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Characterization of cross-resistance patterns in acetyl-CoA carboxylase inhibitor resistant wild oat (*Avena fatua*)

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The purpose of this study was to determine cross-resistance patterns among wild oat lines resistant to acetyl-CoA carboxylase (ACCase) inhibitors and to determine which, if any, cross-resistant type was more common than another. Discriminatory concentrations of two aryloxyphenoxy-propionates (APP) and three cyclohexanediones (CHD) were determined using a petri-dish bioassay. These concentrations were then applied to 82 resistant wild oat lines identified in previous studies. In addition, two resistant standards (UM1 and UM33) and a susceptible standard (UM5) were included in the experiments. Coleoptile lengths expressed as percentages of untreated controls were used to assess the level of resistance to each herbicide. Large variations were observed among wild oat lines and herbicides. However, cluster analysis summarized the relationship between the five herbicides (variables) and the wild oat lines into three main cross-resistance types. Type A included wild oat lines with high resistance to APP herbicides and no or low resistance to CHD herbicides. Types B and C included those with low to moderate resistant and high levels of resistance to all five herbicides, respectively. Type C was the most common cross-resistance type. Relationships among herbicides were determined using pairwise correlation and principal component analysis (PCA). All correlations were high between APP herbicides and between CHD herbicides but not between APP and CHD herbicides. The first two axes of the PCA accounted for 88.4% of the total variance, with the first axis correlated to the CHD herbicides and the second axis correlated to the APP herbicides. In the PCA, wild oat lines were segregated into the three types identified in the cluster analysis. Although CHD and APP herbicides bind at the same region on the ACCase, resistant wild oat lines respond differently to them.

Nomenclature: Clethodim, clodinafop, Diclofop methyl, fenoxaprop-P, sethoxydim, tralkoxydim, blackgrass, *Alopecurus myosuroides* Huds., ALOMY; giant foxtail, *Setaria faberi* Herrm., SETFA; goosegrass, *Eleusine indica* L., ELEIN; green foxtail *Setaria viridis* L., SETVI; Italian ryegrass, *Lolium multiflorum* Lam., LOLMU; large crabgrass, *Digitaria sanguinalis* L., DIGSA; wild oat, *Avena fatua* L. AVEFA.

Key words: Aryloxyphenoxypropionate, cyclohexanedione, ACCase inhibitor, herbicide resistance, cross-resistance.

Aryloxyphenoxypropionate (APP) and cyclohexanedione (CHD) herbicides inhibit acetyl-CoA carboxylase (ACCase), an enzyme essential to fatty acids biosynthesis (Devine and Shimabukuro 1994). Diclofop methyl (an APP herbicide) was the first commercially available ACCase inhibitor to be registered postemergence in western Canada and has been used frequently in Manitoba since 1980. With the registration of additional APP and CHD herbicides, over half of the sprayed acreage in Manitoba was treated with these herbicides by the early 1990s (Bourgeois and Morrison 1997a).

The first reported case of wild oat resistance to ACCase inhibitors occurred in a field that had been repeatedly sprayed with APPs and CHDs over 10 yr (Heap et al. 1993). Resistance to ACCase inhibitors has also been found in a number of other grassy weeds including Italian ryegrass (Stanger and Appleby 1989); blackgrass (Moss 1990); goosegrass (Marshall et al. 1994); large crabgrass (Wiederholt and Stoltenberg 1995); and giant foxtail (Stoltenberg and Wiederholt 1995). In Manitoba, resistance has also been reported in green foxtail (Heap and Morrison 1996).

Physiological evidence indicates that APPs and CHDs both bind to the same region of the target enzyme (Rendina et al. 1989). However, different cross-resistance patterns characterize ACCase inhibitor resistance in weeds (Heap et al. 1993; Moss 1990; Stanger and Appleby 1989). In wild oat populations, the levels of resistance vary from 0- to 300-fold resistance to specific ACCase inhibitors (Heap et al. 1993). Murray et al. (1995) demonstrated that the difference in cross-resistance between two resistant wild oat populations (UM1 and UM33) was genetically controlled by semidominant point mutations at a single nuclear gene locus. An altered ACCase enzyme confers herbicide resistance in these two wild oat populations (M. D. Devine, personal communication). However, the actual site and type of mutation have not been identified on the gene encoding for ACCase.

