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Cross-resistance Patterns to Acetyl-CoA Carboxylase Inhibitors Associated with Different Mutations in Japanese Foxtail (*Alopecurus japonicus*)

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Japanese foxtail is a grass weed in eastern China. This weed is controlled by fenoxaprop-*P*-ethyl, one of the most common acetyl-CoA carboxylase (ACCase)-inhibiting herbicides. Some Japanese foxtail populations have developed resistance to fenoxaprop-*P*-ethyl, owing to target-site mutations (amino acid substitutions) located within the carboxyl transferase domain of ACCase. In the present study, three mutations were detected in three fenoxaprop-*P*-ethyl–resistant Japanese foxtail populations: Ile-1781-Leu in JCJT-2, Ile-2041-Asn in JZJR-1, and Asp-2078-Gly in JCWJ-3. Two copies of *ACCase* (*Acc1-1* and *Acc1-2*) were identified, but mutations were detected only in *Acc1-1*. The derived cleaved amplified polymorphic sequence (dCAPS) method detected these mutations successfully in Japanese foxtail. The mutation frequencies in JCJT-2, JZJR-1, and JCWJ-3 were approximately 98%, 92%, and 87%, respectively. Different cross-resistance patterns to ACCase inhibitors were found in the three resistant populations. JCJT-2 (Ile-1781-Leu) and JZJR-1 (Ile-2041-Asn) showed cross-resistance to haloxyfop-*R*-methyl, clodinafop-propargyl, and pinoxaden, but were susceptible to clethodim. JCWJ-3 (Asp-2078-Gly) showed cross-resistance to all tested ACCase-inhibiting herbicides.

Nomenclature: Clethodim; clodinafop-propargyl; fenoxaprop-*P*-ethyl; pinoxaden; haloxyfop-*R*-methyl; Japanese foxtail, *Alopecurus japonicus* Steud.

Key words: Aryloxyphenoxypropionate (FOP), Asp-2078-Gly, cyclohexanedione (DIM), derived cleaved amplified polymorphic sequence (dCAPs), Ile-1781-Leu, Ile-2041-Asn, phenylpyrazolin (DEN), target-site mutation.

Japanese foxtail is a grass weed infesting in oilseed rape and wheat (*Triticum aestivum* L.) fields in eastern China (Bi et al. 2016; Cui et al. 2015; Tang et al. 2012; Yang et al. 2007). Fenoxaprop-*P*-ethyl, an acetyl-CoA carboxylase (ACCase)-inhibiting herbicide, was firstly registered to control grass weeds in wheat by Bayer AG in 1998 in China (www.chinapesticide.gov.cn). Since then, fenoxaprop-*P*-ethyl has been used continuously by farmers to control grass weeds by farmers continuously. Many Japanese foxtail populations have evolved fenoxaprop-*P*-ethyl resistance, as well as cross-resistance to other ACCase-inhibiting herbicides (Cui et al. 2015; Pan et al. 2015; Tang et al. 2012; Xu et al. 2013; Yang et al. 2007).

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Target-site resistance (TSR) and non–target site resistance are the two categories of mechanisms for resistance to ACCase-inhibiting herbicides in grasses (Kaundun 2014). ACCase-inhibiting herbicides bind to the site of action in the carboxyl transferase (CT) domain of ACCase and inhibit de novo fatty acid synthesis in sensitive grass weeds, leading to necrosis and plant death (Kaundun 2014; Post-Beittenmiller et al. 1992). There are three types of ACCase-inhibiting herbicides: aryloxyphenoxypropionate (FOP), cyclohexanedione (DIM), and phenylpyrazolin (DEN). TSR prevents an herbicide from binding to the target enzyme because of amino acid substitutions at the CT domain of ACCase (Devine 1997). Fourteen target-site mutations (amino acid substitutions) referring to six positions that induce resistance to different ACCase-inhibiting herbicides have been identified within the CT domain of homomeric chloroplastic ACCase. These substitutions include Ile-1781 to Leu, Val, Thr, or Ala (Délye et al. 2011; Kaundun 2014; Zagnitko et al. 2001; Zhang and Powles 2006); Trp-1999 to Cys, Leu, or Ser (Liu et al. 2007); Trp-2027 to Cys (Gherekhloo et al. 2012; Petit et al. 2010; Yu et al. 2007); Ile-2041 to Asn or Val (Délye et al. 2003; Scarabel et al. 2011; Tang et al. 2014); Asn-2078 to Gly (Collavo et al. 2011; Kaundun 2010);

