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Resistance to ACCase-inhibiting herbicides in sprangletop (*Leptochloa chinensis*)

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This study reports evolved resistance to fenoxaprop-P in a population of sprangletop from a rice field in Thailand (BLC1). After eight applications of fenoxaprop-P, the herbicide appeared no longer effective. To confirm herbicide resistance in the BLC1 population, three experiments were conducted. First, glasshouse experiments revealed that the BLC1 population survived 600 g ai ha^{-1} of fenoxaprop-P without visual injury. Second, the BLC1 population was treated with fenoxaprop-P and other acetyl coenzyme A carboxylase (ACCase)–inhibiting herbicides (quizalofop-P, cyhalofop-butyl, and profoxydim) under field conditions; BLC1 exhibited resistance to all of these herbicides. Third, seeds of susceptible SLC1 and resistant BLC1 were germinated on 0.6% (v/v) agar across a range of herbicide concentrations. The resistant BLC1 population exhibited 61-, 44-, 9- and 8-fold resistance to fenoxaprop-P, cyhalofop, quizalofop-P, and profoxydim, respectively, compared with a susceptible SLC1 population. At the enzyme level, ACCase from the resistant BLC1 exhibited 30, 24, 11, 4, and 5 times resistance to fenoxaprop, cyhalofop-butyl, haloxyfop, clethodim, and cycloxydim, respectively. The spectrum of resistance at the whole plant level correlated well with resistance at the ACCase level. Hence, the mechanism of resistance to ACCase-inhibiting herbicides in this biotype of sprangletop is a herbicide-resistant ACCase. The specific mutation(s) of the ACCase gene that endows resistance in this population remains to be investigated.

Nomenclature: Clethodim; cycloxydim; cyhalofop; fenoxaprop-P; haloxyfop; profoxydim; quizalofop-P; sprangletop, *Leptochloa chinensis* (L.) Nees LEPCH; rice, *Oryza sativa* L.

Key words: ACCase-inhibiting herbicides, cross-resistance.

Two chemically distinct classes of herbicides, the aryloxyphenoxypropionate (APP) and cyclohexanedione (CHD) herbicides, are important in world agriculture and have the same mode of action. The APP and CHD herbicides are chemically dissimilar but both inhibit chloroplastic acetyl coenzyme A carboxylase (ACCase; EC 6.4.1.2) (Gronwald 1991). Injury symptoms produced by these herbicides on susceptible plants are necrosis in the meristematic tissue, necrosis or chlorosis in developing leaf tissue, and appearance of purple leaf coloring because of anthocyanin accumulation (Ishikawa et al. 1985; Swisher and Corbin 1982).

Since their commercial introduction from 1977, ACCase herbicides have become widely and very successfully used in world agriculture to control a broad range of grass weeds infesting a range of crops. A repercussion of their widespread and often persistent use has been the evolution of weed biotypes that are resistant to ACCase herbicides. Selection with ACCase herbicides has led to resistance in biotypes of many important weedy plant species (Devine and Shimabukuro 1994). A common feature of evolved resistance to ACCase herbicides is that there can be quite different patterns of resistance within or across both APP and CHD herbicides. Target site–based resistance because of a resistant ACCase enzyme is frequently responsible for resistance (Cocker et al. 1999; Gronwald et al. 1992; Kuk et al. 1999; Leach et al. 1995; Maneechote et al. 1994, 1997; Seefeldt et al. 1996; Shukla et al. 1997a, 1997b). There are now four mutations of the ACCase gene known to endow resistance to certain APP and CHD herbicides (Brown et al. 2002; Christoffers et al. 2002; Delye et al. 2002, 2003; Zagnitko et al. 2001). In addition to target site ACCase-based resistance, there can be non–target site based resistance, in some cases known to be endowed by increased rates of herbicide metabolism (Cocker et al. 2001; Hall et al. 1997; Hidayat and Preston 1997; Holtum et al. 1991; Maneechote et al. 1997; Preston and Powles 1998).

Sprangletop (*Leptochlea chinensis* L. Nees) is a serious grass weed in direct-seeded rice cropping systems in Thailand. At present, four ACCase herbicides, fenoxaprop-P, cyhalofop, quizalofop-P, and profoxydim, have been registered to control sprangletop in rice crops. These herbicides are applied as a spot application to grass weeds infesting rice crops. In Thailand, fenoxaprop-P has been used to control sprangletop for more than 15 yr. However, in 2002, we became aware of a population of sprangletop no longer controlled with fenoxaprop-P on a field with a history of eight once-annual applications of fenoxaprop-P and one application of cyhalofop.

