MAGGOT MANIA!

An OSU Extension Newsletter about Cabbage Maggot Management

Pesticide Resistance | Dept. of Horticulture MagNet Project | Summer 2005-Issue 4

Are cabbage maggots in the Willamette Valley resistant to Lorsban?
How to battle the SuperPests !
Resistance or Not???

Lorsban, an organophosphate pesticide (OP), is currently the most widely used insecticide for controlling cabbage maggot (CM) in the Pacific Northwest. In spring 2005, CM could not be controlled in the northern Valley after using scheduled applications of Lorsban. Many Brassica fields, turnips and rutabagas, had greater than 80% losses due to maggots. The cabbage maggot is proving to be a major and devastating pest problem and has contributed greatly to increases in crop damage since 2001 (**Fig. 1**).

Many questions arise as to why CM could not be controlled with Lorsban such as,

- Was it just a bad year with the right environmental conditions to support CM populations?
- Is the application method not targeting CM eggs adequately?
- Could we be seeing Lorsban-resistant maggot populations? In 2003, CM larvae from BC, Canada were sent to a laboratory for resistance-testing and results concluded that the larvae were 10X resistant to Lorsban (Zimmerman, BC Ministry of Agriculture, personal communication 2004).

Pesticide resistance can greatly affect a grower's ability to produce a crop and control economically-damaging pests.

This newsletter explains pest resistance, how resistance works, and how we might reduce the risks of pest resistance.

In 1995, high crop losses due to the tobacco budworm devastated the cotton growers in the southeast US. In spite of attempts of spraying insecticides and combinations of insecticides nothing could control the budworm. After testing, scientists found the budworms to have high tolerances and resistance to these insecticides (Graves et al. 1993).

Lorsban (chlorpyrifos) has been a most effective and reliable soil insecticide for onion maggot control since it was labeled for that use in the early 80's. In 1987, Lorsban appeared to be slightly less effective in a few fields in western New York. …By 1993, 20% of New York's commercial onion acreage had detectable levels of Lorsban resistance in resident onion maggot populations." (1995 www.nysaes.cornell.edu/pubs/fls/OCRPDF/144.pdf)

What is pest resistance?

Pest resistance is the ability of an insect to survive exposure to a lethal concentration of pesticide due to their behavior or genetics. Chemical field rates (specified on the labels) are determined to kill ~100% of the population and insects that survive may be resistant. Resistant insects have some genetic or behavioral variation compared to the insect population as a whole that allows them to survive pesticide applications. While insects without this genetic variation will die, insects with this genetic variation survive and reproduce, increasing the percentage of insects in the population with the resistant genes. **Fig. 2** (next page) is a good visual explaining how pest resistance accumulates in a pest population over several generations of pest suppression.

How do insects develop resistance?

There are several clever adaptation strategies utilized by insects to become resistant to and escape from insecticides. Here are a few:

<u>Behavioral Resistance</u>: Resistant insects might have the ability to detect or recognize a danger and avoid the toxic chemical that is being used on them. Insects may leave an area or stop feeding when spraying occurs. Depending on the pesticide an insect may also protect itself by simply

Fig. 1. Seasonal assessment of cabbage maggot damage in root crops (turnips and rutabagas) conventionally-treated with Lorsban 4E. The 2005 data set is incomplete, fall harvests are not included.