Cys-2088 to Arg (Yu et al. 2007); and Gly-2096 to Ala or Ser (Cruz-Hipolito et al. 2012; Li et al. 2014).

TSR often causes different cross-resistance to herbicides that possess the same mode of action (Beckie and Tardif 2012). Since 2007, when Japanese foxtail resistance to ACCase-inhibiting herbicides was first reported, multiple amino acid mutations have been identified (Bi et al. 2016; Cui et al. 2015; Xu et al. 2013, 2014a; Yang et al. 2007). Previous studies showed that the Ile-1781-Leu substitution in Japanese foxtail confers high resistance to fenoxaprop-*P*-ethyl and cross-resistance to clodinafop-propargyl, clethodim, and pinoxaden (Bi et al. 2016; Cui et al. 2015; Mohamed et al. 2012). The Ile-2041-Asn substitution confers resistance to haloxyfop-*R*-methyl and cross-resistance to other FOPs in Japanese foxtail (Tang et al. 2012). Additionally, the mutation at position 1999 confers resistance to fenoxaprop-*P*-ethyl, but not to clodinafop-propargyl, haloxyfop-*R*-methyl, or clethodim (Xu et al. 2014a). The Trp-2027-Cys substitution in Japanese foxtail confers resistance to fenoxaprop-*P*-ethyl and cross-resistance to other FOPs and pinoxaden but not to DIMs (Xu et al. 2013).

Therefore, it is important to elucidate the mechanisms of resistance in order to design more effective weed management strategies. We previously studied the cross-resistance patterns of Japanese foxtail populations that harbored mutations at positions 1999, 2027, and 2041 for five ACCase-inhibiting herbicides (Tang et al. 2012; Xu et al. 2013, 2014a). All the Japanese foxtail populations harboring those three mutations were collected from eastern China. In the present study we focused on three populations with high resistance to fenoxaprop-*P*-ethyl also collected from eastern China. Our objectives here were to (1) identify different mutations in fenoxaprop-*P*-ethyl–resistant Japanese foxtail, (2) develop an effective dCAPS protocol for detecting the mutation type and frequency in fenoxaprop-*P*-ethyl–resistant Japanese foxtail populations, and (3) determine cross-resistance patterns to ACCase inhibitors associated with different mutations in Japanese foxtail.

Materials and Methods

Plant Material and Herbicides. Three putative resistant Japanese foxtail populations were collected in 2013 from fields in Jiangsu province, China. A reference sensitive population was collected in 2011 from a field that was never treated with any herbicide in Jiangsu province, China (Figure 1). The collected seeds were kept under dry conditions at room temperature until use. The following herbicides were used for the dose–response tests: fenoxaprop-*P*-ethyl, clodinafop-propargyl, haloxyfop-*R*-methyl, clethodim, and pinoxaden. All the herbicides used in this study were commercial formulations and are listed in Table 1.

