 584', 'Northrup King 9283', 'Ciba 4393 RSC', 'Ciba 4393', 'ICI 8532', 'ICI 8532 IT', 'Pioneer 3377 IR', 'Pioneer 3377'; soybean, *Glycine max* (L.) Merr., 'Elgin '87', 'W20-STS';

Additional index words: tolerance, combination, crop safety, cross-resistance, tank-mixture, antidote.

INTRODUCTION

Commercialization of sulfonylurea and imidazolinone herbicides has accelerated the development of herbicide-resistant crops. Problems associated with some member of these herbicide families include injury to subsequent crops from soil residues and increased crop injury following the use of certain insecticides. Herbicide resistant crops have benefits such as increasing the crop safety margin, reducing crop damage from residual herbicides and widening the choice of herbicides.

Resistant crop genotypes have been introduced by selection from naturally existing populations within crop species, selection of resistant mutants within a cultivar at the cell or whole plant level, and insertion of genes for resistance (1,7,10). With genetic engineering techniques now available, if the mechanisms for resistant and the genetic sequence are known, herbicide resistance can be inserted into crops (6).

Despite much research on herbicide-resistant crops, few herbicide-resistant crops have been released to date. Metribuzin [4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5-(4H)-one] resistant soybean (Glycine max (L.) Merr.) [TracyM], bipyridylium-resistant forage grass species, atrazine [6-chloro-N-ethyl-N'-(1-methylethyl)-1,3,5-triazine-2,4-diamine] resistant canola (Brassica napus L.), have been released (5,8,16).

Cell culture selection for herbicide resistance has achieved a small portion of success with corn (Zea mays L.), and sugarbeet (Beta vulgaris L.) (2). Resistance to the sulfonylurea and imidazolinone group of herbicides appeared to be partially dominant. Greater resistance was achieved when both resistant alleles were present. Resistance is due to a site modification in ALS. The imidazolinone resistant corn hybrid, Pioneer 3343 IR, has cross-resistance to the sulfonylurea herbicides which act by inhibiting the same enzyme.

Recently, private companies and universities developed and released the ALS inhibiting herbicide-resistant crop cultivars. Bauman et al. (4) reported that imazethapyr [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid] resistant corn (ICI 8532 IT) showed no injury to imazethapyr regardless of the presence of terbufos [S-[[(1,1-dimethylethyl) thio] methyl] O,O-diethylphosphorodithioate]. However, they found imazethapyr injury on ICI 8532 was enhanced when terbufos was included in the treatment. Wilcut et al. (17) reported that Pioneer 3343 IR hybrid was not injured by the interaction of either terbufos or carbofuran [2,3-dihydro-2, 2-dimethyl-7-benzofuranyl methylcarbamate] with nicosulfuron [2-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl] -N,N-dimethyl-3-pyridinecarboxamide] (15), imazethapyr, or AC 263,222. Also, Mukaida et al. (9) reported that Pioneer 3343 IR and ICI 8532 IT were unaffected by imazethapyr at 70 g ai/ha. Chlorimuron-ethyl [2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]benzoic acid] at 10 g ai/ha treatment reduced shoot weight of Pioneer 3343, ICI 8532, and ICI 8532 IT. Pioneer 3343 IR was not injured by chlorimuron-ethyl. From the ALS activity study, Mukaida et al. (9) suggested that the mechanism for IR and IT resistance was due to differential sensitivity at the target site. They found Pioneer 3343 IR corn was resistant to both imazethapyr and chlorimuron-ethyl, while ICI 8532 IT was resistant only to imazethapyr.

Barrett et al. (3) reported that PBO, tetcyclasis, and aminobenzothiazole inhibited nicosulfuron metabolism by Pioneer 3343 IR microsomes. Based on the observation that nicosulfuron is metabolized by a PBO sensitive MFO, one can hypothesize that PBO treatments to corn plants should increase injury from sulfonylurea herbicides. Sulfonylurea herbicide tolerant weeds could also be tolerant by metabolizing the herbicide in a manner similar to corn. If PBO were tank-mixed with the sulfonylurea herbicide, this should increase the activity to both corn and weeds, but if the ALS enzyme at the corn were less sensitive as in Pioneer 3343 IR it should have less injury.

The objectives of this study were; a) to determine the effect of the MFO inhibitors on corn safety from application of sulfonylurea or imidazolinone herbicides with and without terbufos; b) to determine the effect of MFO inhibitors on soybean tolerance to several ALS inhibitor herbicides.

Materials and Methods

General greenhouse procedure.

Plants were grown from seed in the greenhouse in 946-ml plastic pot containing air-dried Spinks sandy loam (mixed, mesic Psammentic Hapludalfs) soil consisting of 71.3% sand, 19.4% silt, and 9.4% clay with a pH 6.2. Daytime temperatures were 25 $\pm$ 2 C. Day length was 16 h with 1200 uE./m^2/s with both supplemental and natural sunlight. Terbufos treated soil was used to cover seeds to a 1.5 cm depth. Tank-mixed solutions of herbicide and MFO inhibitors were applied postemergence with a flat-fan 8002E nozzle in a spray volume of 280 L/ha at 240 kPa using a chain link-belt compressed air sprayer. Plant height, injury ratings, and fresh weight were evaluated 14 days after treatments.

Corn hybrids.

The six corn hybrids, Great Lakes 584, resistant to high rate of acetanilide herbicides, Northrup King 9283, sensitive to high rate of acetanilide herbicides (13), ICI 8532 IT and Pioneer 3377 IR, two imazethapyr resistant corn hybrids, ICI 8532 and Pioneer 3377, two imazethapyr sensitive hybrids, were included in this study. Two corn seeds were planted per 946-ml plastic pot. Postemergence herbicides and MFO inhibitors were applied when corn was at the three- to four-leaf stage. In the greenhouse, a completely randomized design was used with four replications per experiment. The data presented are the means of two experiments. Following an analysis of variance, the means were separated using the LSD Test at the 5% probability level.

Soybean varieties.

Two soybean varieties were used in this study. W20-STS hybrid was developed and released by Du Pont Company as having enhanced tolerance to sulfonylurea herbicide, thifensulfuron (14). Elgin '87 variety was used as a standard soybean variety. Two soybean seeds were planted per 946-ml plastic pot. Tank-mixed solutions of herbicides and/or MFO inhibitors were applied postemergence when the soybean reached the three- to four-leaf stage. In the greenhouse, a complete randomized design was used with four replications per experiment. The data presented are the means of two experiments. Following an analysis of variance, the means were separated using the LSD Test at the 5% probability level.

Chemical treatments.

Terbufos insecticide was applied as a preplant incorporated (PPI). Corn seeds were covered to a depth of 1.5 cm with the terbufos treated soil (2.9 kg/ha). Nicosulfuron and primisulfuron herbicides were applied at a rate range from 35 and 40 g ai/ha, respectively to twice that and MON 12000 and MON 12000 + MON 13900 were applied at 34.7 and 84.1 g ai/ha, respectively. PBO was applied at 4,000 g/ha alone, and 1,000, 500, 333 g/ha rates in combination with the herbicide treatments. In the soybean study, two soybean varieties, Elgin '87 and W20-STS, were sprayed with thifensulfuron at 8.8, 17.5, and 35 g ai/ha rates and PBO was applied at 2,000 g/ha. Imazethapyr was applied at 70 and 105 g ai/ha to Elgin '87, and PBO and BHA were applied at 2,000 and 4,000 g ai/ha rates. X-77[1] surfactant (0.25% v/v) was added to all spray solutions.

Field study.