A total of 150 resistant wild oat lines collected in two separate field surveys have been identified in previous studies (Bourgeois and Morrison 1997b, Bourgeois et al. 1997). Resistant wild oat lines occurred primarily in areas where APP and CHD herbicides were used repeatedly. Only a few of these have been characterized based on cross-resistance patterns (Heap et al. 1993). The objectives of this study were (1) to determine the discriminatory rates of the herbicides clodinafop, clethodim, and tralkoxydim on the wild oat line UM5, and (2) to determine cross-resistant patterns

and quantify differences in herbicide resistance patterns among 82 wild oat lines collected in recent field surveys in Manitoba. The main purpose of the study was to describe the range in response types among lines and to determine which type, if any, was most common.

Materials and Methods

Determination of Discriminatory Rates

A discriminatory concentration for a specific herbicide is the minimum herbicide concentration required to distinguish susceptible from resistant lines. Murray et al. (1996) developed seed bioassays for rapid identification of resistance to fenoxaprop-P (an APP herbicide) and sethoxydim (a CHD herbicide). Presence or absence of resistance in wild oat was based on coleoptile and radicle length of seedlings placed on an agar medium[1] containing a discriminatory concentration of herbicide. In this study, additional bioassays were developed for the APP herbicide clodinafop and for the CHD herbicides clethodim and tralkoxydim. The dose–response curves were developed using the susceptible wild oat population UM5 and procedures comparable to those described by Murray et al. (1996). The origin of UM5 is described in Heap et al. (1993).

The bioassays were conducted on agar media with concentrations of 0, 0.05, 0.1, 0.15, 0.25, 0.5, 0.75, 1, 1.5, 3, and 5 μmol for clodinafop and clethodim, and 0, 1, 2, 3, 4, 5, 7.5, 10, 20, and 30 μmol for tralkoxydim. Plexiglas boxes, 13.5 by 12.5 by 3.5 cm deep, contained three rows of five seeds each of UM5 wild oat on a 1-cm-thick agar medium containing the herbicide. Plates were left in the dark for 5 d at 21 C. After 5 d, the length of coleoptiles of 10 seedlings were measured. These 10 seedlings were picked at random from among those that germinated from the 15 plated seeds. The same process was replicated three times, with new batches of agar used each time. A mean coleoptile length was calculated for each dose and herbicide. Dose–response curves were fitted to the means using a sigmoidal model with the NONLIN procedure (SAS, 1985). The model fitted was

$$y = k/(1 + e^{bg}x^{b}) + d \quad [1]$$

where y is the dependent variable (mean coleoptile length in mm), x is the herbicide concentration (mol), e is the base of the natural logarithm, k is the difference between the upper and lower asymptotes, d is the lower asymptote, and b and g determine the shape of the curve. Parameter estimates were considered to be statistically significant at $P = 0.05$ where the standard error was less than half the numerical value of the estimate (Koutsoyiannis 1977). A large standard error of a parameter indicates a poor estimation or that the equation is not representative of the data set.

Screening of Herbicide Resistant Lines

Eighty-two lines were selected from among resistant wild oat populations collected in two field surveys (Bourgeois and Morrison 1997b; Bourgeois et al. 1997). Forty-three of these lines were collected in a field survey conducted in 1993 in a single township (0810W) near Treherne, Manitoba (Bourgeois and Morrison 1997b). The remaining 39 lines were from among those collected in a more extensive survey conducted in 1994 (Bourgeois et al. 1997). These lines were from nine townships located throughout the southern part of the province. In addition, three populations (UM1, UM5, and UM33) were included as standards. UM1 and UM33 are both resistant to ACCase inhibitors but have different cross-resistance patterns (Heap et al. 1993). UM5 was included as a susceptible standard. The origins of UM1, UM5, and UM33 were described by Heap et al. (1993). In total, the 85 wild oat lines were screened at the discriminatory rates of two APP herbicides (fenoxaprop-P and clodinafop) and three CHD herbicides (sethoxydim, clethodim, and tralkoxydim).

Fifteen hand-peeled seeds were placed on media without herbicide and at the discriminatory concentration of each herbicide. A similar procedure to the one used for the determination of the discriminatory concentration was used for each of the wild oat lines. Coleoptile lengths were measured to differentiate responses of the 85 lines to each of the five herbicides. The mean coleoptile length was calculated for each wild oat line and herbicide combination. Values were expressed as a percentage of the coleoptile length of untreated controls, since coleoptile lengths of untreated wild oat varied considerably from one population to another (Murray et al. 1996).

