CERTIFIED CROP ADVISOR (CCA) RESISTANCE MANAGEMENT STUDY GUIDE

MANAGE RESISTANCE Now

Protect your land, one field at a time

CCA Resistance Management Study Guide

WRITERS

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ACKNOWLEDGEMENTS

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Table of Contents

Organization acronyms used in this Guide:

IRAC: Insecticide Resistance Action Committee

FRAC: Fungicide Resistance Action Committee

HRAC: Herbicide Resistance Action Committee

WSSA: Weed Science Society of America

PROFICIENCY AREA I
EVOLUTION OF RESISTANCE

COMPETENCY AREA 1

Development of Resistance

The development of resistance to a pesticide (e.g., insecticide, fungicide or herbicide) is recognized as a decrease or failure in the effectiveness of control of a pest (e.g., insects, fungi, pathogens, weeds, etc.) following its exposure to the appropriate chemical used according to label recommendation. Any pest resistance to an agricultural pesticide is the result of genetic mutation and selection. The change in the pest genetic code (genotype) can manifest itself in different ways (phenotypes) in order to make the pesticide harmless (these mechanisms of resistance are discussed in section 1.c. below). When encountered in the field, pesticide resistance will appear as a measurable reduction in the efficacy of the affected pesticide. Once acquired in a pest population, resistance becomes transmissible genetically; it is a heritable change.

1. Discuss the biology of resistance evolution:

a) Selection pressure;
b) Genetics of resistance;
c) Mechanisms of resistance;
d) Genetic diversity of the target species.

a) Selection pressure

Natural selection associated with the use of pesticides allows initially very rare, naturally occurring, pre-adapted pests bearing resistance genes to survive and pass the resistance trait onto their offspring. Repeated applications of a pesticide with the same mechanism of action, however, select for the resistant individuals and cause their number to increase in the population. This is done at the expense of susceptible individuals that are eliminated by the pesticide. Under this selection pressure, resistant individuals eventually outnumber susceptible ones and the pesticide becomes no longer effective.

Three main factors contribute to the intensity of selection pressure for pesticide resistance: 1) the pesticide effectiveness; 2) its duration after application; and 3) the frequency of use. The efficiency of a pesticide depends on the target site and the relative mortality in the populations caused by the use of the pesticide at a specific dose. In weeds, the effect on the reduction in seed production is also taken into account. The 'duration of the pesticide' is a measure of the time during which the pesticide is toxic to a specific pest. Moreover, the efficacy and duration of an applied pesticide in the field are, in turn, influenced by the region, season and climatic conditions. The 'frequency of use' relates to the number of times the pesticide was sprayed in the field within one growing season and/or the number of years used on the same field.

Note of interest: Pesticide effectiveness is a double-edge sword. This means the more efficient a pesticide is at controlling the pest the 'better' it will be at selecting for resistance. On the other hand, a more effective pesticide is more likely to be used again and again by growers, compared to a pesticide with moderate efficacy. In a way, efficient pesticides are responsible for their own demise; the same factors that make us want to use them more also may render them less effective in the long term.

Resistance is not a new phenomenon. Accounts of pesticides losing their efficacy surfaced shortly after some of the products were introduced to the market. Tables 1 and 2 provide a snapshot of the status of herbicide and insecticide resistance in the province of Ontario. It is apparent, that cases of resistance appeared very early and continue to do so; a variety of modes of actions and a range of pests are affected. This problem affects both field and horticultural crops.

Table 1: State of herbicide resistance in Ontario in 2018

WSSA GROUP	Herbicide mode of action	Weed species		Year reported	Main crops affected
		Scientific name	Common name		
1	ACCase inhibitors	*Avena sativa*	Wild oats	2005	Cereals
		Digitaria sanguinalis	Large crabgrass	2011	Onions
2	ALS inhibitors	*Amaranthus powellii*	Green pigweed	1998	Soybeans; corn
		Amaranthus retroflexus	Redroot pigweed	1998	Soybeans; corn
		Solanum ptychanthum	Eastern-black nightshade	2000	Soybeans; corn
		Ambrosia artemisiifolia	Common ragweed	2000	Soybeans; corn
		Chenopodium album	Common lamb's-quarters	2001	Soybeans; corn
		Setaria viridis	Green foxtail	2001	Soybeans; corn
		Amaranthus tuberculatus (=A. rudis)	Tall waterhemp	2002	Soybeans; corn
		Setaria faberi	Giant foxtail	2003	Soybeans; corn
4	Synthetic auxins	*Daucus carota*	Wild carrot	1957	Roadsides

Table 1. State of herbicide resistance in Ontario in 2018

(continued)

WSSA GROUP	Herbicide mode of action	Weed species		Year reported	Main crops affected
		Scientific name	Common name		
5	Photosystem II inhibitors (Triazines)	*Chenopodium album*	Common lamb's-quarters	1973	Corn
		Ambrosia artemisiifolia	Common ragweed	1976	Corn
		Chenopodium strictum	Late-flowering goosefoot	1976	Corn
		Amaranthus powellii	Green pigweed	1977	Corn
		Senecio vulgaris	Common groundsel	1977	Corn
		Amaranthus retroflexus	Redroot pigweed	1980	Corn
		Echinochloa crus-galli var. *crus-galli*	Barnyardgrass	1981	Corn
		Panicum capillare	Witchgrass	1981	Corn
		Setaria pumila	Yellow foxtail	1981	Corn
		Sinapis arvensis	Wild mustard	1983	Corn
6	PSII inhibitors (Nitriles)	*Amaranthus hybridus*	Smooth pigweed	2004	Seed corn
		Amaranthus retroflexus	Redroot pigweed	2005	Seed corn
7	PSII inhibitor (Ureas)	*Amaranthus retroflexus*	Redroot pigweed	2001	Carrots
9	EPSP synthase inhibitors	*Ambrosia trifida*	Giant ragweed	2008	Soybeans; corn
		Conyza canadensis	Canada fleabane	2010	Soybeans; corn
14	PPO inhibitors	*Amaranthus tuberculatus*	Waterhemp	2018	Soybeans
22	PSI Electron Diverter	*Conyza canadensis*	Canada fleabane	1993	Orchards
		Lepidium virginicum	Virginia pepperweed	1993	Orchards
		Solanum ptychanthum	Eastern-black nightshade	2009	Blueberries

Table 1. State of herbicide resistance in Ontario in 2018

(continued)

WSSA GROUP	Herbicide mode of action	Weed species: Scientific name	Weed species: Common name	Year reported	Main crops affected
Multiple Resistance Two or more resistant sites of action in the same biotype					
2 & 5	ALS inhibitors; Photosystem II inhibitors	*Amaranthus powellii*	Green pigweed	1998	Soybeans; corn
		Amaranthus tuberculatus	Tall waterhemp	2002	Soybeans; corn
2 & 9	ALS inhibitors; EPSP synthase inhibitors	*Ambrosia trifida*	Giant ragweed	2011	Soybeans; corn
		Conyza canadensis	Canada fleabane	2011	Soybeans; corn
		Ambrosia artemisiifolia	Common ragweed	2012	Soybeans; corn
		Amaranthus tuberculatus	Tall waterhemp	2014	Soybeans; corn
5 & 7	Photosystem II inhibitors (Triazines and Ureas)	*Amaranthus powellii*	Green pigweed	1999	Seed corn
2, 5 & 9	ALS inhibitors; Photosystem II inhibitors; EPSP synthase inhibitors	*Amaranthus tuberculatus*	Tall waterhemp	2014	Soybeans
2, 5, 9 & 14	ALS inhibitors; Photosystem II inhibitors; EPSP synthase inhibitors;	*Amaranthus tuberculatus*	Tall waterhemp	2018	Soybeans

Adapted from Heap, I. The International Survey of Herbicide Resistant Weeds. Online. Internet. Wednesday, June 6, 2018. Available at www.weedscience.org

Table 2: Cases of insecticide resistance in Ontario affecting field and horticultural crops

Insect scientific and common names	Crop	Year	Insecticide	IRAC Group
Argyrotaenia velutinana Red-banded leafroller	Apple	1962	DDT	4A
		1965	TDE	2A
Cydia pomonella Codling moth	Fruit trees	1960	DDT	4A
		2008	Methoxyfenozide	18 *
Delia antiqua Onion maggot	Vegetables	1959	BHC/cyclodienes	2A
		1965	Aldrin	2A
		1972	Chlordane	2A
		1976	Carbofuran	1A
		1976	Chlorfenvinphos	1B
Delia brassicae Cabbage root-fly	Crucifers	1965	Aldrin	2A
			BHC/cyclodienes	2A
			Dieldrin	2A
			Heptachlor	2A
Delia florilega Onion fly	Beans	1961	Aldrin	2A
			Dieldrin	2A
Delia platura Seed corn maggot	Cereals, grains, vegetables	1962	BHC/cyclodienes	2A
		1965	Aldrin	2A
			Dieldrin	2A
			Lindane	N/A
		1972	Chlordane	2A
Euxesta notata Marked spotted-wing fly	Onions	1962	BHC/cyclodienes	2A
			DDT	3B
Euxoa detersa Sandhill cutworm	Corn	1965	BHC/cyclodienes	2A

Table 2: Cases of insecticide resistance in Ontario affecting field and horticultural crops

(continued)