Field studies were conducted in 1992 and 1993 at East Lansing, Michigan on a loam soil with 2.6% organic matter and pH of 7.1. The plots were 3 by 10.5 m, with a 75 cm row spacing. Four corn hybrids (ICI 8532 IT, ICI 8532, and Pioneer 3377 IR, Pioneer 3377) in 1992 and six corn hybrids (added Ciba 4393 RSC and Ciba 4393) were planted with 9,713 seeds per ha population on May 11 1992 and May 10 1993. Terbufos 15 G was applied at 74.4 g/100 m of row, in furrow. Metolachlor (8 EC 2.2 kg/ha) and atrazine (4 F 1.1 kg/ha) herbicides were applied as preemergence treatment to control weed species. Postemergence

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[1]X-77 nonionic surfactant is a mixture of alkylarylpolyoxyethylene glycols, free fatty acids, and isopropanol marketed by Valent U.S.A. Corp., 1333 N. California Blvd., Walnut Creek, CA 94596.

treatments were sprayed with a compressed air sprayer with a flat fan 8003 nozzle, 207 kPa, and 206 L/ha. Postemergence herbicides, nicosulfuron, primisulfuron, imazethapyr, chlorimuron were applied at one and/or two times the recommended rates. Preplant incorporation treatment of the herbicides, imazaquin [2-[4,5-dihydro-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-quinolinecarboxylic acid], MON 12000 [methyl 3-chloro-5-(4,6-dimethoxypyrimidin-2-arylcarbamoyl sulfamoyl)-1-methylpyrazde-4-carboxylate], chlorimuron [2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl)amino] carbonyl] amino] sulfonyl] benzoic acid], CGA-152005 [1-(4-methoxy-6-methyl-triazin-2-yl)-3-[2-(3,3,3-trifluoropropyl)-phenylsulfonyl]-urea], and flumetsulam + metolachlor, were at recommendated rates or two times these rates. PBO at 2 kg/ha was applied as tank-mixture with nicosulfuron and primisulfuron. To identify the antidote effect on the corn growth, R 29148 [3-(dichloroacetyl)-2,2,5-trimethyloxazolidine] antidote was applied with the sulfonylurea herbicides (1992 only). All postemergence treatments included 0.25% (v/v) X-77 surfactant. At 2 and 6 (1992), and 2 and 4 (1993) weeks after postemergence treatments, plants were evaluated for visual injury and measured plant height (10 plants/row).

Field experiments were conducted as a randomized complete block design. Each treatment was repeated three times. Following an analysis of variance, means were separated using the LSD Test at the 5% probability level.

Results and Discussion

Applications of 35 g/ha of nicosulfuron, primisulfuron, and 4 kg/ha of PBO alone did not induce injury on the two corn hybrids, Great Lakes 584 and Northrup King 9283 (Table 1). PBO at 1 kg/ha tank-mixed with nicosulfuron and primisulfuron herbicides reduced plant height and fresh weight of both corn hybrids compared to herbicide alone. Addition of PBO at 0.5 kg/ha to nicosulfuron increased injury to Northrup King 9283 corn hybrid, and reduced plant height of Great Lakes 584 hybrid. PBO at 0.5 kg/ha plus primisulfuron caused injury to Great Lakes 584 and Northrup King 9283 hybrids. PBO at 0.33 kg/ha tank-mixed with the two herbicides caused injury to the Northrup King 9283 hybrid. However, addition of 0.33 kg/ha of PBO to primisulfuron did not affect growth of the Great Lakes 584 hybrid. Application of nicosulfuron plus 0.33 kg/ha of PBO decreased plant height of Great Lakes 584, but did not reduce fresh weight (Table 1). From the results, Great Lakes 584 appeared more tolerant to combinations of PBO with sulfonylurea herbicides than Northrup King 9283 hybrid. According to Rowe et al. (13), Great Lakes 584 hybrid also showed more tolerance to high rates of acetanilide herbicides than Northrup King 9283 hybrid. Due to the crop injury, the addition of PBO to sulfonylurea herbicides is not recommended on the normal corn hybrids. The addition of PBO at rates in excess of 0.33 kg/ha was sufficient to cause injury to corn from the nicosulfuron and primisulfuron indicating that these herbicides were metabolized by the MFO system in corn.

PBO tank-mixed with nicosulfuron, primisulfuron, and imazethapyr did not affect the growth of imazethapyr resistance corn hybrid, Pioneer 3377 IR (Table 2). Also, the addition of BHA to imazethapyr did not cause injury to Pioneer 3377 IR hybrid. Barrett et al. (3) found that PBO inhibited metabolism of nicosulfuron. Despite PBO inhibition of sulfonylurea herbicide metabolism, Pioneer 3377 IR corn was not injured by the combination treatments of sulfonylurea herbicides plus PBO or BHA since the ALS in the Pioneer 3377 IR corn hybrid is known to be less sensitive to this class of herbicide (11).

Application of terbufos or PBO at 2 kg/ha alone had no effect on the growth of four corn hybrids, but if the two factors were combined, reduced plant height of ICI 8532IT and ICI 8532 hybrids was observed (Tables 3 and 4). The combination of nicosulfuron plus terbufos with or without PBO reduced corn plant height of Pioneer 3377, ICI 8532, and ICI 8532 IT hybrids compared to nicosulfuron alone, but it did not affect the growth of Pioneer 3377 IR hybrid. Primisulfuron alone, or combined with terbufos, reduced plant height and increased visual injury of ICI 8532 IT, ICI 8532, and Pioneer 3377 hybrids, but not of Pioneer 3377 IR hybrid. PBO tank-mixed with primisulfuron resulted in greater visual injury to both sensitive corn hybrids than observed with nicosulfuron, and decreased plant height of all four corn hybrids. The combinations of primisulfuron, terbufos, and PBO induced greater corn injury of three corn hybrids, except Pioneer 3377 IR hybrid. From the results, Pioneer 3377 IR hybrid appeared resistant to the combination of sulfonylureas, terbufos, and PBO (Tables 3 and 4). According to the Mukaida et al. (9), Pioneer 3343 IR corn showed cross-resistance to sulfonylurea and imidazolinone herbicides, but ICI 8532 IT corn was resistant to only imidazolinone herbicides regardless presence of terbufos.

These it would appear possible to use PBO in combination with nicosulfuron and primisulfuron to increase herbicide activity on weeds without loss of corn safety to Pioneer 3377 IR even if terbufos had been applied for insect control.

MON 12000 applied at 34.7 g ai/ha did not affect the plant height of the four corn hybrids, Pioneer 3377, Pioneer 3377 IR, ICI 8532, and ICI 8532 IT (Table 5). However, application of MON 12000 plus antidote MON 13900 reduced plant height of Pioneer 3377 and ICI 8532 corn hybrids, it might be due to the 1.5 times of recommendated rate. PBO at 2 kg/ha did not increase corn injury from MON 12000. The two imazethapyr resistant corn hybrids, Pioneer 3377 IR and ICI 8532 IT, were resistant to a high rate of MON 12000. Since the addition of PBO to MON 12000 did not enhance corn injury to either imazethapyr resistant and sensitive hybrids, PBO may not inhibit the metabolism of MON 12000 in corn plants.

Field Experiment. All plots, including control plots, received terbufos 15G at 11.9 kg/100 m row, in furrow, metolachlor 8 EC at 2.2 kg ai/ha, atrazine 4F at 1.1 kg ai/ha and X-77 0.25% (v/v), to control insects and weed species.

First year, 1992. Postemergence application of nicosulfuron or primisulfuron reduced plant height, and caused injury 2 WAT to ICI 8532IT, ICI 8532, and Pioneer 3377 hybrids, due to the prior application of terbufos insecticide (Tables 6 and 7). Addition of PBO at 2 kg/ha, even though tank-mixed antidote R29148 was applied enhanced visual injury to corn except for the Pioneer 3377 IR hybrid. The imazethapyr resistance corn hybrids, Pioneer 3377 IR and ICI 8532IT, were not injured by imazethapyr or thifensulfuron herbicides plus terbufos, but the sensitive corn hybrids, Pioneer 3377 and ICI 8532, were injured by that combination treatments (Tables 6 and 7).

By 6 WAT, the injury to corn from the primisulfuron and nicosulfuron interaction with terbufos was much less apparent (Tables 8 and 9). However, if PBO had been applied, the interaction was stable apparent. The imazethapyr interaction effect with terbufos was greater at 6 WAT than at 2 WAT. The two imazethapyr-resistant corn hybrids, ICI 8532 IT and Pioneer 3377 IR, showed resistance to the combination of imazethapyr plus terbufos. Only Pioneer 3377 IR hybrid showed resistance to sulfonylureas and imazethapyr herbicides plus terbufos even with added PBO (Tables 6, 7, 8 and 9). ICI 8532 IT corn hybrid was showed resistant to the interaction of thifensulfuron with terbufos regardless of the presence of PBO, but was very sensitive to the combination of nicosulfuron and primisulfuron herbicides plus terbufos.

Second year, 1993.

