Determination of Antagonism between Cyhalofop-butyl and Other Rice (*Oryza sativa*) Herbicides in Barnyardgrass (*Echinochloa crus-galli*)

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Herbicide antagonism is defined as the reduction of control of certain weeds as the result of applying mixtures of two or more herbicides. Cyhalofop-butyl, a graminicide used for postemergence grass weed control in rice, is antagonized by some rice herbicides when applied simultaneously. The result of this type of antagonism usually results in decreased control of grass weeds. Research has shown that herbicide antagonism between graminicides and other herbicides may be caused by different mechanisms as the result of activity of the tank-mix partner. Using HPLC, the objective of this experiment was to analyze the fate of cyhalofop-butyl in barnyardgrass tissue when applied alone and in combination with halosulfuron, propanil, or triclopyr. Results indicated that absorption of cyhalofop-butyl and hydrolysis to its phytotoxic metabolite, cyhalofop-acid, was rapid and that halosulfuron and triclopyr had no effect. Because of a likely interaction of propanil with an apoplastic esterase enzyme, increased levels of cyhalofop-butyl and cyhalofop-acid were detected in barnyardgrass tissue, indicating that cyhalofop-butyl metabolism was hindered by propanil.

KEYWORDS: Barnyardgrass [*Echinochloa crus-galli* (L.) Beauv.]; cyhalofop-acid; cyhalofop-butyl; herbicide antagonism

INTRODUCTION

Cyhalofop-butyl (CB) [(2*R*)-2-[4-(4-cyano-2-fluorophenoxy)-phenoxy]propanoic acid, butylester], fenoxaprop-ethyl [2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoic acid, ethylester], fluazifop-P [(2*R*)-2-[4-[[5-(trifluoromethyl)-2-pyridinyl]-oxy]phenoxy]propanoic acid, butylester], and quizalofop-P [(2*R*)-2-[4-[(6-chloro-2-quinoxalinyl)oxy]phenoxy]propanoic acid, ethylester] are graminicides and are members of the aryloxyphenoxy propionate family of herbicides. They stop plant growth by inhibiting the acetyl CoA carboxylase (ACCase) enzyme (*1*). Members of this herbicide family are formulated as esters to facilitate movement through the cuticle. Once in the plant, they are rapidly hydrolyzed to the acid form, which in most cases is the herbicidally active form (*2*). In tolerant species, increased ACCase activity (*3*) and rapid metabolism to herbicidally inactive compounds leads to selectivity (*4*).

It has been reported that single postflood applications of fenoxaprop-ethyl controlled propanil-resistant barnyardgrass [*Echinochloa crus-galli* (L.) Beauv.] (*5*). CB is an effective herbicide for barnyardgrass control when applied postemergence alone or in combination with residual rice herbicides (*6*−*7*) and

has been shown to control one- to three-leaf barnyardgrass 91−98% control when applied at a rate of 210 g ha^{-1} (*8*).

Herbicide antagonism is defined as the reduction of control of certain weeds as the result of applying mixtures of two or more herbicides. CB can be antagonized by other rice herbicides, causing a reduction in barnyardgrass control when applied to rice (*9*). Similarly, fenoxaprop-ethyl was antagonized by bensulfuron [2-[[[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]methyl]benzoic acid] and bentazon [3-(1-methylethyl)-1*H*-2,1,3-benzothiadiazin-4(3*H*)-one-2,2-dioxide], causing a reduction in barnyardgrass control of 40% when applied as a tank mixture (*10*). Fenoxaprop-ethyl activity was antagonized by acifluorfen [5-[2-chloro-4-(trifluoromethyl)-phenoxy]-2-nitrobenzoic acid], causing a reduction in barnyardgrass control when applied in soybean [*Glycine max* (L.) Merr.] (*11*).