Insect scientific and common names	Crop	Year	Insecticide	IRAC Group
Euxoa messoria Darksided cutworm	Corn	1965	Aldrin	2A
			Dieldrin	2A
			Heptachlor	2A
Euxoa ochrogaster Red-backed cutworm	Cereals, canola, vegetables, sunflower, alfalfa,	1976	DDT	4A
			Endrin	2A
			Methoxychlor	3B
Euxoa scandens White cutworm		1977	BHC/cyclodienes	2A
Helicoverpa armigera Corn earworm	Corn, sorghum, tomato	1974	Azinphos-methyl	1B
			Carbaryl	1A
Leptinotarsa decimlineata Colorado potato beetle	Eggplant, pepper, potato, tomato	1976	Aldrin	2A
			DDT	4A
			Deltamethrin	3A
			Dieldrin	2A
			Endosulfan	2A
		1984	Cypermethrin	3A
			Endrin	2A
			Fenvalerate	3A
		2008	Imidacloprid	4A †
Liriomyza trifolii American serpentine leafminer	Greenhouse ornamentals and vegetables	1989	Chlorpyrifos	1B
			Demeton	1B
			Pyrazophos	N/A

Table 2: Cases of insecticide resistance in Ontario affecting field and horticultural crops

(continued)

Insect scientific and common names	Crop	Year	Insecticide	IRAC Group
Lissorhoptrus oregonensis Rice water weevil	Carrot	1975	BHC/cyclodienes	2A
Myzus persicae Green peach aphid	Flower, crops, fruit, trees, grains, tobacco, vegetables	1974	Azinphos-methyl	1B
			Malathion	1B
			Naled	1B
			Parathion	1B
Panonychus ulmi European red mite	Fruit trees	1959	Organophosphates	1B
		1973	Tetradifon	12D
		1974	Azinphos-methyl	1B
			Demeton	1B
			Dicrotophos	1B
			Ethion	1B
			Fenthion	1B
			Malathion	1B
			Mevinphos	1B
			Parathion	1B
			Phosphamidon	1B
		1987	Cyhexatin	12B
		1989	Dicofol	UN
Phyllonorycter blancardella Spotted tentiform leafminer	Apple	1980	Azinphos-methyl	1B
		1986	DDT	4A
			Tau-Fluvalinate	3A
		1990	Methomyl	1B
			Oxamyl	1A

Table 2: Cases of insecticide resistance in Ontario affecting field and horticultural crops

(continued)

Insect scientific and common names	Crop	Year	Insecticide	IRAC Group
Psilia rosae Carrot rust fly	Carrot, celery, parsley, parsnip	1962	BHC/cyclodienes	2A
Psylla pyricola Pear psylla	Pear	1965	Parathion	1B
Sitophilus granarius Granary weevil	Stored grain	1972	Ethylene dibromide	8A
			Methyl bromide	8A
Striacosta albicosta Western bean cutworm	Corn	2017	Bt protein Cry1F	11A ‡
Tetranychus urticae Two-spotted spider mite	Cotton, fruits, vegetables, walnut, ornamentals	1965	Parathion	1B
		1957	Organophosphates	1B
Thrips palmi Melon thrips	Melon	2005	Diazinon	1B
Thrips tabaci Onion thrips	Onion	2005	Cyhalothrin-lambda	3A
			Deltamethrin	3A
			Diazinon	1B
Tribolium castaneum Red flour beetle	Stored grain, peanuts, sorghum	1984	Malathion	1B
Tribolium confusum Confused flour beetle	Stored grain	1984	Malathion	1B
Trichoplusia ni Cabbage looper	Crucifers	1965	DDT	4A

Data as reported to the Arthropod Pesticide Resistance Database maintained by the University of Michigan (https://www.pesticideresistance.org/index.php) except where indicated:

* *Grigg-McGuffin, K., Scott, I. M., Bellerose, S., Chouinard, G., Cormier, D., & Scott-Dupree, C. (2015). Susceptibility in field populations of codling moth,* Cydia pomonella *(L.)(Lepidoptera: Tortricidae), in Ontario and Quebec apple orchards to a selection of insecticides. Pest Management Science, 71, 234-242.*

† *Scott, I. M., Tolman, J. H., & MacArthur, D. C. (2015). Insecticide resistance and cross-resistance development in Colorado potato beetle* Leptinotarsa decemlineata *Say (Coleoptera: Chrysomelidae) populations in Canada 2008–2011. Pest Management Science, 71, 712-721.*

‡ *Smith, J. L., Lepping, M. D., Rule, D. M., Farhan, Y., & Schaafsma, A. W. (2017). Evidence for field-evolved resistance of* Striacosta albicosta *(Lepidoptera: Noctuidae) to Cry1F* Bacillus thuringiensis *protein and transgenic corn hybrids in Ontario, Canada. Journal of Economic Entomology, 110, 2217-2228.*

b) Genetics of resistance

The ensemble of genes expressed throughout an organism is the blueprint of when, where and what proteins will be made in each part of this organism. This organism could be a plant, an insect, a fungi or any life present on earth. The genes that form an individual in a species can vary slightly within the species. One of the possible forms of a gene (trait) is called an allele while the phenotype is the physical manifestation of this allele. In general, most genes have two alleles for a given trait. These alleles can be either strong (dominant) or weak (recessive) and are inherited from the parents during sexual reproduction. Homozygous indicates having identical alleles for a single trait. Two dominant alleles are called homozygous dominant and the associated trait is a dominant phenotype. On the other hand, an individual with two recessive alleles is called homozygous recessive and the associated trait is a recessive phenotype. Heterozygous means that the individual has one copy of each allele. In pesticide resistance literature, an allele for resistance is often indicated by R while an allele for susceptibility is indicated by S. In addition, the R allele is most often dominant. If one allele is completely dominant, the phenotype of the heterozygote (RS) will be the same as the phenotype of the homozygote dominant (RR). If the allele is incompletely dominant, the heterozygote demonstrates a phenotype that is intermediate between the homozygote resistant (RR) and the homozygote susceptible (SS). If the resistance allele is recessive, then only the homozygote resistant (RR) individuals would survive and the heterozygotes would have the same response as the homozygous susceptible (SS). In Figure 1, we illustrate the scenario of what happens to the offspring of a butterfly with a dominant allele when it reproduces with an individual with recessive trait. Figure 2 is an example of two heterozygotes reproducing together.

Figure 1

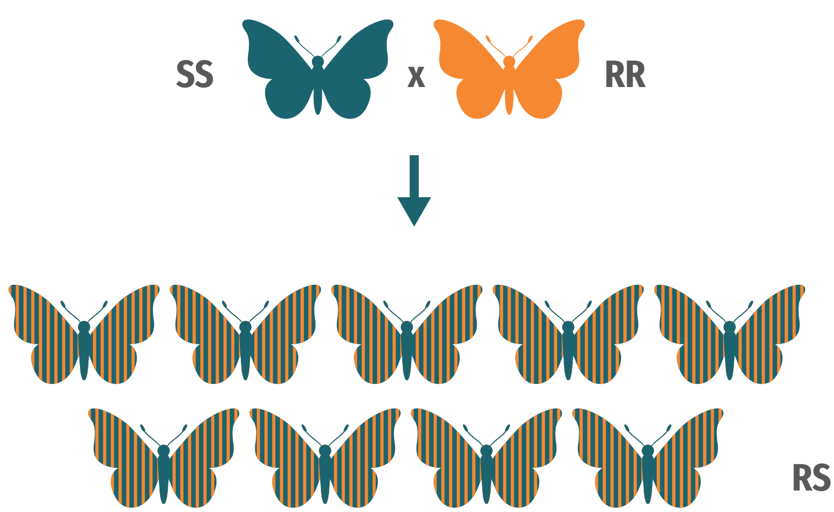

Figure 1: Illustration of the fate of a resistance allele after a homozygous resistant individual (RR) mates with a homozygous susceptible (SS). The progeny inherits one allele from each parent and they are all heterozygous (RS). If the R allele is dominant, the RS individuals will have the same level of resistance as the RR parent; in the case of a semi-dominant allele, the RS individuals will have an intermediate response.

Figure 2

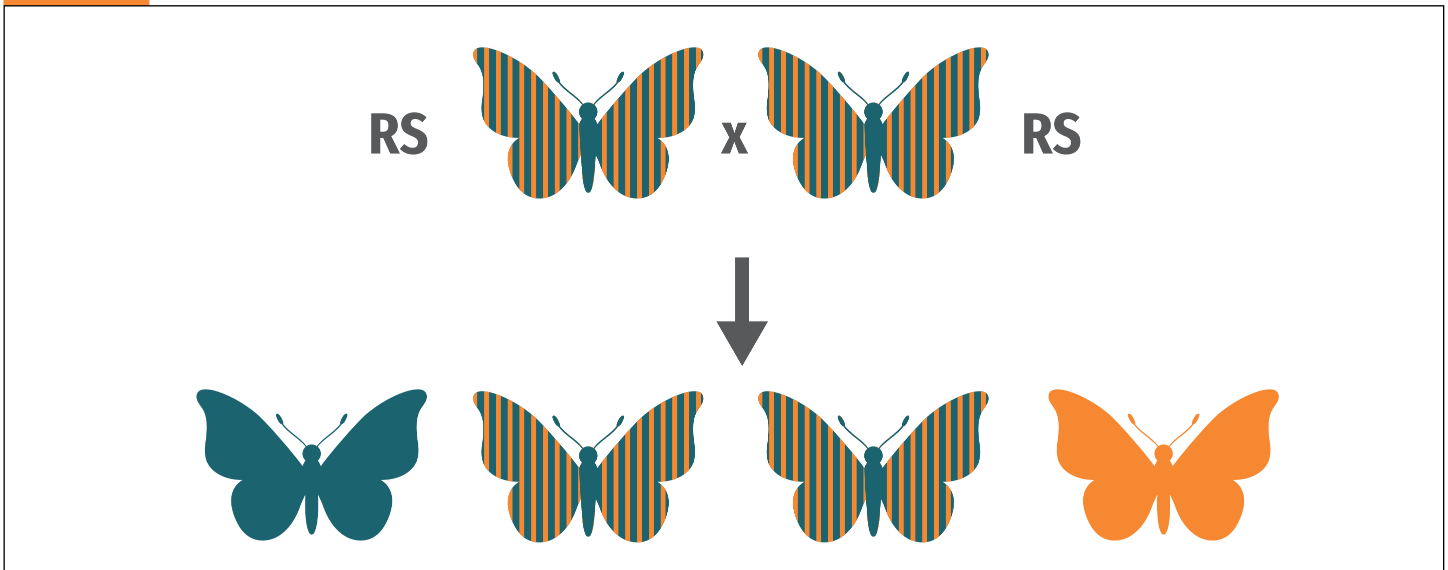

Figure 2: When two individuals that are heterozygous for the resistance trait (RS) reproduce, their progeny shows segregation for that trait. That is, individuals will be either heterozygous, homozygous R or homozygous S. In addition, according to Mendel's Laws of Inheritance, the ratio of the different types will be 1:2:1 (RR:RS:SS).