Statistical Analysis

The wild oat lines were classified into cross-resistance types using a multivariate cluster analysis procedure. Cluster analysis produces a hierarchical dendrogram summarizing the relationships between objects based on measured variables. In our analysis, objects are the 85 wild oat lines, and variables are the five herbicides. Variable values are herbicide resistance expressed as a percentage of the coleoptile length of untreated control. The clustering algorithm minimized the increase in error sum of squares at each fusion (Ward's method, Podani 1994) based on the similarity ratio resemblance measure. Cluster analysis was performed using the SYNTAX multivariate package (Podani 1994).

The herbicide cross-resistant types delineated by cluster analysis were tabulated by township and were mapped on township and field maps for the wild oat lines originating from 0810W. These maps were used to discuss possible relationships among resistant wild oat populations.

Pairwise Pearson product–moment correlations (r) were computed between the five herbicides to examine trends in cross-resistance patterns among the lines. This correlation matrix was also input into principal component analysis (PCA) (Podani 1994). PCA is a linear multivariate ordination method that produces a parsimonious, low-dimensional representation of the variation present in the original five-dimensional variable (herbicide resistance) space. If the original variables are correlated, PCA takes advantage of these correlations to obtain new, derived variables (principal components or PCA axes) that offer a more efficient summarization of the major trends present in the data. A PCA biplot consists of the coordinate position of each individual (wild oat line), together with vectors indicating the direction of variation of each variable (herbicide resistance). PCA was performed using the SYNTAX package (Podani 1994).

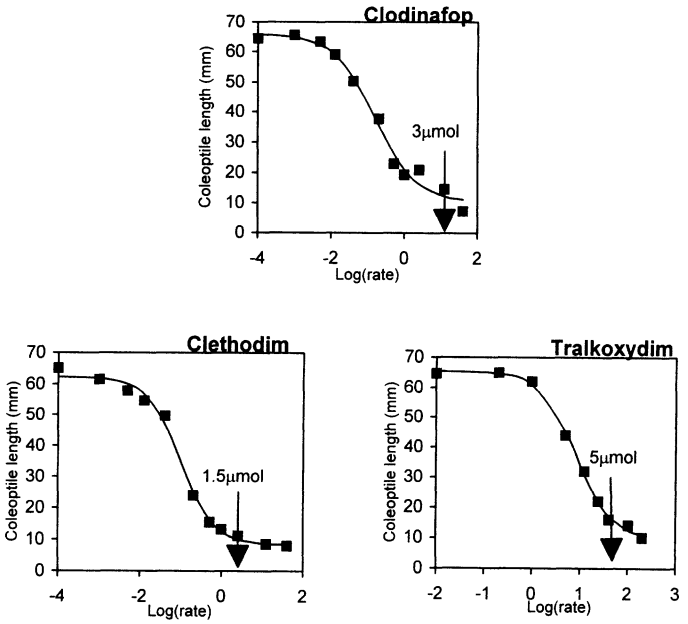

FIGURE 1. Coleoptile growth of UM5 wild oat as influenced by clodinafop, clethodim, and tralkoxydim. See Table 1 for parameter estimates.

Results and Discussion

Determination of Discriminatory Rates

As reported for fenoxaprop-P and sethoxydim (Murray et al. 1996), increasing dosages of clodinafop, clethodim, and tralkoxydim resulted in a reduction of coleoptile length in the susceptible UM5 wild oat line (Figure 1). Parameter estimates of the dose response curves are provided in Table 1. The discriminatory concentration represented the lowest dosage that resulted in at least 80% inhibition of coleoptile elongation compared to the untreated control. Using this criterion, the discriminatory concentrations were 1.5 μmol for clethodim, 3 μmol for clodinafop, and 5 μmol for tralkoxydim. Discriminatory concentrations of 10 μmol for fenoxaprop-P and 5 μmol for sethoxydim were determined using comparable procedures (Murray et al. 1996).