Theories exist as to why antagonism occurs between graminicides and other herbicides. One theory proposed that the uptake of the graminicide sethoxydim [2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one] (cyclohexanedione family) was impeded as the result of bentazon decreasing the activity of plasma membrane ATPases (*12*). Other research found that imazapic [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-methyl-3-pyridinecarboxylic acid] did not alter the absorbance of clethodim [2-[(1*E*)-1-[[[(2*E*)-3-chloro-2-propenyl]oxy]imino]propyl]-5-[2-

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(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one] into goosegrass [*Eleusine indica* (L.) Gaertn.] (*13*); however, they suggested that the presence of imazapic slowed photosynthate transport processes, which affected clethodim movement to its active sites in the plant. A similar study found that the ALS-inhibiting herbicide DPX-PE350 [sodium 2-chloro-6-[(4,6-dimethoxy-pyrimidin-2-yl)thio]benzoate] did not alter the uptake or metabolism of fluazifop-P in large crabgrass [*Digitaria sanguinalis* (L.) Scop.] but did reduce translocation of ^{14}C fluazifop-P out of the treated leaf (*2*). Furthermore, tribenuron [2-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)methylamino] carbonyl]-amino]sulfonyl]benzoic acid] reduced basipetal movement of diclofop in wild oat (*Avena fatua* L.) but had no effect on diclofop [2-[4-(2,4-dichlorophenoxy)phenoxy]propanoic acid, methylester] absorption or metabolism (*14*). A recent report suggested that apoplastic esterases were responsible for the hydrolysis of haloxyfop-methyl [methyl 2-(4-((3-chloro-5-(trifluoromethyl)-2-pyridinyl)oxy)phenoxy) propanoate], fenoxaprop-ethyl, and fluazifop-butyl to their respective acids. It also suggested that organophosphate and carbamate insecticides block esterase activity, which might explain interactions between these compounds and propanil in plants (*15*).

Results from a study examining the basis of antagonism between bromoxynil [3,5-dibromo-4-hydroxybenzonitrile] and quizalofop-P found that the presence of bromoxynil reduced absorption of quizalofop-P into yellow foxtail (*Setaria glauca* L.) leaves (*16*). Other research has shown that aryloxyphenoxy propionate herbicides can serve as competitive inhibitors of synthetic auxin-receptor binding proteins, reducing auxin-like herbicide activity in broadleaf weeds (*17*).

Cyhalofop-acid (CA) [(2*R*)-2-[4-(4-cyano-2-fluorophenoxy)-phenoxy]propanoic acid], the primary metabolite of CB in susceptible grasses, is the herbicidally active metabolite (*4*). It is unknown as to whether there is a reduction of this metabolite when CB is tank-mixed with other herbicides or whether there is competition from these herbicides for an active site within the plant.

Applying graminicides simultaneously with other herbicides would be of great benefit to rice producers because it would allow them to control a wide variety of weeds with one herbicide application; however, because control of barnyardgrass and other grass weeds can be reduced when graminicides are mixed with other herbicides, they must be applied separately to maximize control. This phenomenon necessitates multiple herbicide applications, which increases costs to the producer.

No published research has been done to determine the fate of CB in susceptible plants either applied alone or in a mixture with other herbicides. Using barnyardgrass resistant to both propanil and quinclorac [3,7-dichloro-8-quinolinecarboxylic acid] as a test species, the objective of this research was to elucidate the fate of CB when applied to propanil and quinclorac multiple-resistant barnyardgrass alone and in tank mixtures with halosulfuron [3-chloro-5-[[[[(4,6-dimethoxy-2-pyrimidinyl)ami-no]carbonyl]amino]sulfonyl]-1-methyl-1*H*-pyrazole-4-carboxylic acid], propanil, or triclopyr [[(3,5,6-trichloro-2-pyridinyl)-oxy]acetic acid] using high-performance liquid chromatography (HPLC).

MATERIALS AND METHODS

Chemicals. CB and its metabolites were provided by Dow Agro-Sciences (Indianapolis, IN).