Nowadays, most pesticides are very specific in their modes of action and a single mutation in the pest target site confers, in general, a high level of resistance. The evolutionary aspect of pesticide resistance in a population is closely linked to the understanding of the dominance relationship among the alleles of the gene affected. Pesticide resistance expressed by a dominant allele spreads rapidly throughout a pest population in comparison to a population expressing resistance by the presence of recessive resistant alleles. In this latter case, resistance tends to spread slowly since the susceptible heterozygous and homozygous individuals can be eliminated by application of the pesticide to which resistance was developed. Not surprisingly, the majority of cases of pesticide resistance have evolved from single dominant (mutated) alleles. Resistance due to recessive alleles is rare. Polygenic resistance caused by minor genes can still develop in the field but would do so very slowly due to inadequate genotypic discrimination in favour of resistance. These observations come from lab studies owing to the fact that the degree of dominance of an allele that confers resistance to a pesticide cannot be known until such allele is identified and appropriate crosses are made (Georghiou and Taylor 1977).[1]

Resistance explained by a single gene or 'major gene' mutation is often referred as 'single step' mutation and, once introduced in a population, tends to be stable. This point mutation causes a single amino acid change in the target site protein and is responsible for the phenotype of resistance manifested by the pathogen, insect or weed. When this event occurs, susceptible and resistant pests are clearly distinct. If the pesticide is withdrawn or used rarely in the field, the pathogen, insect or weed population carrying the mutation can still remain resistant for many years. A documented example of 'sustained' resistance in the field is *Cercospora betae* (sugar-beet leafspot) to benzimidazole fungicides in Greece (Dovas et al. 1976).[2]

[1] Georghiou G. P. and Taylor C. E. (1977) Operational influences in the evolution of insecticide resistance. *Journal of Economic Entomology, 70*: 653-658.

[2] Dovas C., Skylakakis G. and Georgopoulos S.G. (1976) The adaptability of the benomyl resistant population of *Cercospora beticola* in Northern Greece. *Phytpathology* 66: 1452-1456.

Resistance develops gradually to some pesticides such as the 2-amino-pyrimidine fungicide ethirimol. A reduction in both control and susceptibility of the pest is revealed by monitoring tests and gradually becomes noticeable at partial and variable degrees. This type of resistance is often called ‘multi-step’ or ‘cumulative’ resistance and can revert rapidly to a more sensitive condition under circumstances where, for example, the fungicide concerned becomes less intensively used and alternative fungicides are applied against the same disease. (Source FRAC online: http://www.frac.info/docs/default-source/publications/monographs/monograph-1.pdf)

The ‘multi-step’ or ‘cumulative’ resistance that is more likely to develop in fungicides, for example, possibly originates from a ‘polygenic’ change at the DNA level of the pathogen. As in the single gene scenario, multi-step resistance happens from the selection of mutants but this time, arising from several different genes each carrying a mutation with a small effect in terms of resistance. On their own, these genetic changes do not have a drastic effect on the pathogen and do not allow them to resist strongly to a given fungicide. When several minor mutations are combined, however, the overall resistance to the pesticide can become much stronger and allow the pathogen to survive. In fact, the more genes that mutate to resistance causing forms, the greater the degree of resistance (*ibid*). There are however few occurrences of cumulative resistance in the field.

One example has been with powdery mildew in cereals where biochemical evidence for polygenic resistance to azole (DMI) fungicides has been documented and involves several resistance mechanisms. (Online source: Fungicide resistance – The assessment of risk. 2007. Brent KJ and Hollomon DW http://www.frac.info/docs/default-source/publications/monographs/monograph-2.pdf)

c) Mechanisms of resistance

Once a single or multi-step mutation has appeared in a pest population, the corresponding physical change (phenotype) can manifest in different ways depending where the mutated allele(s) is expressed in the pest. Mechanisms of resistance represent measurable adaptations used by a weed, insect or pathogen to survive a pesticide assault; they are the cause of resistance. In Table 3, we list all the known mechanisms used by pests to resist pesticides with examples that were found in the field. A variety of resistance mechanisms are possible at the physiological levels and there are striking similarities among weeds, pathogens and insects. The key point is that pests are highly adaptable and selection pressure imposed by pesticides can result in any possible mechanisms that allow survival. In addition, mechanisms can accumulate in pests following continuous selection or crossing, which leads to enhanced levels of resistance and/or multiple resistance (similar to gene stacking).

Table 3. Summary of resistance mechanisms to herbicides, fungicides and insecticides

Mechanisms	Explanation	Examples		
		Herbicides	**Fungicides**	**Insecticides**
Target Site Resistance (TSR)				
Target site modification	A single nucleotide change in the gene coding for the pesticide target enzyme prevents or reduces inhibition conferring protection	Imazethapyr (Pursuit) and other ALS inhibitors (WSSA Group 2) resistance in multiple species (pigweeds, nightshade, foxtail, etc.) in Ontario due to ALS modification Linuron (WSSA Group 7) resistance in pigweeds in Ontario carrots due to alteration in target site gene *psbA*	Pyraclostrobin (Headline) (QoI FRAC Code 11) resistance in *Cercospora* leaf spot in sugar beets in Ontario due to point mutation in *cyt b* gene	Organophosphate and Carbamate insecticides (IRAC Groups 1B and 1A) resistance in Colorado potato beetles in Michigan due to mutation in Acetylecholine esterase Phosmet (Imidan) (*AChE*, IRAC 1B) resistance in oriental fruit moth in Ontario peaches due to *AChE* gene mutation
Target site amplification or over-expression	More target site enzyme is produced overwhelming the inhibitory capacity of the pesticide	Glyphosate (WSSA Group 9) resistance in Palmer amaranth in the USA due to multiple copies of the *EPSPS* gene DIM and FOP herbicides (WSSA Group 1) resistance in large crabgrass from Ontario onion fields due to multiple copies of the ACCase target site	Fenboconazole (Indar) (DMI, FRAC Group 3) resistance in cherry leaf spot pathogen *Blumeriella jaapii* in cherry in Michigan caused by overexpression of the *Cyp51* target site gene	Organophosphate insecticide (IRAC Group 1B) resistance in two-spotted mites due to increased copy number of *AChE* gene **Amplification resistance in uncommon in insects**

Table 3. Summary of resistance mechanisms to herbicides, fungicides and insecticides

(continued)

Mechanisms	Explanation	Examples		
		Herbicides	**Fungicides**	**Insecticides**
Non-target Site Resistance Mechanisms (NTSR)				
Detoxification via pesticide metabolism	**Various energy-dependent processes transform the pesticide molecule to make it non-toxic**			
Degradation of pesticide	Different types of enzymes 'attack' the pesticide molecule. The modified chemical structure makes the pesticide non-toxic.	Muster (ethametsulfuron-methyl) (ALS inhibitor, WSSA Group 2) degraded by Cytochrome P450 enzymes confer resistance in wild mustard in canola (Western Canada)	Propiconazole (Tilt) (DMI, FRAC Group 3) resistance in *Monilinia fructicola* (brown rot of stone fruits) caused by increased cytochrome P450 based fungicide degradation	Cytochrome P450 mediated detoxification of imidacloprid in Colorado potato beetles Resistance to four insecticide classes (IRAC Groups 1A, 1B, 2A and 3A) due to cytochrome P450 enzymes in western flower thrips
		No evidence of esterase-based degradation of herbicides and fungicides		Resistance to pyrethroids and carbamatates (IRAC Groups 4 and 1A) due to esterase activity in western flower thrips
Conjugation of pesticide	Enzymes such as glutathione S transferase (GST) conjugate (add) the peptide glutathione to the pesticide, making it non-toxic	Triazine (WSSA Group 5) resistance in some velvetleaf populations from the USA due to GST based conjugation	**Little or no evidence of GST mediated fungicide resistance in plant pathogens**	Resistance against carbamate insecticides (IRAC Group 1A) conferred by Glutathione S transferase in western flower thrips

Table 3. Summary of resistance mechanisms to herbicides, fungicides and insecticides

(continued)

Mechanisms	Explanation	Examples		
		Herbicides	Fungicides	Insecticides
Exclusion mechanisms	**Reduction in internal cellular or subcellular concentration of the pesticide prevent it from inhibiting the target site**			
Enhanced efflux	Membrane transporters actively pump out the pesticide outside the cell quicker than it can get in	**Theoretical in weeds**	Resistance to multiple fungicides (FRAC groups 1, 2, 3, 7, 9 and 12) in *Botrytis cinerea* caused gray mold of various fruits	Cellular efflux of organophosphates, endosulfan and pyrethroids (IRAC Groups 1B, 2A and 3A) contributes to resistance in American bollworm *(Helicoverpa armigera)*
Sequestration/ compartmentation	The pesticide is stored in a subcellular compartment or outside the cell	Vacuolar sequestration of glyphosate (WSSA Group 9) in Canada fleabane	**Not documented for insecticides and fungicides**	
Reduced absorption/ penetration/ entry	The pesticide cannot reach the target site because its absorption is impeded at the cuticular level. Penetration resistance is frequently present along with other forms of resistance, and reduced penetration intensifies the effects of those other mechanisms	Reduced absorption and retention of glyphosate (WSSA Group 9) on leaves of an Italian ryegrass *(Lolium multiflorum)* from Chile contribute to resistance	**Not documented for fungicides**	Reduced penetration confers resistance to neonicotinoids (IRAC Group 4A) in the green-peach aphid *(Myzus persicae)*