Screening of Herbicide Resistant Lines

Three main herbicide cross-resistant types (denoted A, B, and C) were separated by cluster analysis (Figure 2). Type C, which includes 44 wild oat lines, was separated from types A and B in the first dendrogram dichotomy. A second dichotomy separated types A and B, which included 23 and 17 wild oat lines, respectively. UM33 and UM1 were placed in types A and C, respectively. Each branch of the dendogram further divided into smaller clusters, indicating greater resemblance in cross-resistance patterns among some wild oat lines. Indeed, wild oat lines originating from a similar field often had similar cross-resistance patterns (data not shown).

TABLE 1. Parameter estimates (standard error in parentheses) describing the coleoptile length of UM5 wild oat seedlings on agar medium treated with five ACCase inhibitor herbicides.

	g[a]	b[a]	d[a]	k[a]
Clodinafop	0.8 (0.1)	1.8 (0.3)	10.4 (2.7)	55.5 (3.9)
Fenoxaprop-P[b]	1.8 (0.2)	1.6 (0.4)	6.8 (1.8)	48.2 (4.2)
Clethodim	1.0 (0.1)	2.4 (0.3)	8.3 (1.3)	54.0 (2.0)
Sethoxydim[b]	0.3 (0.1)	2.0 (0.3)	7.8 (0.8)	30.8 (1.2)
Tralkoxydim	−0.9 (0.1)	2.7 (0.3)	9.7 (1.6)	55.9 (2.1)

[a] g and b determine the shape of the curve; d is the lower asymptote; and k is the difference between the upper and lower asymptotes.
[b] From Murray et al. (1996).

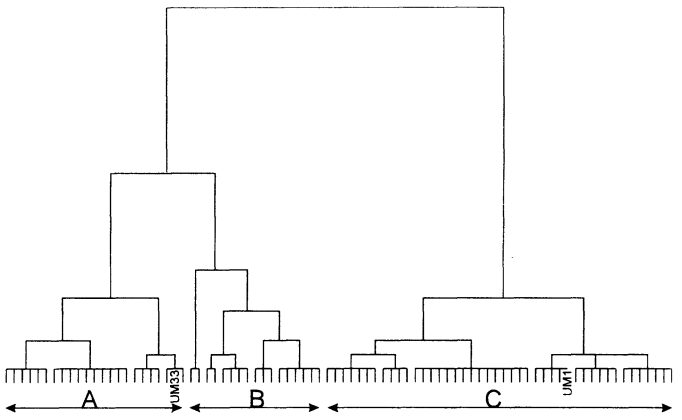

FIGURE 2. Clustering by a dendrogram of 84 wild oat lines according to similarity in cross-resistance to five ACCase inhibitor herbicides.

Mean herbicide cross-resistance for the three types (A, B, and C), the susceptible standard (UM5), and the two known ACCase-resistant lines (UM1 and UM33) are summarized in Table 2. Cross-resistant type C was most common, accounting for 44 of the 84 resistant wild oat lines. This type, characterized by high levels of resistance for both APP and CHD herbicide groups, is more resistant to a broad selection of ACCase inhibitors than the other types. Cross-resistance type A (which includes line UM33) is highly resistant to the APP herbicides but shows little or no resistance to the CHD herbicides. Cross-resistant type B shows low to moderate resistance to both herbicide groups.

In Manitoba, the three wild oat cross-resistance types were often found to evolve within the same locality (Table 3). In township 0810W, all three cross-resistance types occurred together with no discernible spatial pattern (Figure 3). Indeed, different types grew within 100 m of each other in fields 18 and 26 (Figure 4). However, the frequency of use of APP and CHD herbicides may have affected the establishment of wild oat types A and B. Type A occurred in a small patch in field 26, while type C flourished on the

TABLE 2. Mean coleoptile lengths as a percentage of control of wild oat lines assayed with five ACCase inhibitor herbicides in cross-resistance types.

		APP		CHD		
Types of lines	Number of lines	Fenoxaprop-P	Clodinafop	Sethoxydim	Clethodim	Tralkoxydim
		% of control				
A	23	70	81	41	28	14
B	17	43	54	45	33	25
C	44	59	73	76	65	56
UM1 (R)[a]		67	83	102	56	54
UM33 (R)		71	71	31	14	22
UM5 (S)[b]		14	17	15	13	20

[a] R = resistant line.
[b] S = susceptible line.