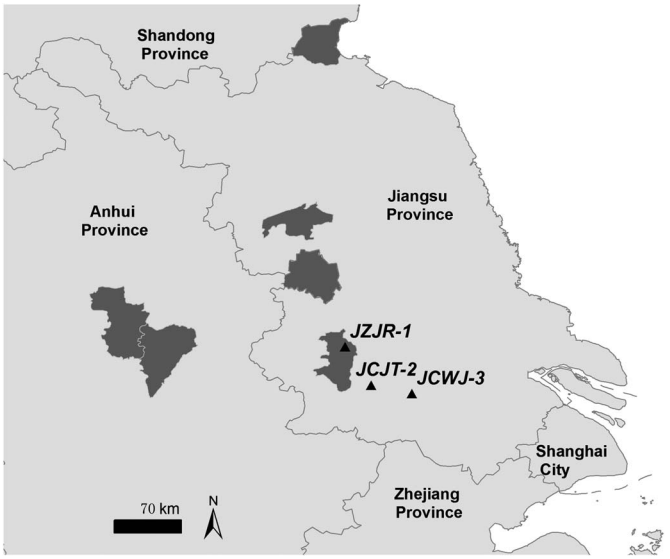

Figure 1. Counties with confirmed resistance to ACCase-inhibitor herbicides in Japanese foxtail in eastern China (Bi et al. 2016; Cui et al. 2015; Mohamed et al. 2012; Xu et al. 2013, 2014; Yang et al. 2007), and the locations of three resistant Japanese foxtail populations used in this study.

Sensitivity to Fenoxaprop-*P*-ethyl. Whole-plant dose–response experiments were conducted under greenhouse conditions at 20/15 C (day/night), as described in Xu et al. (2013). At the 3- to 4-leaf stage, the plants of the susceptible population (JNXW-2) were sprayed with fenoxaprop-*P*-ethyl at a dose of 0.00, 0.81, 3.23, 12.94, 51.75, 207.00, or 828.00 g ai ha^{-1}, whereas those of the putative resistant populations (JCJT-2, JZJR-1, and JCWJ-3) were sprayed at a concentration of 0.00, 3.23, 12.94, 51.75, 207.00, 828.00, or 3312.00 g ha^{-1}. The experiment was conducted as a completely randomized design with four replications (each pot = replication, 20 plants per pot), and the experiment was conducted twice. Three weeks after treatment, the aboveground fresh weight per pot and the effective dose of herbicide causing 50% reduction of fresh weight (GR50) were determined. The resistance index (RI) was calculated as the ratio of GR_{50} values for the resistant and sensitive control populations.

Total DNA Extraction and Plastidic ACCase CT Domain Cloning. Total DNA was extracted from 100 mg of young shoot tissue using the Plant

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Table 1. Herbicides used in this study.

Group	Herbicide	Formulation[a]	Commercial name	Company
FOP	Fenoxaprop-*P*-ethyl	69 g L^{-1} EW	Puma	Bayer China Ltd. (Zhejiang, China)
	Haloxyfop-*R*-methyl	108 g L^{-1} EC	Gallant	Dow AgroSciences (Beijing, China)
	Clodinafop-propargyl	150 g L^{-1} WP	Topic	Syngenta (Shanghai, China)
DIM	Clethodim	120 g L^{-1} EC	Seletone	Changqing Agrochemical (Jiangsu, China)
DEN	Pinoxaden	125 g L^{-1} EC	Axial	Syngenta (Shanghai, China)

[a] Abbreviations: EC, emulsifiable concentrate; EW, water emulsion; WP, wettable powder.

Genomic DNA Kit (Tiangen Biotech, Beijing, China), according to the manufacturer's instructions. Two primer pairs were designed to amplify a 1,230-bp DNA fragment containing the entire CT domain of Japanese foxtail ACCase (Xu et al. 2014a): ACCp1F/ACCp1R to amplify a 553-bp region containing codon 1781, and ACCp2F/ACCp2R to amplify an 873-bp region containing codons 1999, 2027, 2041, 2078, 2088, and 2096 (Xu et al. 2014a). PCR for generating and sequencing *ACCase* was carried out as described by Pan et al. (2015). DNA samples of eight plants from each population were sequenced, and at least five clones of the PCR product for each biological replicate were used to construct the *ACCase* consensus sequence. Fragments were sequenced by Invitrogen Biotechnology (Shanghai, China) in both forward and reverse directions to minimize sequencing errors. The sequence data of putative resistant populations (JCJT-2, JZJR-1, and JCWJ-3) and the sensitive population (JNXW-2) were thus compared.