Table 3. Summary of resistance mechanisms to herbicides, fungicides and insecticides

(continued)

Mechanisms	Explanation	Examples		
		Herbicides	**Fungicides**	**Insecticides**
Exclusion mechanisms	**Reduction in internal cellular or subcellular concentration of the pesticide prevent it from inhibiting the target site**			
Altered translocation	In plants, reduced or altered movement of a systemic herbicide prevents accumulation in growing points	Reduced translocation to the growing points and accumulation in leaf tips confers resistance to glyphosate (WSSA Group 9) in rigid ryegrass *(Lolium rigidum)*	**Not applicable to fungicides and insecticides**	
Behavioural	Pest recognizes the pesticide and altered behaviour ensues or life cycle is modified to reduce exposure	**Experimental demonstration that weeds can modify their emergence patterns and dormancy requirements in response to control measures. No field cases reported**	**Not applicable to fungicides**	Diamondback moth females can lay more eggs at the base of the stem than on leaves of cruciferous crops, allowing reduced insecticide exposure

d) Genetic diversity of the target species

The level of genetic diversity found in a weed species or any other pests can influence its ability to develop resistance under selection pressure. One key factor influencing genetic diversity within a species is the **mating system** of a plant. For example, cross-pollinated plants like common and giant ragweed, annual and perennial ryegrass, or tall waterhemp are naturally more diversified genetically at the population level compared to self-pollinated species such as common lamb's-quarters, wild oats or redroot pigweed. As a result, any pests that are more diversified genetically are often more prone to developing resistance and/or multiple resistance in their populations. In addition to genetic diversity, **gene flow, fitness costs,** and **seed dormancy** all have moderating or accelerating influences on the speed of resistance evolution.

Mating system: Whether the plant is an obligate self-pollinator or cross-pollinator will have a strong impact on the rate of resistance evolution and especially on the accumulation of multiple resistance mechanisms. Cross-pollinated plants recombine their genes each generation while self-pollinated plants have genomes that remain more or less identical from generation to generation.

Gene flow: the efficient transfer of resistance alleles from one population to another. Therefore, it can have a strong impact in the spread of resistance in any given species.

Fitness: an individual's capacity to transmit its genes to the next generation. Very often resistant individuals suffer from a fitness penalty (costs) compared to the wild type as a side effect of their resistance mechanism.

Seed dormancy: the capacity of most weed species to produce seeds that can lay dormant in the soil and retain their viability for an extended period of time allow them to constitute a seed bank. This is a buffer that slows down the development of resistance. Plants with long seed bank life have susceptible individuals germinating at the same time as the resistant ones, thereby diluting the selection process. In contrast, plants with short seed bank life see the rate of resistance increasing more rapidly over time as the susceptible individuals disappear through normal attrition.

Similar to weeds, fungal pathogens and insects that go through sexual recombination are more likely to have a higher level of genetic diversity within a population. In general, fungicide or insecticide resistance occurs more frequently in populations that have a high level of genetic diversity compared to populations with low genetic diversity. The pest generation time also influences the development of fungicide or insecticide resistance. A short generation time means that more individual isolates or insects will appear within a growing season. This will enable more genetic diversity and a higher rate of natural mutations that could result in the occurrence of resistance alleles to the insecticide or fungicide. Fungi that can produce a large number of spores are also more prone to develop resistance.

2. Discuss how the following affect the development and evolution of resistance, including:

a) Rotation and/or combinations of best management practices (BMPs);

b) Pest maturity, pest severity, frequency of control;

c) Pest dispersal mechanisms;

d) Reliance on a single mechanism of action;

e) Reduced or off-label application rates of pesticides;

f) Off-label application practices of pesticides.

a) Rotation and/or combinations of best management practices (BMPs)

By using diverse crop management techniques, growers can reduce the appearance and spread of pesticide-resistant weeds, insects or other pathogens. In general, it is assumed that a diverse rotation with multiple crops will reduce the risk of resistance development, as different products will be used in different phases of the rotation. It is worth noting, however, that this is not always the case; some technologies allow the use of the same product and, hence, that same selection pressure in different phases of the rotation. Therefore, one needs to not only pay attention to the crops used in rotation but to the overall pesticide program and the range of BMPs available. Table 4 shows the relation between the use of BMPs and resistance development. More diversity of BMP methods often means less risk of resistance development to pesticides.

Table 4. Impact of various BMPs on the risks of resistance development

Method	Resistance development risk		
	Low	**Intermediate**	**High**
Crop rotation	Full rotation	Inconsistent or limited	No rotation
Number of pesticide modes of action used in rotation	More than two modes of action	With two modes of action	Only one mode of action
Number of methods used to control pests	Cultural, mechanical and chemical	Mechanical and chemical	Chemical only
Pesticide usage with same mode of action per season	One application	More than one application	Several times within the growing season
Resistance status to mode of action	Undetermined	Limited	Common
Level of pest infestation	Low	Average	High
Pest control in last three years	Good	Reduced	Poor

Adapted from HRAC Global: http://hracglobal.com/prevention-management/best-management-practices

b) Pest maturity, pest severity, frequency of control

- There is a strong correlation between the frequency of control and the development of resistance as described in the previous table.
- Pest maturity can also have an impact on resistance evolution. For example, in insects, DDT resistance in *Anopheles gambiae* (mosquito) declines with age, which has implications for testing and diagnostics as false negatives may be detected. In the case of weeds, the more mature that plants are at spraying time, the less sensitive they tend to be to most POST herbicides; this will therefore exert a lesser selection pressure. Late treatments are also not desirable in most situations since the negative impact due to competition would have taken place and the weeds would be harder to control anyway.

  Pest maturity affects insecticides as well. For example, whitefly larvae are more susceptible to organophosphate insecticides than adult whiteflies. At the same dose, the insecticide applied on an immature population will impose a greater selection pressure for resistance.

- The speed at which resistance develops also depends on other factors; including how fast the insects reproduce, the migration and host range of the pest, or the availability of nearby susceptible populations. Indeed, resistance increases fastest in situations such as greenhouses, where insects or mites reproduce quickly and there is little or no immigration of susceptible individuals. (Source IRAC)

c) Pest dispersal mechanisms

Dispersal plays a key role in the evolution and spread of resistance. In insects, dispersal is a fundamental mechanism by which resistance genes are transported across different fields. On the other hand, when resistance is located in one field, its spread could be slowed down by the immigration of susceptible individuals in the affected field. This is also the reason behind planting a refuge of susceptible corn along with Bt corn. In this case, the presence of susceptible corn hybrids help pests survive and mate with the resistant organisms, thus delaying the ability of the pests to develop a resistant population. In both scenarios, decreasing the frequency of resistance alleles in the local pest population is desirable.

With fungi, spores carrying resistance alleles can travel long distances when released in the air and blown by winds or any air movement caused by humans or animals. Inoculum can also be carried by farm machinery or by crop seeds.

In weeds, dispersal mechanisms also have a significant role to play in the development of resistance. For this type of pest, dispersal is done in four possible ways:

1. **Equipment:** The use of field machinery is the most common way to spread herbicide-resistant seeds from field to field. It is easy for weed seeds to stick to any farm equipment used during the growing season.

2. **Seed, grain or seed mixes:** If seed crops are purchased from agricultural areas where there are resistant weeds, the crops can be contaminated by the resistant weed seed that were transported in the purchased bags. Similarly, producers bin-running their own seed may help spread resistance from affected parts of their property to other parts that were clean.
3. **Animals and feed:** Herbivorous animals feed on a variety of plants. Their manure has been identified as a cause of resistant weed transmission over long distances. For example, pigweed can pass through an animal's digestive system and germinate afterward.
4. **Wind dispersal or movement dispersal:** Resistant seeds or pollen can be wind-dispersed from one field to the neighbouring one. Kochia is a typical example of a tumbling weed that can travel over long distances by wind, dropping seeds on its path. Seeds of resistant populations can also travel on animal fur to other fields.

d) Reliance on a single mechanism of action

For any type of pesticide, reliance on a single mode of action is the most significant driver of resistance development. It is also important to realize that different pesticides belonging to the same mode of action group will impose the same selection pressure as if only one product had been used repeatedly. For example, the sequential use of four herbicides such as Pinnacle (thifensulfuron), Classic (chlorimuron), Accent (nicosufuron) and Broadstrike (flumetsulam) would be equivalent to using only one of them four times as they are all herbicides that affect the same mode of action, acetolactate synthase (ALS; WSSA Group 2). Lack of diversity of mode of action is identified as one of the main determinants of resistance risk in the BMPs table (Table 4).

e) Reduced or off-label application rates of pesticides

Reduced or off-label application rates that provide less than desirable kill of the target species may select for resistance in some species. This resistance might be polygenic and will be more easily selected in cross-pollinated plants. For example, weeds species that are obligate cross-pollinators such as ryegrass, waterhemp or ragweed are capable of exchanging minor resistance genes that can accumulate in the progeny to give an overall greater level of resistance. Spraying a reduced herbicide rate on ragweed may allow a few plants that have a low level of resistance to survive. In contrast, a higher rate of herbicide application would eliminate these plants and avoid a buildup of weaker resistance alleles in the offspring. In the situation where progeny plants have accumulated enough weak resistant alleles in their genetic makeup, the plants will be able to survive higher doses of application. In contrast to cross-pollinator weeds, self-pollinators like wild oats, lamb's-quarters or green foxtail do not have this potential of building resistance gradually.