Plant Material. Propanil and quinclorac multiple-resistant barn-yardgrass seeds obtained from a population in Louisiana were planted in 40- by 60-cm plastic trays containing a commercial potting media (Sunshine Mix #1, Sun Gro Horticulture, Bellevue, WA). Seeds from multiple-resistant plants were chosen because of their exceptional germination ability, and their resistance to both propanil and quinclorac had no bearing on the experiment. Plants were maintained in a greenhouse with approximate daily minimum and maximum temperatures of 20−30 °C. A 13-h photoperiod of natural light was provided. All trays received 250 mL of a 25-g/L commercial fertilizer (Miracle-Gro, Marysville, OH) at emergence and 10 days after emergence.

The study was conducted using a split-plot design with four replications per treatment. Main plots were herbicide treatments, and subplots were harvest timings. Plot size was one tray (40 by 60 cm) of plants per herbicide treatment. At the three-leaf growth stage, CB at 280 g ha^{-1} was applied alone or in combination with propanil (3.36 kg ha^{-1}), halosulfuron (53 g ha^{-1}), or triclopyr (280 g ha^{-1}) using a spray chamber calibrated to deliver 93 L ha^{-1}. Crop oil concentrate (Agri-Dex, Helena Chemical, Memphis, TN) at 2.5% v/v was included in all treatments. After application, plants were placed in the greenhouse.

One-fourth of the plants in each plot were randomly harvested at 0.5, 4, 10, and 18 h after application (HAA) by excising the upper one-half with scissors. Harvest samples were placed in 18- by 20-cm resealable plastic bags and stored at −20 °C until further analysis.

Barnyardgrass Extraction. Samples were homogenized in 20 mL of liquid nitrogen using a mortar and pestle. An approximately 5-g portion of the homogenate was removed, weighed, and placed in a 250-mL plastic bottle with a cap containing 80 mL of methanol and 20 mL of 1 N hydrochloric acid. Samples were shaken for 1 h on a reciprocating shaker table at a speed of 180 cycles min^{-1}. The homogenate was then filtered with vacuum through #1 qualitative filter paper (Whatman, Inc., Clifton, NJ) into a 250-mL filter flask. The plastic bottle was rinsed with 50 mL of methanol, and the rinse was filtered through the same filter. The combined filtrate and rinse was transferred into a 500-mL separatory funnel containing 150 mL of a 6% sodium chloride solution in deionized (DI) water. The solution was extracted with three 50-mL additions of methylene chloride, shaking the separatory funnel for 30 s extraction^{-1}. The combined methylene chloride portions were reduced to dryness under a stream of nitrogen in a water bath set at 45 °C.

The dried samples were redissolved with 10 mL of methanol/pH 3 DI water (7:3, v/v), vortexed for 10 s, and sonicated for 10 s. A 1.5-mL aliquot was removed and placed in a 2-mL centrifuge vial. Samples were centrifuged at 8500 rpm for 10 min and transferred to a vial (Miniuniprep 0.2 μm nylon syringeless filter, Whatman, Inc., Clifton, NJ). A plunger, consisting of a nylon filter at the base with a cap and septum, was pushed through the liquid in the vial as a secondary means of filtration, thereby preparing samples for analysis.

Untreated homogenized plant samples were fortified prior to extraction with 300 μL of a 94.9% acetone/5% water/0.1% acetic acid solution containing 1000 μg/mL standards of CB, CA, cyhalofop-diacid [4-[4-(1-carboxyethoxy)phenoxy]-3-fluorobenzoic acid] (CD), and cy-halofop-amide [2-[4-[4-(aminocarbonyl)-2-fluorophenoxy]phenoxy]pro-panoic acid] (CM) (**Figure 1**). CD and CM standards were included in fortified samples as references in the event that the presence of another herbicide caused formation of these metabolites in barnyardgrass. It was understood that these metabolites had only been detected in resistant rice plants upon application of CB (*4*).

HPLC Conditions. The HPLC system consisted of a Hitachi L-7450A diode array detector, an L-7200 autosampler, an L-7100 pump, and Hitachi HSM software for data processing. The column (Gemini 5μ, 250 by 2.0 mm, Phenomenex, Torrance, CA) consisted of a C18 stationary phase and was maintained at a consistent temperature of 40 °C. The gradient used pH 2 DI water with hydrochloric acid and HPLC-grade acetonitrile. The program was 20% acetonitrile at 0.35 mL min^{-1} for 1 min, increased to 85% acetonitrile over 0.1 min, held for 14.9 min at 0.35 mL min^{-1}, and re-equilibrated for 12 min. The total run time was 28 min, and data were collected for the first 16 min. The injection volume was 10 μL, and quantitation was at 250 nm.