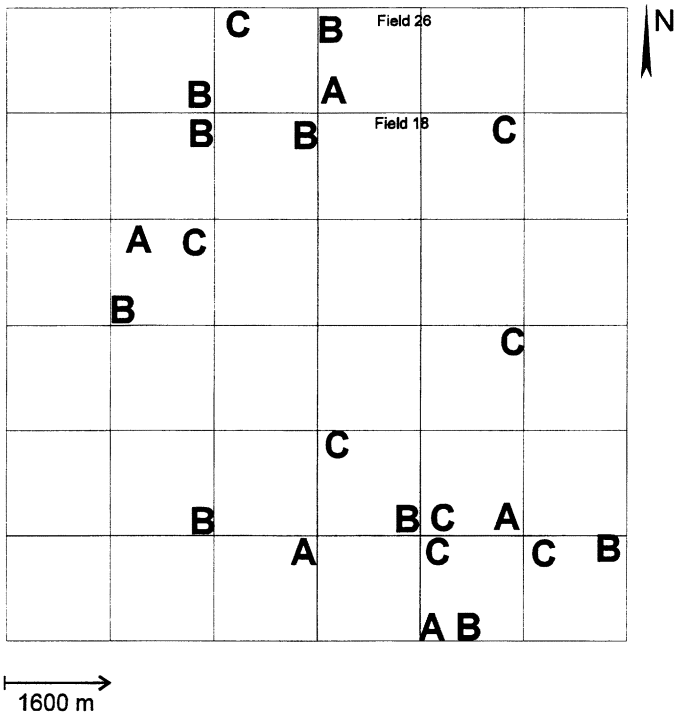

FIGURE 3. Sampling locations of resistant wild oat lines in township 0810W with reference to cross-resistance types A, B, and C.

north part of the field. On the other hand, wild oat of all three types was scattered across field 18. Past herbicide use histories may explain these differences in distribution. Both fields 18 and 26 were sprayed six times with APP herbicides, but field 18 was never sprayed with a CHD herbicide while field 26 was sprayed twice with sethoxydim (data not shown). Sethoxydim may have impeded the evolution of type A wild oat in field 26, and type C wild oat was not affected by this CHD herbicide. The low level of resistance to both CHD and APP herbicides may explain a slower evolution of type B wild oat compared to type A and C. Injury resulting from the application of the herbicides may reduce seed set in type B wild oat compared to the other types.

TABLE 3. Classification of the resistant wild oat lines by townships of origin and cross-resistance types A, B, and C.

	Cross-resistance types		
Township	A	B	C
0802E	0	0	1
0609W	1	2	6
0810W	17	13	31
1208W	1	0	0
1318W	0	1	3
1415W	1	0	0
1416W	1	0	0
2422W	1	0	2
3726W	0	1	0
Total	22	17	43

Pairwise product-moment correlations of herbicide resistance are presented in Table 4. All correlations are positive. Patterns of resistance indicate a high correlation between the two APP herbicides (fenoxaprop-P and clodinafop) and high correlations among the three CHD herbicides (sethoxydim, clethodim and tralkoxydim). However, all cross-correlations between the APP and CHD herbicides are low. Therefore, wild oat that is resistant to fenoxaprop is generally resistant to clodinafop but not always to the CHD herbicides. These correlations are in agreement with results of the cluster analysis where cross-resistance types were defined according to level of resistance to either APP or CHD herbicides.

In the PCA analysis, the first PCA axis is strongly correlated with CHD herbicide resistance and the second axis with APP herbicide resistance (Figure 5). The first two axes account for 88.4% of the total variance observed (69.5 and 18.9% on the first and second axes, respectively). The importance of the first axis indicates that cross-resistance patterns in the 85 lines are based primarily on levels of resistance to CHD herbicides. The three cross-resistance types A, B, and C delineated by cluster analysis are well separated

