Resistant Pest Management Newsletter

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Resistance Management from around the Globe

Baseline Susceptibility to Abamectin in the Colorado Potato Beetle *Leptinotarsa decemlineata* (Say)

ABSTRACT. Baseline susceptibility to abamectin in thirty-four Colorado potato beetle (CPB) populations was determined by diet-incorporated assays. Also, filter-paper exposure assays quantified esfenvalerate and azinphosmethyl resistance. LC50 values ranged from 0.09 to 2.69 ppm abamectin and differed by 9-fold in 1993 and 21-fold in 1994. Based on the composite response of this assay, the predicted LC50, LC95, and LC99 of 0.62, 15.8, and 60.3 ppm, respectively, represent the best estimate of the overall baseline susceptibility in CPB populations before field exposure to abamectin. Susceptibility to abamectin significantly decreased as the levels of resistance to esfenvalerate and azinphosmethyl increased.

INTRODUCTION: Colorado potato beetle (CPB), *Leptinotarsa decemlineata* (Say), has developed resistance to virtually every class of insecticides. Because of this resistance problem, insecticides with novel modes of toxic action are needed to deploy in pesticide rotations to minimize selection pressure to any one pesticide. Abamectin exhibits a uniquely different toxic action and is highly active on CPB. Because registration of this insecticide for CPB control may be forthcoming, we determined baseline susceptibility in CPB populations to abamectin as a reference to detect shifts in susceptibility following commercial use. We also determined if abamectin showed any potential cross resistance to esfenvalerate and azinphosmethyl to provide insight into how resistance may occur and how the product should be used to minimize the risks of resistance.

MATERIALS and METHODS: Thirty-two geographically distinct populations of post-diapause CPB adults were collected by cooperators from nine states in the U.S. and one province in Canada in 1993 and 1994. During May through July, roughly 200~300 adults from each population were shipped to a bioassay facility at the University of Maryland Wye Research and Education Center, Queenstown, MD. Questionnaires documented the insecticide use patterns used by the grower at each collection site. In 1994, three laboratory colonies of CPB were also tested: 1) a standard New Jersey (NJ) strain from the NJ Department of Agriculture Beneficial Insects Rearing Laboratory (Trenton, NJ) that was maintained continuously for 12 years without introgression of new adults. This population was never exposed to abamectin and served as a standard for future comparisons with field populations and as a measure of variation in our bioassay technique; 2) an abamectin-resistant strain (~25-fold) from the University of Massachusetts induced field selection in large cages over two growing seasons (Argentine and Clark 1990); and 3) a susceptible strain that was used to generate the resistant one. Adults from each population were segregated and reared in large field cages on potato plants. Initially, a limited number of adults were available in the abamectin-resistant strain, so this population was reared through two generations and tests were performed on the F2 progeny. Egg masses were collected daily, surface-sterilized in a 5% chlorox solution, and stored attached to leaf discs at 13°C to synchronize development. Several days prior to each test, eggs were removed from storage and reared at 24°C until eclosion. Neonates were allowed to feed on the remaining chorion then transferred directly to bioassay trays.

A feeding bioassay was conducted on each population with modified potato leaf-agar diet developed by Tom Forrester, USDA-APHIS Methods Development Center, Mission, TX. On each test day, a stock solution of a 15% emulsified concentration of abamectin was prepared in de-ionized water with surfactant added (0.5 ml/liter Triton 100). Five solutions of a 0.5-fold dilution series plus a water control were prepared. Diet was freshly prepared each day, divided into six portions, and maintained in a water bath at 55oC. An aliquot of each solution was added to diet to achieve a final 0.1-fold dilution. The final abamectin diet concentrations in the diet ranged from 0.125 to 10 ppm. After agitation for 10 seconds, treated diet of each concentration was dispensed into five wells of a bioassay tray, allowed to cool and set. Five wells of untreated diet per tray were also prepared. Ten active neonates with red body color were transferred with a camel-hair brush to each diet well. Vented mylar was then glued to the top of the tray to prevent escape. Larvae were allowed to feed on the treated diet in a growth chamber under 16:8 (light:dark) photoperiod at 24°C. After 48 h, larvae were counted as dead if they could not move any leg after being flipped over on their dorsal side. Fifty larvae were tested per

concentration per replicate tray. For each population, at least three replicates were conducted on separate days with fresh larvae. Concentration-mortality responses were calculated with the POLO-PC probit regression program (LeOra Software 1987).

A standard filter-paper assay (Heim et al. 1990) was used to quantify resistance for each population to organophosphates and pyrethroids. Insecticide concentrations in acetone were applied to separate filter papers and air-dried. A total of 300 neonate larvae (30 larvae per replicate) were exposed to discriminating concentrations of 100 mg/ml of esfenvalerate and 2,500 mg/ml of azinphosmethyl. Mortality was observed after 24 h and averaged over the 10 tests. The degree of association the LC50 for abamectin and the LC50s for esfenvalerate and azinphos methyl were determined by correlation analysis.

RESULTS and DISCUSSION: Significant variations in sensitivity and toxicity to abamectin existed among the 35 CPB populations. Of the 12 populations tested in 1993, LC50 values ranged 9-fold from 0.31-2.68 ppm and averaged 1.24 ppm (Table 1). Of the 23 populations tested in 1994, LC50 values ranged 21-fold from 0.09-1.89 ppm and averaged 0.44 ppm (Table 2). Only three populations were collected from the same field locations in both years and the relative ranking in susceptibility of these populations was similar. Response slopes ranged from 1.0 to 2.5 and tended to become shallower as the sensitivity to abamectin decreased. Concentration-mortality responses of populations tested in 1993 showed significantly less sensitivity to abamectin. Although the assay procedure was the same, this difference in susceptibility may be partly due to technician biases in assessing end-point mortality between years. However, because the NJ standard susceptible strain was not included in 1993 tests, it was impossible to document shifts in assay consistency. Another reason for the difference in lethal concentrations between years may be attributed to the history of insecticide use at the collection sites. Nine of the 12 populations tested in 1993 were collected on research farms where abamectin was used in small-scale testing for several years, whereas populations in 1994 came primarily from commercial potato farms without any previous exposure to abamectin. Due to prior exposure, the 1993 populations may have been more tolerant to abamectin.

Table 1. Susceptibility of *Leptinotarsa decemlineata* (Say) populations to azinphosmethyl, esfenvalerate, and abamectin. Data are listed in order of ascending LC_{50} levels. 1993.

Location	Farm	% mortality of neonates exposed to:		Concentration-mortality responses of abamectin incorporated-diet feeding assay			
		Azinphos-methyl	Esfenvalerate	No. of insects tested	Slope ± SE	LC_{50} (95% CL) ppm	LC_{95} (95% CL) ppm
Roper, NC	Jones	41.7 ± 11.4	100.0	1200	1.82 ± 0.31	0.31 (0.02 - 0.86)	2.88 (1.02 - 89.1)
Salisbury, MD	Res. Farm	35.3 ± 3.9	77.3 ± 4.1	1200	1.03 ± 0.09	0.41 (0.19 - 0.70)	7.00 (3.52 - 25.13)
Upper Marlboro, MD	Res. Farm	56.3 ± 5.1	88.3 ± 3.3	1500	1.52 ± 0.10	0.64 (0.37 - 1.00)	4.48 (2.60 - 11.23)
Delmar, DE	Res. Farm	36.3 ± 2.9	69.7 ± 4.9	1500	1.45 ± 0.11	0.65 (0.29 - 1.16)	5.01 (2.64 - 16.04)
Deerfield, MA	Res. Farm	44.0 ± 3.8	12.3 ± 3.3	1500	2.04 ± 0.17	1.38 (0.78 - 2.13)	15.83 (8.08 - 51.11)
Bridgeton, NJ	Res. Farm	29.0 ± 4.2	33.0 ± 5.2	2100	1.50 ± 0.13	1.36 (0.70 - 2.17)	9.65 (4.99 - 24.19)
State College, PA	Res. Farm	35.7 ± 6.0	12.3 ± 2.4	1500	1.39 ± 0.10	1.38 (0.90 - 1.93)	11.43 (7.07 - 23.93)
Phelps, NY	Res. Farm	24.3 ± 4.2	34.3 ± 3.0	1500	1.12 ± 0.09	1.64 (1.00 - 2.60)	22.92 (10.98 - 94.7)
Stockton, MD	Ward	16.0 ± 5.1	38.0 ± 3.1	1500	1.47 ± 0.12	1.98 (1.51 - 2.55)	14.82 (10.0 - 26.0)
St. Paul, MN	Res. Farm	missing	84.7 ± 2.3	1500	1.73 ± 0.11	2.09 (1.69 - 2.57)	11.48 (8.41 - 17.44)
Macungie, PA	Lichtenwalner	7.7 ± 3.4	27.0 ± 3.4	1200	1.35 ± 0.12	3.31 (1.63 - 5.36)	20.50 (11.98 - 49.2)
Germansville, PA	Res. Farm	12.0 ± 1.6	23.3 ± 2.3	1500	1.31 ± 0.14	2.68 (1.55 - 4.11)	25.62 (13.84 - 81.9)

Table 2. Susceptibility of *Leptinotarsa decemlineata* (Say) populations to azinphosmethyl, esfenvalerate, and abamectin. Data are listed in order of ascending LC_{50} levels. 1994.

Location	Farm	% mortality of neonates exposed to:		Concentration-mortality responses of abamectin incorporated-diet feeding assay			
		Azinphos-methyl	Esfenvalerate	No. of insects tested	Slope ± SE	LC_{50} (95% CL) ppm	LC_{95} (95% CL) ppm
Aberdeen, ID	Yancy	96.5 ± 2.1	100	900	2.1 ± 0.3	0.09 (0.05 - 0.13)	0.36 (0.27 - 0.57)
Paco, WA	T&B Farms	100	100	900	1.7 ± 0.2	0.13 (0.09 - 0.18)	0.52 (0.55 - 1.03)
UMass Susc. Strain	Lab Colony	100	99.5 ± 0.5	1800	1.7 ± 0.1	0.13 (0.06 - 0.20)	0.55 (0.51 - 1.48)
Alliance, NC	Daniels	40.0 ± 6.6	73.0 ± 4.8	900	1.9 ± 0.1	0.18 (0.14 - 0.22)	0.83(0.66 - 1.13)
Aberdeen, ID	Backer	99.5 ± 0.5	100	900	2.5 ± 0.2	0.22 (0.16 - 0.27)	0.72(0.56 - 1.00)
Hatfield, MA	Swastowski	60.5 ± 12.0	38.5 ± 3.4	900	2.1 ± 0.1	0.37 (0.31 - 0.45)	1.50(1.09 - 2.35)
Fairbrook, PA	Campbell	32.5 ± 4.2	38.0 ± 3.1	900	1.9 ± 0.2	0.41(0.34 - 0.49)	1.97(1.47 - 2.96)
Allston, ON	Dorsey	68.5 ± 3.8	47.0 ± 4.7	900	1.6 ± 0.2	0.50(0.33 - 0.68)	3.12(2.01 - 7.11)
Stockton, MD	Holland	33.8 ± 3.2	6.3 ± 3.8	900	1.9 ± 0.2	0.50(0.31 - 0.74)	2.38(1.39 - 7.51)
Presque Isle,ME	Res. Farm	85.5 ± 2.8	76.5 ± 4.2	600	1.1 ± 0.2	0.53(0.32 - 1.10)	7.20(2.51 - 117.8)
Roper, NC	James	88.5 ± 2.5	44.0 ± 3.6	1200	1.8 ± 0.1	0.55(0.44 - 0.70)	2.81(1.90 - 5.10)
Hamburg, PA	Lichtenwalner	34.5 ± 5.7	14.0 ± 1.8	1200	1.8 ± 0.2	0.56(0.35 - 0.83)	3.80(1.65 - 8.57)
NJ Dept of Ag	Lab Colony	68.5 ± 11.6	84.5 ± 1.9	900	2.1 ± 0.2	0.58(0.43 - 0.77)	2.86(1.66 - 4.26)
UMass Resist. Strain	Lab Colony	82.5 ± 5.4	93.5 ± 2.6	1200	1.3 ± 0.8	0.60(0.39 - 0.97)	5.71(2.67 - 28.17)
Savannah, NY	Malone	76.0 ± 3.6	56.0 ± 4.1	900	1.4 ± 0.2	0.60(0.32 - 0.93)	4.71(2.41 - 23.25)
Hadley, MA	Tudryn	61.0 ± 4.9	17.0 ± 5.7	1200	1.5 ± 0.2	0.62(0.39 - 0.92)	4.38(2.41 - 14.81)
Upper Marlboro, MD	Res. Farm	96.5 ± 1.1	77.0 ± 3.5	900	2.1 ± 0.2	0.63(0.53 - 0.79)	2.59(1.94 - 3.93)
Savannah, NY	Jackson	66.8 ± 4.6	58.5 ± 4.6	900	2.4 ± 0.4	0.70(0.57 - 0.97)	2.44(1.67 - 5.31)
Allston, ON	MacKenzie	53.5 ± 5.7	23.0 ± 3.4	600	1.4 ± 0.3	0.76	-
Shrewsbury,MD	Thompson	90.5 ± 2.0	58.0 ± 6.2	1800	1.7 ± 0.2	0.93(0.11 - 1.73)	5.52(2.75 - 69.48)
Germansville,PA	Res. Farm	31.5 ± 4.7	36.7 ± 8.8	300	1.0 ± 0.2	0.99	-
Riverhead, NY	Wells	30.5 ± 4.5	39.5 ± 5.4	1200	1.4 ± 0.2	1.69(0.91 - 5.08)	14.85(4.99 - 1641)
Riverhead, NY	Zaweski	83.5 ± 3.5	34.5 ± 7.5	900	1.0 ± 0.1	1.89(1.11 - 5.12)	36.47(10.15 - 1052)