In this study, we document the first case of sprangletop with evolved resistance to fenoxaprop-P and cross-resistance to other ACCase-inhibiting herbicides. A herbicide-resistant ACCase was determined to be the resistance mechanism in this population.

Materials and Methods

Plant Materials

A coding system was used to classify the sprangletop seed samples. The first letter is taken from the name of the prov-

ince of Thailand from where the population was collected. The subsequent letters are the genus and species of sprangletop. The number is the order of collection from each province. For example, population BLC1 represents sprangletop from Bangkok province, collection No. 1.

In May 2002, a farmer in Minburi District, Bangkok province, Thailand (latitude 13°44′42″N, longitude 100°42′30″E) reported that fenoxaprop-P failed to control a population of sprangletop. During the previous 4 yr, this putative resistant BLC1 population had been exposed to eight applications of fenoxaprop-P and one application of cyhalofop. Because this was the first report of fenoxaprop failure to control sprangletop in Thailand, the BLC1 field was treated twice with fenoxaprop-P at 12 and 19 d after sowing rice (DAS) at the rates of 37.5 and 75 g ai ha^{-1}, respectively. No phytotoxic symptom was evident on BLC1 plants (whereas the herbicide severely damaged rice plants) indicating resistance and therefore seeds of this putative resistant BLC1 were collected in July 2002. A susceptible population from Supanburi province known to be susceptible to all ACCase-inhibiting herbicides (SLC1) was used as a control in all experiments. Hereinafter, the herbicide-resistant BLC1 population will be referred to as R and the herbicide susceptible SLC1 population as S.

Verification of Resistance to Fenoxaprop-P

Glasshouse Experiments

Because of the herbicide failure in BLC1 field, resistance was investigated initially using the Syngenta Quick-Test (Boutsalis 2001). Plants surviving herbicide in the BLC1 field at 26 DAS were collected during May–June 2002 and transplanted into pots containing clay soil and thereafter maintained in a glasshouse using the protocol described by Boutsalis (2001). Each 15-cm-diam pot contained five cuttings taken from the field and after 7 d, the cuttings had produced new leaves 5 to 10 cm long. These plants were then sprayed with fenoxaprop-P[1] at 0, 75, 150, 300, and 600 g ai ha^{-1}, respectively. A knapsack sprayer with Teejet 8002 flat-fan nozzle delivering a spray volume of 375 L ha^{-1} at 275 kPa was used. For each herbicide rate, six replicate pots were used. At 21 d after application, survival plants were cut at soil level and dried at 70 C for 48 h before dry weight measurement.

Laboratory Experiment

A total of 400 seeds of the R and S were sown separately on 50 ml of 0.5% (wt/v) agar solidified water in 400-ml plastic containers (11 cm diameter). Various concentrations (0, 0.06, 0.12, 0.48, 0.96, 1.2, and 2.4 mg ai L^{-1}) of fenoxaprop-P[2] had been added to the solidified water agar. At each herbicide concentration, four replicate containers were used. These sealed containers were kept in a growth room at a light density of 50 µmol photon flux density with a 12-h photoperiod at 27 C. Seven days after treatment, seedlings with green, healthy shoots extended from the coleoptiles were counted as survivors. Total dry weight (TDW), shoot and root length of both R and S plants were also measured. The LD_{50} (dose giving 50% mortality) and GR_{50} (dose giving 50% reduction in growth) values of both R and S populations were calculated from the graph data.

Verification of Target Site Cross-resistance across APP and CHD Herbicides

All four ACCase herbicides registered for controlling sprangletop in Thailand (fenoxaprop-P,[1] cyhalofop-butyl,[3] quizalofop-P,[4] and profoxydim[5]) were tested on the R sprangletop to assess resistance across chemically dissimilar ACCase-inhibiting herbicides.

