

MANAGING NEONICOTINOID RESISTANCE IN COLORADO POTATO BEETLE

Russell L. Groves^{1/} and Scott A. Chapman^{2/}

Cooperator (s):

Ed Grafius and Adam Byrne, Department of Entomology, Michigan State University, 243 Natural Science, East Lansing, MI 48824. grafius@msu.edu, byrne@msu.edu.

Background and Rationale

The Colorado potato beetle (CPB), *Leptinotarsa decemlineata*, the potato leafhopper, *Empoasca fabae*, and colonizing aphid species remain as the major insect pests of potato in commercial as well as seed production in Wisconsin. If left unchecked, feeding by both the larvae and adults of the CPB alone will completely defoliate plants. Potato growers have struggled to control this problematic insect pest since 1865 when the first broad-spectrum insecticide, Paris green (lead arsenate), was dusted onto potato leaves to protect the foliage from CPB. Since that time, maintaining control of this insect remains at the forefront of our efforts to protect potato from damaging insect pests.

The Colorado potato beetle is truly one on a short list of “super” pests in commercial agriculture that includes, among others, the diamondback moth. The CPB has developed resistance to most insecticides and great care must be used when targeting this insect and making choices to select an appropriate material, or sequence of materials for its control. It is important to recognize that once you begin to observe reductions in the time interval over which a particular material works effectively against CPB or you must increase the rate to get adequate control, insensitivity (resistance) to that insecticide is likely present in the population. Because of the difficulty faced in controlling populations of CPB, future production and revenue increases in Wisconsin will remain challenged by these persist insect pest populations. Recurrent populations of these insect pests demand the development of efficient and effective integrated pest management (IPM) and insecticide resistance management (IRM) strategies which out of necessity need to not only enhance environmental quality but also reduce risks in potato production systems.

The Wisconsin Eco-Potato Collaboration between the Wisconsin Potato and Vegetable Growers Association, the World Wildlife Fund, and the University of Wisconsin has embraced this concept and has taken a leadership role in the promotion and support of research to encourage the adoption of pest management alternatives to minimize the reliance on high-risk pesticides and incorporate safer and more selective pest control products which have less adverse environmental impacts. The goals of the collaboration have been directly focused on the development of biologically-based, pest management strategies that produce a safer food supply for consumers, reduce the risk to agricultural producers, and enhance environmental quality.

Wisconsin potato growers rely heavily on neonicotinoid insecticides for the control of damaging populations of the Colorado potato beetle. Reported at-plant applications of soil-applied neonicotinoid insecticides have occurred on greater than 80% of all acres planted through the five year interval 2002-2006 under the Wisconsin Eco-Potato Collaboration and likely reflect use rates on the remaining potato acreage in the state. The potential for resistance due to the frequent and now widespread use of these

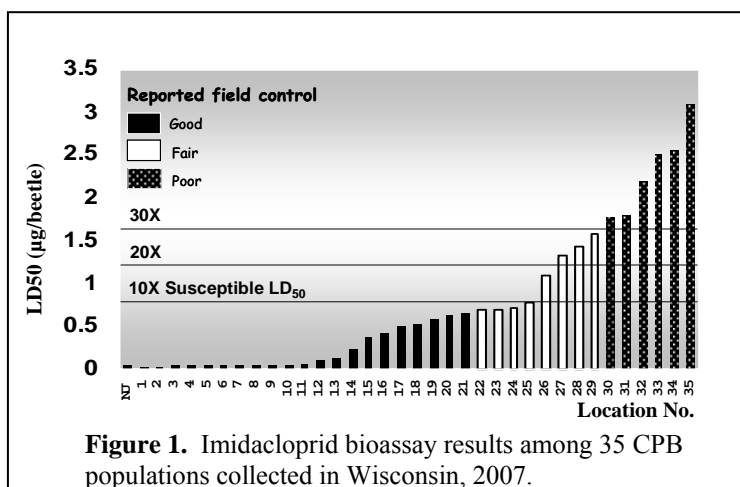
^{1/}Entomology Extension Specialist, 537 Russell Labs, Univ. of Wisconsin-Madison, Madison, WI, 53706 groves@entomology.wisc.edu (608) 262-3229.