dCAPS for Mutation Detection and Genotype Analysis. Three new dCAPS markers were developed for detecting three different mutations (Ile-1781-Leu, Ile-2041-Asn, and Asp-2078-Gly) according to methods reported by Délye et al. (2011) and Xu et al. (2014a). The primers and corresponding restriction enzymes were designed using *dCAPS* Finder (http://helix.wustl.edu/dcaps/dcaps.html). They are shown in Tables 2 and 3, and restriction enzymes *Eco*O109I (Thermo Fisher Scientific, Waltham, MA), *Eco*RV (Thermo Fisher Scientific), and *Kpn*I (Thermo Fisher Scientific) were used for Ile-1781-Leu, Ile-2041-Ala, and Asp-2078-Gly mutations, respectively. PCR was performed as described by Xu et al. (2014a), and PCR products were separated on 3% agarose gels, stained with ethidium bromide, and visualized using UV.

Sensitivity to Other ACCase-inhibiting Herbicides. Four ACCase-inhibiting herbicides representing three different chemical classes were selected for whole-plant bioassays, including two FOPs, one DIM, and one DEN. The whole-plant pot bioassays were conducted to determine the sensitivity to different herbicides. Herbicide doses (Table 4) were determined based on preliminary experiments. The experiment was conducted as a completely randomized design with four replications, and the experiment was conducted twice.

Statistical Analysis. The fresh weight data were expressed as percentages of the nontreated control. Regression analysis and the three-parameter logistic function (LL.3) were performed using the 'drc' add-on package (Ritz and Streibig 2007) in R v. 3.1.3 (R Core Team 2015) to pool and fit the data to a nonlinear logistic model (Chen et al. 2016; Valverde et al. 2014). Thus, the effective rate of herbicide

Table 2. dCAPS primers used in this study.

Primer	Sequence (5′ → 3′)[a]	Product size (bp)	Annealing temperature (C)	Target mutation
I1781LF	GGTTATCGATTCTGTTGTGGGAAAAGAGGAT GGACTAGGTGTGGGGACC	320	58	Ile-1781-Leu
I1781LR	CTGGAACAGTCAGATGG			
I2041NF	AGAGGCTTCTCTGGAGGGCAAAGAGATCTTTTTGAAGGAT	391	58	Ile-2041-Asn
I2041NR	GCAGCAACTGTTTCTTC			
D2078GF	AGCCTGATTCCCACGAGCGG	331	58	Asp-2078-Gly
D2078GR	GCAGTCCTCTCAGCATAGCACTCGATGCGAT CTGGGTTTATCTTGGTA			

[a] The forced mismatches introduced to create a restriction site are underlined.

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Table 3. dCAPS markers and restriction enzymes.

Target mutation	Primer pair	Restriction enzyme	Recognition site (5′ → 3′)	Fragment size (bp)	
				WT	Mutant
Ile-1781-Leu	I1781LF/I1781LR	*Eco*O109I	RG^GNCCY	320	271, 49
Ile-2041-Asn	I2041NF/I2041NR	*Eco*RV	GAT^ATC	391	351, 40
Asp-2078-Gly	D2078GF/D2078GR	*Kpn*I	GGTAC^C	331	283, 48

causing 50% inhibition in fresh weight (GR50) was determined for each population referring to each herbicide treated. Resistance factors (RFs) were calculated as the ratio between the GR50 of the resistant population and the GR50 of the susceptible population: S, not resistant (<2); L, low resistance (2–5); M, moderate resistance (6–10); and H, high resistance (>10) (Beckie and Tardif 2012).