Figure 1 gives the composite concentration-mortality response based on pooled data from both years. The predicted LC50, LC95, and LC99 of 0.62, 15.8, and 60.3 ppm, respectively, represent the best estimate of

the overall baseline susceptibility in CPB populations before field exposure to abamectin. Thus, the response can be used as a reference for tracking changes in CPB susceptibility to abamectin. The LC50 value for the susceptible and resistant strains differed only by 4-fold and fell within the normal range of baseline susceptibility. This difference was much lower than the expected ratio of resistance determined by a topical assay of 4th instars (Argentine et al. 1992) from the same laboratory strains. Because the laboratory-induced abamectin resistance is unstable and associated with high fitness costs (Clark, personal communication), the resistant strain, reared for two generations without selection pressure, may have reverted to a more susceptible level. Alternatively, this resistance, induced in the laboratory by applying a topical dose to late instars, may not be fully expressed in an ingestion assay with neonates. Additional testing will be necessary to clarify this interpretation.

Since most populations had little or no history of abamectin exposure, differences in susceptibility were probably due to variation in general vigor. However, correlative responses to abamectin, esfenvalerate, and azinphosmethyl were demonstrated. Of the 35 populations tested, 13 were highly resistant to esfenvalerate and azinphosmethyl, 13 exhibited moderate levels of resistance to at least one insecticide, and 9 were susceptible to both insecticides. Populations showing <40% mortality to discriminating concentrations were considered resistant, whereas those showing >80% mortality were susceptible. Susceptibility to abamectin significantly decreased as the levels of resistance to esfenvalerate and azinphosmethyl increased (Figures 2 & 3). In 1993, the two most tolerant populations (LC50s of 1.69 and 1.89 ppm) were collected from Long Island potato farms where multi-class insecticide resistance was well documented. The metabolic mechanisms involved in the resistance of CPB to insecticides are generally polyfactorial (Forgash 1985, Clark et al. 1992, Ioannidis et al. 1991), so an altered molecular factor in organophosphate and pyrethroid resistant beetles may increase their sensitivity to abamectin. Argentine et al. (1992) determined that abamectin resistance in CPB may in part be attributed to increased levels of carboxylesterases - - a common biochemical mechanism of pesticide resistance. Whether such interactions actually enhanced tolerance to abamectin in populations tested in this study is unknown. Another explanation for the correlative responses may be related to predisposition response associated with the general fitness of the resistant populations. Proactive implementation of the findings of this study will help monitor shifts in susceptibility to abamectin and design the most appropriate resistance management strategies for this novel insecticide.

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Changes in Resistance Status of *Rhyzopertha dominica* (F.) (Coleoptera: Bostrychidae), A Pest of Stored Grain in Brazil, With and Without Deltamethrin Selection

INTRODUCTION: *Rhyzopertha dominica* (F.) (Coleoptera: Bostrychidae) is the most serious pest in stored wheat in Brazil and deltamethrin is one of the most important pyrethroid insecticides recommended as a grain protectant. Failure to control this pest by deltamethrin was reported in 1993 by Lorini and Schneider and resistant strains were found in 1996. Pest susceptibility to insecticides may change depending on the selection pressure from these compounds on populations. Strains of *R. dominica* developed resistance to deltamethrin following continuous treatment of wheat grain with deltamethrin for many years. Laboratory selection experiments provide a useful tool to predict the probability of a pest developing resistance to an insecticide in storage and this can be done by selecting the pest population with the insecticide over many generations.

To verify the extent of susceptibility changes in certain strains of *R. dominica*, selection experiments were carried out over several generations of this species.

MATERIALS and METHODS:

Strains and selection
R. dominica from Brazil (BR4, BR2, BR6 and BR7) and from United Kingdom (UK1) were used to determine changing patterns of resistance with and without insecticide selection pressure. BR4 and UK1 represent the normal susceptible strains, BR2 had intermediate tolerance, and BR6 and BR7 were resistance strains (Lorini & Gallery 1996). Subcultures of each strain were reared with and without deltamethrin pressure at each generation. Selection exposed each population to filter paper treated with an appropriate LC50 on filter paper, the survivors cultured and selected again at the next generation.

Bioassay procedure
Every third generation, each strain was bioassayed with a filter paper recommended by the FAO (Anonymous 1969, 1974) with some modifications. Each strain was bioassayed on filter paper with five concentrations of deltamethrin and a control. A stock solution (27 g a.i./liter) of deltamethrin (K-otec 25 EC) was diluted with petroleum ether solvent to the required concentrations and 1.0 ml of each concentration applied to separate 9.0 cm diameter filters. The filter papers were left to dry before placing them in petri dishes. Ten adult *R. dominica*, 01-10 days old, were released onto each treated filter paper. They were left in an incubator with temperature and humidity controls at 27 ± 1 oC and 70 ± 5% RH for 24 h before assessment.

Mortality assessment and statistical analysis
R. dominica larvae that were unable to walk during a two-minute observation period were considered dead. In order to obtain the LC50 values, data was analysed in GLIM (Statistical Package published by Royal Statistical Society) version 3.77 (Crawley 1993), and resistance ratios calculated. All data analysis was based on four treatment replications.

RESULTS: Resistant strains BR2, BR6 and BR7 of *R. dominica* (F.) reared without selection increased in susceptibility to deltamethrin insecticide as much as 14-fold after nine generations while the susceptible strains BR4 and UK1 showed no statistical change in over nine generations (Table 1). The most resistant strain, BR7, showed a significant ($p < 0.05$) reduction in LC50 after the third generation. No further significant reduction occurred by the ninth generation.

Table 1. Response (LC_{50} values in g/cm^2) of *Rhyzopertha dominica* (F.) to deltamethrin based on adult bioassays after nine generations without selection.

Strains	$LC_{50}(F_0)$[1]	$LC_{50}(F_n)$[1]	Ratio[2]	$LC_{50}(F_0)$[1]	Ratio[2]	$LC_{50}(F_n)$[1]	Ratio[2]
BR4	0.0252 a	0.0111 a	0.44	0.0117 a	0.46	0.0208 a	0.82
UK1	0.0497 a	0.0443 a	0.89	0.0547 a	1.10	0.0379 a	0.76
BR2	1.5340 a	0.5473 b	0.36	0.5571 b	0.36	0.4331 b	0.28
BR6	3.2240 a	1.2740 ab	0.39	0.9079 b	0.24	0.5195 b	0.16
BR7	22.020 a	2.2610 b	0.10	2.0456 b	0.09	1.5380 b	0.07

[1] LC_{50}s followed with the same letter are not significantly different ($p < 0.05$) within the same strain; [2] Ratio = $LC_{50}(F_n)$ divided by $LC_{50}(F_0)$ where n = 3,6, or 9 generations.

When the strains were selected with deltamethrin the susceptible strains quickly increased their tolerance to this insecticide, by 158-fold after six generations. Meanwhile, the tolerance of the resistant strains increased slowly to five-fold by the sixth generation of selection. Again, the first three generations of selection were the most important invoking the highest changes in LC50 values. When the mean LC50 of the most susceptible strain (BR4) after nine generations of non-selection is compared to the most resistant strain (BR7) after six generations of selection, the resistance level increased from 1,280 to 6,948 fold (Tables 1 & 2).

Table 2. Response (LC_{50} values in µg/cm2) of *Rhyzopertha dominica* (F) to deltamethrin based on adult bioassays after six generations without selection.

Strains	$LC_{50}(F_1)$[1]	$LC_{50}(F_x)$[1]	Ratio[2]	$LC_{50}(F_x)$[1]	Ratio[2]
BR4	0.0283 a	1.4660 b	50.2	3.9950 c	150.5
UR1	0.0497 a	0.5957 b	12.0	0.8963 b	18.0
BR2	1.5340 a	8.0710 b	5.3	11.720 c	7.6
BR6	3.3240 a	20.360 b	6.1	67.460 c	20.3
BR7	22.020 a	85.220 b	3.9	119.50 c	5.4

[1] LC_{50}s followed with the same letter are not significantly different ($p < 0.05$) within the same strain; [2] Ratio = $LC_{50}(F_x)$ divided by $LC_{50}(F_1)$ where x = 5 or 6 generations.

DISCUSSION: Considerable changes in *R. dominica* tolerance to deltamethrin insecticide occurred quickly. Thus, *R. dominica* has a high potential to develop resistance in the laboratory. Also, these laboratory studies show that the resistance factor declines slowly when the insecticide pressure is removed. This suggests that deltamethrin-resistant strains in the field should be left without pyrethroid treatments for as long as possible until a more susceptible population is restored. However, the potential for cross-resistance to other compounds as demonstrated by Prickett (1980) must be assessed. Any compounds demonstrating cross-resistance must be avoided. Since deltamethrin will continue to be used as a grain protectant in Brazil, careful management is urgently required to avoid the spread of resistant strains and losses in grain quantity and quality.

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Insecticide-Stimulated Reproduction of Cotton Aphid, *Aphis gossypii* Glover, Resistant to Pirimicarb

INTRODUCTION: Resistance to insecticides is found in at least 20 aphid species (Georghiou 1981). In regards to *Aphis gossypii*, resistance to organophosphoric products was first reported in 1964 (Kung et al. 1964). Subsequently, resistance to carbamates (Furk et al. 1980) and pyrethroids (Zil'bermints & Zhuravleva 1984) was reported. For several years, severe infestations of *A. gossypii* were observed on seed potato crops in northern Italy. A preliminary investigation indicated the presence of strains resistant to Pirimicarb (Rongai & Cerato 1994). Non-lethal doses of Sulprofos stimulated aphid mobility (Kerns & Gaylor 1992). O'Brien and Graves (1992) noted that *A. gossypii* resistant to organophosphates had a higher reproduction rate on the first and second day after birth than susceptible individuals.

Our objectives were to assess the degree of resistance in *A. gossypii* to Pirimicarb (Pirimor), Lambda-Cyalothrin (Karate) and Imidacloprid (Confidor) and to investigate whether Pirimicarb is a physiological stimulus that modifies aphid reproduction capacity.

MATERIALS and METHODS: Surveys carried out between 1992-1995 determined aphid infestation in seed potato fields located in the plains and in the Apennines at 800 m above sea level in northern Italy. Every 10 days following plant emergence, an aphid count was made on 100 leaves from randomly selected plants. Concurrently, two yellow traps were sampled twice a week to monitor aphid flight activity (Moericke 1951).