Quality Control. Quality control measures included a lab fortified and lab blank per 10 samples. Percent recovery and method limit of detection were based on lab-fortified samples ($n = 10$). Recovery of all analytes was >98%, and samples were not corrected for percent
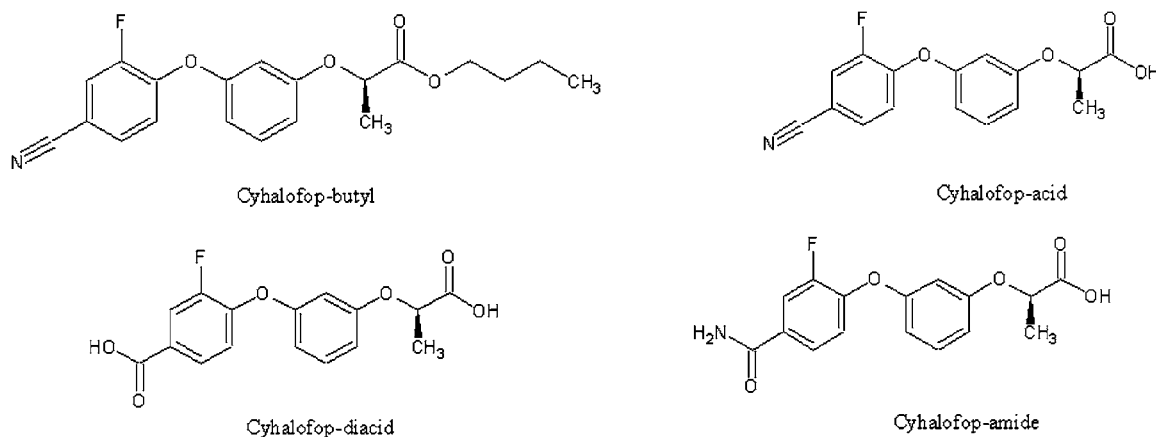
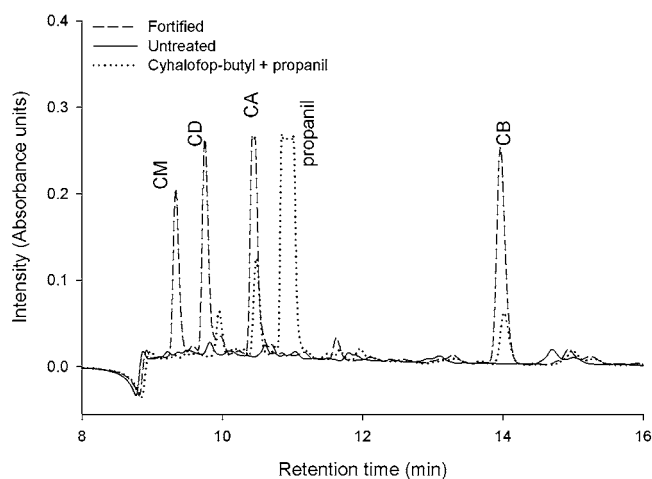**Figure 1.** Structures of CB and its metabolites, CA, CD, and CM.