Most insect species have sexual reproduction. Resistance to insecticides is often due to genes that give full resistance when homozygous (RR) but not as strongly when heterozygous (RS). In the early phase of selection, most resistant insects are heterozygous (RS) and are eliminated by the use of a high (full) dose of insecticide application. Applying insecticides at a reduced rate allows some heterozygous insects to survive and reproduce. In turn, this gives rise to homozygous (RR) progeny that is not killed when the grower applies the insecticide at a higher dose. While most insects are diploid and reproduce in a sexual manner, there are some species like aphids that can also reproduce in a clonal manner and are always in the RR state. In this event, a higher or lower dose insecticide application will not change the outcome.

f) Off-label application practices of pesticides

It is possible that application at a stage outside of the label recommendation may affect resistance. However, this would likely slow down resistance evolution rather than speed it up, especially with resistance endowed by major genes. The theory is that resistance is most effectively selected when there is a large differential in response between individuals that have the resistance alleles (RR and RS) and those without (SS). This differential allows only the resistant individuals to survive and enrich the next generation with their R alleles. If the pesticide is applied at a stage when the pest is less susceptible, then there is higher survival of SS plants that is not due to resistance. As a result, the grower will observe lesser weed control and the pest population will preserve more SS individuals.

In contrast, with cross-fertile species (insects and cross-pollinated plants) and resistance endowed by minor genes (polygenic resistance), it is possible that use of off-label applications at stages when the pesticide is less effective may lead some individuals to survive – whereas they would have been controlled were the pesticide been applied at the right stage. This can only contribute to resistance development if there is a genetic basis for the survival.

Other off-label scenarios:

- **Use of a pesticide on a pest that is not on the label;** this may or may not lead to resistance depending on susceptibility.
- **Use of wrong additive (e.g., adjuvant):** if this reduces overall efficacy, this could lead to individuals with minor resistance genes to survive, therefore leading to quantitative resistance (see above).

COMPETENCY AREA 2

Identifying resistance

1. Identify the possible reason(s) for pest control failures.

In major crops, whether grown in fields or in greenhouses, there are several reasons for a lack of control by pesticides other than resistance. This includes:

- Failure to properly monitor for pests in the crop;
- Incorrect pest identification;
- Wrong pesticide choice;
- Incorrect use of pesticide (e.g., wrong rate, off-label use or degradation of active ingredient due to long storage, etc.);
- Inadequate preparation of pesticide (e.g., tank mix, adjuvants, pH, concentration, etc.);
- Incorrect spraying/application time (e.g., time of day, environmental conditions, etc.);
- Faulty equipment (e.g., incorrect calibration, poor maintenance, inconsistency, worn nozzles, low pressure, etc.);
- Insufficient or improper pesticide coverage;
- Long delay before addressing problem/issue (e.g., pest population has become too high or pest is not at the right stage); or
- Lack of implementing an integrated pest management program.

2. Identify the possible reasons for genetic plant and trait resistance failures.

Genetic and plant trait failures (breakdown)

Due to their constant exposure to pathogens and insects, plants have evolved natural defense mechanisms in order to survive and thrive. Some traits present in plants have allowed them to resist or tolerate these constant attacks. The distinction between tolerance and resistance differs between insects and pathogens and is made clearer in Table 5.

Table 5. Difference between resistance and tolerance in pests (pathogens, insects) in crops.

	Pathogens	Insects
Resistance	Plant uses physical or biochemical mechanisms to actively cause a reduction in pathogen growth (e.g., toxins, extra pubescence, thick cuticle, etc.). The pathogen cannot infect the plant.	**Active plant action:** has an adverse effect on insects. **Antibiosis:** biochemical or biophysical factors (e.g., toxins, lack of nutrients, thick cuticle, hairs, etc.) of the host plant have a negative effect on the insect growth. **Antixenosis:** factors that affect the behavior of an insect pest and usually are expressed as non-preference of the insect for a resistant plant compared with a susceptible plant.
Tolerance	High pathogen load in the plant has little to no effect even when pathogen goes in the plant and multiplies.	Plant is able to survive despite insect attacks. Plant has capacity to regrow or compensate by producing new shoots or branches from its reserves.

Resistance traits are resource dependent and therefore require an investment tradeoff for the plants: by investing in resistance traits, there are less resources allocated for overall growth and reproduction. However, investment in defense traits allows survival while their absence might result in plant death. Thus, there is a strong evolutionary advantage to develop (invest) in these types of traits if the pathogen or insect attacks are sustained and/or intense.

These resistance traits (Host Plant Resistance or HPR) can be controlled by major genes (qualitative resistance) or by multiple genes (quantitative resistance). Qualitative resistance generally confers specific defense with a strong effect, which is inherited in a Mendelian fashion. In contrast, multiple genes confer quantitative resistance, each with a small effect, but with a strong reaction when combined. Qualitative resistance is also referred to as vertical resistance (effective against specific biotypes or strains of the pest) while quantitative resistance is also named horizontal resistance (effective against all strains or biotypes, e.g., non-specific response)

Breeders have attempted to improve HPR on various crops for insects or pathogens by using a plant breeding approach. Defense traits are inserted in the crop of interest while other desirable traits (e.g., yield, quality, etc.) are maintained. Breeders have traditionally favoured monogenic qualitative resistance because these traits have a strong effect and a simple inheritance pattern.

The benefits of this pattern are:

- Specificity to a single pest
- Remains effective for many successive generations
- Reduces pesticide usage
- Easy to adopt as trait is present in the seed
- Effective

While these are great benefits, there are also disadvantages to this approach:

- Time consuming: breeding for a new trait takes three to 10 years
- Trait might not be present in the current genetic pool of species; need to find trait in another related species
- Biotypes or strains of pest can develop immunity to the trait present in the plant and can attack/infect it

This last disadvantage is the most concerning and is the cause for breakdown/failure of the plant trait. This breakdown is similar to plant pests developing resistance to insecticides or fungicides.

Major HPR traits can exert a strong selection pressure for variants in a pest population that have the genetic capacity to overcome or defeat the resistance trait. The following example illustrates the need to treat HPR as an exhaustible resource that can be defeated by variable plant pest populations.

Phytophthora root rot (PRR) in soybeans is a major disease caused by *Phytophthora sojae*. It is responsible for severe yield loss in this crop and breeders have used different resistance gene (*Rps*) to confer HPR in various soybean cultivars. *Rps* genes differ in their ability to give protection against specific races of P. *sojae*. These *Rps* genes are identified by various numbers and letters: 1a, 1b, 3a, 6, 7, etc.

In addition, *P. sojae* has different races defined by their ability to 'defeat' specific *Rps* genes and thus causing infection in the soybean plant. This situation is also complicated by the fact that some races of *P. sojae* can defeat more than one *Rps* and, in any given field, there is often multiples races present.

Surveys conducted in Ontario in 2010 to 2012 revealed the presence of 22 races in various fields. Results show that race 25 was the most prominent in Ontario. It can defeat soybean cultivars with *Rps* 1a, 1b, 1k and 7. This means a soybean cultivar containing one of these genes would get infected by *P. sojae* race 25. In contrast, using a different soybean variety containing *Rps* 1d or 6 would prevent disease development in the crop. While some *P. sojae* races have virulence against two *Rps* genes, others can have virulence against 6, 7 or 8 *Rps*, which mean they can infect a large range of cultivars.

Genetically Engineered HPR

While the examples above relate to HPR and traditionally bred crops, the fact that major genes control many of these traits makes it possible to use genetic engineering for crop improvement. One such example is the development of Bt crops.

Genes isolated from the bacterium *Bacillus thuringiensis* code for entomotoxic proteins (Bt proteins) that are lethal to many insects upon ingestion. Plants that have been transformed through molecular techniques can express these entomotoxic proteins in their tissue. In fact, a crop that was previously susceptible to an insect infestation is able, once transformed, to fight back by producing its own insecticide. Bt crops such as Bt corn are very effective against insect infestations and were rapidly adopted by growers. The downside of Bt crops being used intensively year after year creates a very strong selection pressure on pests to develop resistance. As insects can develop resistance to conventional insecticides they can likewise become resistant to Bt proteins.

Awareness to potential resistance development was recognized early in the development of Bt crops and regulations were implemented to assist growers in the reduction of selection pressure. To comply with the regulations, any crop carrying the Bt trait is mixed with non-carrier plants. This is called the refuge strategy to insecticide resistance management. (See Table 6) It is based on the premise that initial individuals that have developed resistance to Bt in an insect population will only have one copy of the resistance allele. These individuals are termed RS, meaning that they are a carrier of one resistance (R) allele and one susceptible (S) allele. In comparison, normal susceptible individuals have two copies of the susceptible allele and are deemed SS.

The allele conferring resistance to the Bt trait is recessive. It confers only a low level of protection. In practice, if the Bt corn expresses a high level of the Bt protein, the high toxicity will severely affect the performance of RS individuals. On the other hand, resistant homozygous (RR) insects will be completely immune to the Bt toxins. This is why the refuge strategy is essential to maintain a high proportion of SS individuals so that newly erupted RS individuals will likely mate with susceptible insect populations. This approach will ensure that the resistance trait stays in the heterozygous (RS) state in the insects and causes minimal damage to the crop. If a grower were to plant only a Bt crop without any susceptible plants in the field, all SS insects would be killed and the few remaining RS individuals would reproduce among themselves. The resulting progeny would have 25% of individuals in the RR homozygous state expressing a high level of resistance to Bt and thus allowing an exponential explosion of insect resistance.