Laboratory assessment of resistance to insecticides A strain of *A. gossypii* and of *Myzus persicae* were reared on potato plants in Plexiglas cages (40 x 60 x 40

cm). The cages were kept in a chamber with a photoperiod of 16:8 h (light:dark), RH between 60 and 80% and at 24°C. The "slide-dip-test", with a few modifications, was used for resistance bioassays (Harlow & Lampert 1990). The aphids were immersed for 30 sec. in insecticide solutions. Each insecticide was tested at seven concentrations (1/8X, 1/4X, 1/2X, X, 2X, 4X and 8X, where X was the recommended field rate expressed in µg a.i./ml). The insecticides tested were Pirimicarb (Pirimor, 17.5% a.i., X=300 µg a.i./ml), Lambda-Cyhalothrin (Karate, 2.8% a.i., X=12 µg a.i./ml) and Imidacloprid (Confidor, 20% a.i., X=150 µg a.i./ml). For both aphid species, 15 aphids were placed on a slide and dipped in one of seven concentrations. Four replications per concentration per species was performed. In 1994 and 1995, the slide-dip test was repeated on *A. gossypii* with the maximum concentration of Pirimicarb.

Effect of Pirimicarb on reproduction rate of A. gossypii
A. gossypii offspring were placed in Petri dishes on young potato leaves on filter paper dipped in water. The Petri dishes were incubated at 25°C and 95% RH. The experiment included three groups of ten aphids with three replicates each. Group A was the control without any treatment, group B was treated with Pirimicarb (300 µg a.i./ml) on the second day after birth and group C was also treated on the second day and then at 7 and 14 day intervals. Each day, new born aphids were counted and removed. The data were subjected to analysis of variance and multi comparison of groups according to the Student-Newman-Keuls method (SigmaStat, version 1.01).

RESULTS: The massive infestations that occurred in recent years in potato crops in northern Italy can be largely attributed to *A. gossypii* insensitivity to Pirimicarb. Data from the yellow traps in 20 fields from the mountain and plains of the north revealed a progressive increase in *A. gossypii* abundance as compared with *M. persicae*, *M. euphorbiae* and *A. fabae* (Figure 1) over a four-year period (1992-1995). Resistance bioassays in the laboratory revealed that *A. gossypii* was insensitive to Pirimicarb, even at the maximum concentration tested (2,400 µg a.i./ml). Pirimicarb was still effective in controlling *M. persicae*. Lambda-Cyhalothrin and Imidacloprid, on the other hand, were very effective against *A. gossypii* (LC50 of 3.6 and 107.4 µg a.i./ml, respectively) (Figure 2).

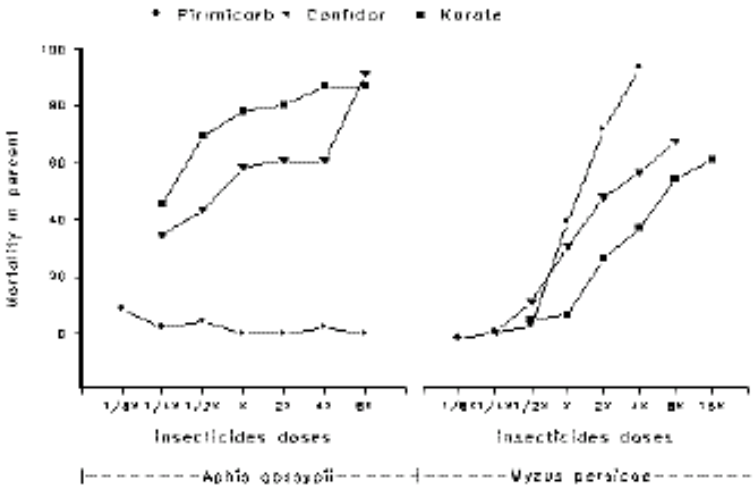

Figure 1. Percentage of A. gossypii lineraly regressed against the total number of M. persicae, M. euphorbiae, A. fabae and A. gossypii, captured between 1992-95. Each point represents the percentage determined for one field.

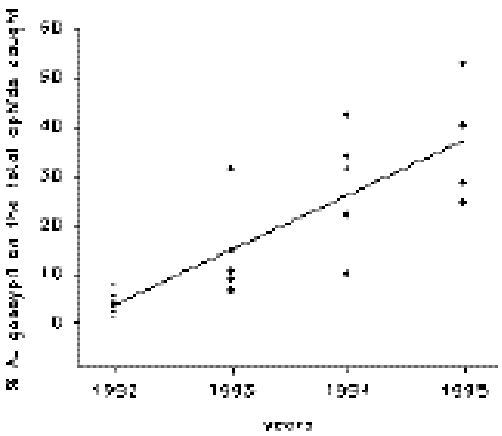

Figure 2. Mortality of A. gossypii and M. persicae to increasing doses of Pirimicarb, Confidor and Karate.

There was a very significant difference in 3- to 12-day-old *A. gossypii* in the reproductive capacity of aphids exposed to, and not exposed to, Pirimicarb (Figures 3 & 4). Furthermore, in the untreated group, the new born aphids begin to appear on the 5th day as reported by O'Brien & Graves (1992); whereas in the treated groups, the first born begin to appear on the 3rd day. Control mortality of individuals always tended to be higher in the untreated group of aphids than in the treated groups, but the difference was not statistically significant.

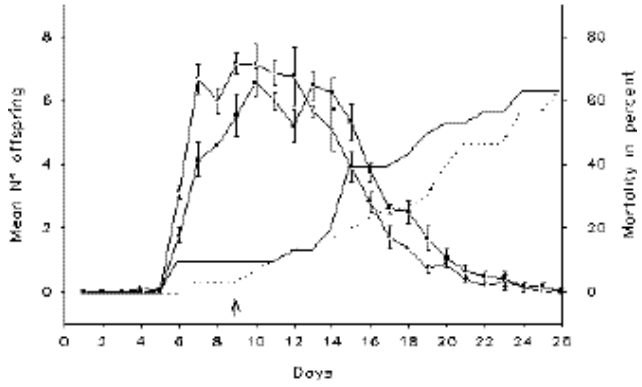

Figure 3. A. gossypii offspring produced in the untreated group (square) compared to a group treated with Pirimicarb (circle, treatment date indicated by the arrow). Control mortality is over time for the untreated group (solid line) and the treated group (dashed line). Error bars represent the 95% confidence limits .

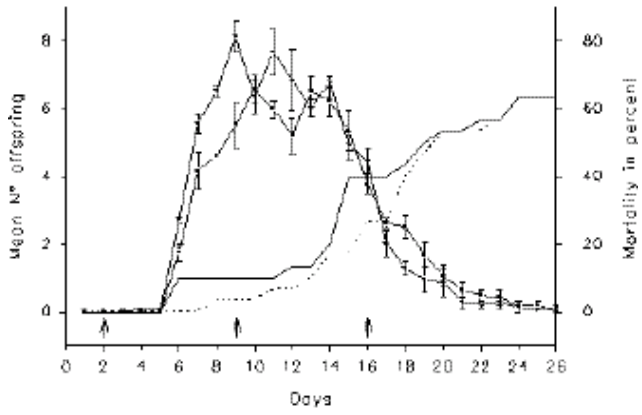

Figure 4. A. gossypii offspring produced in the untreated group (square) and compared to treated three times with Pirimicarb (triangle, treatments indicated by arrows). Control mortality over study period in the untreated group (solid line) and the treated group (dashed line). Error bars represent

In our experiments, the mean increase in reproduction rate in *A. gossypii* exposed to Pirimicarb was about 30% (Table 1). As an example, on day 9 the reproduction rate reached 8.1 aphids born over 24 h for the treated individuals compared with an average of 5.6 for the untreated aphids (44% difference). Enhanced rates of reproduction were clearly evident at the beginning of the reproduction cycle. The same pattern was observed, although to a less degree, when *A. gossypii* was exposed to multiple treatments of Pirimicarb (Figure 4).

Table 1. The effect of Pirimicarb on the reproductive activity of *A. gossypii*.

Pirimicarb treatment	Number of offspring over period		
	3-12 day	13-26 day	life long
Group A (control)	3.75(0.214)a	2.26(0.026)a	65.40(2.21)a
Group B (single treatment)	4.88(0.221)b	1.65(0.085)b	67.19(2.99)
Group C (multiple treatments)	4.88(0.303)b	2.03(0.147)a	72.25(4.28)a

Values in parentheses represent standard errors. Values within a column followed by different letters are significantly different ($P < 0.05$).

These laboratory data are probably lower than those obtained in the field. In fact, Lowers & Sears (1986) found that in the field, the increase in the reproduction rate was almost 3-fold higher than the increase in rate in the laboratory. Considering what we observed in the laboratory, we conclude that in just a few days after treatment, crops can be completely infested by Pirimicarb-resistant *A. gossypii*. Such high levels of infestation will cause a marked drop in production due to direct damage as well as the spread of virus diseases. In the fields with the highest infestation, potato virus Y was detected with ELISA in approximately 80% of the plants.

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Possible Impact of *Barbarea vulgaris* on Insecticide-Resistant Diamondback Moth Management Program

Diamondback moth (DBM), *Plutella xylostella* (L.), is an important pest of *Brassica* crops worldwide and is also found on many wild *Brassicaceae* plants (Marsh 1917, Torsteinson 1953, Harcourt 1986). Host plant abundance and natural enemies are two key factors that regulate DBM populations in the field (Harcourt 1986, Fox et al. 1990, Ooi 1992). In Michigan and southern Ontario, DBM is commonly found on the weed species, *Barbarea vulgaris* R. Br., in early spring in fields and pastures (Idris & Grafius 1994, Harcourt 1986).

The objectives of our study were to: 1) observe the effect of *B. vulgaris* on DBM and 2) evaluate its possible role in management of insecticide-resistant DBM.

The diamondback moths used were a Geneva strain, G88 F97, susceptible to all pesticides applied to control DBM. DBM were reared in the laboratory on broccoli, *Brassica oleracea* L., leaves. Nine wild and six cultivated *Brassicaceae* species were used as host plants (treatments). The leaves of host plants were collected on Michigan State University's campus. Choice and no-choice tests were used for a DBM oviposition study. In this study, we cut a piece of leaf (2 x 2 cm) from each host plant (four replications per host) and randomly put one piece of each host on moist filter paper around inside edge of petri dish (15 cm diameter) with a screen opening (8 cm diameter) in the lid. Leaf pieces were placed 1.0 cm from the dish edge and 4.5 cm from the center, with 1.5 cm between the leaf pieces. The dishes were placed 60 cm under a 160-W white florescent light at 25 ± 2°C. One 2-day-old mated DBM female was released at the center of each dish. Eggs laid were recorded after a 4 h oviposition period. For a no-choice test, we followed procedures similar to the choice test except three leaf pieces of each host plant were put around the edge of each petri dish, 4.0 cm from the dish center. We took 50 eggs per replicate per plant from choice and no-choice tests to measure percentage of egg hatch. The eggs laid on a particular *Brassica* plants were placed on a fresh leaf of the same plant in a petri dish. Leaves were replaced with fresh ones after 12-15 h. Leaves and eggs were kept in the growth chamber [23 ± 2°C, 50-75% RH, 16:8 h (L:D) photoperiod] for four days and the numbers of eggs hatched were recorded. Larvae from this experiment were saved and used for the larval survival and development studies. Forty newly-hatched 1st instars were selected randomly (10 per replicate) and survival through 4th instar recorded on each host plant. In addition, five randomly selected newly hatched 1st instars for each host plant were used to measure the developmental time from hatch through pupation. Larvae for both experiments were placed in a petri dish and fed field-collected leaves of the respective host plants. Leaves were replaced with fresh ones as needed. Numbers of surviving larvae at the end of the 2nd and 4th instars were recorded in the first experiment and the time to pupation was recorded in the second experiment. Numbers of eggs laid, percentage of egg hatch, larval survival and developmental times were analyzed with a one-way analysis of variance (ANOVA).