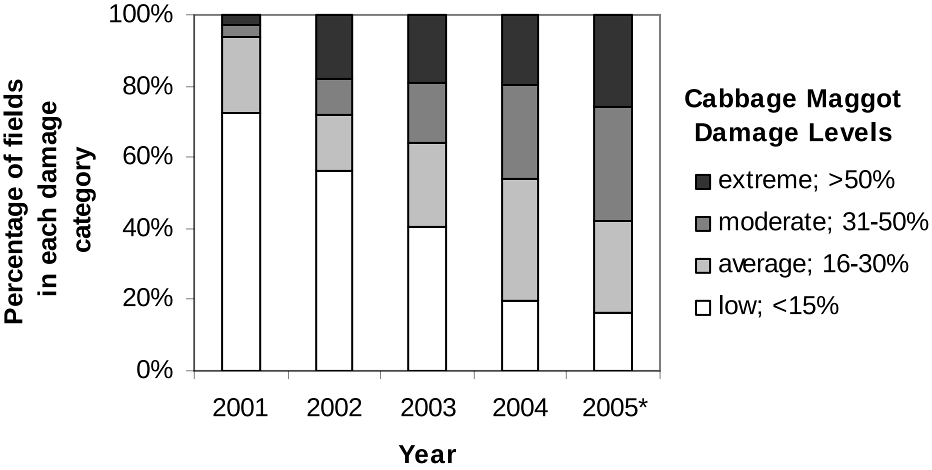
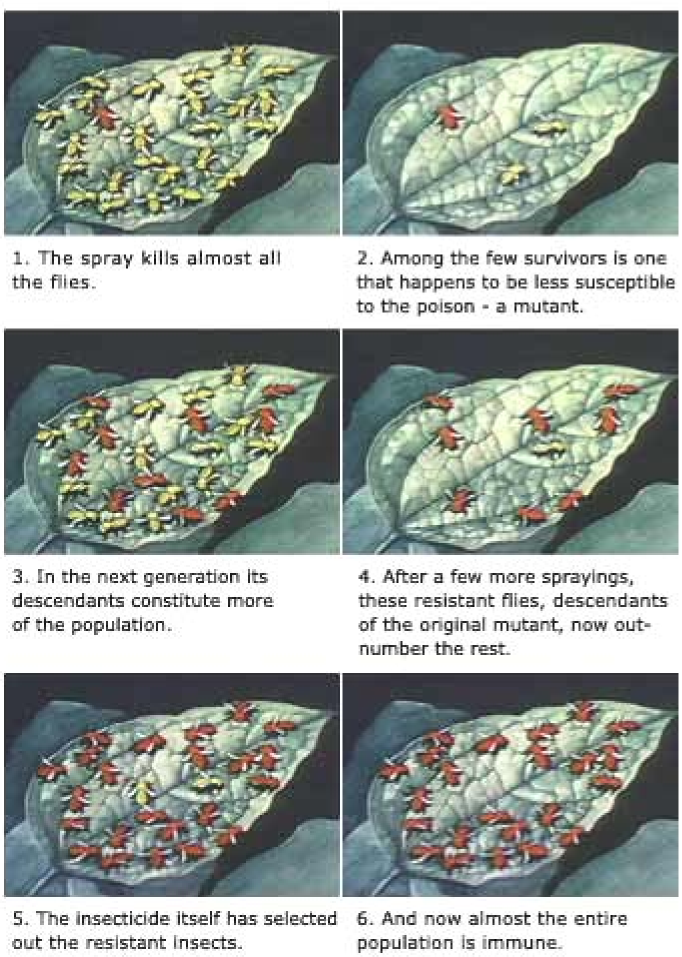


Fig. 2. A visual explaining how pesticide resistance works. Source: http://www.pbs.org/wgbh/evolution/library/10/1/l_101_02.html



moving underneath a leaf or deeper into the crop canopy.

Barrier Resistance: Resistant insects may absorb the toxic chemical slower than non-resistant insects due to a thicker outer skin that creates some form of barrier. The quantity of toxin reaching the “blood stream” may be insufficient to harm the insect and allow the insect to survive.

Biochemical Resistance: Resistant insects can break down or metabolize a toxic chemical that normally would be fatal. The resistant insects “may produce large amounts of enzymes, such as esterases, which either break down or metabolize the insecticide molecule or bind to it so tightly that it cannot function (a process known as sequestration).” For example, the green peach aphid, *Myzus persicae*, has a biochemical resistance to pyrethroids (Aldridge 2005).