Nicosulfuron at 35 or 70 g ai/ha following prior application of terbufos caused corn injury to ICI 8532, Pioneer 3377 and Ciba 4393. The addition of PBO to these combination increased injury to these corn hybrids including the ICI 8532 IT hybrid (Tables 10 and 11). Pioneer 3377 IR and Ciba 4393 RSC showed resistance to the combination of nicosulfuron plus terbufos even with PBO at 2 WAT. Primisulfuron after terbufos with/without PBO caused injury to the ICI 8532 IT, ICI 8532, and Ciba 4393 hybrids. Pioneer 3377 IR, Pioneer 3377 and Ciba 4393 RSC showed no injury from primisulfuron plus terbufos and/or PBO. The Ciba 4393 hybrid was the most sensitive to combination of primisulfuron, terbufos, and PBO. All three imazethapyr sensitive corn hybrids showed sensitivity to the combination of imazethapyr plus terbufos. Chlorimuron postemergence plus terbufos caused injury to ICI 8532 IT, ICI 8532, Pioneer 3377, Ciba 4393, but not Pioneer 3377 IR or Ciba 4393 RSC. MON 12000 applied at 168 g ai/ha prior to terbufos treatment similarly caused injury to ICI 8532 IT, ICI 8532, Pioneer 3377, and Ciba 4393, but not on Pioneer 3377 IR and Ciba 4393 RSC. Flumetsulam/metolachlor, CGA-152005, and imazaquin plus terbufos caused injury to the imazethapyr sensitive corn hybrids.

Injury to ICI 8532 IT, ICI 8532, Pioneer 3377, and Ciba 4393 corn hybrids from nicosulfuron and primisulfuron plus terbufos $\pm$ PBO was less evident at 4 WAT compared to 2 WAT (Tables 12 and 13). But corn injury 4 WAT from imazethapyr was greater or similar to that observed at 2 WAT. From the results, it appear safe to plant Pioneer 3377 IR and Ciba 4393 RSC and apply combination treatments of ALS inhibitor herbicides plus terbufos. Also, PBO tank-mixed with these herbicides did not increase injury to these corn hybrids. The ICI 8532 IT corn hybrid was sensitive to nicosulfuron and primisulfuron with/without PBO, MON 12000, and CGA 152005 plus terbufos, and to chlorimuron ethyl. However, ICI 8532 IT showed resistance to imazethapyr plus terbufos treatment.

Soybean Study.

Thifensulfuron alone, even at two times the recommended rate, did not affect the growth of the two soybean hybrids, Elgin '87 and W20-STS (Table 9). Application of tank-mixed PBO at 2 kg/ha with thifensulfuron at 17.5 and 35 g/ha reduced soybean plant height and induced visual injury to Elgin '87 hybrid, but it did not affect the growth of the W20-STS hybrid. W20-STS hybrid showed excellent tolerance to application of thifensulfuron 35 g/ha with PBO 2 kg/ha compared to Elgin '87 hybrid (Table 14). Several researchers have reported that PBO inhibited the metabolism of sulfonylurea herbicides in corn (9,12). The inhibition of metabolism of thifensulfuron by PBO would be expected in both soybean hybrids, Elgin '87 and W20-STS. Thus, the difference in tolerance to the combination effect of thifensulfuron and PBO may be due to differential sensitivity at the target site of action. W20-STS was shown to have a less sensitive target site (14). PBO tank-mixed with thifensulfuron applied to W20-STS soybean field could increase weed control without soybean injury.

The additions of PBO or BHA to imazethapyr showed no effect on the growth of Elgin '87 soybean hybrid even at 1.5 fold recommended rate (Table 15). These results suggest that PBO and BHA do not inhibit the metabolism of imazethapyr herbicide in soybean.

From the results, the sulfonylurea and imidazolinone herbicides are metabolized by a different MFO, and MFO that metabolize imazethapyr is not sensitive to PBO. But, MFO that metabolize sulfonylurea herbicides is very sensitive to PBO. Certain corn hybrids, Pioneer 3377 IR, Ciba 4393 RSC, are not affected by the combination treatments of PBO or/and terbufos. Because these corn hybrids changed the sensitivity at the site of action of sulfonylurea herbicides.

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Table 1. The effect of PBO on the response of two corn hybrids to nicosulfuron and primisulfuron in the greenhouse 2 WAT.

		Great Lakes 584		Northrup King 9283	
Treatment	Rate	Plant ht	Fresh wt	Plant ht	Fresh wt
	(g/ha)	(cm/plant)	(g/plant)	(cm/plant)	(cm/plant)
Control	-	65.7	17.3	70.3	18.3
PBO	4,000	67.0	16.7	62.5	16.2
Nicosulfuron	35	65.6	17.3	70.6	16.7
+ PBO	35 + 1000	48.0	11.3	46.4	8.6
+ PBO	35 + 500	60.1	16.8	55.6	13.1
+ PBO	35 + 333	59.0	16.1	60.2	13.9
Primisulfuron	35	63.5	16.7	67.0	16.4
+ PBO	35 + 1000	51.8	12.3	48.5	11.4
+ PBO	35 + 500	56.8	13.7	56.3	12.5
+ PBO	35 + 333	63.4	15.4	62.9	15.3
LSD at 0.05		**4.2**	**2.7**	**7.0**	**2.2**

Table 2. The effect of MFO inhibitors on the response of imazethapyr resistance Pioneer 3377 IR corn hybrids to sulfonylurea herbicides in the greenhouse 2 WAT.

Treatment	Rate	Plant ht	Fresh wt
	(g/ha)	(cm/plant)	(g/plant)
Control	-	72.3	31.1
PBO	4000	74.7	33.8
PBO	2000	77.4	36.3
BHA	4000	77.9	35.5
BHA	2000	76.8	35.3
Nicosulfuron	35	74.1	32.0
+ PBO	35 + 2000	74.4	34.1
Primisulfuron	35	72.3	31.6
+ PBO	35 + 2000	75.0	36.0
Imazethapyr	70	71.1	31.4
+ PBO	70 + 4000	69.3	29.6
+ PBO	70 + 2000	68.6	31.1
+ BHA	70 + 4000	73.5	32.9
+ BHA	70 + 2000	74.4	36.9
LSD at 0.05		**N.S.**	**3.6**

Table 3. The effect of PBO and terbufos on the responses of imazethapyr resistant and sensitive corn hybrids to nicosulfuron and primisulfuron herbicides in the greenhouse 2 WAT.

		PR[a]		PS[b]		IT[c]		IS[d]	
		Terbufos[e]							
Treatment	Rate	-	+	-	+	-	+	-	+
	(g/ha)	plant ht (cm/plant)							
Control	-	63.3	63.1	62.1	61.6	62.5	61.8	59.8	57.6
PBO	2000	64.2	60.8	61.1	59.1	62.8	58.5	59.0	55.7
Nicosulfuron	35	59.0	57.7	59.9	51.0	62.7	58.1	57.3	51.1
+ PBO	35 + 2000	57.3	57.5	38.0	30.6	51.5	30.4	37.9	28.5
Primisulfuron	40	61.2	58.8	54.5	42.2	56.3	50.9	52.9	42.8
+ PBO	40 + 2000	58.1	58.8	39.2	31.4	51.7	32.5	43.4	28.6
LSD at 0.05		**3.0**		**3.6**		**3.6**		**3.6**	

[a] PR : Pioneer 3377IR, imazethapyr resistant
[b] PS : Pioneer 3377, imazethapyr sensitive
[c] IT : ICI 8532IT, imazethapyr resistant
[d] IS : ICI 8532, imazethapyr sensitive
[e] Terbufos : Terbufos at 2.9 kg ha^{-1} applied PPI.

Table 4. The effect of PBO and terbufos on responses of imazethapyr resistant and sensitive corn hybrids to nicosulfuron and primisulfuron herbicides in the greenhouse 2 WAT.

		PR[a]		PS[b]		IT[c]		IS[d]	
		Terbufos[e]							
Treatment	Rate	-	+	-	+	-	+	-	+
	(g/ha)	(% injury)							
Control	-	0	0	0	0	0	0	0	0
PBO	2000	0	0	0	0	0	0	0	0
Nicosulfuron	35	0	2	0	3	0	2	1	5
+ PBO	35+2000	1	2	32	56	12	71	34	64
Primisulfuron	40	0	1	1	18	1	7	2	11
+ PBO	40+2000	1	1	24	54	4	58	11	62
LSD at 0.05		2		6		5		5	

[a] PR : Pioneer 3377IR, imazethapyr resistant
[b] PS : Pioneer 3377, imazethapyr sensitive
[c] IT : ICI 8532IT, imazethapyr resistant
[d] IS : ICI 8532, imazethapyr sensitive
[e] Terbufos : Terbufos at 2.9 kg ha^{-1} applied PPI.