Results and Discussion

Sensitivity to Fenoxaprop-*P*-ethyl. The GR50 values of JCJT-2, JZJR-1, and JCWJ-3 were higher than the recommended field dose for fenoxaprop-*P*-ethyl, whereas that of JNXW-2 was lower than the field dose (Table 5). The GR50 values of JCJT-2, JZJR-1, and JCWJ-3 were 55-fold, 52-fold, and 80-fold higher, respectively, than those of JNXW-2 (Table 5; Figure 2). In China, fenoxaprop-*P*-ethyl has been used to control grass weeds in wheat and rapeseed fields since 1998. Two counties in Anhui province and four counties in Jiangsu province have been confirmed to have fenoxaprop-*P*-ethyl–resistant Japanese foxtail (Figure 1). Meanwhile, a group of grass weeds in Chinese wheat lands have also evolved fenoxaprop-*P*-ethyl resistance, including American sloughgrass [*Beckmannia syzigachne* (Steud.) Fernald] (Li et al. 2014; Pan et al. 2015), shortawn foxtail (*Alopecurus aequalis* Sobol.) (Guo et al. 2017; Xia et al. 2015), Asia Minor bluegrass (*Polypogon fugax* Nees ex Steud.) (Tang et al. 2014), and barnyardgrass [*Echinochloa crus-galli* (L.) Beauv.] (Chen et al. 2016). Therefore, some herbicides have been introduced as alternatives to fenoxaprop-*P*-ethyl, such as pyroxsulam, mesosulfuron-methyl, and tralkoxydim.

Identification of ACCase Mutations. Sequences of the entire CT domain of Japanese foxtail ACCase were obtained from all four populations. Two copies of *ACCase* (*Acc1-1* and *Acc1-2*) were identified, but mutations were detected only in *Acc1-1*. Sequence comparison revealed that an A (JNXW-2) to C (JCJT-2) substitution resulted in an Ile (ATA) to Leu (CTA) substitution at codon position 1781 (Ile-1781-Leu); a T (JNXW-2) to A (JZJR-1) substitution resulted in an Ile (ATT) to Asn (AAT) substitution at codon position 2041 (Ile-2041-Asn); and an A (JNXW-2) to G (JCWJ-3) substitution resulted in an Asp (GAT) to Gly (GGT) substitution at codon position 2078 (Asp-2078-Gly).

Multiple copies of genes encoding plastidic ACCase have been identified in rice barnyardgrass [*Echinochloa phyllopogon* (Stapf) Koso-Pol.], sterile oat (*Avena sterilis* L.), Japanese foxtail, and wild oat (*Avena fatua* L.) (Christoffers et al. 2002; Iwakami et al. 2012; Xu et al. 2014b; Yu et al. 2013), and it has been demonstrated that any of the three Acc1 homologues (*Acc1-1*, *Acc1-2*, and *Acc1-3*) in wild oat and two *Acc1* homologues (*Acc1-1* and *Acc1-2*) in Japanese foxtail, can also host ACCase resistance mutations (Christoffers et al. 2002; Xu et al. 2014a; Yu et al. 2013). Mutations Ile-1781-Leu and Ile-2041-Asn in Japanese foxtail have been reported (Bi et al. 2016; Cui et al. 2015; Mohamed et al. 2012; Tang et al. 2012), but the Asp-2078-Gly mutation in Japanese foxtail has not been reported.

dCAPS for Mutation Detection and Genotype Analysis. The dCAPS method detected these mutations successfully in Japanese foxtail. Of the 96 plants tested in JCJT-2, two were wild type and 94

Table 4. Herbicide treatments applied for the dose–response treatments to the resistant and susceptible Japanese foxtail populations.