Field Experiments

Without rice. In August 2002, after rice harvest, the BLC1 field was densely covered with sprangletop seedlings. At the tillering stage, these seedlings were sprayed with each of three APP and one CHD herbicide. Fenoxaprop-P at 150, 300, and 1,500 g ai ha^{-1}, cyhalofop at 300 and 1,500 g ai ha^{-1}, quizalofop-P at 30 and 150 g ai ha^{-1}, and profoxydim at 100 g ai ha^{-1} were applied. Treatments were arranged in a complete block design (CRD) with four replicates. Plot size was 1 by 1 m. Two weeks after herbicide application, visual estimates of plant injury were recorded using a scale of 0 to 10 (0 = no injury, 1 to 3 = slightly toxic, 4 to 6 = moderately toxic, 7 to 9 = severely toxic, 10 = completely killed). Stem and panicle length of 10 plants was measured in each replicate. Data were analyzed by analysis of variance (ANOVA) and $LSD_{0.05}$ was used for mean comparison.

With rice. In January–April 2003, two identical field experiments were conducted in Bangkok and Supanburi provinces, where the seeds of R and S were originally collected. Rice seeds were pregerminated 2 d before sowing at the rate of 19 kg ha^{-1}. After sowing rice, the land was not irrigated for 15 d to allow most of the sprangletop seeds to emerge. At 15 DAS, the plants were at the two- to three-leaf stage and ACCase-inhibiting herbicides fenoxaprop-P, cyhalofop, quizalofop-P, and profoxydim were sprayed at their recommended rates of 150, 150, 30, and 100 g ha^{-1}, respectively. A knapsack sprayer with a Teejet 8002 flat-fan nozzle delivering a spray volume of 375 L ha^{-1} at 275 kPa was used in both field experiments. Treatments were arranged in a randomized complete block design with four replicates. Plot size was 4 by 4 m with 0.5-m guard rows. Two weeks after treatment, visual estimates of plant injury were recorded using a scale of 0 to 10 (0 = no injury, 1 to 3 = slightly toxic, 4 to 6 = moderately toxic, 7 to 9 = severely toxic, 10 = completely killed). The number of rice and sprangletop R plants in an area of 1 m^2 of each replicate was also counted. Data were analyzed by ANOVA, and $LSD_{0.05}$ was used for mean comparison.

Laboratory Experiment

R and S seeds were tested with four ACCase-inhibiting herbicides contained in agar as described previously. Fenoxaprop-P, cyhalofop-butyl, quizalofop-P, and profoxydim at a range of concentrations of 0 to 4.8, 0 to 1.2, 0 to 1.0, and 0 to 1.2 mg ai L^{-1}, respectively, were used. After 7 d, the numbers of seedlings with green shoots extended above coleoptiles were counted as survivors. TDW and shoot and root length of both R and S seedlings were also measured. The LD_{50} and GR_{50} were calculated. Difference in resistance between two populations was expressed as the ratio of the value of R divided by that of S. For each herbicide

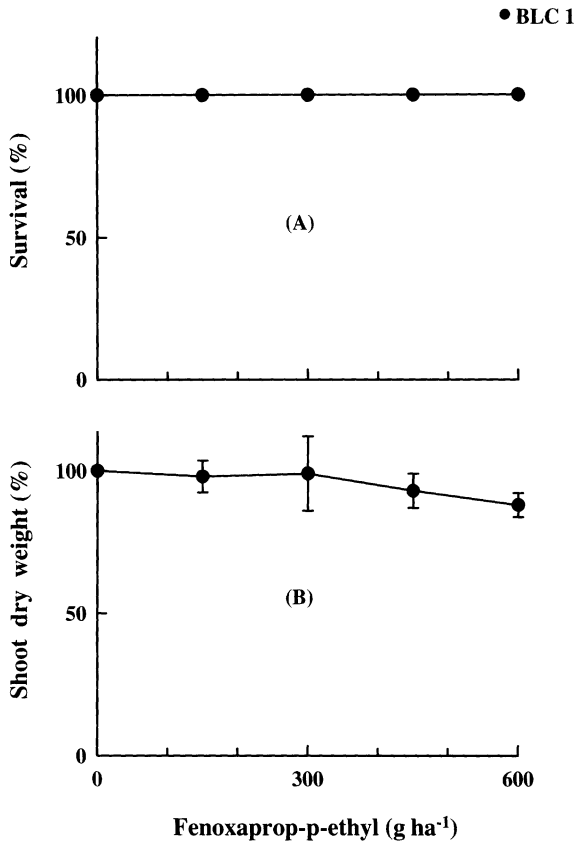

FIGURE 1. Survival (A) and shoot dry weight (B) of putative resistant sprangletop BLC1 population at 21 d after herbicide application. Points are the mean of the average of six replicates ± standard errors.