Table 5. The effects of PBO and MON 13900 on plant height of imazethapyr resistant and sensitive corn hybrids treated with MON 12000 in the greenhouse 2 WAT.

			Corn hybrids		
Treatment	Rate	PS[a]	PR[b]	IS[c]	IT[d]
	(g/ha)		Plant ht (cm/plant)		
Control	-	73.9	72.8	76.5	75.0
MON 12000	34.7	71.0	72.5	74.2	74.1
+ PBO	34.7+2000	70.6	73.4	72.8	73.8
MON 12000 MON 13900	84.1	67.4	71.1	69.8	72.4
+ PBO	84.1+2000	67.0	71.5	67.8	72.1
LSD at 0.05		**5.7**	**N.S.**	**3.5**	**N.S.**

[a] PS: Pioneer 3377, imazethapyr sensitive
[b] PR: Pioneer 3377 IR, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] IT: ICI 8532 IT, imazethapyr resistant

Table 6. The effects of PBO, terbufos, and the antidote R-29148 on plant height of imazethapyr resistant and sensitive corn hybrids treated with ALS inhibitor herbicides at 2 WAT in the field[a] study in 1992.

		Corn hybrids			
Treatment	Rate	IT[b]	IS[c]	PR[d]	PS[e]
		Plant ht (cm/plant)			
Control	-	63.4	64.3	62.0	64.9
Nicosulfuron	35	50.9	45.1	60.7	41.9
+ PBO	35 + 2000	34.6	28.6	58.9	28.6
+PBO+R29148	35 + 2000 + 605	34.2	27.7	58.1	27.2
Nicosulfuron	70	49.0	39.3	57.9	39.0
Primisulfuron	40	50.3	44.1	55.6	39.2
+ PBO	40 + 2000	36.7	27.0	59.7	28.5
+PBO+R29148	40 + 2000 + 605	32.7	27.4	54.5	27.2
Primisulfuron	80	48.6	41.8	61.4	38.2
Imazethapyr[f]	70	58.4	28.7	58.3	28.4
Imazethapyr[f]	140	54.9	25.9	57.3	26.6
Thifensulfuron	4.4	56.8	49.9	57.2	45.2
LSD at 0.05		**7.9**	**7.0**	**5.4**	**6.9**

[a] All plots were treated with terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8 EC 2.2 kg/ha, Atrazine 4 F 1.1 kg/ha, and X-77 0.25% (v/v)
[b] IT: ICI 8532IT, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] Tank-mixed 28% UAN

Table 7. The effects of PBO, terbufos, and the antidote R-29148 on visual injury of imazethapyr resistant and sensitive corn hybrids treated with ALS inhibitor herbicides at 2 WAT in the field[a] study in 1992.

		Corn hybrids			
Treatment	Rate	IT[b]	IS[c]	PR[d]	PS[e]
		(% injury)			
Control	-	0	0	0	0
Nicosulfuron	35	18	25	2	32
+ PBO	35 + 2000	43	53	0	57
+PBO+R29148	35 + 2000 + 605	43	52	0	55
Nicosulfuron	70	20	30	2	42
Primisulfuron	40	25	32	2	38
+ PBO	40 + 2000	43	55	0	60
+PBO+R29148	40 + 2000 + 605	48	50	0	60
Primisulfuron	80	23	27	0	35
Imazethapyr[f]	70	3	53	0	55
Imazethapyr[f]	140	3	63	0	60
Thifensulfuron	4.4	3	13	0	20
LSD at 0.05		**11**	**10**	**2**	**13**

[a] All plots were treated with terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8 EC 2.2 kg/ha, and atrazine 4 F 1.1 kg/ha, and X-77 0.25% (v/v).
[b] IT: ICI 8532IT, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] Tank-mixed 28% UAN

Table 8. The effects of PBO, terbufos, and the antidote R-29148 on plant height of imazethapyr resistant and sensitive corn hybrids treated with ALS inhibitor herbicides at 6 WAT in the field[a] study in 1992.

		Corn hybrids			
Treatment	Rate	IT[b]	IS[c]	PR[d]	PS[e]
		Plant ht (cm/plant)			
Control	-	179.1	177.0	186.1	185.0
Nicosulfuron	35	179.1	169.8	183.7	175.5
+ PBO	35 + 2000	144.7	90.9	170.9	113.7
+ PBO+R29148	35 + 2000 + 605	140.4	96.5	174.7	115.1
Nicosulfuron	70	167.8	160.9	183.4	150.0
Primisulfuron	40	167.7	151.0	174.2	159.5
+ PBO	40 + 2000	138.5	97.7	175.5	125.5
+ PBO+R29148	40 + 2000 + 605	135.3	127.7	179.1	125.9
Primisulfuron	80	162.6	154.8	175.4	155.1
Imazethapyr[f]	70	173.1	67.6	170.5	116.8
Imazethapyr[f]	140	164.9	33.1	172.7	63.2
Thifensulfuron	4.4	172.7	174.9	179.5	174.4
LSD at 0.05		**10.6**	**23.4**	**11.9**	**28.8**

[a] All plots were treated preemergence with Terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8 EC 2.2 kg/ha, atrazine 4 F 1.1 kg/ha, and X-77 0.25% (v/v).
[b] IT: ICI 8532IT, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] Tank-mixed 28% UAN

Table 9. The effect of PBO, terbufos, and the antidote R-29148 on visual injury of imazethapyr resistant and sensitive corn hybrids treated ALS inhibitor herbicides at 6 WAT in the field[a] study in 1992.

		Corn hybrids			
Treatment	Rate	IT[b]	IS[c]	PR[d]	PS[e]
		(% injury)			
Control		0	0	0	0
Nicosulfuron	35	0	0	0	0
+ PBO	35 + 2000	13	47	0	37
+ PBO+R29148	35 + 2000 + 605	17	37	0	43
Nicosulfuron	70	0	3	0	7
Primisulfuron	40	3	3	0	0
+ PBO	40 + 2000	20	37	0	33
+ PBO+R29148	40 + 2000 + 605	33	40	0	43
Primisulfuron	80	0	0	0	3
Imazethapyr[f]	70	0	73	0	40
Imazethapyr[f]	140	0	93	0	77
Thifensulfuron	4.4	0	0	0	0
LSD at 0.05		**10**	**14**	**NS**	**15**

[a] All plots were treated preemergence treatments with Terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8EC 2.2 kg/ha, atrazine 4F 1.1 kg/ha, and X-77 0.25% (v/v).
[b] IT: ICI 8532IT, imazethapyr resistant
[c] IR: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] Tank-mixed 28% UAN

Table 10. The effect of PBO, terbufos on imazethapyr resistant and sensitive corn hybrids treated with ALS inhibitor herbicides 2 WAT in a field[a] study in 1993.

Treatment	Rate	Corn hybrids IT[b]	IS[c]	PR[d]	PS[e]	CR[f]	CS[g]
	(g/ha)	Plant ht (cm/plant)					
Control	-	73.9	75.3	85.6	82.9	85.2	84.0
POST							
Nicosulfuron	35	81.8	59.1	86.0	60.3	86.1	74.3
+ PBO	35+2000	57.0	47.0	77.5	54.2	81.0	47.6
Nicosulfuron	70	68.5	54.8	77.5	55.2	87.0	55.0
Primisulfuron	40	71.9	77.8	77.8	74.2	78.7	72.1
+ PBO	40+2000	61.9	60.7	75.4	72.6	81.8	39.0
Primisulfuron	80	77.9	73.5	76.4	71.2	86.8	70.4
Imazethapyr	70	76.1	33.5	75.2	36.1	78.9	38.7
Imazethapyr	140	81.2	31.8	72.6	31.0	73.9	28.3
Chlorimuron	12	33.6	32.7	80.4	36.6	81.7	31.6
PPI							
Chlorimuron	14	45.1	55.8	79.8	47.1	80.7	57.3
Chlorimuron	28	40.5	42.7	73.6	27.1	77.4	40.9
MON 12000	168	56.7	59.8	77.4	38.0	81.3	37.8
Flumetsulam/ metolachlor	2417	70.0	60.5	72.2	67.7	79.9	44.8
CGA-152005	40	61.7	60.9	72.2	57.3	72.0	44.0
Imazaquin	70	79.6	47.5	74.6	26.3	81.8	64.0
LSD at 0.05		**17.5**	**21.7**	**9.5**	**19.0**	**10.0**	**20.4**

[a] All plots were treated PRE treatments with Terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8EC 2.2 kg/ha, atrazine 4F 1.1 kg/ha, and X-77 0.25% (v/v).
[b] IT: ICI 8532 IT, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377 IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] CR: Ciba 4393 RSC, sulfonylurea resistant
[g] CS: Ciba 4393, sulfonylurea sensitive

Table 11. The effect of PBO, terbufos on imazethapyr resistant and sensitive corn hybrids treated with ALS inhibitor herbicides 2 WAT in a field[a] study in 1993.