^{2/}Associate Research Specialist, 537 Russell Labs, Univ. of Wisconsin-Madison, Madison, WI, 53706 chapman@entomology.wisc.edu (608) 262-9914.

compounds has recently been investigated by our laboratory as well as cooperating scientists at Michigan State University, East Lansing, Michigan. The development of insecticide resistance is one of the key factors which limit the range of available products for controlling insect pests in potato. In Wisconsin, localized resistance by Colorado potato beetle to some organophosphates, carbamates, pyrethroids, and organochlorines has resulted in sporadic control failures. Although this resistance is very discrete in its distribution, it stresses the need for continued vigilance in the management of insecticide resistance and strict adherence to IRM strategies which reduce the likelihood and onset of resistance development. Resistance monitoring among Wisconsin populations of CPB began with limited assays through the interval 2003-2005 in which no detectable increases in product insensitivity were observed. In 2005, notable increases in insensitivity to both imidacloprid and thiamethoxam were observed in two populations in Michigan. Although the magnitude of estimated resistance levels was not suggestive of a total control failure, the emerging, widespread insensitivity to neonicotinoid compounds raises serious concerns. Recent topical bioassays performed on five adult populations in 2006 revealed a single population with measurable insensitivity.

In 2007, we conducted a comprehensive, statewide survey of potential insensitivity to the neonicotinoids, specifically imidacloprid. A letter was sent out in 2007 to request University and Extension personnel, consultants, agrichemical representatives, and growers to identify and send in suspect populations for testing. The response received from the potato growing community was truly excellent. Requests were made of agricultural practitioners representing 1) potato seed production, 2) commercial potato production, 3) Certified Healthy Grown production, and 4) sites where CPB populations were showing some level of insensitivity. Furthermore, adult CPB were collected from a select number of sites (N=5) representing successive, within-season generations to compare changes in insensitivity between overwintered beetles and summer adults.

Briefly, adult bioassays were performed by treatments with a topical application of technical grade insecticide dissolved in acetone, 30 beetles/dose, 5 doses causing between 0 and 100% mortality, plus 30 beetles treated only with acetone. Mortality was assessed after 7 days and data analyzed using log-probit regression analysis. A highly susceptible CPB strain obtained from a New Jersey population was used as a reference control. A subset of adults were also forwarded to cooperators at Michigan State University for confirmatory topical bioassays for direct comparison to reference control populations.



In 2007, a total of 27 overwintered, CPB populations were assayed for imidacloprid sensitivity and an additional 8 populations of summer adult CPB populations were re-sampled for a total of 35 site/season samples (Fig. 1). These samples were distributed among 6 potato growing Counties in Wisconsin including Adams, Columbia, Langlade, Oconto, Portage, and Waushara counties. It is important to note that sites were not selected at random across these most areas. A portion of candidate populations evaluated in this survey were included with the prior knowledge that some were difficult to control with neonicotinoid tools. A total of nine populations were observed with estimated resistance ratios (LD_{50} test population / LD_{50} New Jersey reference population) exceeding 20-fold. Of these, six populations resulted in estimated resistance ratios exceeding 30-fold. Again in 2008, we will reinstitute this survey to

determine what, if any changes, have taken place to measure annual increases in neonicotinoid insensitivity.

Managing Resistance

Resistance develops over time in a pest population as a result of successive external population stressors (e.g. insecticide application(s)) resulting in a physiological or behavioral adaptation that can be passed to subsequent generations. Pest management practitioners then continue to select for these individuals in the population when they fail to discontinue the use same insecticide or materials with very similar modes of action. Key components of a comprehensive insecticide resistance management program should attempt, where possible, to embrace the following recommendations. In turn, it is necessary to avoid the consecutive use of a particular product and/or products which possess similar modes of action.