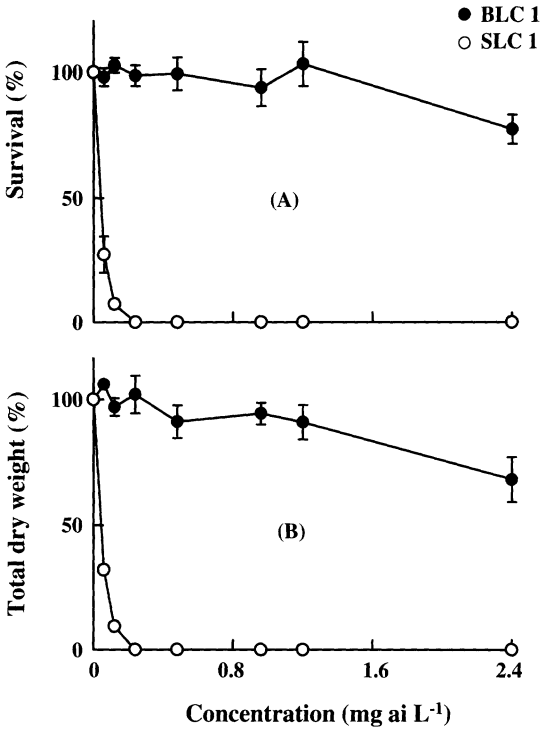

FIGURE 2. Survival (A) and total dry weight (B) of resistant BLC1 (●) and susceptible SLC1 (○) sprangletop populations when grown in 0.6% agar containing various concentrations of fenoxaprop-P (0, 0.06, 0.12, 0.48, 0.96, 1.2, 2.4, and 4.8 mg ai L^{-1}). Data represented in percent of untreated control. Points are the average ± standard error of four replicates.

concentration, there were four replicates and each experiment was repeated three times.

Inhibition of ACCase Activity by APP and CHD Herbicides

Seedlings at the three- to four-leaf stage were collected and kept at − 80 C until assayed. ACCase was extracted from seedlings using a mortar and pestle in liquid nitrogen. Partially purified ACCase of R and S populations were assayed in the presence of herbicides (fenoxaprop, haloxyfop, cyhalofop, clethodim, and cycloxydim) as described previously by Maneechote et al. (1994), with minor modification. All stock herbicide solutions were made up at 1 mM in 10% (v/v) acetone except cyhalofop-butyl,[5] which was dissolved in 50% (v/v) ethanol at 10 mM. All solutions were diluted with 100 mM Tricine before use. The I_{50} values (the herbicide dose required to inhibit the ACCase activity by 50%) were computed by regression analysis using SigmaPlot software.

Results

Verification of Resistance to Fenoxaprop

Glasshouse Experiment

Fenoxaprop resistance in the R population was evident with the Syngenta Quick-Test method (Boutsalis 2001). All plants from the BLC1 field survived at all rates of herbicide (75 to 600 g ha^{-1}) (Figure 1A). Shoot dry weight was not significantly reduced even when exposed to high rates of the herbicide (Figure 1B) and no visual phytotoxic symptoms were evident.

Laboratory Experiment

The R biotype exhibited a high level of resistance to fenoxaprop-P when seeds were germinated on agar containing herbicide. Fenoxaprop-P at 0.24 mg ai L^{-1} killed all S, whereas all R plants survived. At the highest concentration of 2.4 mg ai L^{-1}, 77% of the R population survived (Figure 2A). TDW of the S was reduced by 90% when germinated at a very low concentration of 0.12 mg ai L^{-1}. In contrast, at the highest concentration of 2.4 mg ai L^{-1}, the dry weight of the R plants was reduced by only 20% (Figure 2B). In addition, the shoot and root length of R plants were reduced only 13 and 27% when exposed to the highest concentration of 2.4 mg ai L^{-1} (data not shown).