In both choice and no-choice tests, we found that the numbers of eggs laid per host plant were significantly different among the plants (Table 1). Among the cultivated host plants, DBM laid significantly more eggs on broccoli than on other hosts, especially in the choice test. *Thlaspi arvense* L. and *Lepidium campestre* (L.) R. Br. seemed to be the preferred, while the *B. vulgaris*, *Berteroa incana* L. DC, *Erysimum cheiranthoides* L. and *Raphanus raphanistrum* L. were the less preferred host plants for DBM oviposition. DBM oviposition on *T. arvense* and *L. campestre* were not different significantly from other wild host plants except *E. cheiranthoides* and *B. incana* in no-choice test. We also found that DBM preferred to lay eggs on cultivated plants rather than wild plants in no-choice tests. Furthermore, in choice tests, DBM generally preferred the cultivated host plants over the wild host

plants. There was a significantly higher survival rate of early instar (hatch through 2nd) on cultivated *Brassica* plants and some wild *Brassica* plants (*Brassica kaber* D.C. Wheeler, *Brassica nigra* L. and *R. raphanistrum*). Interestingly, there was a very low survival rate of DBM on *B. incana* and no survival on *B. vulgaris*. We observed that most 1st instar DBM refused to feed or stayed away from *B. vulgaris* leaves. This indicates the presence of an antifeedent in the leaf of *B. vulgaris*. Similar trends were displayed by the percentage of survival of 3rd through 4th instar DBM. The development time of DBM larvae was significantly longer on *L. campestre* than the rest of the host plants, especially the cultivated *Brassicaceae*.

Table 1. Effect of host plant on the diamondback moth female oviposition, percentage of eggs hatching, larval survival and developmental time.

Host plants	Common name	Eggs laid per Female (± SE)		Percent survival (± SE)		Developmental time
		Choice	no-choice	hatch - 2nd	3rd - 4th	(hatch - pupation) (± SE)
CULTIVATED						
Brassica oleracea L. var. *italica*	Broccoli	25.7 ± 8.5a	72.8 ± 7.2a	77.2 ± 6.8abc	83.3 ± 6.4 abc	11.5 ± 0.8f
B. oleracea L. var. *acephala*	Flowering kale	NT[a]	NT	86.1 ± 7.1ab	83.1 ± 7.3abc	12.1 ± 0.9ef
B. oleracea L. var. *botrytis*	Cauliflower	NT	67.5 ± 6.3abc	87.6 ± 9.4a	85.2 ± 6.8ab	11.4 ± 1.1f
B. oleracea L. var. *capitata*	Green cabbage	NT	NT	68.2 ± 7.3c	71.2 ± 5.1bcde	11.8 ± 0.9f
B. oleracea L. var. *capitata*	Red cabbage	NT	NT	53.1 ± 10.5d	66.5 ± 5.7de	12.6 ± 0.5def
B. napus L.	Canola	20.2 ± 4.5ab	69.2 ± 6.5abc	75.1 ± 6.7bc	84.5 ± 5.2ab	11.5 ± 0.7f
WILD						
Brassica kaber D.C. Wheeler	Wild mustard	16.6 ± 2.7bc	70.2 ± 5.1ab	70.1 ± 5.4c	78.7 ± 5.9abcd	11.6 ± 0.6f
B. nigra L. Koch	Black mustard	13.6 ± 3.1cd	62.0 ± 4.5abcd	65.2 ± 4.8c	74.6 ± 5.6abcd	11.4 ± 1.5f
Raphanus raphanistrum L.	Wild radish	6.8 ± 3.4ef	36.7 ± 5.2d	65.8 ± 5.3c	66.3 ± 4.2de	12.5 ± 0.8cdef
Erysimum cheiranthoides L.	Wormseed mustard	5.6 ± 3.3ef	30.3 ± 5.6e	50.4 ± 7.8d	69.4 ± 3.6cde	13.5 ± 1.0bc
Thlaspi arvense L.	Field pennycress	15.6 ± 3.6bc	62.1 ± 7.3abcd	40.3 ± 9.5d	87.3 ± 6.5a	13.3 ± 0.7bcd
Hesperis incana L. D.C.	Hoary rocket	3.4 ± 0.9f	40.1 ± 3.4e	7.5 ± 0.4e	34.4 ± 10.2e	14.5 ± 2.2b
Lepidium campestre (L.) R. Br.	Field pepperweed	15.6 ± 3.4bc	60.3 ± 3.5cd	40.1 ± 5.3d	67.4 ± 5.3de	10.3 ± 5.4a
Capsella bursa-pastoris (L.) Medic	Shepherd's purse	8.3 ± 3.2de	60.8 ± 4.6bcd	44.8 ± 8.7d	65.2 ± 5.6de	12.5 ± 0.8bcde
Barbarea vulgaris R. Br.	Yellow Rocket	5.1 ± 2.5ef	38.8 ± 4.3 cd	0	-[b]	-

Means in columns followed by the same letter are not significantly different based on Tukey's test.
[a] NT = not tested
[b] - = no survivors

Wild host plants for diamondback moth are especially important early in the year in temperate climates before planting cultivated *Brassica* plants (Talekar & Shelton 1993). Results of this study indicated that susceptible DBM larvae can not survive on *B. vulgaris* (Table 1). However, about 13-16% of DBM larvae reared from field population were able to survive and become adults (Idris & Grafius 1994). The mustard glucosides content of *B. vulgaris* was higher than in the other *Brassica* plants tested. Mustard glucosides were toxic to DBM larvae, especially to the susceptible individual as well as the later instars of more tolerant individual. Although some of the individuals reared from field population successfully developed to adults, their development time was significantly prolonged on *B. vulgaris* than on the other wild host plants (Idris & Grafius 1994). This effect could expose individual larva longer to parasitism by *Diadegma insulare* (Cresson), the major parasitoid. We suggest that *B. vulgaris* be used for insecticide-resistant management of DBM. *B. vulgaris* seeds can be sown in the field before winter. They germinate in late April in the northern U.S. and the plant will be abundant throughout May and early June prior to planting of mid-to late-season cole crops. As the most dominant wild host in early spring (Idris, personal field observation), *B. vulgaris* will be the primary host available to DBM females for oviposition. Any surviving larvae will go through an extended developmental period, and thus, are likely to be parasitized by *D. insulare* or sprayed with selective pesticides such as *Bacillus thuringiensis* that have no effect on the parasitoid adults (Idris & Grafius 1993). Alternatively, *B. vulgaris* could serve as a trap crop and be killed by cultivation or herbicides before DBM larvae mature. These tactics will reduce the number of insecticide-resistant DBM before the cropping season begins. *B. vulgaris* also serves as excellent nectar source for *D. insulare* adults (Idris & Grafius 1995) and could enhance their numbers in the field.

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Resistance Monitoring of Helicoverpa armigera to Bacillus thuringiensis in North China

Commercial formulations of *Bacillus thuringiensis* subsp. *kurstaki* (Btk) have been used for a number of years to control cotton bollworm (CBW), *Helicoverpa armigera* in China. The outbreak of CBW in 1992-1995 in North China and its resistance development to almost all extensively used chemical insecticides in cotton fields promoted the application of Bt. In 1994, about 1,000 tons of Bt formulations were used on 160,000 ha cotton in Hadan, Hebei province. An average of four to six sprays were applied annually (1-1.5 kg per ha each spray). In China, there are 24 and 28 pesticide factories that produce powder and liquid Bt formulations (mostly Btk), respectively. It is estimated that the annual production of Bt formulations in China was about 20,000 tons. In recent years, research and demonstration of transgenic Bt cotton developed quickly in USA, Australia and China. There were at least three sources of Bt cotton demonstrated in North China in 1996. Bt cotton for the control of CBW should be commercially available in China in two to three years. In anticipation, the aims of our research were (1) to develop suitable bioassays for monitoring the potential resistance of *H. armigera* to Bt and transgenic cottons, and (2) to investigate the geographic variability in susceptibility of *H. armigera* to Bt in North China.

Five and six field populations of *H. armigera* were collected in late June of 1995 and 1996, respectively, in the Hebei, Henan and Shandong provinces of North China. A diet incorporation bioassay was used in this study to compare toxicities of field populations over both years. F1 or F2 larvae from the field populations were used for bioassays. A laboratory strain collected in 1992 from the Hebei province was also used. The Bt sample, provided by Wuhan BtRDC of China, was calibrated at 15,000 IU/mg. Second instars (2.0 - 4.0 mg/ individual) were held on treated diet in 24-well trays for five to seven days at 25°C. In mortality assessments, larvae were considered dead if unable to move in a coordinated manner. Two populations were considered significantly different if the 95% FL about the LC50s did not overlap. The best time for investigating the mortality was 7 days after the second instars were treated when the slope of the LD-p line was highest and the LC50 became stable (Table 1). Control mortality in some bioassays were much higher on the seventh day after treatment compared to 5 days. So, LC50s were expressed in mg(Bt)/ml diet at seven days after treatment in 1995 and mg(Bt)/ml diet at five days after treatment in 1996.

Table 1. Toxicity of *Bt* on a laboratory strain of *H. armigera* evaluated on different days after treatment.

Days after treatment	Slope b(SE)	LC_{50} (95%FL) (mg/g)	LC_{50} Ratio *
3	2.16(0.25)	0.3789(0.3066-0.4684)	1.00
5	2.63(0.29)	0.1712(0.1300-0.2256)	45.18
7	3.16(0.29)	0.1018(0.0830-0.1249)	26.87
9	3.04(0.28)	0.0783(0.0680-0.0900)	20.67

*LC_{50} Day/ LC_{50} Day 3.

In 1995, the resistance ratios (RRs) of *H. armigera* to Bt ranged from 0.72 - 1.30-fold in five populations when compared with the laboratory strain (Table 2). The Guan population was significantly more susceptible to Bt than most of the other field populations and the laboratory strain. Historically, few Bt sprays have been applied for the control of *H. armigera* in the Guan area. The susceptibility to Bt in Qiuxian of Hebei and in Xihua of Henan province, where Bt were more extensively used, were 1.73- and 1.80-fold, respectively, compared with Guan strain. LC50s of all field populations were not significantly higher than that of the laboratory strain.

Table 2. Resistance monitoring of *H. armigera* to *Bt* in 1995.

Populations	b(SE)	LC_{50}(95%FL) (mg/g)	Resistance ratio		
			A[1]	B[2]	
Laboratory	3.79(0.41)	0.0878(0.0773-0.0988)	1.00	1.38	a
Guan, HB	3.04(0.39)	0.0635(0.0547-0.0736)	0.72	1.00	b
Qiuxian, HB	2.64(0.51)	0.1098(0.0863-0.1388)	1.25	1.73	a
Xihua, HN	2.94(0.38)	0.1140(0.0958-0.1355)	1.30	1.80	a
Gaomi, SD	1.38(0.28)	0.0973(0.0643-0.1471)	1.11	1.53	ab
Dezhou, SD	2.65(0.31)	0.0897(0.0762-0.1577)	1.02	1.41	a

HB=Hebei province, HN=Henan province, SD=Shandong province; [1] LC_{50}s population/ LC_{50} laboratory strain; [2] LC_{50}s population/ LC_{50} Guan population.

The RRs were 3.47, 2.80, 2.13, 4.20, 2.36 and 1.23 folds in 1996 in *H. armigera* populations from Qiuxian and Jizhou (Hebei province), Anyang and Xihua (Henan province), Gaomi and Dezhou (Shandong province), respectively, compared with the laboratory strain (Table 3). Four of the six populations were significantly more tolerant to Bt than the laboratory strain, indicating that the susceptibilities of certain field populations in North China increased in 1996 compared with 1995. The potential of *H. armigera* resistance to Bt toxins is most threatening to the sustainable application of both Bt formulations and transgenic Bt cotton. Therefore, it is necessary for research on the monitoring and management of Bt resistance in *H. armigera* in China to continue.

Table 3. Resistance monitoring of *H. armigera* to *Bt* in 1996.