I. Problem Identification: If you suspect resistance, first eliminate other possible causes. In many instances, lack of control can be attributed to application error, equipment failure, or less-than-optimal environmental conditions. If these possibilities have been ruled out, work with local agricultural advisors and the manufacturer to confirm actual resistance to the compound applied. In the event of a control failure due to resistance, do not repeat the application with an insecticide of the same chemical class and consult a pest control advisor or area extension specialist for up-to-date recommendations and advice on IPM and IRM options.

II. Product Rotation: Avoid the consecutive use of a single product, or multiple products with similar modes of action. To assist farmers, growers, advisors, extension staff, consultants and crop protection professionals, the Insecticide Resistance Action Committee (IRAC) has developed and updates a Mode of Action (MoA) classification system with a guide to the selection of insecticides or acaricides in an effective and sustainable insecticide or acaricide resistance management (IRM) strategy (Table 1). This program was the direct result of the EPA's voluntary pesticide labeling proposal (2001) for all registered insecticides to include the MoA in a particular numbered group. To implement an effective IRM program, growers can consult this information and select a sequence of insecticides that represent different groups with unique MoA's.

III. Cultural Control(s): Where possible, consider selecting early-maturing or pest-tolerant varieties of crop plants. Adopt all non-chemical techniques known to control or suppress pest populations, including crop rotation in particular for managing resistance with the Colorado potato beetle. Specifically, potato crops should be rotated > 400 m (¼ mile) away from a previous potato crop. It has been well documented that overwintering adult Colorado potato beetles disperse only short distances after emergence from the soil.

IV. Preserve Natural Control(s): Where possible, select insecticides and other pest management tools which preserve beneficial insects. Natural mortality factors other than insecticides can significantly delay the onset and development of resistance. The use of selective insecticides (e.g., spinosad, *Bacillus thuringiensis*) and the selective, well-timed use of low-dose strategies are feasible for specific pest targets.

V. Pest Surveillance and Scouting: Monitor the pest population during the growing season. Regularly monitor fields to identify pests and natural enemies, estimate insect populations and track stage of development. Insecticides and acaricides generally should be used only if insect counts exceed action thresholds or the point where economic losses exceed the costs of insecticide plus application. Time applications against the most susceptible life stages to gain maximum benefit from the product. Where larval stages are being controlled, target younger larval instars where possible because these are usually much more susceptible and therefore much more effectively controlled by insecticides than older stages

VI. Rates and Spray Intervals: Use insecticides at labeled rates and follow prescribed spray intervals. Do not reduce or increase rates from manufacturer recommendations as this can hasten resistance development. Use products at their full, recommended doses. Reduced (sub-lethal) doses quickly select populations with average levels of tolerance, whereas doses that are too high may impose excessive selection pressures.

VII. Product Application: Appropriate, well-maintained equipment should be used to apply insecticides. Recommended water volumes, spray pressures and optimal temperatures should be used to obtain optimal coverage. Sprayer nozzles should be checked for blockage and wear, and be able to handle pressure adequate for good coverage. Spray equipment should be properly calibrated and checked on a regular basis. Use application volumes and techniques recommended by the manufacturers and local advisors.

VIII. Tank Mixes: Mixtures may offer a short-term solution to resistance problems, but it is essential to ensure that each component of a mixture belongs to a different insecticide mode of action class, and that each component is used at its full rate. Under certain circumstances, tank mix different chemicals for improved or broader spectrum pest control. It is equally crucial that compounds should persist on the crop or surface for similar periods in order to expose insects to both modes of action for the same length of time. Use of multiple products of the same mode of action in the spray tank will do little more than using an increased rate of a single compound of the same chemical class.

IX. Resistance Monitoring: Consideration should be given to monitoring for the incidence of resistance in the most commercially important situations and gauge levels of control obtained. Developing baseline susceptibility information for a particular population will provide a basis upon which further decisions can be made to extend the functional life of pest control products and minimize the onset of resistance.

Table 1. Insecticide Resistance Action Committee Mode of Action Classification, May 2004.