Verification of Target Site Cross-resistance Across APP and CHD Herbicides

Field Experiments

Without rice. The R population was found to be highly resistant to fenoxaprop-P. Plants were unaffected when treated with fenoxaprop-P at four times commercial rates (Table 1). However, the shoot length of R plants was significantly inhibited when treated with the extreme dose of 40 times commercial rates. Slight phytotoxicity was observed only on older leaves, but the young leaves did not show symptoms. In addition, the R population exhibited resistance to other APP herbicides cyhalofop and quizalop-p-tefuryl. At high

TABLE 1. Phytotoxicity, shoot length, and panicle length of sprangletop BLC1 plants at 14 d after treatment (DAA) with four ACCase-inhibiting herbicides.

Treatment	Rate	Factor of CR[a]	Phytotoxicity[b] 14 DAA	Shoot length	Panicle length
	g ai ha^{-1}			cm	
Fenoxaprop-P	150	4×	0	88.3 a	40.4 a
Fenxaprop-P	300	8×	1	78.9 ab	39.5 a
Fenoxapro-P	1,500	40×	2	75.7 b	36.6 a
Cyhalofop-butyl	300	3×	2	63.8 c	26.9 b
Cyhalofop-butyl	1,500	15×	5	49.4 d	18.2 c
Quizalofop-P	30	1×	3	73.5 bc	35.9 a
Quizalofop-P	150	5×	6	49.7 d	22.8 bc
Profoxydim	100	1×	1	83.8 b	37.3 a
Untreated control	—	—	0	96.3 a	38.1 a
$LSD_{0.05}$				8.0	5.8

[a] Abbreviation: CR, commercial rate.
[b] Phytotoxicity level: 0 = no injury, 1 to 3 = slightly toxic, 4 to 6 = moderately toxic, 7 to 9 = severely toxic, 10 = completely killed.

rates of both APP herbicides, moderately phytotoxic symptoms appeared on the older leaves; however, the newly emerged leaves were unaffected. The lengths of shoots and panicle were inhibited by 40 to 50%. R sprangletop plants showed slight symptoms only when treated with the CHD herbicide profoxydim (Table 1).

With rice. The density of sprangletop in the Bangkok and Supanburi field experiments were 3,053 and 726 plants m^{-2}, respectively (Table 2). In Supanburi, the results showed that all the S plants were killed by all ACCase-inhibiting herbicides (Table 2), whereas at four times commercial rate of fenoxaprop-P, the R plants showed no mortality. At this dose, 63% of the rice plants were killed. Similarly, the number of sprangletop in the plots treated with cyhalofop and quizalofop-P were not significantly reduced. Profoxydim at the commercial rate reduced the density of sprangletop by 50% (Table 2). After the rice harvest, seeds of the BLC1 population were collected for laboratory use and then the farmer took dramatic action to try to eradicate the resistant weed problem to prevent future yield loss and prevent dispersion to neighbors. Thus it was not possible to repeat the experiment.

Laboratory Experiment

The laboratory experiment provided further evidence that the R plants exhibited a high level of resistance to fenoxaprop-P and cyhalofop and a moderate level of resistance to quizalofop-P and profoxydim (Table 3). The R population was 61 times more resistant to fenoxaprop-P than S. In addition, R exhibited cross-resistance to other APP and CHD herbicides. The LD_{50} values of R were 44, 9, and 8 times higher than S in the presence of cyhalofop, quizalofop-P, and profoxydim, respectively. The GR_{50} values were 38, 28, 6, and 5 times higher for R than S when treated with fenoxaprop-P, cyhalofop, quizalofop-P, and profoxydim, respectively.

Inhibition of ACCase Activity by APP and CHD Herbicides

To establish the mechanism of resistance, ACCase was extracted from leaves of R and S plants. Without herbicide, the amount of ACCase activity from both R and S biotypes were not different (data not shown). At the enzyme level, R showed a high level of resistance (30-fold) to the APP herbicide fenoxaprop but exhibited a lower level of resistance (fivefold) to the CHD herbicide cycloxydim (Figure 3; Table 4). ACCase enzyme from R plants also displayed resistance to other ACCase inhibitors. The I_{50} values of cyhalofop, haloxyfop, clethodim, and cycloxydim on ACCase activity from R were 24, 11, 4, and 5 times greater than that from S plants, respectively (Table 4).

Discussion

In Thailand, fenoxaprop-P has been used to successfully control grass weeds in rice fields for more than 15 yr. This

TABLE 2. Number of sprangletop and rice in 1 m^{-2} in two locations, Bangkok and Supanburi, at 15 d after treatment with herbicides. Sprangletop plants were at the two- to four-leaf stage when fields were sprayed at 15 d after sowing rice.