Altered Target-site Resistance: Resistant insects can modify the target site where the chemical toxin would normally bind to, making the toxin ineffective. The target site for organophosphates and carbamates is acetylcholinesterase. Maggot populations in Canada altered the target site that Lorsban normally binds to and this enabled them to be resistant to chemical applications.

How long does it take insects to become resistant?

The introduction of synthetic chemicals for use in agriculture in the 1940's put enormous selective pressure on insect pests. Pests can develop resistance to synthetic chemicals, just like viruses and bacteria in humans can develop resistance to antibiotics. Some insects take only a few years to become resistant while others take 40-50 years (See **Table 1**). As with most natural processes, an insects' ability to develop pesticide resistance depends on many variables. History and research have shown that no single pesticide or IPM tool can escape resistance forever. Below are SOME FACTORS THAT INCREASE THE RATE OF PEST RESISTANCE:

- Insect species with high reproduction potential (produce lots of young every generation). For example: aphids, fruit flies.
- Insect species with a short time period between generations having multiple generations every year.
- Insect populations with naturally high genetic variability. When there is high genetic variability within a population there is an increased possibility that some individual insects are already resistant to the pesticide and their numbers just need to be increased.
- Frequent applications of non-persistent chemicals. For example: Malathion
- Lack of non-exposed susceptible insect pests or little immigration from other insect pest populations that can breed with resistant pests therefore reversing selective evolution towards a resistant population.

Table 1. Here are a few chemicals and chemical families and the number of years after introduction that resistance was detected.

Chemical	Year Introduced	Year Resistant	Resistant Pests
Bacillus thuringiensis (B.t.)	1961	1989 1993	Diamondback moth (*Plutella xylstella*) in vegetable crops Tobacco budworm (*Heliothis virescens*) in cotton plus 15 other species
Chlorpyrifos	1966	1993 2003	Onion maggot (*Delia antiqua*) Cabbage maggot (*Delia radicum* in Canada)
Cyromazine (Insect Growth Regulator)	1979	No resistance yet?	Australian sheep blowfly (*Lucilia cuprina*)
DDT	1942	1946 1959 1966	House flies (Denmark) Mosquitoes (*Culex and Anopheles* sp.; India) Diamondback moth (*Plutella xylostella* (L.))
Dieldrine	1955	1958	Australian sheep blowfly (*Lucilia cuprina*)
Malathion	1965	1968	Australian sheep blowfly (*Lucilia cuprina*) Mosquitoes
Organophosphates (OP's)	Various yrs	1991	Houseflies
Pyrethroid (Cypermethrin)	1977	1986	Cotton bollworm (*Helicoverpa armigera)*
71 Chemicals	Various yrs	Various yrs	Green peach aphid (*Myzus persicae)*

How can a grower reduce the risk of pest resistance?

1) **ROTATE CHEMICALS:** When selecting pesticides to control insects, choose different pesticide families (with different mode of actions or metabolic pathways) if you are controlling the same pest over a long period of time. Sometimes cross-resistance can be a problem when pesticides from different families are metabolized by the same enzyme system or have the same target site. Research has shown that it is best to use different chemicals over a period longer than a single pest generation. Thus, when an insecticide must be used, it is probably best to use the same insecticide over a 1 to 2 month period rather than weeks before switching to another insecticide with a different mode of action. (See **Table 2** (next page) for list of main chemical families and their modes of action).
2) **AVOID TANK MIXES:** When using tank mixes, chemicals must have different modes of action and very short degradation times. Tank mixes should be avoided due to the danger that resistance will occur even more rapidly to the mixtures than to rotations of chemical families.
3) **CONSIDER APPLICATION METHOD:** Provided that they are used only sporadically, fogs are generally less likely to select for resistance than overhead sprays, and overhead sprays are less likely to select for resistance than soil drenches of the same insecticide. In addition, if you treat "hot" pest areas instead of broadcast spraying an entire field this decreases resistance.
4) **CHOOSE LOWER DOSAGES:** Lower dose applications are less likely to select gene resistance because often susceptible genotypes insects are preserved.
5) **CREATE REFUGE:** Keep a refuge of susceptible insects to dilute resistant population.
6) **MONITOR PESTS:** Scouting and monitoring pests is a good way to

stay on top of pest abundance and development. Research has shown that many insect population flight and egg layers can be predicted by using either soil or air temperatures. (See Maggot Degree-Day Model in the fall issue of MagNet Mania. Learn how to identify low and high pest risk periods.