Populations	b(SE)	LC_{50}(95%FL) (mg/ml)	Resistance ratio*
Laboratory	1.82 (0.25)	0.0447(0.027-0.075)	1.00 c
Qiuxiah, HB	2.15 (0.24)	0.1550(0.092-0.261)	3.47 ab
Jizhou, HB	2.87 (0.32)	0.1251(0.080-0.196)	2.80 ab
Anyang, HN	1.60 (0.21)	0.0951(0.074-0.123)	2.13 bc
Xihua, HN	2.52 (0.35)	0.1877(0.154-0.229)	4.20 a
Gaomi, SD	1.64 (0.30)	0.1054(0.078-0.141)	2.36 b
Dezhou, SD	2.44 (0.38)	0.0550(0.045-0.067)	1.23 bc

HB=Hebei province, HN=Henan province, SD=Shandong province; * LC_{50}s population/ LC_{50} laboratory strain

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Resistance to Imidacloprid in Colorado Potato Beetles from Michigan

Imidacloprid applied as Admire® at planting, or Provado® as a foliar spray was registered by Bayer Corp. for control of Colorado potato beetle on Michigan potatoes in 1995. Because of its effectiveness and the high level of beetle resistance to most other insecticides, over 80% of the potato acreage in Michigan was treated with Admire in 1995 and nearly 90% treated in 1996. Such high use levels raise serious concerns about resistance development. The objectives of our research were to survey fields for Colorado potato beetle adults and larvae that survived Admire treatments and test them for resistance. Characterization of resistance may help to design effective resistance management strategies.

Resistance to Imidacloprid - "Montcalm-R" Strain
Twenty Colorado potato beetle adults were found on June 24 in an Admire-treated commercial potato field in Montcalm County, Michigan. An additional fifteen adults were collected from this site on June 27. These were not found on the border row or on row ends (as might be expected if they were new arrivals), but were two to five rows in from a field border, on plants scattered the length of the field. They were fed field-collected foliage for a day, then fed foliage from greenhouse potato plants (cv. `Snowden') treated with Admire at normal field rate at planting. On both foliage types, many beetles went through periods of intoxication (laying on their back, legs extended to the side, uncontrolled movement of legs, inability to walk or right themselves) followed by periods of normal activity and feeding. Approximately two-thirds (21 out of 35) of the beetles survived on foliage from Admire-treated plants and after five days were placed on untreated greenhouse potato foliage. Eggs were collected and larvae were reared on untreated potato foliage. Adults from these larvae were used in topical insecticide trials described below.

Thirty-five larvae (4th instar) and 14 adults were collected from the same field on July 19 and returned to the lab for testing. Levels of imidacloprid in the foliage in the field had declined by this time (80% of susceptible Colorado potato beetle adults survived on it). However, these larvae must have been present for 2-3 weeks earlier to be in the final instar. When fed on foliage from greenhouse plants treated with Admire, approximately 95% of the field-collected larvae and 85% of the adults survived.

Beetles that survived the dose of > 0.1 mg in topical assays were kept for rearing and future testing. A single female from the Montcalm-R strain survived a dose of 1.0 mg (over 10 times the average lethal dose) in preliminary tests. A male that survived a dose of 0.1 mg was put on the cage with the female and eggs collected for future rearing of a separate strain.

"Admire-selected" Strain
Adults were collected from commercial potatoes treated with Admire in 1995 and fed on Admire-treated greenhouse plants for 2-3 days. Survivors were reared on greenhouse plants. Adults of the next generation were tested for resistance to imidacloprid in topical assays (no significant resistance was found in early generations) and survivors of the higher insecticide doses were used for the next generation.

"Montcalm" Strain
Susceptible beetles were collected from a university field research site at the Montcalm Potato Research Farm, Entrican, Michigan. They were summer generation adults collected one week prior to this experiment, and fed on potato foliage to ensure that they were at least one-week-old, healthy and well fed. This strain is maintained in laboratory culture for use as a susceptible strain.

"Bt Susceptible" Strain
This strain was reared in the laboratory for seven years (approximately 30 generations) without exposure to insecticides and fed on greenhouse-grown potato foliage. Only 30 adults were available for treatment at the time of these experiments (too few for statistical calculations), but mortality data is included for comparison with results for the other strains.

Topical insecticide test
To determine resistance levels, adults from each strain (1- to 2-weeks-old) were treated on the underside of the abdomen with technical grade imidacloprid in acetone (1 ml solution /beetles, 0.001 to 1.0 mg imidacloprid/ml solution). They were placed in petri dishes in groups of 5-10 adults per dish and kept at 25°C, photoperiod 16:8 h (light:dark). Intoxication was evaluated at 24, 48, 72 and 96 h after treatment. Affected beetles were unable to walk forward for a distance greater than their own body length and unable to hold/ climb a pencil or pen. Often, they merely laid on their backs, with legs out to the sides and twitching. If upright, they showed abnormal leg movement and were unable to walk forward. Beetles still intoxicated after 72 h were considered dead. Log dose-probit mortality regressions were used to analyze the data and calculate the dose (LD50) required to kill 50% of beetles of each strain.

Mortality was higher for beetles from the susceptible and Montcalm strains than for beetles from the Montcalm-R and Admire-selected strains (Figure 1). Differences were largest at doses of 0.05 mg and 0.1 mg/beetle. Mortality at 0.05 mg/beetle was over 40% in the two susceptible strains and 10% or less in the Montcalm-R and Admire-selected strains (Figure 2). Beetles collected from the University's Collins Entomology Farm, where Admire was never used, were also tested at this dose and mortality was 69%. In the Admire-selected strain, 100% mortality did not occur until a dose of 1.0 mg/beetle and, as mentioned above, a single Montcalm-R female survived a dose of 1.0 mg/beetle in preliminary test.

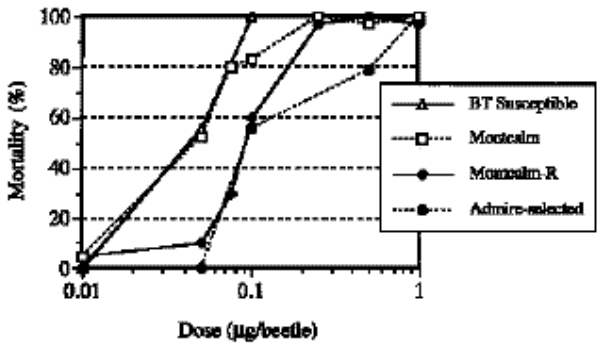

Figure 1. Colorado potato beetle mortality after 72 hours with a range of imidacloprid doses. Beetle mortality was defined as an inability to walk forward more than one body length. Other symptoms included beetles on their back, legs rigid out to the side and twitching.

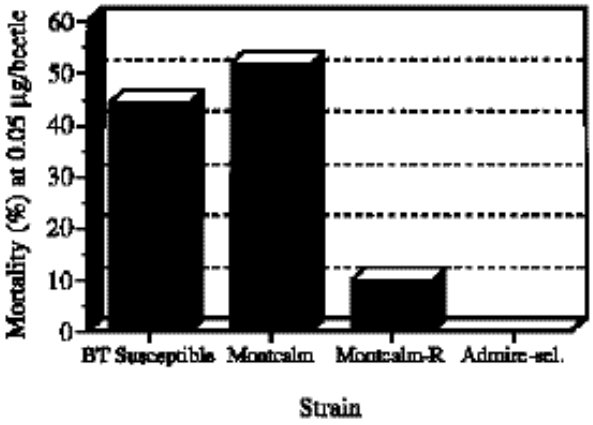

Figure 2. Colorado potato beetle mortality 72 hours after topical treatment with 0.05 mg imidacloprid per beetle.

LD50 values for the Montcalm-R and Admire-selected strains were significantly higher than the LD50 value for beetles collected from the Montcalm Research Farm (Figure 3). The Montcalm-R and Admire-selected strains were 3.8- and 4.5-fold resistant, respectively, to imidacloprid compared with the field-collected strain. Although resistance to insecticides is commonly 10- to 50-fold or greater, this low level of resistance is apparently sufficient to allow survival in the field, at least under some conditions.

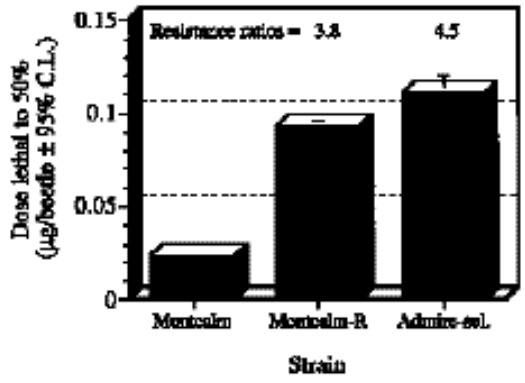

Figure 3. LD 50 values (dose lethal to 50% of the population) for Colorado potato beetle adults 72 hours after treatment with imidacloprid. Resistance ratios are the ratio of the LD50 for the resistant strain (Montaclam-R or Admire-selected) divided by the LD50 for the susceptible (Montcalm) strain.

Recovery from toxic effects

Some beetles in the Montcalm-R and Admire-selected strains recovered from topical treatment after 2 days of intoxication and after expressing symptoms that led to death in other beetles (Figure 4). One group of Montcalm-R beetles treated with 0.25 mg/beetle was kept for observation beyond the 4-day experiment because they were still moving, but unable to walk and expressed other symptoms of intoxication. One day after treatment, all ten beetles were scored as intoxicated. After the first 4 days, one had recovered and the other nine were scored as intoxicated. At the end of 8 days, six out of ten of the beetles had recovered completely from toxic effects and were feeding, mating and laying eggs. This level of recovery indicates that future resistance assays need to be run for a longer than 72-96 h typical for topical treatment studies. The above LD50 values, calculated after 72 h, would be higher if more time was allowed for recovery of the intoxicated beetles.

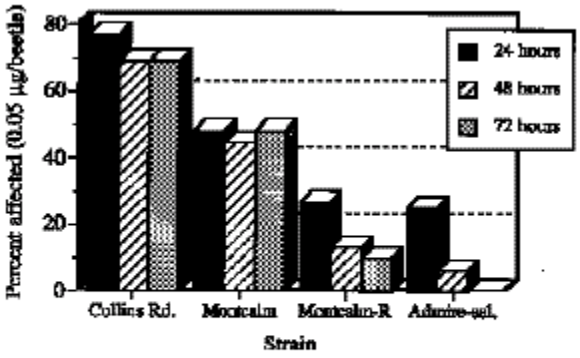

Figure 4. Percent of beetles affected 24, 48 and 72 hours after topical application of 0.05 mg imidacloprid per beetle.

Figures 5 and 6.

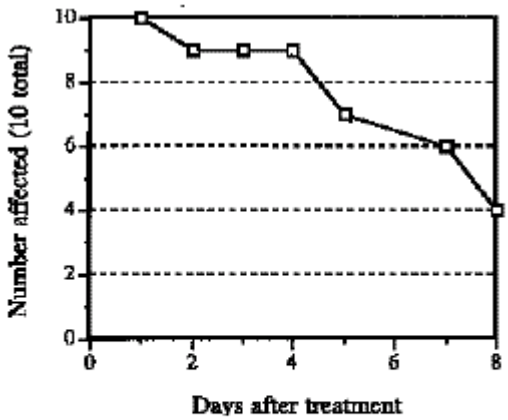

Figure 5. Recovery from intoxication of Montcalm-R strain beetles 1 to 8 days after treatment topically with 0.25 mg imidacloprid per beetle. By 7 days after treatment beetles had either recovered or were dead (no movement, darkened color, abdomen sunken and dry).

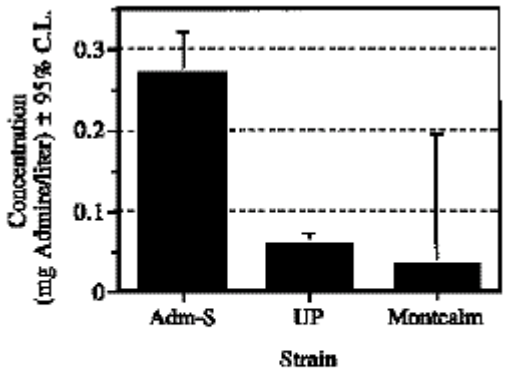

Figure 6. LD50 after 7 days for Admire-selected resistant strain (n=89) and two susceptible strains in a leaf dip assay (n=23 for UP and 60 for Montcalm strains).