Group	Herbicide	Recommended field dose (g ai ha^{-1})	JNXW-2 (dose in g ai ha^{-1})	JCWJ-3, JCJT-2, JZJR-1 (dose in g ai ha^{-1})
FOP	Haloxyfop-*R*-methyl	30	0.47, 1.87, 7.5, 30, 120, 480	1.87, 7.5, 30, 120, 480, 1,920
	Clodinafop-propargyl	45	0.7, 2.81, 11.25, 45, 180, 720	2.81, 11.25, 45, 180, 720, 2,880
DIM	Clethodim	72	1.125, 4.5, 18, 72, 288, 1,152	4.5, 18, 72, 288, 1152, 4,608
DEN	Pinoxaden	45	0.7, 2.81, 11.25, 45, 180, 720	2.81, 11.25, 45, 180, 720, 2,880

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Table 5. Summary of fenoxaprop-*P*-ethyl dose–response analyses and target-site resistance (TSR) mutations for resistant and susceptible Japanese foxtail populations.

Population	GR_{50} (g ai ha^{-1}) (SE)	RF[a]	Mutation
JCJT-2	269.87(52.71)	54.74	Ile-1781-Leu
JZJR-1	258.47 (44.23)	52.43	Ile-2041-Asn
JCWJ-3	395.30 (44.93)	80.18	Asp-2078-Gly
JNXW-2	4.92(2.42)	1.00	

[a] RF, resistance factor.

were mutants with the Ile-1781-Leu substitution (Table 6); of the 61 plants tested in JZJR-1, five were wild type and 56 were mutants with the Ile-2041-Asn substitution; and of the 23 plants tested in JCWJ-3, three were wild type and 20 were mutant with the Asp-2078-Gly substitution. The mutation frequencies in JCJT-2, JZJR-1, and JCWJ-3 were approximately 98%, 92%, and 87%, respectively. dCAPS is a high-throughput PCR-based method to detect known resistance-endowing mutations, and this method was developed to easily determine resistance caused by mutations of ACCase or ALS in different weed species (Délye et al. 2011; Pan et al. 2015).

Cross-resistance to Other ACCase-inhibiting Herbicides. JCJT-2 (Ile-1781-Leu) was highly resistant to clodinafop-propargyl, slightly resistant to haloxyfop-*R*-methyl and pinoxaden, but susceptible to clethodim (Table 7; Figure 3). JZJR-1 (Ile-2041-Asn) was highly resistant to clodinafop-propargyl and haloxyfop-*R*-methyl, slightly resistant to pinoxaden, but susceptible to clethodim (Table 7; Figure 3). Previous studies have shown that the Ile-1781-Leu substitution confers high resistance to FOPs, DIMs, and pinoxaden in blackgrass (*Alopecurus myosuroides* Huds.), wild oat, sterile oat, green foxtail [*Setaria viridis* (L.) Beauv.], and rigid

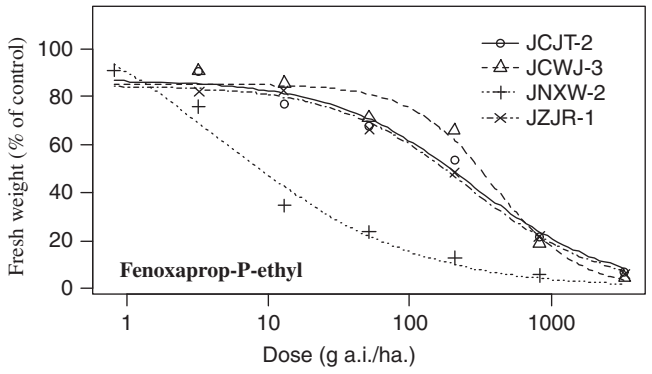

Figure 2. Fenoxaprop-*P*-ethyl dose–response tests for the four Japanese foxtail populations.

Table 6. The derived cleaved amplified polymorphic sequence (dCAPS) detection of mutations in the resistant populations of Japanese foxtail.