Treatments	Rate	Factor of CR[a]	Bangkok		Supanburi	
			BLC1	Rice	SLC1	Rice
	g ai ha^{-1}					
Fenoxaprop-P	150	4×	3,199 a	57 c	0 b	213 b
Cyhalofop-butyl	300	3×	2,280 ab	163 a	0 b	305 a
Quizalofop-P	30	1×	2,151 ab	81 bc	0 b	255 ab
Profoxydim	100	1×	1,695 b	131 ab	0 b	229 b
Untreated check	—		3,053 a	154 a	726 a	304 a
$LSD_{0.05}$			1,188	145	56	54

[a] Abbreviation: CR, commercial rate.

TABLE 3. LD_{50} and GR_{50} values of resistant sprangletop BLC1 (R) and susceptible SLC1 (S) grown in agar containing ACCase-inhibiting herbicides. Ratios are the value for resistant BLC1 divided by the value of susceptible SLC1.[a]

Herbicides	LD_{50} (mg ai L^{-1})			GR_{50} (mg ai L^{-1})		
	R	S	R/S ratio	R	S	R/S ratio
Aryloxyphenoxypropionates						
Fenoxaprop-P	4.9 ± 0.4	0.08 ± 0.0	61	3.42 ± 0.5	0.07 ± 0.0	38
Cyhalofop-butyl	3.1 ± 0.4	0.07 ± 0.1	44	1.96 ± 0.1	0.06 ± 0.0	28
Quizalofop-P	0.6 ± 0.1	0.07 ± 0.1	9	0.53 ± 0.2	0.09 ± 0.0	6
Cyclohexanediones						
Profoxydim	0.8 ± 0.1	0.11 ± 0.1	8	0.6 ± 0.1	0.11 ± 0.0	5

[a] Abbreviations: LD_{50}, dose giving 50% mortality; GR_{50}, dose giving 50% reduction in growth; ACCase, acetyl coenzyme A carboxylase.

herbicide is usually applied 20 to 30 DAS. At this time, most sprangletop is in the three- to four-leaf stage with one to two tillers and fenoxaprop-P at 42 g ha^{-1} has provided good control. Yokohama et al. (2001) reported that fenoxaprop-P at the rate of 7.5 to 10 g ha^{-1} gave 95 to 97% sprangletop control when sprayed at 14 DAS.

In field experiments, the R plants were found to be unaffected by fenoxaprop-P at the rate of 150 g ha^{-1} (about four times the commercial rate), a rate lethal to rice. When seedlings were germinated on agar containing fenoxaprop-P, the R plants were 61-fold resistant to fenoxaprop-P compared with S. At the enzyme level, the activity of ACCase from R plants was also less sensitive to fenoxaprop than the S. The I_{50} value of fenoxaprop on ACCase of R was 30 times greater than of S. Hence, resistance to fenoxaprop in the R population is because of a herbicide-resistant ACCase enzyme.

In many other weed species, a herbicide-resistant ACCase is the mechanism conferring resistance to APP or CHD herbicides (or both). For example, the presence of an insensitive form of ACCase has been documented in blackgrass (*Alopercurus myosuroides*) (Cocker et al. 1999), wild oat (*Avena fatua*) (Seefeldt et al. 1996), sterile oat (*Avena sterilis* spp. *ludoviciana*) (Maneechote et al. 1994, 1997; Shukla et al. 1997a), smooth crabgrass (*Digitaria ischaemum*) (Kuk et al. 1999), goosegrass (*Eleusine indica*) (Leach et al. 1995), Italian ryegrass (*Lolium multiforum*) (Gronwald et al. 1992), rigid ryegrass (*Lolium rigidum*) (Tardif et al. 1993), littleseed canarygrass (*Phalaris minor*) (Tal et al. 1996), giant foxtail (*Setaria faberi*) (Shukla et al. 1997b), and green foxtail (*Setaria viridis*) (Shukla et al. 1997b).