For example, if two RS corn borers mate and the female lays 200 eggs, 50 of those eggs will be RR, 100 RS and 50 SS. The 50 RR individuals (assuming a balanced sex ratio) now have the potential to produce 5,000 RR individuals in the next generation and 500,000 in the one following. This exponential augmentation of resistance illustrates the need for proactive management measures.

Current regulation demands that growers plant 20% of their field with a hybrid that does not carry the Bt trait. These susceptible plants form the refuge upon which SS individuals can grow and reproduce, thereby maintaining a sufficient pool of S alleles. Another approach is to have refuge seeds incorporated in the bag along with Bt seeds. In this case, the proportion of seeds with SS trait is about five to 10%. Compliance with the refuge strategy ensures no breakdown in the Bt trait is seen in other jurisdictions with corn or other Bt crops. Another current approach is to have corn hybrids stacked with various Bt genes, assuring a reduced chance of resistance developing. More information is available at www.cornpest.com.

Table 6. Refuge strategy

Benefits	Disadvantages
Ensure resistance traits remain at low levels in the insect population.	Five to 20% of the area planted to corn (or other crop) is susceptible to insect damage and yield loss.
Preserve susceptible alleles in the insect population.	Require high overall compliance: if growers do not follow strategy, resistance may develop and RR insects will spread to neighbouring fields where refuge was implemented.
Easy to implement, especially with 'refuge seeds' incorporated in Bt crop bags.	

3. Discuss the processes for identifying pest resistance.

If resistance is still suspected following a detailed inquiry of the possible reasons for control failure listed previously listed on page 22, the Crop Advisor needs to work with the farmer to review the field history with the following questions in mind:

- Did the farmer use the same pesticide or pesticides that have the same mechanism (mode) of action over a period of several growing seasons?
- Has the uncontrolled pest been managed efficiently by the same pesticide in past years?
- After the pesticide application, is the surviving pest (weed, insect, pathogen) growing and thriving thoroughly?
- Can the farmer see some mortality besides the surviving pest?
- Has a decline in the control of this particular pest been noticed in recent years?
- Is there any known report of pest resistance around the farm area?
- Is the pest being well managed in neighbouring farms?

If most answers are affirmative, the Crop Advisor should take samples of the pest (trap insects, collect eggs, whole plants, seeds, diseased plant parts, etc.) to send for further testing to confirm the presence of resistance.

Figure 3

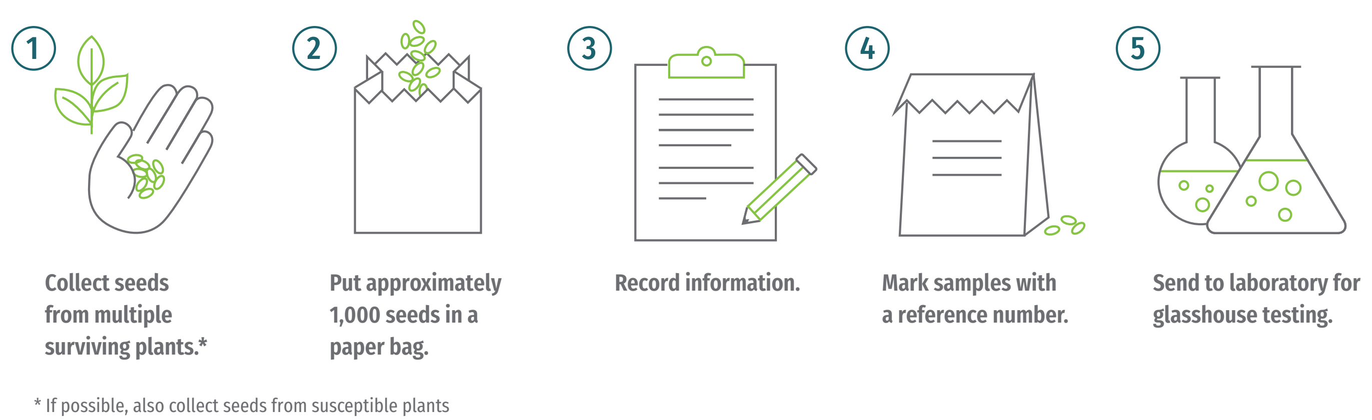

Figure 3: Steps for the collection and submission of samples for testing resistance. (Source: http://hracglobal.com/prevention-management/best-management-practices)

4. Discuss the methodologies for testing and confirming suspected resistant pests.

To confirm weed resistance, lab tests need to be performed in a scientifically and unbiased manner. Resistant and susceptible plants of the same species are tested in a dose response experiment conducted in a controlled environment using whole plants. Several replications are carried out to eliminate possible errors. Moreover, in cases where resistance has already been found in a given species, diagnostic doses or molecular tests (by PCRs and specific primers) are used to confirm resistance in the farmer's field.

Ideally, when resistance is suspected in a new combination of pest and pesticide, a dose response experiment needs to be conducted in the conditions under which future diagnostic tests will be done. A dose response implies the pesticide is applied (e.g., sprayed, put on Petri dish surface or in growth media, dipped, etc.) at different doses to multiple individuals of the target pest and the response is measured. Depending on the pest, the response may be survival/mortality, growth rate, biomass accumulation, etc. In the majority of cases, dose response follows a sigmoidal curve and the results are best described according to a log-logistic equation (Figure 4). Basically, this means that at very low doses, a pesticide has barely any effect (see Figure 4 when the response trends around the 100% mark), and at very high doses the response trends towards 0 where the effect of the pesticide is maximum. Obviously, there is a range of doses where the response changes dramatically, going rapidly from the upper limit to the lower limit. This is the range that is the most interesting biologically as it allows the most precise comparison between different pests, biotypes, populations or pesticides. The dose at which a 50% in response is observed is often used as a benchmark for comparisons. That dose is often referred to with terms such as LD_{50}, LC_{50}, ED_{50}, GR_{50}, I_{50}[3] depending on the variable used to measure response.

Figure 4

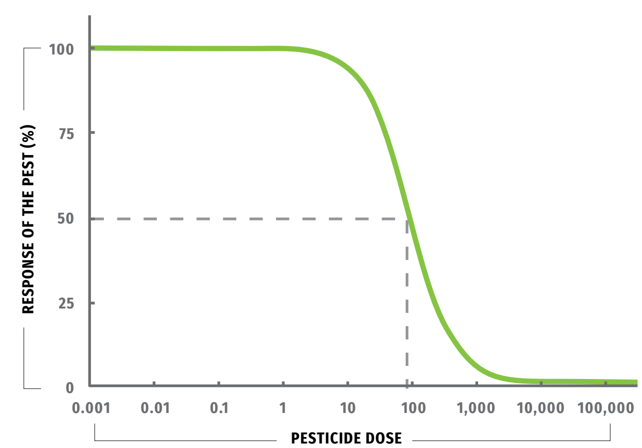

Figure 4: Example of a typical dose response curve measuring the effect of a pesticide on a pest. With increasing doses, we see a steep response once a threshold dose is reached. There is virtually no response at very low doses, while there is complete response at high doses. The point of inflexion of the curve (half way between upper and lower limits) helps determine parameters such as LD_{50}. In this example, the 50% response is attained at a dose of almost 100.

[3] LD_{50} stands lethal dose 50, LC_{50} for lethal concentration, ED_{50} for effective dose, GR_{50} is growth reduction and I_{50} stands for inhibition.

A standard procedure in a diagnostic lab is the establishment of dose response curves for a susceptible (sensitive) population or line and one or more resistant lines (Figure 5). Comparison of the doses providing 50% response allows the determination of the resistance level compared to the standard susceptible. In Figure 5, the susceptible line (S) has a 50% response at a dose of 0.5, the resistant 1 line (R1) of 90 and R2 of 500. By dividing the 50% dose for the R1 and R2 by that of the S we can obtain a resistance index. In this case, R1 has 180-fold resistance and R2 has 1000-fold resistance over S.

In addition, the finding of these dose response curves allows an establishment of a diagnostic dose. This is a dose that can be used in further testing and gives a much more rapid diagnosis without having to complete a dose response. The diagnostic dose provides the largest vertical difference between the response of S and R. Ideally, it would be a dose that completely controls or inhibits an S line while having little or no effect on R lines. In the example given here, the diagnostic dose would be slightly more than 10 (Figure 5).

Figure 5

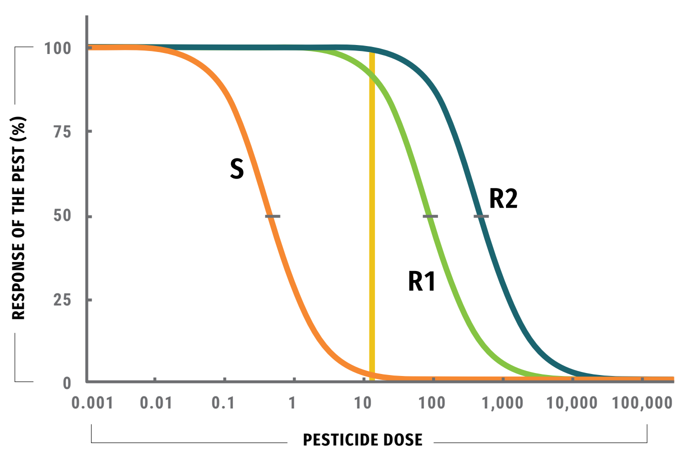

Figure 5: Theoretical dose response curves for a susceptible (S) and two resistant (R1 ad R2) lines. The short horizontal lines represent the 50% response. The vertical line shows the diagnostic dose.

PROFICIENCY AREA II

BEST MANAGEMENT PRACTICES (BMPs) FOR RESISTANCE MANAGEMENT

COMPETENCY AREA 1

Site/Mechanism of Action's Role

1. Discuss an effective site/ mechanism of action for pest control in:

a) Insects;

b) Weeds;

c) Diseases.