Group	Sub-group	Primary Target Site of Action	Chemical Sub-Group or Active Ingredient (product names)
1 *	A	Acetylcholine esterase inhibitors	Carbamates (Temik, Vydate, Lannate, Sevin, Furadan)
	B		Organophosphates (Dimethoate, Diazinon, Di-Syston, Mocap, Malathion, Methyl Parathion, PennCap-M, Thimet, Phorate, Monitor, Guthion, Imidan/Phosmet)
2 *	A	GABA-gated chloride channel antagonists	Cyclodiene organochlorines (Phaser, Endosulfan, Thiodan)
	B		Fipronil
3		Sodium channel modulators	Pyrethroids, Pyrethrins, Esfenvalerate (Asana, Ambush, Baythroid, Delta Gold, Leverage, Pounce, etc)
4 *	A	Nicotinic Acetylcholine receptor agonists / antagonists	Neonicotinoids (Platinum, Admire, Assail, Cruiser, Gaucho, Genesis, Leverage, Actara, Provado)
	B		Nicotine
	C		Cartap, Bensultap
5		Nicotinic Acetylcholine receptor agonists (not group 4)	Spinosyns (Success, SpinTor, Entrust) Spinetoram (Radiant)
6		Chloride channel activators	Avermectins, Milbemycins (Agri-mek)
7 *	A	Juvenile hormone mimics	Juvenile hormone analogues
	B		Fenoxycarb
	C		Pyriproxyfen
8 *	A	Compounds of unknown or non-specific mode of action (fumigants)	Methyl bromide
	B		Aluminium phosphide
	C		Sulfuryl fluoride
9 *	A	Compounds of unknown or non-specific mode of action (selective feeding blockers)	Prokil Cryolite (Kryocide)
	B		Pymetrozine (Fulfill)
	C		Flonicamid (Turbine)
10 *	A	Compounds of unknown or non-specific mode of action (mite growth inhibitors)	Clofentezine, Hexythiazox
	B		Etoxazole
11 *	A1	Microbial disruptors of insect midgut membranes (includes transgenic crops expressing <i>Bacillus thuringiensis</i> toxins)	<i>Bacillus thuringiensis</i> var. <i>israelensis</i>
	A2		<i>Bacillus thuringiensis</i> var. <i>sphaericus</i>
	B1		<i>Bacillus thuringiensis</i> var. <i>aizawai</i>
	B2		<i>Bacillus thuringiensis</i> var. <i>kurstaki</i> (Agree, Biobit, Dipel, Javelin, Lepinox)
	C		<i>Bacillus thuringiensis</i> var. <i>tenebrionensis</i> (Novodor)
12 *	A	Inhibitors of oxidative phosphorylation, disruptors of ATP formation	Diafenthiuron
	B		Organotin miticides
13		Uncoupler of oxidative phosphorylation via disruption of H ⁺ proton gradient	Chlorfenapyr, DNOC
14		Inhibition of magnesium-stimulated ATPase	Propargite (Comite, Omite)
15		Inhibitors of chitin biosynthesis, type 0, Lepidopteran	Benzoylureas Novaluron (Rimon)
16		Inhibitors of chitin biosynthesis, type 1, Homopteran	Buprofezin
17		Inhibitors of chitin biosynthesis, type 2, Dipteran	Cyromazine
18		Ecdysone agonist / disruptor	Diacylhydrazines
19		Octopaminergic agonist	Amitraz
20		Site II electron transport inhibitors	Hydramethylnon, Dicofol
21		Site I electron transport inhibitors	METI acaricides, Rotenone
22		Voltage-dependent sodium channel blocker	Indoxacarb (Avaunt)
23		Inhibitors of lipid synthesis	Tetronic acid derivatives
24		Site III electron transport inhibitors	Acequinocyl, Flucyprym
25		Neuroactive (unknown mode of action)	Bifenazate (Acramite)
26		Unknown mode of action	Azadirachtin (Azadirect, Ecozin)