		Corn hybrids					
Treatment	Rate	IT[b]	IS[c]	PR[d]	PS[e]	CR[f]	CS[g]
	(g/ha)	Visual injury (%)					
Control	-	0	0	0	0	0	0
POST							
Nicosulfuron	35	18	32	0	32	2	23
+ PBO	35+2000	37	57	2	43	5	55
Nicosulfuron	70	18	37	0	33	2	37
Primisulfuron	40	20	20	0	7	3	15
+ PBO	40+2000	23	28	2	5	3	57
Primisulfuron	80	20	22	2	5	2	23
Imazethapyr	70	2	70	2	65	7	65
Imazethapyr	140	5	75	5	75	8	75
Chlorimuron	12	78	78	7	70	5	75
PPI							
Chlorimuron	14	48	33	0	42	3	40
Chlorimuron	28	48	52	23	47	2	42
MON 12000	168	32	32	3	50	5	47
Flumetsulam/ metolachlor	2417	17	23	3	23	3	33
CGA-152005	40	27	23	15	33	7	38
Imazaquin	70	2	35	5	57	3	33
LSD at 0.05		**20**	**16**	**15**	**24**	**6**	**21**

[a] All plots were treated PRE treatments with Terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8EC 2.2 kg/ha, atrazine 4F 1.1 kg/ha, and X-77 0.25% v/v.
[b] IT: ICI 8532 IT, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377 IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] CR: Ciba 4393 RSC, sulfonylurea resistant
[g] CS: Ciba 4393, sulfonylurea sensitive

Table 12. The effect of PBO, terbufos on imazethapyr resistant and sensitive corn hybrids treated with ALS inhibitor herbicides 4 WAT in a field[a] study in 1993.

Treatment	Rate	Corn hybrids IT[b]	IS[c]	PR[d]	PS[e]	CR[f]	CS[g]
	(g/ha)	Plant ht (cm/plant)					
Control	-	142.1	154.8	162.9	164.9	164.7	158.0
POST							
Nicosulfuron	35	157.2	133.6	161.4	147.0	164.5	155.5
+ PBO	35+2000	123.9	83.9	148.1	137.3	160.8	114.0
Nicosulfuron	70	149.3	118.1	149.6	129.6	166.2	140.0
Primisulfuron	40	142.2	156.6	155.7	153.1	161.1	156.2
+ PBO	40+2000	135.7	141.3	145.1	155.8	154.9	102.1
Primisulfuron	80	142.5	139.4	154.5	150.7	170.3	159.3
Imazethapyr	70	137.4	50.8	141.2	68.1	148.5	78.5
Imazethapyr	140	153.1	45.7	130.0	40.6	150.8	45.4
Chlorimuron	12	46.8	44.0	148.2	71.0	151.9	73.9
PPI							
Chlorimuron	14	94.7	136.7	156.7	108.6	160.1	106.7
Chlorimuron	28	74.5	91.0	141.6	52.1	155.9	95.4
MON 12000	168	114.2	106.2	144.0	93.4	145.9	79.6
Flumetsulam/ metolachlor	2417	126.2	127.7	145.6	138.8	149.4	104.6
CGA-152005	40	130.3	137.7	139.6	121.4	149.7	108.7
Imazaquin	70	149.4	105.5	145.9	65.5	154.5	129.6
LSD at 0.05		**31.7**	**40.8**	**19.0**	**38.4**	**18.2**	**44.1**

[a] All plots were treated PRE treatments with Terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8EC 2.2 kg/ha, atrazine 4F 1.1 kg/ha, and X-77 0.25% v/v.
[b] IT: ICI 8532 IT, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377 IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] CR: Ciba 4393 RSC, sulfonylurea resistant
[g] CS: Ciba 4393, sulfonylurea sensitive

Table 13. The effect of PBO, terbufos on imazethapyr resistant and sensitive corn hybrids treated with ALS inhibitor herbicides 4 WAT in a field[a] study in 1993.

		Corn hybrids					
Treatment	Rate	IT[b]	IS[c]	PR[d]	PS[e]	CR[f]	CS[g]
	(g/ha)	Visual injury (%)					
Control	-	0	0	0	0	0	0
POST							
Nicosulfuron	35	0	20	0	13	0	15
+ PBO	35+2000	23	45	0	17	0	33
Nicosulfuron	70	8	25	0	20	0	22
Primisulfuron	40	15	17	0	7	0	7
+ PBO	40+2000	15	17	0	8	0	33
Primisulfuron	80	8	8	0	3	0	12
Imazethapyr	70	2	82	3	68	0	60
Imazethapyr	140	0	58	7	88	0	82
Chlorimuron	12	78	80	3	58	2	68
PPI							
Chlorimuron	14	18	23	0	37	0	32
Chlorimuron	28	45	47	7	72	0	48
MON 12000	168	37	35	7	47	0	52
Flumetsulam/ metolachlor	2417	13	13	2	15	0	20
CGA-152005	40	25	20	7	20	3	33
Imazaquin	70	5	35	0	65	2	33
LSD at 0.05		**17**	**29**	**7**	**23**	**3**	**29**

[a] All plots were treated PRE treatments with Terbufos 15G 74.4 g pr/100 m row, in furrow, metolachlor 8EC 2.2 kg/ha, atrazine 4F 1.1 kg/ha, and X-77 0.25% v/v.
[b] IT: ICI 8532 IT, imazethapyr resistant
[c] IS: ICI 8532, imazethapyr sensitive
[d] PR: Pioneer 3377 IR, imazethapyr resistant
[e] PS: Pioneer 3377, imazethapyr sensitive
[f] CR: Ciba 4393 RSC, sulfonylurea resistant
[g] CS: Ciba 4393, sulfonylurea sensitive

Table 14. The effect of PBO on thifensulfuron tolerance by two soybean varieties in the greenhouse 2 WAT.

		Elgin '87		W20-STS	
Treatment	Rate	Plant ht	Visual injury	Plant ht	Visual injury
	(g ai/ha)	(cm/plant)	(%)	(cm/plant)	(%)
Control		15.3	0	13.0	0
PBO	2000	15.4	0	13.0	0
Thifensulfuron	8.8	15.4	0	12.8	0
+ PBO	8.8+2000	14.5	0	13.6	0
Thifensulfuron	17.5	15.4	0	13.1	0
+ PBO	17.5+2000	14.1	30	12.7	1
Thifensulfuron	35	15.8	0	12.9	0
+ PBO	35+2000	11.0	44	11.9	6
LSD at 0.05		1.0	4	1.1	2

Table 15. The effects of PBO and BHA on imazethapyr tolerance of Elgin '87 in the greenhouse 2 WAT.

Treatment	Rate	Plant ht	Fresh wt
	(g ai/ha)	(cm/plant)	(g/plant)
Control		11.4	5.0
PBO	4000	11.5	5.7
BHA	4000	11.3	5.6
Imazethapyr	70	11.2	4.7
+ PBO	70 + 2000	11.3	4.8
+ PBO	70 + 4000	11.9	5.3
+ BHA	70 + 2000	11.5	4.6
+ BHA	70 + 4000	11.1	5.0
Imazethapyr	105	11.3	5.1
+ PBO	105 + 2000	11.8	4.8
+ PBO	105 + 4000	11.5	5.0
+ BHA	105 + 2000	11.3	5.0
+ BHA	105 + 4000	12.0	6.1
LSD at 0.05		**N.S.**	**0.9**

Chapter 5

Response of a Chlorsulfuron-Resistant Biotype of Kochia scoparia to ALS Inhibitor Herbicides and Piperonyl Butoxide

ABSTRACT

Greenhouse studies were conducted to determine kochia resistance to a spectrum of ALS-inhibiting herbicides. The chlorsulfuron resistant biotype was resistant to six herbicides; triflusulfuron, thifensulfuron, MON 12037, imazamethabenz, chlorsulfuron, and nicosulfuron. But, the resistant biotype showed sensitivity similar to the susceptible biotype to three herbicides; metsulfuron, imazethapyr, and imazaquin. The resistant biotype was slightly less sensitive to primisulfuron, chlorimuron and flumetsulam than the sensitive biotype.