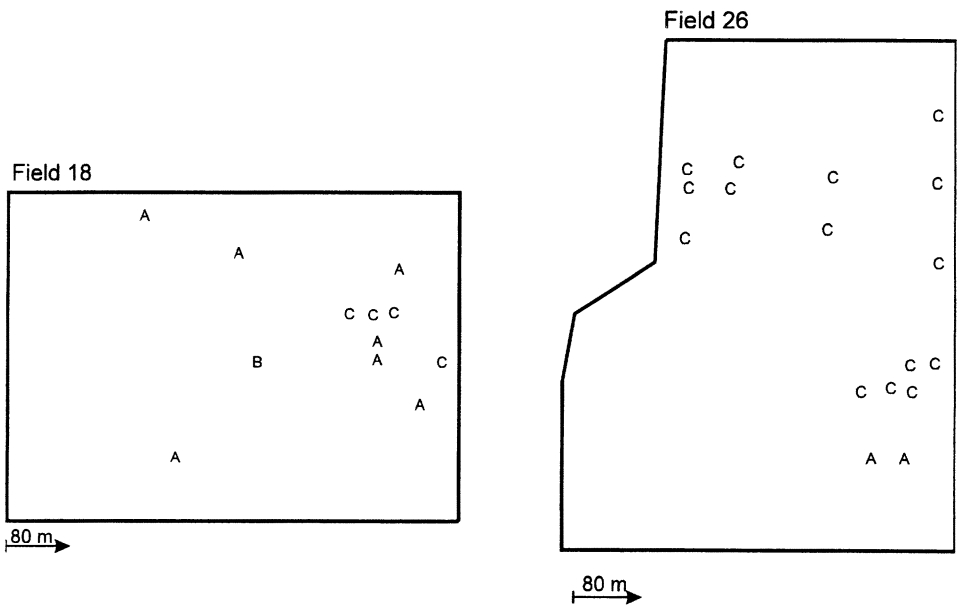

FIGURE 4. Sampling locations of resistant wild oat lines in two fields with reference to cross-resistance types A, B, and C.

TABLE 4. Pairwise Pearson product–moment correlation coefficients between herbicide responses of 85 wild oat lines.

	APP		CHD		
	Fenoxaprop-P	Clodinafop	Sethoxydim	Clethodim	Tralkoxydim
	r^2				
Fenoxaprop-P	1.0				
Clodinafop	0.7	1.0			
Sethoxydim	0.2	0.3	1.0		
Clethodim	0.2	0.3	0.9	1.0	
Tralkoxydim	0.0	0.2	0.8	0.8	1.0

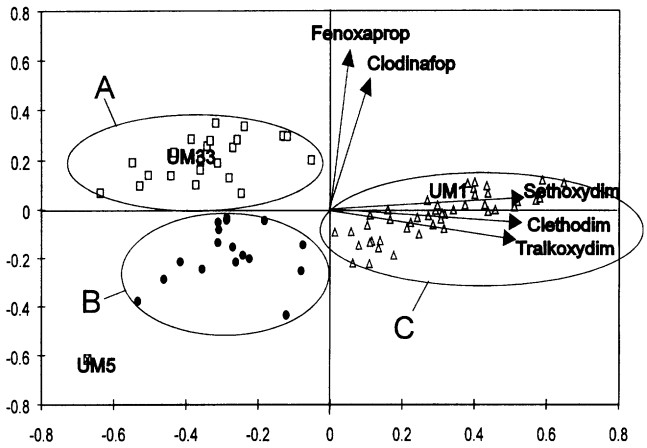

FIGURE 5. PCA biplot for the five ACCase inhibitor herbicides and the 85 wild oat lines.

in the two-dimensional ordination space. Type A lines are positively weighted on the second ordination axis, indicating that they have the highest resistance to APP herbicides. Type C lines are weighted positively on the first axis, indicating high resistance to the CHD herbicides compared to types A and B. The type B lines and the susceptible line UM5 are negatively weighted on both ordination axes, indicating that they have comparatively low levels of resistance (or no resistance) to all five herbicides. The scattering of the wild oat lines in the two-dimensional space highlights the variation in cross-resistance even within the defined types A, B, and C. These variations are not caused by vigor differences among lines, as resistance was established as a percentage of coleoptile length compared to untreated control. Therefore, the variations within cross-resistance types may indicate that each type includes several mutations conferring somewhat comparable cross-resistance patterns.

The apparent untrended distribution of wild oat patches with different cross-resistant patterns indicates that resistance in wild oat evolves from independent sources, at least during the early stages of evolution. As previously reported (Bourgeois and Morrison 1997b; Bourgeois et al. 1997), wild oat lines were collected from clearly defined patches within fields and, with few exceptions, did not constitute a serious problem in terms of major crop losses. The frequent application of ACCase inhibitors in all fields in 0810W (Bourgeois and Morrison 1997a) would appear to have provided sufficient selection pressure to support the evolution of several types of ACCase mutants at random locations in the township.