This recovery after exposure to imidacloprid observed in the topical assay is similar to the recovery exhibited by the field-collected Montcalm-R strain beetles that fed on foliage from Admire-treated plants. This may indicate that detoxification allows beetles to survive in the field until Admire levels in the plants decline to sublethal levels.

Leaf dip assay

In a leaf-dip assay, 2- to 4-week-old beetles were fed potato foliage dipped in a range of concentrations of Admire 2E. Foliage was allowed to dry for one hour and then fed to beetles for 24 h. Uneaten foliage was removed after 24 h and new foliage provided every one to two days. Number of beetles affected were assessed daily for 7 days; a portion of the beetles were followed for 10 days. The LC50 value after 7 days was significantly higher (4.5-fold) for the Admire-selected strain compared to the UP strain. As in the previous study, significant recovery from intoxication (up to 10 days after treatment) occurred at low concentrations of Admire (Figure 7).

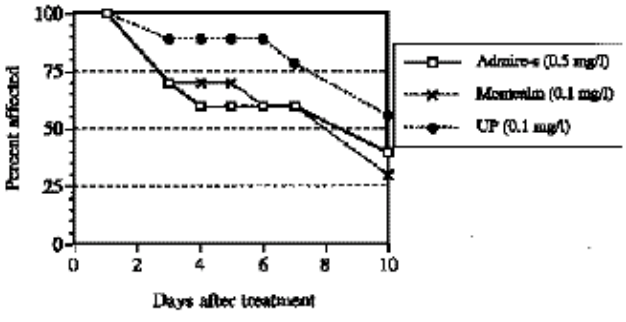

Figure 7. Recovery after feeding with Admire treated foliage, 1 to 10 days after feeding.

These resistance levels are relatively low, but they are similar to the resistance levels to Temik in a Colorado potato beetle population from Monroe County, Michigan, in 1984 (4.4-fold). This resistance level allows beetles to recover from intoxication after feeding on foliage from Admire-treated plants (especially if time for full recovery is allowed). Behavioral resistance may also add to the beetle's ability to survive an Admire treatment. For example, slow-feeding beetles would eat less imidacloprid before getting "sick" and may stop feeding before consuming a toxic dose. Late beetle emergence from overwintering may enhance survival on potatoes treated at planting with Admire. A low resistance level, as reported here, could be especially important when combined with either late emergence of Colorado potato beetle from their overwintering sites or a growing season that allows early potato planting.

Further studies on the effects of imidacloprid ingestion by beetles from the three strains, long-term beetle recovery studies, the interaction between resistance and feeding behavior, and the effects of non-lethal doses of imidacloprid on mating and egg laying are planned. Ongoing studies of these resistant strains will predict the frequency and magnitude of resistance development, the inheritance of resistance and potential resistance mechanism(s).

Management implications

Management strategies to prevent beetle resistance were widely discussed when imidacloprid was first introduced. Research results on these two resistant strains may help determine optimal management strategies. While it is disturbing that resistance occurred after only two years of imidacloprid use, resistance levels are low and immediate research may help prevent the further development of resistance. Survival differences between populations of first instars fed artificial diet treated with imidacloprid were recently reported, but no field survival was not indicated (Olsen et al. 1996).

Insecticide alternation as a resistance management strategy assumes that resistance decreases in the absence of exposure to each insecticide. Unfortunately, Colorado potato beetle resistance to insecticides is often very stable. For example, Michigan beetles are still resistant to DDT although DDT has not been used since 1968. In some cases, resistance may be unstable initially, but become stable with continued use of the insecticide. Research results on the stability of imidacloprid resistance in Colorado potato beetle will be important.

A high-dose strategy for resistance management assumes that resistance is inherited as a recessive gene and any beetle carrying one copy of the gene will not

survive a high insecticide dose. Unfortunately, many insecticide resistance factors in Colorado potato beetle involve dominant genes and even very high doses are unable to kill beetles carrying one copy of the resistance gene (Bishop & Grafius 1996, Ioannidis 1990, Ioannidis et al. 1992). Also, a potato plant treated with Admire at planting accumulates as a high dose early in the season and then degrades to a low dose. Studies with these resistant strains will determine if a high dose strategy is useful at all.

There are advantages and disadvantages associated with the use of Admire at planting versus Provado (as a foliar spray applied later in the season). Admire will always degrade to a low dose over time. A foliar spray of Provado will also degrade into a low dose, but in considerably less time (a few days). The use of Admire on late-planted potatoes would only increase the likelihood that late-emerging beetles will find high doses of Admire in the plants. Provado applied in early-planted fields, at peak beetle activity would reduce beetle exposure to and survival at low doses of the insecticide. Provado rather than Admire applications would also reduce the proportion of the population selected by imidacloprid and some beetles in every field would remain susceptible to Admire/Provado.

Combining Admire treatment with another insecticide such as Agri-Mek (abamectin, Merck) might result in little or no survival of Admire-resistant beetles. However, beware, a few beetles with resistance to both insecticides might survive and create the worst possible outcome.

At this time, the most conservative strategy would be to avoid applying Admire or Provado in 1997 at or near the locations where it was applied in 1996. Adding other mortality agents to the management program (such as crop rotation and propane flaming) would reduce the numbers of imidacloprid-resistant beetles surviving (although not necessarily the proportion of imidacloprid-resistant beetles in the population). We hope to produce preliminary research results next spring to help make better management decisions.

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Resistant *Phalaris minor* Mimics Wheat in Detoxifying Ioproturon

The evolution of weed resistance to herbicides was first predicted in the mid fifties based on microbe and pest resistance to drugs and insecticides (Able 1954, Harper 1956). However, this was not fully realized until the first case of triazine resistance was reported in *Senecio vulgaris* (Ryan 1970). Over the next two decades,113 species (excluding *Phalaris minor*) evolved resistance -- 58 species resistant to triazine alone (Jutsum & Graham 1995). Increased selection pressure due to cultural practices and continued herbicide usage were the main factors leading to resistance in the majority of these cases (Gressel 1990).

In India, adoption of chemical weed control practices allowed wheat farmers to better harness potential yield and India became self sufficient in food production as a result of the `green revolution'. Among the grass weeds, *Phalaris minor* (Littleseed canarygrass), is the major, most prevalent annual weed of wheat cultivated in winter under high fertility and irrigation. While other weed seeds may be killed during the summer or by rainy season flooding under rice cultivation, *Phalaris minor* remains unaffected due to an impermeable seed coat.

The evolution of resistance in *Phalaris minor* to the photosynthesis-inhibiting herbicide, isoproturon, in rice-wheat cropping systems is due to a number of factors. These factors include continuous use of isoproturon for many years, under-dosing and inappropriate application methods (Singh et al. 1993, Gressel et al. 1994, Malik & Singh 1995). Isoproturon resistance was first confirmed in 1992 in biotypes from rice-wheat growing areas of Haryana State, India (Malik & Singh 1994) and since then resistance spread

rapidly in both range and magnitude. The situation in the adjoining state of Punjab is no better (Brar, Pers. Comm.). The dose of isoproturon required for 50% reduction in growth (GR50) in the resistant (R) biotype is greater than that in wheat; and hence, makes isoproturon unsuitable for weed management in wheat. Isoproturon resistant biotypes were also cross-resistant to diclofop-methyl (Singh et al. 1995a). During the last growing season, increasing the dose of diclofop-methyl to twice the recommended dose (2.0 kg a.i/ha) failed to provide any relief to the farmers in affected areas (Balyan, Per. Comm). Fenoxaprop-P-ethyl shows promising results under controlled conditions, but the results need to be verified in the field. A mixture of fenoxaprop-P-ethyl with isoproturon or an addition of a surfactant (Silwet L-77, 0.05%) did not greatly improve the control of the R biotype (Singh et al. 1995b). This article describes the metabolism of isoproturon in wheat and in the R and S biotypes of *Phalaris minor*.

MATERIALS and METHODS: Plants were sprayed at the 2-3 leaf stage with isoproturon (Sabre 55.3% SC) at rates ranging from 0 to 4 kg a.i./ha. Each treatment pot had 5-6 plants and each treatment was replicated three times. Treated plants were harvested 3 weeks later and fresh/dry weights recorded.

For the metabolism studies, single plants were treated with 0.052 mCi 14C-ring labelled isoproturon (sp. Activity = 12 Mbq/mg). Untreated portions of the plants were sprayed with cold isoproturon at 0.25 kg a.i./ha (GR50 for the S biotype). The plants were harvested 48 hours after treatment. The surface residues were removed with 80% methanol and the plants were homogenised in 80% methanol, centrifuged and the supernatant collected. The pellet was re-dissolved, centrifuged and the collected supernatant pooled and dried in vacuum. The extract was filtered with C-18 Sep-Pak cartridges and filtered aliquots of the extracts were spotted on thin layer chromatography (tlc) plates of 25 mm thickness (Sil 60G-25/UV254) along with the 14C isoproturon (standard) for comparison. The tlc plates were eluted with toluene: methanol: acetone: acetic acid (75:15:5:1) and later subjected to phosphor image analysis. Four plants per species were treated and analyzed.

RESULTS and DISCUSSION: Isoproturon had no effect on the growth of the resistant (R) biotype except at the highest dose, whereas the dry weight the of susceptible (S) biotype was significantly reduced at 0.25 kg/ha. The R biotype required 8- to 12-fold higher dose of isoproturon compared to the S biotype for the same level of control (Figure 1). GR50 for wheat were 2.0, 6.0 and 0.5 kg ioproturon for wheat, R and S biotypes, respectively. Degradation of the 14C isoproturon was most rapid in the R biotype compared to the S biotype and wheat (Table 1). The metabolites and Rf values formed by the R biotypes were similar to those of wheat; and thus, the resistance mechanism evolved seems to mimic that of wheat (Table 1).

Table 1. Relative percentages and Rf values for ^{14}C isoproturon metabolites formed by wheat, R & S biotypes of *P. minor*.

Species	% ^{14}C isoproturon metabolites at six Rf values					
	0.01	0.09	0.11	0.21	0.30	0.40
Standard ^{14}C IPU	2	-	-	-	-	98
Wheat	12	8	-	5	21	54
P. minor S biotype	10	8	3	4	9	66
P. minor R. biotype	13	14	-	7	16	49

In wheat, isoproturon is not selective at the doses required to control the R biotype. *In vitro* photosynthesis of S and R biotypes was inhibited equally by isoproturon indicating no alteration at the target site. The recovery of photosynthesis was most rapid in the R biotype after isoproturon application at 4.0 kg a.i./ha, whereas the S biotype did not recover and wheat took longer to recover (Singh et al. 1996a). There was no difference in the uptake and translocation of the 14C isoproturon in the R and S biotypes of *Phalaris*, whereas the degradation of 14C isoproturon was faster in the R biotype (Singh et al 1996b).

Wheat is known to degrade isoproturon by P-450 monooxygenase enzymes. A similar mechanism appears to be involved in *P. minor*, but at a higher rate of activity. Isoproturon toxicity to wheat and the R biotype was increased several fold when P-450 inhibitors were added to the herbicide solution. The effect of the combination, however, was less on the R biotype at lower dose of P-450 inhibitors in the mixture. This indicates that the R biotype has evolved a defensive mechanism similar to, but possibly more efficient than, wheat (Singh et al. unpublished data). It is apparent that degradation of isoproturon in *P. minor* occurs by a mechanism that may be similar, if not identical, to that in wheat. Researchers should look for a selective inhibitor of this degradation process; i.e., a compound that inhibits herbicide degradation in *Phalaris* but not in wheat. Such selective synergists have been found in analogous systems where the herbicide is degraded by similar enzymes in weeds and crops; e.g., atrazine degradation in maize and Poaceae (Gressel 1993) and propanil degradation in rice and *Echinochloa* (Caseley & Leach 1996, Valverde 1996).