Addition of a mixed function oxidase inhibitor, PBO at 2 kg/ha, to primisulfuron and thifensulfuron increased visual injury and reduced plant height of chlorsulfuron sensitive kochia biotype. And PBO tank-mixing to primisulfuron enhanced R biotype control at low rate of primisulfuron.

Nomenclature: chlorimuron, 2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl] benzoic acid; chlorsulfuron, 2-chloro-N-[[(4-methoxy-

6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] benzenesulfonamide; flumetsulam, N-[2,6-difluorophenyl]-5-methyl(1,2,4)triazolo-[1,5a]-pyrimidine-2-sulfonamide; imazamethabenz, (±)-2-[4,5-dihydro-4-methyl]-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-4(and 5)-methylbenzoic acid(3:2); imazaquin, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-quinolinecarboxylic acid; imazethapyr, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid; metsulfuron, 2-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] amino] sulfonyl] benzoic acid; MON 12037, methyl 3-chloro-5-(4,6-dimethoxypyrimidin-2-ylcarbamoyl sulfamoyl)-1-methylpyrazole-4-carboxylate; nicosulfuron, 2-[[[[(4,6-dimethoxy-2-pyrimidinyl) amino] carbonyl] amino] sulfonyl]-N,N-dimethyl-3-pyridinecarboxamide; primisulfuron, 2-[[[[[4,6-bis (difluoromethoxy)-2-pyrimidinyl] amino] carbonyl] amino] sulfonyl] benzoic acid; Thifensulfuron, 3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) amino] carbonyl] amino] sulfonyl]-2-thiophenecarboxylic acid; triflusulfuron, 2-[[[[[4-(dimethylamino)-6-(2,2,2-trifluoroethoxy)-1,3,5-triazin-2-yl] amino] carbonyl]-amino] sulfonyl]-3-methylbenzoic acid; piperonyl butoxide (PBO), α-(2-(2-butoxyethoxy) ethoxy)-4,5-methyl enedioxy-2-propyltolune; kochia, Kochia scoparia (L.) Schrad.

Additional index words: Cross resistance, MFO inhibitor, interaction.

INTRODUCTION

Repeated use of herbicides with the same mode of action on the same site has been implicated in the emergence of resistant weed populations. Resistance to pesticides is a world-wide phenomenon and exists for fungicides, insecticides, and herbicides (5). Since 1970, herbicide resistance has become well known in scientific and agricultural communities (15).

Newer types of herbicides with high specific activity are sulfonylureas and imidazolinones. The mode of action of these compounds have been demonstrated to be the inhibition of acetolactate synthase (ALS), also known as acetohydroxyacid synthase (AHAS) (2,12,17).

Recently, chlorsulfuron-resistant biotypes of four weed species have appeared in the USA, Canada, and Australia. These include prickly lettuce (Lactuca serriola L.), kochia (Kochia scoparia (L.) Schrad), and Russian thistle (Salsola iberica Sennen & Pan), in the U.S., chickweed (Stellaria media (L.) Vill), in Canada, and rigid ryegrass (Lolium rigidum Gaud.), in Australia (3,4,7,9,10,11,16). Many reports concluded that the mechanism for sulfonylurea resistance is a less sulfonylurea-sensitive ALS enzyme (3,11,13,16). Devine et al. (3) reported that the altered ALS in chlorsulfuron resistance-biotypes did not confer the same level of resistance to other ALS-inhibiting herbicides. Also, Primiani et al. (10) reported that the degree of cross resistance of a chlorsulfuron resistant kochia biotype to ALS-inhibiting herbicides varied.

Piperonyl butoxide (PBO), a widely used as a pesticide synergist, has been effective with many organophosphate, carbamate, and pyrethroid insecticides, increasing insecticidal activity against resistant insects (1,8). PBO has shown potential to increase activity of several herbicides, such as EPTC (S-ethyl dipropyl carbamothioate) (6), and bentazon (3-(1-methylethyl)-(1H)-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide) (14).

The objectives of this study were a) to determine the cross-resistance of chlorsulfuron resistant kochia biotype to various ALS-inhibiting herbicides, and b) to evaluate potential synergistic effects of PBO with sulfonylurea herbicides to the chlorsulfuron-resistant kochia biotype.

MATERIALS AND METHODS

Plant Materials.

Seeds of kochia resistant and sensitive to chlorsulfuron were obtained from du Pont de Nemours & Co., Inc. Seeds were planted in 27 by 53 cm plastic boxes which contained BACCTO soil mix, and germinated in the greenhouse. The plants were grown at 24 C $\pm$ 2 C with supplemental lighting from high pressure sodium lights to provide a midday light intensity of 1200 μ E $m^{-2}s^{-1}$ for both supplemental and natural light. The day length was 18 h. After emergence, the 2-cm seedlings were transplanted one per pot to 945-ml plastic pots containing BACCTO soil. Uniform plants 4 to 5 cm tall were selected for postemergence herbicide treatments.

Chemical Treatments.

To determine the cross-resistance to various ALS inhibiting-herbicides, nicosulfuron (70 g/ha), primisulfuron (80 g/ha), thifensulfuron (8.8 g/ha), chlorimuron (24 g/ha), metsulfuron (8.4 g/ha), chlorsulfuron (28 g/ha), imazethapyr (135 g/ha), imazamethabenz (1008 g/ha), imazaquin (280 g/ha), MON 12307 (100 g/ha), flumetsulam (100 g/ha), and triflusulfuron (35 g/ha) herbicides were applied to both kochia biotypes. All treatments were applied as postemergence, and included X-77[1] (0.25% v/v). All herbicide treatments were applied with a flat-fan 8002E nozzle in a spray volume of 280 L/ha at 240 kPa using a chain link-belt compressed air sprayer. To evaluate PBO effect on ALS inhibiting herbicide on the growth of chlorsulfuron-resistant and -sensitive kochia biotypes, thifensulfuron (1.1 and 4.4 g/ha) and primisulfuron (20 and 40 g/ha) herbicides were applied to both kochia biotypes with 2 kg/ha PBO.

Data Analysis.

Plant height and visual injury ratings were evaluated 14 days after postemergence treatments. Data presented are the means of two experiments with four replication in each. Experiments were conducted as a completely randomize design. Two (herbicide, biotype) and three (treatment, PBO, biotype) factorial design were used in cross-resistance and PBO effects experiments. Means were separated by LSD Test at the 5% level using the MSTAT program.

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[1]X-77 nonionic surfactant is a mixture of alkylarylpolyoxyethylene glycols, free fatty acids, and isopropanols marketed by Valent U.S.A. Corp., 1333 N. California Blvd., Walnut Creek, CA 94596.

RESULTS AND DISCUSSION

Cross-resistance study.

All two factors, kochia biotypes and herbicides, affected the response on plant height and visual injury. All ALS inhibiting-herbicides applied at two times the usual use rates reduced plant height, and caused more than 50% injury to the chlorsulfuron sensitive kochia biotype (Table 1). Response of this sensitive kochia biotype to the various ALS-inhibiting-herbicides, ranked from the highest to the lowest level of sensitivity, imazethapyr $\geq$ chlorsulfuron $\geq$ metsulfuron $\geq$ flumetsulam $\geq$ nicosulfuron, primisulfuron $\geq$ imazaquin $\geq$ MON 12037 $\geq$ thifensulfuron $\geq$ chlorimuron $\geq$ triflusulfuron $\geq$ imazamethabenz. Application of imazethapyr and metsulfuron at two times field use rates controlled chlorsulfuron resistant kochia 99%, and 89%,respectively. The chlorsulfuron resistant biotype was very resistant to thifensulfuron, MON 12037, and triflusulfuron herbicides applied at two times the usual use rates causing less than 10 % injury. The magnitude of resistance of the chlorsulfuron-resistant kochia biotype to ALS-inhibiting herbicides ranked from the highest to the lowest levels, triflusulfuron, thifensulfuron $\geq$ MON 12037 > imazamethabenz $\geq$ chlorsulfuron $\geq$ nicosulfuron $\geq$ chlorimuron $\geq$ primisulfuron > imazaquin, flumetsulam $\geq$ metsulfuron $\geq$ imazethapyr (Table 1).