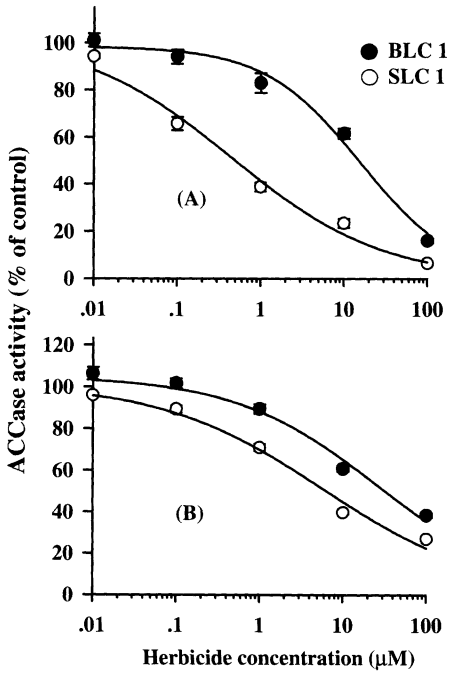

FIGURE 3. Inhibition of ACCase activity in resistant BLC1 (●) and susceptible SLC1 (○) sprangletop populations by fenoxaprop (A) and cycloxydim (B). Each point is the mean of three experiments with three replicates. Vertical bars represent standard error, which are sometimes obscured by point symbols.

In vivo, the R plants also exhibited a high level of resistance to cyhalofop and low level of resistance to quizalofop-P and profoxydim (Tables 1–3). In vitro, ACCase activity of R plants was highly less sensitive to cyhalofop and haloxyfop and slightly less sensitive to clethodim and profoxydim (Table 4). Clearly, R plants showed a certain level of target site cross-resistance to other APP and CHD herbicides.

To date, herbicide resistance has not been reported in sprangletop. The R population had been selected with eight applications of fenoxaprop-P and one application of cyhalofop before the development of high resistance to both herbicides. Currently, farmers in Thailand are relying on ACCase-inhibiting herbicides for sprangletop control because the application is more convenient than preemergence and early postemergence herbicides. Hence, the occurrence of resistance to ACCase inhibitors in sprangletop populations

TABLE 4. Concentrations of ACCase-inhibiting herbicides giving 50% inhibition of partially purified ACCase from resistant BLC1 and susceptible SLC1 populations. Ratios are the value for the resistant divided by the value for the susceptible. Values are the average ± standard error of four separate experiments.[a]

Herbicides	R[b]	S[b]	R/S ratio
Aryloxyphenoxypropionates			
Fenoxaprop	14.8 ± 0.4	0.5 ± 0.4	30
Cyhalofop-butyl	480.3 ± 46.6	20.4 ± 2.2	24
Haloxyfop	15.2 ± 0.8	1.4 ± 0.5	11
Cyclohexanediones			
Clethodim	5.0 ± 0.4	1.3 ± 0.1	4
Cycloxydim	26.5 ± 1.1	5.8 ± 0.2	5

[a] Abbreviations: ACCase, acetyl coenzyme A carboxylase; R, resistant; S, susceptible; I_{50}, herbicide dose required to inhibit the ACCase activity by 50%.

[b] I_{50} (μM) values.

in rice-growing areas in central Thailand is of economic significance.

In conclusion, we have documented evolved resistance to fenoxaprop-P and cross-resistance to cyhalofop, quizalofop-P, and profoxydim in a population of sprangletop from a rice field in Thailand. An altered ACCase that is less sensitive to these herbicides is a resistance mechanism in this biotype. The specific resistance endowing mutation of the ACCase gene remains to be identified.

Sources of Materials

[1] Whip at 75 g ai L^{-1}, Bayer CropScience (Thailand) Co. Ltd., 130/1 North Sathorn Road, Silom, Bangrak, Bangkok 10500, Thailand.

[2] Ricestar at 69 g ai L^{-1}, Bayer CropScience (Thailand) Co. Ltd., 130/1 North Sathorn Road, Silom, Bangrak, Bangkok 10500, Thailand.

[3] Grandstand at 100 g ai L^{-1}, Dow AgroSciences (Thailand) Co. Ltd., 14th–16th Floor, White Group Building II, 75 Soi Rubia, Sukhumvit 42 Road, Klongtoey, Bangkok 10110, Thailand.

[4] Zotus 40 at 40 g ai L^{-1}, Zotus Company, 26th Floor Jasmine International Tower, 200 Chaengwattana Road, Pakred, Nontaburi 11120, Thailand.

[5] Tetris at 75 g ai L^{-1}, BASF (Thai) Co. Ltd, 23rd Floor, Emporium Tower, 662 Sukhumvit 24 Road, Klongtoey, Bangkok 10110, Thailand.

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