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Tobacco Budworm: Seasonal Field Control Versus LD50 of Methyl Parathion in Lower Rio Grande Valley

Methyl parathion has been the most widely used organo-phosphorus insecticide for control of the tobacco budworm on cotton in Mexico and the United States since the early 1950's. There is no recent information on response of budworm populations to methyl parathion in the Lower Rio Grande Valley from either country. We do not know if populations still respond in 1992 as they did in 1967-1968. In addition to the response levels, field control of tobacco budworm with methyl parathion was also determined.

Between 1967 and 1968, larvae bioassayed weighed 30-40 mg and mortalities were determined after 48 h. In 1992, larvae weighed 15-26 mg and mortalities were determined after 72 h. Topical applications of methyl parathion diluted in acetone were made as described by Wolfenbarger (1970) and Wolfenbarger et al. (1982). LD50s (µg/larva) were determined by Probit analysis for data collected in 1967, 1968 and 1992. In 1992, the slope + standard error and the 95% confidence intervals were also determined.

To control larval feeding damage on squared and small bolls, methyl parathion was applied in cotton plots 11 and 15 times in 1967 and 1968, respectively (Wolfenberger 1970). Plots were sampled 2-3 days after each application or as needed. Tobacco budworm accounted for > 90% larval population in these plots.

Control plots were sampled to estimate percent damage in untreated cotton.

Field control was better in 1967 (Figure 1) than 1968 (Figure 2). In 1967, 100% control was observed on three of 11 sample days while complete control was not observed in 1968. The lowest level of control in 1967 was 75% (day 173), while in 1968 it was 6% (day 196). No loss in control was determined in 1967 while 6 to 22% control was lost by midseason in 1968. In 1968, control ranged from 45% to 85% in early and late season. (Percent damaged squares/bolls in the untreated check was 9.5, 4.0, 4.5, 2.2, 7.5, 2.0, 4.0, 3.0, 4.0, 17.0 and 19.0% in 1967 on calendar days 170, 173, 177, 179, 184, 188, 193, 199, 207, 215 and 222, respectively, and 4.3, 4.0, 4.5, 6.5, 11.8, 16.0, 23.0, 19.8, 5.3, 7.0, 7.8 6.8, 8.5, 8.3 and 14.0 in 1968 on calendar days 165, 168, 170, 175, 183, 186, 190, 193, 196, 198, 205, 211, 213, 218 and 225, respectively.)

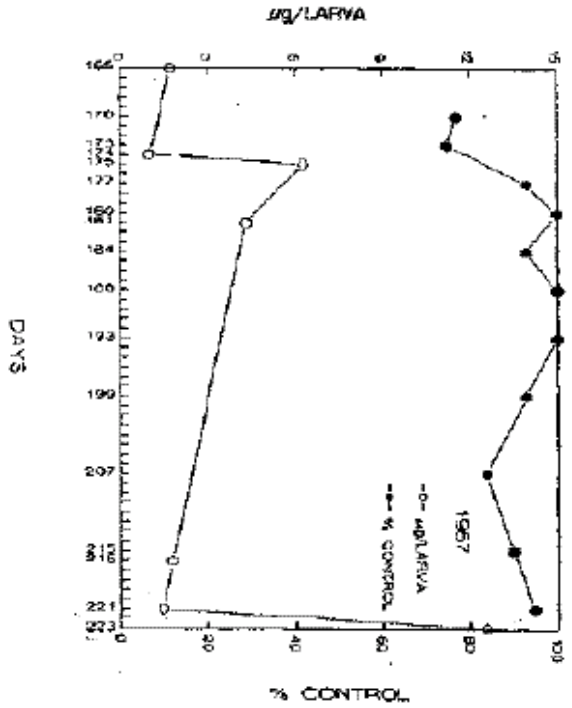

Figure 1. Control (%) of damaged square and small bolls in cotton treated with methyl parathion (1.68 kg a.i./ha) and LD50s. Lower Rio Grande Valley, TX. 1967.

LD50 values in 1967 and 1968 are also presented in Figures 1 and 2. Figure 1 shows LD50s ranged from a low of 0.97 mg/larva (day 174) to 12.6 mg/larva (day 223) during the 1967 season. One month after collecting larvae and obtaining the lowest LD50 value, another collection of larvae from the same plots revealed an LD50 of 6.29. This difference was significant since the 95% confidence intervals about these LD50 values did not overlap. Thus, there was differences in budworm response to methyl parathion, but no seasonal trend was indicated. Following the high LD50 value (day 175), the later larval collections showed decreasing LD50s that dropped to 1.43. Nevertheless, percent control of budworm remained excellent throughout the season.

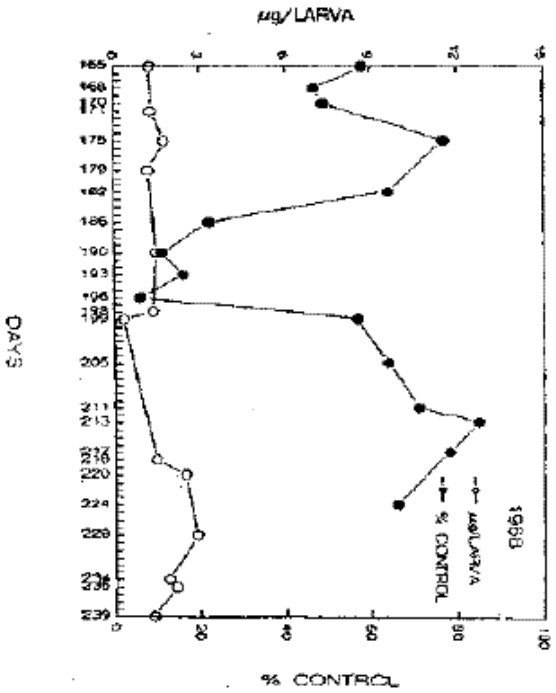

Figure 2. Control (%) of damaged squares and small bolls in cotton treated with methyl parathion (2.24 kg a.i./ha) and LD50s. Lower Rio Grande Valley, TX. 1968

In 1968, the fifteen LD50 values for budworms exposed to methyl parathion ranged from 0.33 (day 199) to 2.87 (day 228). Again no trend indicated in these fluctuations. Both of these values were lower than shown for the lowest and greatest LD50s in 1967. As in 1967, the lowest and the greatest LD50 were significantly different based on non-overlapping of the 95% confidence intervals.

In 1992, nine LD50s from budworm populations in the Lower Rio Grande Valley of Texas, USA and Tamaulipas, Mexico (Table 1) fell within the range of LD50s determined in 1967 and 1968. LD50s ranged from a low of 0.36 mg/larva to a high of 5.45mg/larva and these values were significantly different from each other.

Table 1. Relative toxicity of methylparathion against larvae of tobacco budworm in lower Rio Grande Valley, Texas and Mexico, 1992.

Location	Number of larvae tested	Slope ± SE	LD_{50} mg/larvae	95% Confidence Level
La Blanca (Field 1)	166	0.5 ± 0.12	2.36	0.23-8.73
La Blanca (Field 2)	237	0.7 ± 0.13	5.45	3.09 - 12.39
La Blanca (Field 3)	195	0.5 ± 0.17	0.8	0.02 - 2.06
La Blanca (Field 4)	84	0.95 ± 0.24	0.36	0.095 - 0.74
Brownsville	156	0.96 ± 0.21	1.01	0.68 - 5.33
San Perlita *	87	0.83 ± 0.15	0.76	0.31 - 2.16
Rio Bravo (Sample 1) **	234	0.83 ± 0.10	1.07	0.39 - 3.17
Rio Bravo (Sample 2) **	220	1.03 ± 0.15	2.55	1.69 - 4.21
Valle Hermosa **	286	1.02 ± 0.17	2.02	1.24 - 5.52

*Taken from Norman *et al.* (1993); **Taken from Vargas *et al.* (1993).

Most budworm LD50 values in 1967, 1968 and 1992 ranged from 1.00 to 3.00. LD50s in this range comprised 57, 93 and 50% of the values determined while 50% of the remaining LD50s were < 1.00. Thus, these data show that the response to methyl parathion was variable, but equally toxic to larvae of the tobacco budworm in 1967, 1968 and 1992.

In 1967, the LD50 value of the Brownsville laboratory strain of the tobacco budworm to methyl parathion was 0.0015mg/larva (0.043 mg/g after 48 hours) (Wolfenbarger et al. 1970). This value was 240-fold less than the lowest LD50 (0.36) determined in 1992. This colony, obtained from the USDA laboratory in Tucson, AZ, has been reared continuously since the 1966. Field-collected insects were never added to the colony to avoid contamination by disease organisms. In the future, experiments need to relate LD50s to field control as shown in 1967-1968.

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Resistance Management Reviews

Fewer Constraints than Proclaimed to the Evolution of Glyphosate-Resistant Weeds

Glyphosate is a general herbicide with excellent properties. It has very low mammalian toxicity, low soil persistence and is used widely to knock down weeds in conservation tillage systems just prior to planting. It can also be used non-selectively with directed sprays in orchards, selectively after crop emergence with genetically engineered glyphosate-resistant crops (Padgette et al. 1996), and even killing intransigent underground parasitic weeds (Joel et al. 1995).

A spate of short papers/abstracts have appeared at national (Bradshaw et al. 1995), international (Padgette et al. 1995) and at regional meetings, as well as in the pages of A Good Weed (Jasieniuk 1995) that left most listeners/readers with the impression that it is nearly impossible for glyphosate resistance to evolve in weeds. The evidence presented is based on the basic unfitness of the target enzyme coded on a resistant bacterial gene. Successful and elegant molecular biology was required to modify this gene to enhance the natural activity of its product, as well as to increase its expression to obtain productive, glyphosate-resistant crops. For more acceptable productivity, another bacterial gene coding for an enzyme that degrades glyphosate was engineered into crops. It was assumed that weeds could not repeat that complicated feat. All these "what, me worry?" papers contained a common motif: Evolution " *of weed resistance to glyphosate appears to be an unlikely event, based on the lack of weeds or crops that are inherently tolerant to glyphosate and the long history of extensive use of the herbicide resulting in no resistant weeds. Unique properties of glyphosate such as its mode of action,*

chemical structure, limited metabolism in plants, and lack of residual activity in soil indicates that the herbicide exerts low selection pressure on weed populations" (Padgette et al. 1995). The impression of invincibility from resistance was enhanced by not citing the growing literature on the known inter-, and especially intra-specific, genetic variability in quantitative levels of glyphosate resistance. This literature was known to the various authors, yet must have been considered irrelevant. In turn, this led to dismissing the need to set resistance management strategies in motion, and the ensuing appearance of a glyphosate-resistant population in the management system and the weed where it was most likely to occur. The major objective of this article is to collate this uncited data.

Herbologists know that it is easier to kill some species with glyphosate that others; legumes have always been especially hard to control. This may well be because enhanced degradation (despite the quotation above) occurs in soybean leaves much faster that the other, non-legume species tested (Komossa et al. 1993).

There is considerable intraspecific biotype variability in susceptibility to glyphosate at the whole plant and cellular level (Table 1). It is not wise to consider weeds as distinct from crops in this respect; genetic variation occurring in some crops will surely occur in some weeds. More efforts are spent selecting for resistance in crops than weeds to this excellent herbicide. In the case of Lotus, variability was correlated with elevated levels of the target enzyme, EPSP-synthase (Figure 1) and higher enzyme levels in some strains would confer resistance to low field rates.

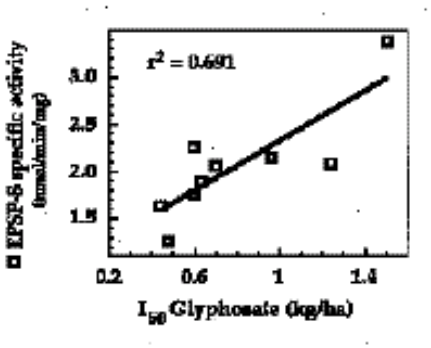

Figure 1. Relationship between the I50 for glyphosate and the specific activity of EPSP-sythase in strains of Lotus corniculatus. (plotted from data in Table 4 of Boerboom et al. 1990).