Primiani et al. (4) found that the 50% growth reduction (GR_{50}) value of chlorsulfuron and metsulfuron to the chlorsulfuron resistant kochia was 30 and 8 times higher than that of susceptible kochia biotype, respectively. Reed et al. (13) reported that 2 and 46 g/ha of metsulfuron were needed to obtain 90% control of susceptible and resistant kochia, respectively. The results cannot be compared to each other directly, due to the difference in methods. But, other researchers reported that the chlorsulfuron resistant kochia was more resistant than the susceptible biotype to metsulfuron, whereas this study showed the same responses, 90% and 89% injury, for both biotypes to metsulfuron. Either the metsulfuron rate was too high or the mutation was different. The kochia response to chlorsulfuron in this study was similar to that reported by others (10,13,16). Friesen et al. (4) ranked the levels of chlorsulfuron resistance of kochia as following ; thifensulfuron > > chlorsulfuron > imazethapyr > metsulfuron. The two most effective herbicides, imazethapyr and metsulfuron, showed no differential herbicide activity to the both biotypes in this study. The mutation in one particular ALS gene affects the binding of each ALS inhibiting herbicide differently and it is not possible to predict degrees of cross-resistance to other herbicides acting on the same target enzyme. According to Sivakumaran et al. (18), the magnitudes of ALS resistance and cross-resistance were highly variable among kochia populations. They explained that the differences might be due to the type of mutation in the gene encoding ALS.

PBO effect on the activity of two sulfonylurea herbicides to the kochia biotypes.

All three factors, kochia biotypes, herbicide treatment and PBO, affected differential responses on the growth of kochia biotypes. Application of PBO at 2 kg/ha alone had no effect on the growth of either biotypes of kochia (Table 2). The herbicide treatments, 1.1 and 4.4 g ai/ha of thifensulfuron, and 20 and 40 g ai ha of primisulfuron, reduced plant height of both chlorsulfuron resistant and sensitive biotypes. PBO tank-mixed to primisulfuron at 20 g/ha enhanced visual injury of R biotype from 44 (no PBO) to 71% (with PBO). The chlorsulfuron resistant biotype appeared more sensitive to primisulfuron than the thifensulfuron. Addition of PBO increased visual injury indicating that both herbicides are metabolized by a mixed function oxidase sensitive to PBO (Table 2). The addition of PBO to sulfonylurea herbicides may provide farmers with a tool to increase activity of these herbicides against kochia.

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Table 1. The responses of chlorsulfuron-resistant and sensitive biotypes of kochia to the ALS inhibiting-herbicides in the greenhouse study 2 WAT.

		Plant height		Visible injury	
Treatment	Rate	S biotype	R biotype	S biotype	R biotype
	(g ai/ha)	------- (cm/plant) -------		-------- (%) -------	
Control		22.8	21.3	0	0
Nicosulfuron	70	6.3	9.9	84	46
Primisulfuron	80	5.9	6.9	84	66
Thifensulfuron	8.8	8.9	18.3	66	3
Chlorimuron	23	9.3	10.3	62	51
Metsulfuron	8.4	5.6	5.6	90	89
Chlorsulfuron	28	4.9	12.9	96	38
Imazethapyr	135	5.0	4.6	100	99
Imazamethabenz	1008	10.3	13.2	50	27
Imazaquin	280	7.1	5.9	74	73
MON 12037	100	7.8	17.1	71	9
Flumetsulam	100	6.4	6.2	89	74
Triflusulfuron	35	9.3	21.4	56	3
LSD at 0.05		**2.9**		**18**	

Table 2. The responses of chlorsulfuron-resistant and sensitive biotypes of kochia to the interaction of PBO and sulfonylurea herbicides in the greenhouse 2 WAT.

		Plant height		Visual injury	
Treatment	Rate	S biotype	R biotype	S biotype	R biotype
	(g ai/ha)	----- (cm/plant) -----		------ (%) ------	
Control		26.4	24.9	0	0
PBO	2000	26.3	24.4	0	0
Thifensulfuron	1.1	17.8	21.3	21	3
" + PBO	1.1+2000	14.9	21.3	45	6
Thifensulfuron	4.4	13.5	19.3	43	10
" + PBO	4.4+2000	11.0	16.3	58	19
Primisulfuron	20	11.6	11.9	52	44
" + PBO	20+2000	7.9	7.8	79	71
Primisulfuron	40	11.0	9.8	58	59
" + PBO	40+2000	8.8	6.3	76	77
LSD at 0.05		4.4		21	

SUMMARY AND CONCLUSION

The effects of antidotes, mixed function oxidases (MFO) inhibitors, insecticides, and corn hybrids on the activity of acetolactate synthase (ALS) inhibiting herbicides were evaluated in greenhouse and field studies. Chlorsulfuron-resistant kochia was evaluated in greenhouse for cross-resistance.

Northrup King 9283 and Cargill 7567 hybrids were sensitive to the interaction of chlorimuron, nicosulfuron and primisulfuron with terbufos. Cargill 7567 hybrid corn was more tolerant to acetanilide herbicides and to the interactions of sulfonylurea herbicides with terbufos than Northrup King 9283. But, there was no interaction of imazaquin with terbufos to corn hybrids. The antidotes, CGA-154281 and NA reduced corn injury from metolachlor, nicosulfuron and primisulfuron applied with or after terbufos treatment, respectively. NA stimulates MFO activity and can partially overcome the sulfonylurea herbicide-terbufos interaction. The combination of primisulfuron and terbufos did not enhance herbicidal activity to weed species.

Tank-mixed piperonyl butoxide (PBO) enhanced nicosulfuron and thifensulfuron activity on barnyardgrass, velvetleaf and common lambsquarters, respectively. Also, butylated hydroxyanisole (BHA) and PBO enhanced nicosulfuron and primisulfuron activity on common lambsquarters and green foxtail.

All three factors, PBO, nonionic adjuvants and 28% UAN, enhanced activity of nicosulfuron on common lambsquarters, velvetleaf, barnyardgrass, and primisulfuron on giant foxtail and velvetleaf, and thifensulfuron on common lambsquarters and velvetleaf. But, addition of 28% UAN to primisulfuron did not enhance activity to common lambsquarters and barnyardgrass. Effective adjuvants with nicosulfuron were K-3000 on common lambsquarters, and SYLGARD 309 on velvetleaf, K-2000, K-3000 and SCOIL on barnyardgrass. Effective adjuvant with primisulfuron were K-2000, SCOIL, and SYLGARD 309 on giant foxtail, X-77, K-2000, K-3000, SCOIL, and SYLGARD 309 on velvetleaf, K-3000 and SYLGARD 309 on common lambsquarters. Effective adjuvants for thifensulfuron were SCOIL on common lambsquarters, and SCOIL and SYLGARD 309 on velvetleaf.

PBO at 0.33 kg/ha tank-mixed with nicosulfuron and primisulfuron caused injury to the Northrup King 9283 corn hybrid. Pioneer 3377 IR corn hybrid was tolerant to the combination of nicosulfuron, primisulfuron plus PBO 2 kg/ha, and also to the combination treatments of imazethapyr or thifensulfuron with terbufos.

In the field study, Pioneer 3377 IR and Ciba 4393 RSC hybrids showed cross-resistance to sulfonylurea and imidazolinone herbicides even treatments with PBO regardless of the presence of terbufos. ICI 8532 IT was cross-resistant to thifensulfuron and imidazolinone herbicides plus terbufos.

The combination of thifensulfuron with PBO caused injury to Elgin '87 soybean, but the W20-STS soybean was tolerant to this combination treatment. Combination of imazethapyr with PBO or BHA had no effect on the growth of Elgin '87 soybean variety.

The chlorsulfuron resistant-kochia biotype was resistant to six herbicides: triflusulfuron, thifensulfuron, MON 12037, imazamethabenz, chlorsulfuron, and nicosulfuron. But, the resistant kochia biotype showed sensitivity similar to the susceptible biotype to three herbicides: metsulfuron, imazethapyr, and imazaquin. Addition of PBO at 2 kg/ha to primisulfuron and thifensulfuron increased injury and reduced plant height of chlorsulfuron resistant kochia biotype.