Figure 2. HPLC chromatograms of extracts of propanil and quinclorac multiple-resistant barnyardgrass tissue receiving no treatment (—), fortified with 300 μL of a 1000 μg/mL solution of CB (- - -), CA, CD, and CM, or treated with CB and propanil (···).
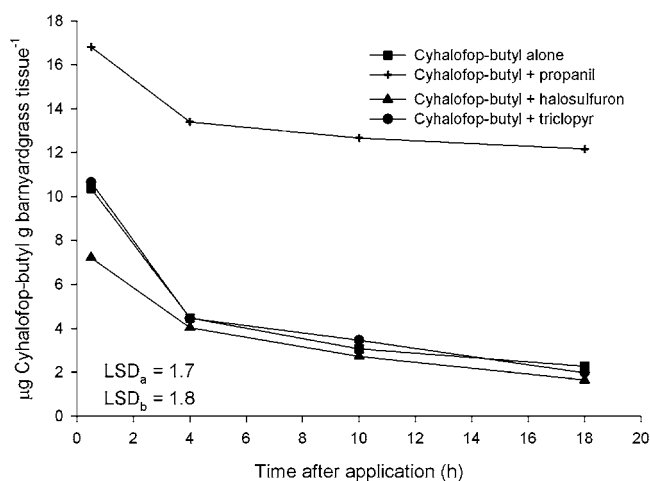

Figure 3. Detection of CB in propanil and quinclorac multiple-resistant barnyardgrass over time following applications of CB alone (■) and in combination with halosulfuron (▲), propanil (+), or triclopyr (●). LSD_a represents the difference within a herbicide treatment across harvest times, and LSD_b represents the difference across herbicide treatments within a harvest time.

recovery. The method limit of detection for CB, CA, CD, and CM was 1.5, 1.1, 1.0, and 1.0 μg/mL, respectively.

Statistical Analysis. All data were subjected to ANOVA with sums of squares partitioned to reflect a split-plot treatment structure using the general linear model in SAS (version 8.02, Statistical Analysis Systems Institute, Inc., Cary, NC). Metabolism data of CB as affected by the four treatments and four timings were separated using Fisher's protected LSD test at the 5% level. LSD values were calculated as appropriate for a split-plot design.

RESULTS AND DISCUSSION

HPLC determination did not reveal the CD or CM metabolites in barnyardgrass tissue samples with any treatment or harvest time (**Figure 2**). Therefore, only the butyl (CB) and acid (CA) forms of cyhalofop were analyzed. Because our recovery of the metabolites was consistent across runs, we concluded that the formation of the CD and CM metabolites was not a cause of antagonism in otherwise susceptible barnyardgrass in the presence of other herbicides and must only occur in resistant plants (*4*). HPLC analysis of the herbicide mixtures prior to barnyardgrass application was conducted to ensure that hydrolysis from the CB to CA did not take place prior to application. Samples were run repeatedly over an 8-h period, and no increases in the CA metabolite were noted over that time.

CB metabolism in barnyardgrass over time was similar for all herbicide treatments (**Figure 3**). More CB was detected when

CB was applied with propanil at all harvest times; however, metabolism of CB in combination with propanil over time appeared to be similar to the other herbicide treatments. Between 0.5 and 4 HAA, there were reductions in CB for all herbicide treatments. At 4 HAA, CB metabolism was slow, with no differences in CB detected between 4 and 18 h within a herbicide treatment. At 0.5 HAA, CB within the CB and halosulfuron treatment was detected at lower amounts compared to the other treatments; however, by 4 HAA, there were no differences among treatments of CB alone or in combination with halosulfuron or triclopyr.