Why is knowing site of action important?

Pesticides grouped within a particular class according to HRAC, FRAC or IRAC, or other organizations like WSSA or Pest Management Regulatory Agency (PMRA), have similar chemical structures or properties and are active through a common mode of action (target site). The industry has developed these groups to facilitate learning and be familiar with modes of action (MoA) and their meaning. The mode of action of any pesticide represents how this pesticide interferes with the physiological functions of the pest. Different classes of chemicals (or pesticides) work in different ways and present different risks and problems. On the other hand, chemicals within the same class function similarly and often share the same advantages or disadvantages.

Knowing the site of action of a pesticide is very important since it helps to reduce or prevent the repetitive use of pesticides that share the same mechanism of action over several years or even within the same growing season. The more familiar a grower or an advisor is with the different classes of pesticides, the higher the chance the decision made about treatment of a problematic pest will be the best under the circumstances.

Is there any value in knowing that some pesticides are more prone to have resistance developed than others?

Some pesticides are more prone to generate resistance in pests than others. For example, FRAC and HRAC have established mechanisms of action risks levels according to what type of MoA is being used by growers (Tables 7 and 8). This has led to the suggestion of voluntary labelling in Canada by PMRA. Manufacturers are encouraged to use FRAC, IRAC or WSSA groups on their labels to facilitate proper resistance prevention and management practices. (See Figure 6)

Figure 6

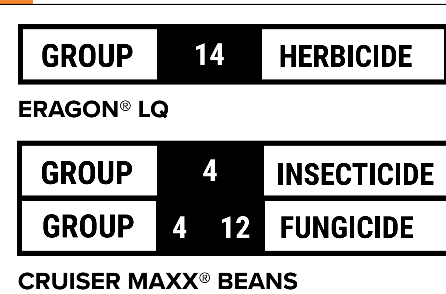

Figure 6: Examples of MoA group labelling for two pesticides used in Canada.

In order to try and prevent the development of resistance, herbicides and fungicides are assigned risk factors. In this system, a pesticide, that is observed to develop resistance after only a few applications would be termed 'high risk.' An example in herbicides would be Group 1 or Group 2 that are often observed selecting for resistance after four or five applications. In contrast, 'low risk' pesticides are those for which resistance tends to appear after multiple applications – sometimes 20 or more. Risk factors for herbicides and fungicides are summarized in Tables 7 and 8.

Table 7. Risk of potential resistance development by herbicide group

Herbicide Group	Frequency of herbicide application				
	6 or less	7-10	11-20 X		>20 X
	High	Moderate-High	Moderate	Low-Moderate	Low
1	✔				
2	✔				
3			✔		
4					✔
5				✔	
6					✔
7		✔			
8			✔		
9					✔
10					✔
22		✔			
27		✔			

Adapted from Beckie H.J. Herbicide resistance weeds: management and practice (2006) Weed Technol. 20:793-814

Table 8. Risk of potential resistance development by fungicide code

FRAC Code	High	Moderate-High	Medium	Low-Moderate	Low	Unknown
1	✔					
2		✔				
3			✔			
4	✔					
5				✔		
6				✔		
7		✔				
8			✔			
9			✔			
10	✔					
11	✔					
12				✔		
13			✔			
14			✔			
16.1						✔
16.2			✔			
16.3						✔
17				✔		
18						✔
19			✔			
20						✔
21		✔				
22				✔		
23				✔		
24			✔			
25	✔					
27				✔		
28				✔		
29					✔	

Table 8. Risk of potential resistance development by fungicide code *(continued)*

FRAC Code	High	Moderate-High	Medium	Low-Moderate	Low	Unknown
30				✔		
31						✔
32						✔
34						✔
35						✔
36						✔
37						✔
38					✔	
39						✔
40				✔		
41	✔					
43						✔
44						✔
45		✔				
46						✔
47		✔				
48						✔
49		✔				
50			✔			
M01-M12					✔	
P01-P06						✔
P07					✔	
U06					✔	
U12				✔		
U13						✔
U14						✔
U16			✔			
U17						✔
U18						✔

Knowledge of resistance risk allows the planning of more efficient rotations and a better use of pesticides in sequences or in mixtures. It is unreasonable to recommend excessive use of high resistance risk pesticides due only to their initial efficiency. Efficiency increases resistance risks. For instance, a pesticide working extremely well in getting rid of a problem pest is a strong indicator that it will probably select for resistance in the near future.

In addition, even low-risk pesticides do not equal 'no-risks.' Under the right circumstances any pesticide can select for resistance. High-risk pesticides are those for which resistance happens more rapidly.

It is worth remembering that risk is unknown when a new pesticide class is brought to the market. Risk is established *a posteriori* – in other words, after observing how quickly resistance appears in a given field and/or region after the first use. As mentioned, herbicides, which select for resistance in a few applications, are termed high risk (e.g., Group 1 and Group 2). Similarly, fungicides belonging to FRAC Groups 1, 4, 10, 11 and 25 easily select for resistance and are termed 'high risk.'

It is also important to acknowledge that these classifications are not set in stone and only serve as guidelines. The assessment of a product may change after significant change in its use pattern. Many are now familiar with the example of risk assessment for glyphosate. This herbicide was introduced to the market in 1974, and there was no case of resistance for more than 20 years despite widespread use. With the introduction of glyphosate-resistant crop technology in the mid to late 1990s, concerns arose as to whether this would lead to resistance developing in weeds. While some stated that glyphosate was considered low risk, as no resistance cases had been yet recorded, others rightly pointed out that the changing pattern of use would increase the selection pressure and the low-risk nature of glyphosate was not accurate. It is also true that many sites have been treated multiple times with this product and, in these cases, the risk threshold has been passed. Remember that 'low risk' is not 'no risk.'

2. Discuss site/mechanism of action's role in:

a) Delaying; or

b) Accelerating pest resistance.

a) Discuss how the knowledge of site/mechanism of action of a pesticide can contribute to delaying the evolution of resistance.

Pesticides with different mechanisms of action used in rotation or tank mixed can significantly delay the evolution of resistance. It has been demonstrated that using two or more pesticides with a different MoA with similar efficacy against the pest can help eliminate any potential surviving individual containing a resistant allele. Each MoA corresponds to a unique target site in the pest that interferes with its physiological functions and results in its death. The knowledge of the MoA or active ingredient in a pesticide helps growers to differentiate each pesticide. The classification of MoA into groups among several marketed pesticides sold by the industry allows the grower to decide on the best course of action when planning which pesticides to apply on a problem pest when they are used in mixtures, in sequence, or rotation.

b) Discuss how misuse or disregard of pesticides MoA can accelerate the evolution of resistance.

Repeated use of pesticides with the same mechanism of action has been shown to be the largest contributing factor to the fast evolution of pesticide resistance throughout the world. Using different brands of pesticides that contain the same active ingredient, or different actives with the same MoA, increases the selection pressure on the pest since the same targeted site is affected regardless of the pesticide brand name. Reducing selection pressure is key to delay the development of resistance in any pest. This is why it is paramount for the grower to understand the classifications of MoA created by the different organizations (e.g., HRAC/WSSA, IRAC and FRAC).

3. For pesticides, evaluate the importance of rotating effective IRAC, FRAC and WSSA or HRAC code or group designations for sites/ mechanisms of action.

See Table of Contents for acronym definitions.

IRAC, FRAC and WSSA/HRAC are committees/organizations that have each developed a classification system for insecticides, fungicides and herbicides respectively, to ensure their sustainable uses. In the case of herbicides, there are two classification systems commonly used. In the mid-1990s, WSSA developed its own classification system on herbicides being used in North America. Later on, HRAC adopted a similar classification system to unify herbicide labeling internationally. These two systems are now being accepted and are very similar in principle – the main difference is that WSSA adopted a number system while HRAC relies on letters. (https://pesticidestewardship.org/wp-content/uploads/sites/4/2016/07/HerbicideMOAClassification.pdf) The aim of all these pesticide classification systems is to harmonize the classification of pesticide sites of action in as many countries as possible. The classification is based on the site or mechanism of action that each pesticide uses. The general guideline is to select pesticides from groups (classes) that use different sites of action to control the same pest and use them in successive applications or in mixtures. Basically, changing chemicals does not necessarily mean that you are changing the mechanism of action. For example, the fungicide propiconazole and the fungicide triadamefon both have the same mode of action. If a grower applies these fungicides in a mix or in rotation, there is no real change in the pest targeted site. Therefore, instead of delaying a potential resistance development, the selection pressure will be increased.

The main advantage to having these classification systems is that rotating pesticides mechanisms of action can easily be done without remembering the specific MoA for each fungicide, herbicide or insecticide that needs to be used on the crop. For example, each herbicide label has a number and/or letter designation that represents a specific MoA. In the event that a grower has two herbicides with the same code on their labels, they should not be used together or one after the other on the problem weed if one would like to delay resistance development in the field. Obviously, this approach would be the same for fungicides and insecticides since FRAC and IRAC have also given number and/or letter designation to their respective pesticides that correspond to specific MoA. Moreover, each one of these committees has other specific recommendations on how to rotate pesticide groups. Tables 9, 10 and 11 – one for each pesticide (fungicides, insecticides and herbicides) – list the classification systems per MoA created by the three action committees. In addition to the codes created by HRAC, a separate column in the table represents the grouping created by WSSA as a reference. (The WSSA codes are more commonly used in North America, while HRAC groups tend to be used in Europe, Asia and South America; Australia has developed its own system for herbicides.)