From the my research, I would like to recommend several methods to protect corn from the interaction of ALS-inhibiting herbicides with corn insecticides, and to enhance activity of these herbicides.

First, selection of an appropriate corn hybrid, resistant to the interaction of ALS-inhibiting herbicides with terbufos can protect corn from interaction effects. The degree of corn injury from the interaction of ALS-inhibiting herbicides with corn insecticides showed greatly variability with hybrid. Use of Pioneer 3377 IR and Ciba 4393 RSC hybrids appear to allow use of sulfonylurea herbicides POST with terbufos, the use of imazethapyr, and the use of PBO. Also, the W20-STS soybean appear to allow use of thifensulfuron plus PBO to increase weed control.

Second, I would recommend the addition of PBO and/or 28% UAN to sulfonylurea herbicides to enhance weed control. Tank-mixing PBO and/or 28% UAN to certain sulfonylurea herbicides increased the activity on several weed species, especially broad-leaf weed species.

Third, herbicide activity can be increased by the selection of an appropriate adjuvant. Efficacy of the nonionic adjuvants was herbicide and weed specific.

Fourth, a weed biotype resistant to one inhibitor of ALS may be effectively controlled by other inhibitors of ALS.

APPENDIX : Analysis of Variance Table

Chapter 2.

Table 1.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
1	Rep.	7	1033	148	2.7
2	Hybrid	1	2642	2642	48.5
4	Insect.	2	122	61	1.1
6	H x I	2	136	68	1.2
8	Herb.	6	22547	3757	68.9
10	H x H	6	4472	745	13.7
12	I x H	12	704	59	1.1
14	HxIxH	12	468	39	.7
-15	Error	287	15646	55	
	Total	335	47771		

c.v.: 18.58%

Table 2.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
1	Rep.	7	969	138	0.6
2	Hybrid	1	28527	28527	118.7
4	Insect.	2	161	80	0.3
6	H x I	2	740	370	1.5
8	Herb.	6	101176	16863	70.2
10	H x H	6	25341	4223	17.6
12	I x H	12	2281	190	0.8
14	HxIxH	12	1535	128	0.5
-15	Error	287	68981	240	
	Total	335	47771		

c.v.: 90.69

Table 3.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
1	Rep.	7	969	138	0.6
2	Hybrid	1	28527	28527	118.7
4	Insect.	2	161	80	0.3
6	H x I	2	740	370	1.5
8	Herb.	6	101176	16863	70.2
10	H x H	6	25341	4223	17.6
12	I x H	12	2281	190	0.8
14	HxIxH	12	1535	128	0.5
-15	Error	287	68981	240	
	Total	335	47771		

c.v.: 90.69%

Table 5.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
1	Rep.	7	325	46	1.5
2	Hybrid	1	453	453	14.5
4	Antidote	1	5472	5472	175.6
6	HA	1	366	366	11.7
8	Insect.	1	17537	17537	562.9
10	HI	1	339	339	10.9
12	AI	1	3545	3545	113.8
14	HAI	1	313	313	10.0
16	Herbi	2	19252	9626	309.0
18	HH	2	378	189	6.1
20	AH	2	3561	1781	57.2
22	HAH	2	180	90	2.9
24	IH	2	14049	7024	225.5
26	HIH	2	354	177	5.7
28	AIH	2	1859	929	29.8
30	HAIH	2	232	116	3.7
-31	Error	161	5016	31	
	Total	191	73230		

c.v.: 52.15%

Chapter 3.

Table 6. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	Herbicide	2	2605	1303	16.3
4	PBO	4	74868	18717	233.6
6	Herb x PBO	8	60251	7531	94.0
-7	Error	105	8413	80	

c.v.: 19.39%

Table 7. Visual injury.

K Value	Source	DF	Sum of Square	Mean Square	F Value
2	Herbicide	2	2383	1191	24.5
4	PBO	4	56672	14168	291.3
6	Herb. x PBO	8	45775	5722	117.7
-7	Error	105	5106	49	

c.v.: 17.58%

Table 8. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	3	2148	716	7.0
4	Adjuvant	5	249166	49833	485
6	PA	15	11324	755	7.3
8	Nitrogen	1	2395	2395	23.3
10	PN	3	299	100	1.0
12	AN	5	2143	429	4.2
14	PAN	15	4908	327	3.2
-15	Error	336	34551	103	

c.v.: 14.40%

Table 9. Visual injury

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	3	6955	2318	12.3
4	Adjuvant	5	178043	35609	189.6
6	PA	15	7457	497	2.7
8	Nitrogen	1	18634	18634	99.2
10	PN	3	793	264	1.4
12	AN	5	4443	889	4.7
14	PAN	15	3343	223	1.2
-15	Error	336	63103	188	

c.v. : 22.30%

Table 10. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	3	952	317	7.7
4	Adjuvant	5	244766	48953	1183.3
6	PA	15	6496	433	10.5
8	Nitrogen	1	6017	6017	145.4
10	PN	3	171	57	1.4
12	AN	5	10065	2013	48.7
14	PAN	15	1217	81	2.0
-15	Error	336	13900	41	

c.v. : 11.15%

Table 11. Visual injury

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	3	5833	1945	31.7
4	Adjuvant	5	138244	27649	451.2
6	PA	15	19663	1311	21.4
8	Nitrogen	1	1258	1258	20.5
10	PN	3	81	27	0.4
12	AN	5	313	63	1.0
14	PAN	15	662	44	0.7
-15	Error	336	20591	61	

c.v. : 12.13%

Table 12. Visual injury

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	2	565	282	0.8
4	Adjuvant	6	17004	2834	7.7
6	PA	12	18652	1554	4.3
8	Nitrogen	1	107	107	0.3
10	PN	2	833	416	1.1
12	AN	6	4215	703	1.9
14	PAN	12	7451	621	1.7
-15	Error	294	107638	366	

c.v. : 25.77%

Table 13. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	2	24810	12405	79.7
4	Adjuvant	6	86033	14339	92.2
6	PA	12	29462	2455	15.8
8	Nitrogen	1	62022	62022	398.7
10	PN	2	7484	3742	24.1
12	AN	6	22510	3752	24.1
14	PAN	12	16242	1353	8.7
-15	Error	294	45741	156	

c.v. : 16.32%

Table 14. Plant height.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	2	1807	904	12.9
4	Adjuvant	6	11022	1837	26.3
6	PA	12	3772	314	4.5
8	Nitrogen	1	85	85	1.2
10	PN	2	114	57	0.8
12	AN	6	316	53	0.8
14	PAN	12	445	37	0.5
-15	Error	294	20574	70	

c.v. : 8.67%

Table 15. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	2	11884	5942	24.0
4	Adjuvant	6	24645	4108	16.6
6	PA	12	4518	376	1.5
8	Nitrogen	1	1189	1189	4.8
10	PN	2	48	24	0.1
12	AN	6	1133	189	0.8
14	PAN	12	860	72	0.3
-15	Error	294	72818	248	

c.v. : 30.02%

Table 16. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	2	17668	8834	132
4	Adjuvant	6	39722	6620	99.3
6	PA	12	34674	2889	43.3
8	Nitrogen	1	1277	1277	19.1
10	PN	2	648	324	4.9
12	AN	6	2248	375	5.6
14	PAN	12	1216	101	1.5
-15	Error	294	19609	67	

c.v. : 11.60%

Table 17. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	PBO	2	11318	5659	45.3
4	Adjuvant	6	48795	8132	65.1
6	PA	12	10274	856	6.9
8	Nitrogen	1	123050	123050	985.1
10	PN	2	2622	1311	10.5
12	AN	6	13161	2194	17.6
14	PAN	12	3254	271	2.2
-15	Error	294	36725	125	

c.v.: 17.19%

Chapter 5.

Table 1. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	Biotype	1	37156	37156	106.0
4	Herbicide	12	151911	12659	36.1
6	BH	12	31185	2599	7.4
-7	Error	182	63974	351	

c.v.: 32.48%

Table 2. Visual injury.

K Value	Source	DF	Sum of Squares	Mean Square	F Value
2	Biotype	1	8023	8023	18.3
4	Herbicide	4	103750	25938	59.0
6	BH	4	9300	2325	5.3
8	PBO	1	7659	7659	17.4
10	BP	1	328	328	0.8
12	HP	4	3003	751	1.7
14	BHP	4	695	174	0.4
-15	Error	140	61538	440	

c.v.: 58.33%