There are many known mechanisms that confer tolerance (Table 2). A major change in any one mechanism (usually coded for on a single major gene) might confer resistance to field rates. Such major changes are likely to occur where there is high selection pressure. Under low selection pressure, the genes conferring a low-level resistance could amplify, increasing the resistance level at each cycle of selection. Also the recombination of different genes, each giving an increment of resistance, may lead to polygenic resistance (Gressel 1995a, Gressel et al. 1996). This may be the case in an open-pollinated weed, more so if there are many such mutants. The resistance level both with gene amplification and polygenic resistances would creep up (Gressel 1995b) with recurrent selection. Additionally, one of the selectable marker genes, commonly used in genetically engineering crops, was claimed to confer glyphosate resistance (Peñaloza-Vásquez et al. 1995).

Table 2. Known mechanisms that could lead to glyphosate resistance.

Mechanism	Reference
Target site modifications (known in bacteria)	Padgette *et al.* 1996
Overexpression of EPSP-synthase	Hollander-Czytko *et al.* 1992
Amplification of the EPSP-synthase gene	Wang *et al.* 1991, Suh *et al.* 1993
Degradation by plant genes	Komossa *et al.* 1992
Accumulation in lactifers	Foley 1997
Modified translocation	Wallace & Hollinder 1995
Modified phosphate carrier glyphosate	Denis & Delrot 1993
Growth regulators overcoming effect	Kishin 1995

The argument that the low persistence of glyphosate would preclude resistance is specious: there are many cases of Paraquat resistance and Paraquat is less environmentally persistent than glyphosate. It is the persistence relative to the way a weed germinates through a season that is important. An ephemeral herbicide has season-long persistence for a weed that germinates predominantly in a single flush and is standing when the herbicide is used (Wrubel & Gressel 1994).

Thus it was no surprise when Australian press reports, corroborated by discussions with Australian researchers at the International Weed Control Congress in June 1996, stating that a glyphosate-resistant annual ryegrass (*Lolium rigidum*) population appeared. According to the reports, the farmer used 260 g a.i./ha glyphosate mixed with 2,4-D for preplant weed control in conservation tillage for ten consecutive years. While the mixture may have additive or synergistic effects on some weeds or enhance the spectrum of weeds controlled, only glyphosate effects a grass weed such as *Lolium* that is inherently resistant to 2,4-D (Wrubel

& Gressel 1994). The rate of glyphosate used was much lower than the 0.34-1.12 kg/ha normally recommended in the U.S. Now the shift in resistance (Figure 2) is high enough to leave some plants resistant to the highest U.S. rates. Resistance, due to gene amplification or a combination of polygenes, probably would not have evolved in this population if the highest rate was used at least once every three applications in mono-pesticide culture (Gressel 1995a, Gressel et al. 1996). Resistance may not have evolved as quickly if the pre-sowing glyphosate was rotated with other contact grass killers such as Paraquat or an inhibitor to protoporphyrinogen oxidase. A good post-emergence grass killer might have controlled the glyphosate-resistant biotype, unless it was too mature to be susceptible or it was already resistant to the post-emergence herbicides used (as is the case for 40% of Australia). Annual ryegrass is open pollinated (facilitating gene transfer and recombination) and is a prolific seed producer. Furthermore, annual ryegrass has a track record for evolving resistance to virtually all herbicides used for its control (Gressel 1988, Heap 1988, Powles & Matthews 1992).

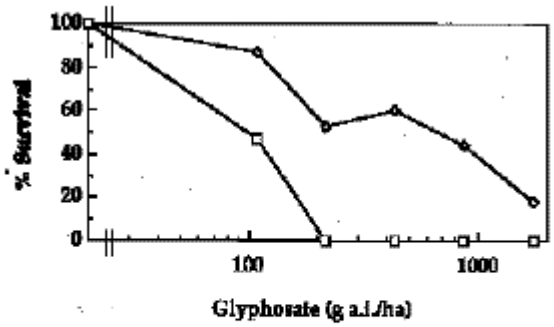

Figure 2. Shift in dose response for Lolium rigidum subjected to recurrent field selections in Australia. Putatively-resistant (diamonds) and never treated (squares) ryegrass plants were sprayed at the 3-4 leaf stage. (replotted from data of Pratley et. al. 1996).

This happenstance in wheat forces us to question the utility of genetically engineered glyphosate-resistant wheat. There have been at least eight applications for testing transgenic wheat in five U.S. states since 1994. Glyphosate use would be clearly contraindicated wherever any given weed species is found in large quantities at both preplant and post-emergence. Such a persistent use of the herbicide would simulate a biological persistence akin to other herbicide (e.g. atrazine) that selected for resistance, despite very low initial frequencies of resistant individuals displaying poor fitness. We should consider similar precautions against multiple uses within a crop season with other glyphosate-resistant crops.

Thus, there are few constraints to weeds evolving resistance to glyphosate. By definition weeds possess the biological characters that allow resistance evolution in most management systems that rely on a single pesticide. We should discuss how to institute resistance management strategies rather than why it will take a long time for resistance to evolve. Farmers should need excellent herbicide such as glyphosate in their arsenal for the future. Industries releasing the first glyphosate-resistant crops after a long a expensive period of research development should have the same interest.

When proclaiming to evolution of pesticide resistance, one should remember the advice of Thurber (1956). "Get it right or let it alone; the conclusion you jump to, may be your own."

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Glyphosate Resistance Discovered in Annual Ryegrass

Herbicide resistance in Australia is an increasing problem and, according to experts, Australia has more resistance than any other country in the world. It is not surprising that the first case of glyphosate (Roundup) resistance worldwide was discovered recently in Australia. While use patterns and mode of action suggest that resistance development to this non-selective knockdown herbicide is unlikely (see *A Good Weed*, No. 5), history now shows that it was not impossible. A case of glyphosate resistance was discovered in Australia. In Echuca, northern Victoria, a farmer found annual ryegrass that was not controlled by glyphosate (Roundup) for the first time in 15 years. The farmer had sprayed this paddock with glyphosate ten times over the past 15 years.

Researchers at the Centre for Conservation Farming at Charles Sturt University at Wagga Wagga confirmed that the ryegrass was resistant to glyphosate. Jim Pratley reported two sets of tests that documented ryegrass resistance to the herbicide. Some of the ryegrass survived almost five times the recommended field dose. Monsanto Australia's technical director, Bill Blowes, said Monsanto was working with the University and conducting trials to confirm the cause of this resistance.

Glyphosate is a broad-spectrum herbicide used to kill unwanted plants both in agriculture and in non-agricultural situations. It is viewed as a key component of conservation or "no-till" farming. In the United

States, glyphosate is the eighth most commonly used herbicide in agriculture and the second most commonly used herbicide in non-agricultural situations. Earlier this year, Monsanto had announced plans to invest nearly US$200 million over the next three years to expand manufacturing and formulation capacity for Roundup. This herbicide played a key role in the 11% increase in Monsanto's agrochemical sales in 1995.

According to Roger Cousens from Latrobe University, high-technology crop production systems that rely entirely on herbicides are "in danger of crashing down around our ears" due to weeds developing resistant to herbicides. Australia is said to have more herbicide resistance than any other major crop-producing region in the world. Ryegrass, the most commonly resistant weed in Australia, is resistant to various herbicides across 40% of the country's agricultural region.

The delay in the buildup of resistance to glyphosate relative to other herbicides is due in part to its use pattern. When used as a pre-sowing knockdown herbicide, glyphosate's failure to control weeds for any reason is masked when complete soil disturbance is achieved during sowing. Survivors of the sowing process may subsequently be controlled by the application of selective post-emergent herbicides. According to Jim Pratley, a glyphosate-resistant weed therefore needs to survive a three-phase mortality process before it can add seed to the soil for the next generation of resistant plants.

The discovery of glyphosate resistance could also affect the introduction of genetically engineered glyphosate-resistant crops in Australia. Monsanto has genetically engineered glyphosate-resistant cotton and soybeans approved for use in the US, the company is currently seeking US approval for glyphosate-resistant canola and is developing glyphosate-resistant corn. In addition, five other genetically engineered glyphosate-resistant crops were tested in US fields - wheat, sugar beets, lettuce, potato and poplar.

At this point, the discovery of glyphosate resistant ryegrass is not a disaster, but rather a single confirmed case over hundreds of thousands of farms. Glyphosate is effective on a wide spectrum of weeds and is considered relatively safe because it breaks down rapidly and is largely inactivated on contact with soil. Thus, glyphosate must be retained as an effective herbicide. Integrated weed management, a combination of weed control techniques, is promoted to avoid the further emergence of herbicide resistance.

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Mutations in *Para* Sodium Channel Protein of Insects Associated with Knockdown Resistance (*kdr*) to Pyrethroids

Since the introduction of the first potent and photostable derivative, permithrin in the early 1970s, synthetic pyrethroids have been widely used in controlling many insect pests. However, the heavy use of synthetic pyrethroids in the last 20 years led to the development of pyrethroid resistance in many insect pest populations. An important resistance mechanism is characterized by a reduced sensitivity of the insect nervous system to pyrethroids and DDT, known as knockdown resistance (*kdr*) (Refs. in Soderlund and Bloomquist 1990). The *kdr* phenomenon was first documented in DDT-resistant house flies. Later it was detected in several other important insect pests, such as *Blatella germanica, Anopheles stephensi, Heliothis virescens* and *Leptinotarsa decemlineata*. Several alleles of *kdr* were identified in house fly including super-*kdr*, that confer up to 500-fold higher resistance to type II pyrethroids such as deltamethrin. Because *kdr* insects are cross-resistant to all pyrethroids and also to DDT, virtually the entire pyrethroid class will not be effective once a pest population has developed the resistance.

Pyrethroid insecticides affect various neuronal components such as ion channels, GABA receptors and ATPases. However, the major site of action of these neurotoxins is the voltage-dependent sodium channels in *kdr* insects as first implicated by electrophysiological studies (Osborne & Hart 1979, Salgado et al. 1983). These studies show that the nervous system of *kdr* house flies was not only less sensitive to pyrethroids, but also to sodium channel site 2 neurotoxins, such as veratridine and aconitine. Subsequent cross-resistance and inheritance studies

with *kdr*-type German cockroach suggest that the resistance to pyrethroids and aconitine was caused by the same mechanism (Dong & Scott 1991). In *Drosophila* two putative sodium channel genes, *para* and DSC1, were identified. Interestingly, the *kdr* resistance trait in *Heliothis virescens, Musca domestica* and *Blatella germanica* was genetically linked to the *para* sodium channel gene (Taylor et al. 1993, Williamson et al. 1993, Knipple et al. 1994, Dong & Scott 1994).

Recently, cDNA sequences of para sodium channel genes, isolated from susceptible and *kdr* or *kdr*-type strains, were reported in both the house fly (Williamson et al. 1996) and the German cockroach (Miyazaki et al. 1996, Dong 1996). Comparison of deduced amino acid sequences revealed only one amino acid difference, leucine in all susceptible strains and phenylalanine in *kdr* and *kdr*-type strains. This amino acid difference resides in the sixth membrane-spanning segment of the domain II of the *para* sodium channel protein. The cDNA sequence of the *para* sodium channel gene was also determined from a super-*kdr* house fly strain (Williamson et al. 1996). Comparative sequence analysis of super-*kdr* and susceptible strains revealed a second amino acid change from methionine (in susceptible stains) to threonine (in super-kdr strains) in the region between the fourth and fifth segments of the domain II of the *para* sodium channel protein. These findings strongly indicate that *kdr* resistance is associated with *para* mutations.

Where do we go from here? Functional analysis of the *para* genes in an expression system, such as *Xenopus oocyte* expression system, is a logical next step to provide further evidence that these putative *kdr* mutations alter the sensitivity of the *para* sodium channel protein to pyrethroids. From the resistance management point of view, the identified putative *kdr* mutations could be used as molecular markers to detect *kdr* individuals in insect populations. For example, *kdr* allele-specific primers can be designed in the polymerase chain reaction to identify *kdr* insects, as well demonstrated by ffrench-Constant's group in detecting cyclodiene resistant insects in pest populations (Steichen & ffrench-Constant 1994).

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