CA metabolism in barnyardgrass tissue over time is depicted in **Figure 4**. On the basis of the findings from the preliminary analysis of the herbicide mixtures prior to application to barnyardgrass, increases in CA in the unsprayed solution were not detected over time (data not shown). Therefore, we have concluded that the CA detected in barnyardgrass plant samples was formed on the plant surface or inside the plant. At 0.5 HAA, CA was detected at similar levels among the CB alone, CB and halosulfuron, and CB and propanil treatments. CA detection with the CB and propanil treatment was lower than the other treatments at 0.5 HAA but did not show the same dissipation trend as did CA in other treatments. CA levels decreased for the CB alone and CB and halosulfuron treatments at 4 and 10
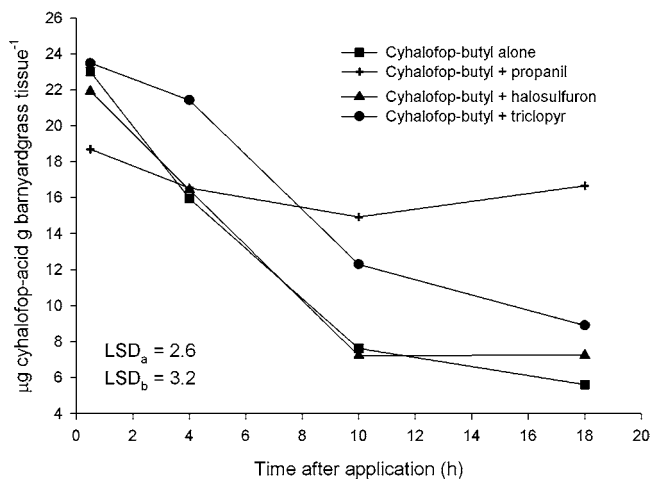
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Figure 4. Detection of CA in propanil and quinclorac multiple-resistant barnyardgrass over time following applications of CB alone (■) and in combination with halosulfuron (▲), propanil (+), or triclopyr (●). LSD$_a$ represents the difference within a herbicide treatment across harvest times, and LSD$_b$ represents the difference across herbicide treatments within a harvest time.

HAA, whereas CA levels from the CB and triclopyr treatment did not decrease until after 4 HAA.

The amount of CA was similar within each harvest time for the CB alone and CB and halosulfuron treatments. A similar relationship between the imidazolinone herbicide, imazapic, and the graminicide, clethodim, has been reported (*13*). It was determined that imazapic did not alter the absorption, translocation, or metabolism of clethodim in goosegrass leaves. Others have suggested that translocation of graminicides to the site of action in the meristem may be reduced by the effects of ALS-inhibiting herbicides on photosynthate transport processes in plants (*18*), while others have proposed that tribenuron impeded the basipetal movement of diclofop (*14*). Translocation of CB and its metabolites in the presence of other herbicides was beyond the scope of this study; however, it appeared that halosulfuron had no affect on the absorption and metabolism of CB and CA in barnyardgrass prior to 18 HAA and that antagonism must be the result of some other process.

Metabolism studies for CB in rice straw and susceptible grasses indicated that CB was rapidly converted to the CD metabolite in tolerant rice plants and was 50% of the total applied less than 10 HAA. In susceptible grasses, CA represented 80% of the total applied in the same time frame (*4*). In our experiment, CD and CM were not detected with any treatment, and CA comprised nearly 80% of the total cyhalofop recovered 4 HAA and was maintained at a consistent level of the total at 71% 10 and 18 HAA (**Table 1**). Although they did

not show results at the harvest times that we tested, it was concluded that fluazifop-P was 90% hydrolyzed to the acid form in large crabgrass within 24 HAA (*4*).

At 0.5 HAA, the herbicidally active CA metabolite comprised nearly 70% of the total cyhalofop recovered from barnyardgrass tissue, regardless of the presence of another herbicide (**Table 1**), indicating that hydrolysis from CB to CA occurred rapidly. These findings are supported by results from a study showing that application of CB at 180 g ha^{-1} to barnyardgrass after simulated rainfall 1 h later provided 85% barnyardgrass control (*19*).

It was reported that bromoxynil initially increased absorption of quizalofop-P in yellow foxtail 0.5 HAA and that the increased absorption of quizalofop-P might have been due to the formulated product serving as a solvent, aiding movement of quizalofop-P through the cuticle prior to cell-membrane destruction by bromoxynil (*16*). Our findings were similar with propanil. CB and CA were detected at higher levels within the CB and propanil treatment initially possibly because of the high solvent load in the propanil formulation; however, hydrolysis of CB appeared to cease beyond 0.5 HAA. Although we used propanil and quinclorac multiple-resistant barnyardgrass in our study, researchers have noted greater than 60% control of propanil-resistant barnyardgrass with sequential propanil applications (*5*). Therefore, propanil showed some initial activity on propanil-resistant barnyardgrass, probably because barnyardgrass resistance to propanil is due to increased metabolism from overexpression of the aryl acylamidase enzyme (*20*) and not a mutated binding site.