The incidence of several cross-resistant patterns in a single field reduces the chances of finding one ACCase inhibitor that will be active on all wild oat present in the field. In field 18, most wild oats were type A, which had no resistance or low resistance to CHD herbicides (Figure 4). Although the use of a CHD herbicide killed type A wild oat, types B and C were not controlled and expanded in the field. Clearly, fields with high ACCase inhibitor use histories should not be sprayed with these herbicides more than once every 3 yr. The use of rotating alternative modes of action is recommended, as are nonchemical weed control methods such as mowing or cultivating dense wild oat patches.

The evolution of different cross-resistance types within a field increases the complexity of weed resistance management because each case of resistance can be different. Moreover, weed populations benefit from selection of several cross-resistance types by maintaining genetic variability through mutations in the population. A high genetic variability within a population increases the probability of some individual plants being resistant to new stresses after the occurrence of selection. Wild oat is known for its ability to maintain genetic diversity under strong selection pressure (Jana and Naylor 1982). The ability of wild oat to maintain genetic variability is probably a key factor in the weediness of the species.

Grassy weeds with ACCase inhibitor resistance have a variety of cross-resistance patterns. Some of these cross-resistance patterns resemble the ones described in this study. For example, populations of Italian ryegrass (Gronwald et al. 1992) and oat species (Maneechote et al. 1994; Mansooji et al. 1992) have high resistance to APP herbicides and almost no resistance to CHD herbicides. Cross-resistance in green foxtail (Heap and Morrison 1996), large crabgrass (Wiederholt and Stoltenberg 1995), giant foxtail (Stoltenberg and Wiederholt 1995), and goosegrass (Leach et al. 1995) resembles the patterns that characterize types B and C.

Physiological and biochemical evidence suggests that the evolution of ACCase resistance in grassy weeds resulted from point mutation altering the ACCase binding sites (Betts et al. 1992; Leach et al. 1995; Marles et al. 1993; Marshall et al. 1994). Furthermore, alteration in binding sites on the ACCase enzyme was described as the mechanism of resistance in UM1 wild oat (M. D. Devine, personal communication). Based on genetic studies, the difference in cross-resistance patterns between UM1 and UM33 resulted from two different alleles occurring at the same gene locus (Murray et al. 1995). Marshall et al. (1994) reported that at least three different alleles conferred resistance to ACCase inhibitors in corn (*Zea mays* L.). Therefore, the basis of the difference between cross-resistance types may be related to specific point mutations that alter the ACCase binding sites in different ways.

Variation in cross-resistance patterns induced by point mutations was documented for imidazolinone and sulfonylurea resistance. Resistance to imidazolinone and sulfonylurea herbicides is conferred by point mutation on the gene encoding for the acetolactate synthase (ALS) (Mazur and Falco 1989; Newhouse et al. 1991). In yeast, Mazur and Falco (1989) identified 24 different amino acid substitutions on the ALS enzyme responsible for resistance to the sulfonylurea herbicides. Three mutations of the ALS enzyme in corn resulted in distinct cross-resistance patterns to imidazolinone and sulfonylurea herbicides (Newhouse et al.

1991). The situation of ACCase resistance in wild oat may be comparable to ALS resistance.

This study clearly indicated similar responses of ACCase resistant wild oat among APP herbicides and among CHD herbicides but not between CHD and APP herbicides. Three major types of cross-resistance were identified: type A was resistant to APP and susceptible to CHD herbicides; type B had low levels of resistance to all herbicides; and type C had comparatively high levels of resistance to all herbicides. However, no wild oat line exhibited high CHD herbicide resistance and low APP herbicide resistance. Since APPs and CHDs bind in the same region of the ACCase enzyme (Rendina et al. 1989), the clear differentiation between type A and C indicates that binding of APP herbicides may be more sensitive to changes in the ACCase than binding of CHD herbicides. Although unproven, it may well be that some mutations or conformational changes in the vicinity of the ACCase inhibitor binding site may prevent APP herbicides from binding, but not CHD herbicides.

Further work is necessary to understand the mechanisms of action of the ACCase inhibitors as well as the mechanisms of resistance. However, the difference in cross-resistance found in wild oat at the phenotypic level should provide interesting leads in the study of these mechanisms at the molecular level.

Sources of Materials

[1] Gum agar, catalog no. 7002, Sigma Chemical Co., P.O. Box 14508, St. Louis, MO 63178.

Acknowledgments

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