7) **RECORD PESTICIDE USE:** Adequate record keeping of pesticide use is critical when knowing how to rotate pesticides over years to minimize resistance development.

8) **REDUCE PESTICIDE USE:** Last but not least, the most effective method to lessen the risk of pest resistance to insecticides is by reducing the usage of pesticides. Resistance may develop from excessive use. Avoid unnecessary spraying. The less an insect population is exposed to a chemical the less likely it will select for resistant traits. The unique and most valuable property of pesticides is that insecticide are fast acting and reliably effective. Proper application procedures to effectively target the pest of interest is important. As such, they should be held in reserve for use only when IPM strategies fail.

Some References:

Aldridge 2005, Insecticide resistance: from mechanisms to management on website: http://www.absw.org.uk/Briefings/insecticide_resistance.htm

Insect Resistance in Green Peach Aphids: http://www.hortnet.co.nz/publications/nzpps/resistance/peachaphid.htm and http://www.biology.mcgill.ca/Phytotron/ipm_protocols.htm#Aphids

IRAC Mode of Action at: http://www.irac-online.org/documents/moa/moa.pdf

Know how a pesticide works, so you don't double up on the same chemical family. Rotating pesticides is ineffective if all the chemicals activate the same resistant mechanism. Rotate pesticides with different modes of action.

Table 2. Some main chemical families and their mode of action.

CHEMICAL FAMILIES	ACTIVE INGREDIENTS (BRAND NAMES)	What is the chemical's MODE OF ACTION or Target Site
Organophosphates Carbamates	Chlorpyrifos (Lorsban, Pilot, Vexter, etc.) Diazinon (Cekuzinon, Basudin, Terminator, etc.) Malathion (Rion, Atrapa, Zithol, etc.) Carbaryl (Sevin and Thiodan) Methomyl (Lannate)	Inhibits an enzyme called Acetylcholine esterase, which is vital to the nervous system of insects.
DDT Pyrethroids Pyrethrins	DDT Esfenvalerate (Conquer, Halmark, Mandrin, Mustang, etc.) Pyrethrins (Pronto, Rid, etc.)	Interferes with the nervous system by opening Na+ gate in the nerve membrane.
Cyclodiene organochlorines Fipronil or Phenylpyrazole	Chlorodane Fipronil (Regent)	Affects the GABA-gated chloride channel used in the nervous system of insects.
Neonicotinoids	Imidacloprid (Admire, Confidor, Impower, etc.)	Excites the insect's nervous system by targeting the insect's nicotinic acetylcholine receptor agonists / antagonists, leading to involuntary muscle contractions, prostration with tremors, and paralysis.
Spinosyns (bacterial by-products)	Spinosad (Success, Entrust, Tracer, etc.)	Excites the insect's nervous system targeting the nicotinic acetylcholine receptor agonists, leading to involuntary muscle contractions, prostration with tremors, and paralysis.
Bacterial endotoxin	B.t. (*Bacillus thunengiensis;* Thuracide, Dipel, etc.)	Endotoxin binds to the midgut epithelial cells, creating pores in the cell membranes and immobilizes the insect's gut. Bacteria spores germinate and invade the host.

Development of resistance to existing chemical products drives the need for new methods and integrated practices to control insect pests. Stay tuned for the Degree-Day Modeling newsletter to help predict cabbage maggot activity in a field.