Population	Mutation	Individuals Tested	Wild type	Mutant type	Mutation frequency (%)
JCJT-2	Ile-1781-Leu	96	2	94	98
JZJR-1	Ile-2041-Asn	61	5	56	92
JCWJ-3	Asp-2078-Gly	23	3	20	87

ryegrass (*Lolium rigidum* Gaudin) (Beckie and Tardif 2012; Brown et al. 2002; Christoffers et al. 2002; Délye et al. 2011; Tal and Rubin 2004). In the present study, JCJT-2, harboring the Ile-1781-Leu substitution, showed resistance to haloxyfop-*R*-methyl, clodinafop-propargyl, and pinoxaden, and was susceptible to clethodim. Our previous study found that American sloughgrass with the Ile-1781-Leu mutation showed resistance to all ACCase-inhibiting herbicides tested; while the cross-resistance varied for different DIMs: specifically, high resistance (RF = 31) to sethoxydim and low resistance (RF = 5) to clethodim (Pan et al. 2015). Yu et al. (2013) also found the Ile-1781-Leu mutation in wild oat ACCase confers high resistance to sethoxydim (RI = 11) and low resistance to clethodim (RI = 4). Moreover, two blackgrass populations harboring Ile-1781-Leu mutation collected in Denmark were found to be sensitive (RF = 1.3) or to have low resistance (RF = 2.4) to a DIM herbicide, cycloxydim (Keshtkar et al. 2015). It is reported that blackgrass, hood canarygrass (*Phalaris paradoxa* L.), and rigid ryegrass with the Ile-2041-Asn substitution were highly resistant to

Table 7. The sensitivities of the resistant and sensitive Japanese foxtail populations to ACCase-inhibiting herbicides.

Herbicide	Population	GR_{50} (g ai ha^{-1}) (SE)	RF[a]
Haloxyfop-*R*-methyl	JCJT-2	40.46 (7.17)	6.36
	JZJR-1	234.9 (46.91)	36.93
	JCWJ-3	67.36 (21.99)	10.59
	JNXW-2	6.36 (1.68)	1.00
Clodinafop-propargyl	JCJT-2	791.38 (105.09)	33.65
	JZJR-1	1,570.2 (147.72)	66.76
	JCWJ-3	1,101.91 (131.36)	46.85
	JNXW-2	23.52 (3.74)	1.00
Clethodim	JCJT-2	27.98 (9.28)	1.22
	JZJR-1	23.64 (8.46)	1.03
	JCWJ-3	283.46 (64.49)	12.37
	JNXW-2	22.92 (4.84)	1.00
Pinoxaden	JCJT-2	62.94 (23.6)	5.33
	JZJR-1	43.04 (19.24)	3.65
	JCWJ-3	548.66 (176.37)	46.50
	JNXW-2	11.8 (0.99)	1.00

[a] RF, resistance factor.