The stagnant detection levels of CB that we observed in tissue samples with the CB and propanil treatment might indicate that CB is not being metabolized at the same rate as the other treatments because of an inhibition of the enzyme responsible for the hydrolysis of CB to CA. Researchers have suggested that apoplastic esterases were responsible for the hydrolysis of haloxyfop-methyl, fenoxaprop-ethyl, and fluazifop-butyl to their respective acids (*15*). They also suggested that organophosphate and carbamate insecticides block esterase activity, which might explain interactions between these compounds and propanil in plants. On the basis of the fact that CB is in the same herbicide family, it is likely that these enzymes are responsible for the hydrolysis of CB and that propanil interacts with them in barnyardgrass, rendering them less effective.

Halosulfuron and triclopyr were antagonistic with CB when applied to barnyardgrass 3 or 5 days prior to CB or tank-mixed with CB, with control ranging from 29 to 81% and from 23 to 51%, respectively (*21*). Moreover, when halosulfuron or triclopyr was applied 5 days after CB, barnyardgrass control was 100%. In a laboratory study evaluating the antagonistic effect of 2,4-D on diclofop-methyl activity on wild oat, 2,4-D inhibited

Table 1. Percentages of CB and CA Detected in Propanil and Quinclorac Multiple-Resistant Barnyardgrass with CB Applied Alone or in Tank Mixtures with Halosulfuron, Propanil, or Triclopyr

| | percentages of recovered CB and CA as affected by time after treatment (h) | | | | | | | |
| | 0.5 | | 4 | | 10 | | 18 | |
herbicide treatment	CB (%)	CA (%)	CB (%)	CA (%)	CB (%)	CA (%)	CB (%)	CA (%)
cyhalofop-butyl alone	31	69	22	78	29	71	29	71
cyhalofop-butyl and halosulfuron	25	75	20	80	27	73	18	82
cyhalofop-butyl and propanil	53	47	55	45	54	46	58	42
cyhalofop-butyl and triclopyr	31	69	17	83	22	78	18	82
LSD$_a$[a] ($p = 0.05$)				5.1				
LSD$_b$[b] ($p = 0.05$)				4.9				

[a] Represents the difference within a herbicide treatment across harvest times. [b] Represents the difference across herbicide treatments within a harvest time.

hydrolysis of diclofop-methyl to the herbicidally active acid metabolite, which ultimately led to reduced movement to the meristematic site of action (*22*). Triclopyr, which has a similar mode of action to 2,4-D in susceptible species, did not appear to alter uptake or metabolism of CB within 18 HAA. Beyond 0.5 HAA, CA was detected at higher levels in barnyardgrass tissue, but it appeared that fate of CA within the CB and triclopyr treatment followed a similar trend to that of CB applied alone. Similarities were observed in the proportions of CB and CA at harvest times among the CB alone, CB and halosulfuron, or CB and triclopyr treatments indicating that the presence of halosulfuron or triclopyr did not appear to alter the absorption of CB or the hydrolysis of CB to CA (**Table 1**).

On the basis of these findings, it appears that antagonism of CB by triclopyr occurs beyond 18 HAA and is not likely associated with reduced hydrolysis of CB to CA as a result of the presence of triclopyr. Research has shown that the auxinic herbicide, 2,4-D, inhibited the production of active oxygen species induced by the effects of diclofop-methyl in oat, thereby reversing phytotoxicity associated with diclofop-methyl (*23*). It may be possible that the auxinic herbicide, triclopyr, has a similar effect on CB activity in barnyardgrass.

Herbicide antagonism is a problem for crop producers and continues to be a field of interest among herbicide researchers. The results of this study indicate that increased levels of CB in barnyardgrass when applied with propanil are likely due to the inhibition of an esterase enzyme by propanil. As reported with herbicides similar to CB, antagonism by halosulfuron and triclopyr is not due to reduced absorption or hydrolysis and occurs beyond 18 HAA. Future research will hopefully bring about remedies to antagonism, so that producers can apply a graminicide in combination with other herbicides and maintain adequate weed control with a single herbicide application.

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