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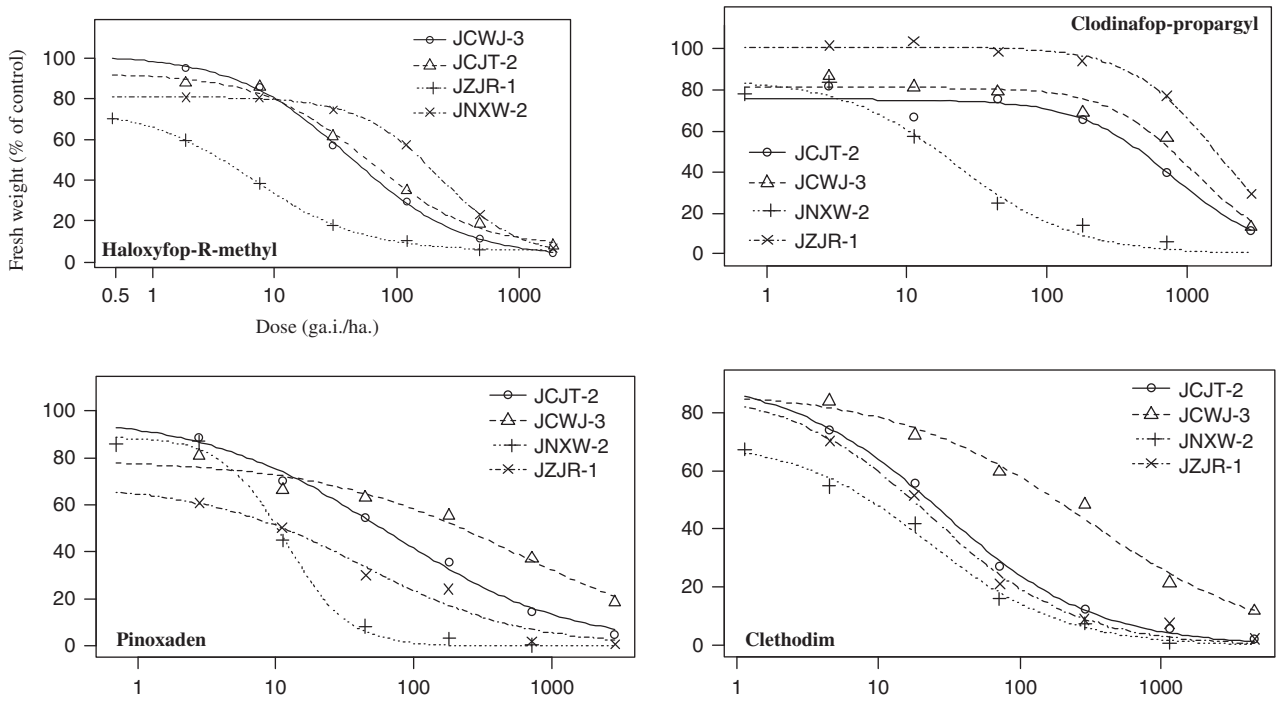

Figure 3. Dose-dependent response of the four Japanese foxtail populations to four ACCase-inhibiting herbicides.

FOPs, but not to DIMs, and showed either no resistance or moderate resistance to DENs (Beckie and Tardif 2012; Hochberg et al. 2009). These findings were consistent with those reported in the present study. JZJR-1, which harbors the Ile-2041-Asn substitution, was highly resistant to the two FOPs, slightly resistant to pinoxaden (DEN herbicide), but susceptible to clethodim (DIM herbicide), indicating that the mutation confers high resistance to FOPs, but not to DIMs or DENs.

JCWJ-3 (Asp-2078-Gly) was highly resistant to all four ACCase-inhibiting herbicides, and the RFs for clodinafop-propargyl and haloxyfop-*R*-methyl, pinoxaden and clethodim were 46.85, 10.59, 46.50, and 12.37, respectively (Table 7; Figure 3). Previous studies have shown that blackgrass, rigid ryegrass, sterile oat, and hood canarygrass harboring the Asp-2078-Gly substitution are resistant to all three classes of ACCase-inhibiting herbicides (Beckie and Tardif 2012; Liu et al. 2007; Yu et al. 2007).

Conclusions. An effective dCAPS protocol was developed to detect the resistance-endowing mutations in the ACCase gene of Japanese foxtail. Ile-1781-Leu, Ile-2041-Asn, and Asp-2078-Gly were detected in the three Japanese foxtail populations studied. JCJT-2 (Ile-1781-Leu) and JZJR-1 (Ile-2041-Asn) showed cross-resistance to haloxyfop-*R*-methyl, clodinafop-propargyl, and pinoxaden, but not to clethodim. JCWJ-3 (Asp-2078-Gly) showed cross-resistance to all tested ACCase-inhibiting herbicides. Our results might allow farmers to select the most appropriate herbicides to manage fenoxaprop-*P*-ethyl–resistant Japanese foxtail populations.

Acknowledgments

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