Table 9. Classification of fungicides according to mode of actions:

FRAC codes are included along with the mode of action, specific chemical groups and an example of a commercial product. (Note: only groups for which there is at least one commercial product registered in field, vegetable or fruit crops in Ontario are included.)

Group #	**Mode of action** (Target site)	**Group name**	**Chemical name** (Product name)
1	Mitosis and cell division (ß-tubulin assembly)	MBC (Methyl- Benzimidazole Carbamates)	Thiophanate-methyl (Senator)
2	Signal transduction (MAP/Histidine-kinase)	Dicarboximides	Iprodione (Rovral)
3	Sterol synthesis in membranes (C14-demethylase)	DMI (demethylation inhibitors)	Propiconazole (Tilt)
4	Nucleic acids synthesis (RNA polymerase I)	Phenylamides	Metalaxyl (Ridomil Gold)
5	Δ14-reductase and Δ8→Δ7 - isomerase in sterol biosynthesis	Amines (morpholines)	Spiroxamine (Priwen)
7	Respiration (complex II: succinate-dehydrogenase)	SDHI (succinate dehydrogenase inhibitors)	Boscalid (Cantus)
9	Amino acids and protein synthesis (methionine biosynthesis)	AP (anilino-pyrimidines)	Pyriminethanil (Scala)
11	Respiration (complex III: cytochrome bc1)	QoI (quinone outside inhibitors)	Pyraclostrobine (Cabrio)

Table 9. Classification of fungicides according to mode of actions *(continued)*

Group #	**Mode of action** (Target site)	**Group name**	**Chemical name** (Product name)
12	Signal transduction (MAP/Histidine-kinase)	PP (phenylpyrroles)	Fludioxonil (Scholar)
13	Signal transduction	Aza-naphthalenes	Quinoxyfen (Quintec)
17	Sterol biosynthesis in membranes (3-keto reductase, C4-demethylation)	Hydroxyanilides	Fenhexamid (Elevate)
21	Respiration (complex III: cytochrome bc1)	Qii (quinone inside inhibitors)	Cyazofamide (Ranman)
22	Mitosis and cell division (ß-tubulin assembly)	Benzamides	Zoxamide (Gavel)
24	Amino acids and protein synthesis (protein synthesis)	Hexopyranosyl antibiotic	Kasugamycin (Kasumin)
25	Protein synthesis (ribosome, initiation step)	Glucopyranosyl antibiotic	Streptomycin (Streptomycin)
27	Unknown	Cyanoacetamide oxime	Cymoxanil (Tanos)
29	Respiration (uncoupler of oxidative phosphorylation)	2,6-Dinitroanilines	Fluazinam (Allegro)
P07	Unknown	Phosphonates	Fosetyl-Al (Aliette)
40	Cell wall synthesis (cellulose synthase)	Carboxylic acid amides	Dimethomorph (Acrobat)
43	Mitosis and cell division (delocalization of spectrin-like proteins)	Benzamides	Fluopicolide (Presidio)

Table 9. Classification of fungicides according to mode of actions

(continued)

Group #	**Mode of action** (Target site)	**Group name**	**Chemical name** (Product name)
44	Lipid synthesis and membrane integrity (microbial disrupters of pathogen cell membranes)	Microbial	*Bacillus subtillis* strain QST 713 (Serenade OPTI)
45	Respiration (complex iii: cytochrome bc1)	Qosi (quinone outside inhibitor)	Ametoctradin (Zampro)
46	Cell membrane disruption (proposed)	Plant extract	Tea tree oil (Timorex Gold)
49	Oxysterol binding protein (osbp) inhibition (proposed)	Piperidinyl-thiazole - isoxazolines	Oxathiapiproline (Orondis Ultra B)
M1	Multi-site contact	Inorganic	Tri-basic copper sulphate (Copper 53W)
M2	Multi-site contact	Inorganic	Sulphur (Microscopic Sulphur)
M3	Multi-site contact	Dithiocarbamates	Mancozeb (Dithane Rainshield)
M4	Multi-site contact	Phthalimides	Captan (Maestro)
M5	Multi-site contact	Chloronitriles	Chlorothalonil (Bravo ZN)
U8	Actin disruption (proposed)	Aryl-phenyl-ketone	Metrafenone (Vivando)
P1	Host plant defense induction (salicylic acid pathway)	Benzo-thiadiazole	Acibenzolar-S-methyl (Actigard)
P5	Host plant defense induction	Plant extract	Reynoutria sachalinensis extract (Regalia Maxx)
NC	Not classified (unknown)	Diverse (biological, bicarbonate, oil)	Streptomyces lydicus (Actinovate), potassium bicarbonate (MilStop), mineral oil (Green Spray Oil 13E)

Table 10. Classification of insecticides according to mode of actions:

IRAC codes are included along with the mode of action, specific chemical groups and an example of a commercial product. (Note: only groups for which there is at least one commercial product registered in field, vegetable or fruit crops in Ontario are included.)

Main group and primary site of action	Chemical sub-group or exemplifying active ingredient	Examples of trade name (Active ingredient)
1 Acetylcholinesterase (*AChE*) inhibitors Nerve action	**1A** Carbamates	Sevin (Carbaryl) Lannate (Methomyl)
	1B Organophosphates	Lorsban (Chlorpyrifos) Cygon (Dimethoate)
2 GABA-gated chloride channel blockers Nerve action	**2A** Cyclodiene organochlorines	Thionex (Endosulfan)
3 Sodium channel modulators Nerve action	**3A** Pyrethroids	Ambush (Permethrin) Force (Tefluthrin)
4 Nicotinic acetylcholine receptor (nAChR) competitive modulators Nerve action	**4A** Neonicotinoids	Poncho (Clothianidin) Admire (Imidacloprid)
	4C Sulfoximines	Closer (Sulfoxaflor)
	4D Butenolides	Sivanto Prime (Flupyradifurone)
5 Nicotinic acetylcholine receptor (nAChR) allosteric modulators Nerve action	Spinosyns	Radiant (Spinetoram) Success (Spinosad)
6 Glutamate-gated chloride channel (GluCl) allosteric modulators Nerve and muscle action	Avermectins, Milbemycins	Agri-Mek (Abamectin)

Table 10. Classification of insecticides according to mode of actions *(continued)*

Main group and primary site of action	Chemical sub-group or exemplifying active ingredient	Examples of trade name (Active ingredient)
8 Non-specific (multi-site) inhibitors	**8B** Chloropicrin	Chloropicrin 100 (Chlorpicrin)
	8F Methyl isothiocyanate generators	Basamid (Dazomet)
9 Chordotonal organ TRPV channel modulators Nerve action	**9B** Pyridine azomethine derivatives	Fulfill (Pymetrozine)
10 Mite growth inhibitors Growth regulation	**10A** Clofentezine	Apollo SC (Clofentezine)
11 Microbial disruptors of insect midgut membranes	**11A** *Bacillus thuringiensis* and the insecticidal proteins they produce	Dipel (*Bacillus thuringiensis* var kurstaki)
15 Inhibitors of chitin biosynthesis, type 0 Growth regulation	Benzoylureas	Rimon (Novaluron)
17 Moulting disruptors, Dipteran	Cyromazine	Citation (Cyromazine)
18 Ecdysone receptor agonists Growth regulation	Diacylhydrazines	Intrepid (Methoxyfenozide)
20 Mitochondrial complex III electron transport inhibitors Energy metabolism	**20B** Acequinocyl	Kanemite (Acequinocyl)
	20D Bifenazate	Acramite (Bifenazate)

Table 10. Classification of insecticides according to mode of actions *(continued)*

Main group and primary site of action	Chemical sub-group or exemplifying active ingredient	Examples of trade name (Active ingredient)
21 Mitochondrial complex I electron transport inhibitors Energy metabolism	**21A** METI acaricides and insecticides	Nexter (Pyridaben)
23 Inhibitors of acetyl CoA carboxylase Lipid synthesis, growth regulation	Tetronic and Tetramic acid derivatives	Movento (Spirotetramat)
25 Mitochondrial complex II electron transport inhibitors Energy metabolism	**25A** Beta-ketonitrile derivatives	Nealta (Cyflumetofen)
28 Ryanodine receptor modulators Nerve and muscle action	Diamides	Altacor (Chlorantraniliprole)
29 Chordotonal organ Modulators — undefined target site Nerve action (distinct from Group 9)	Flonicamid	Beleaf (Flonicamid)
NC Not classified	N/A	Opal Insecticidal Soap (Potassium salts of fatty acids)

Table 11. Classification of herbicides according to mode of actions:

WSSA and HRAC codes are included along with the mode of action, specific chemical groups and an example of a commercial product. (Note: only groups for which there is at least one commercial product registered in field, vegetable or fruit crops in Ontario are included.)

WSSA Group	HRAC Group	Mode of action	Chemical family (Group)	Example of commercial product (Active ingredient)
1	A	Inhibition of acetyl CoA carboxylase (ACCase)	Aryloxyphenoxypropionate ‘FOPs’	Excel Super (Fenoxaprop-ethyl)
			Cyclohexanedione ‘DIMs’	Poast Ultra (Sethoxydim)
			Phenylpyrazoline 'DEN'	Axial (Pinoxaden)
2	B	Inhibition of acetolactate synthase ALS (acetohydroxyacid synthase AHAS)	Sulfonylurea ‘SUs’	Accent (Nicosulfuron)
			Imidazolinone ‘IMIs’	Pursuit (Imazethapyr)
			Triazolopyrimidine	FirstRate (Cloransulam)
			Pyrimidinyl(thio)benzoate	Velocity (Bispyribac)
			Sulfonylaminocarbonyl-triazolinone	Vios G3 (in part) (Thiencarbazone)
3	K1	Microtubule assembly inhibition	Dinitroaniline	Prowl (Pendimethalin)
			Benzamide	Kerb (Pronamide)
			Benzoic acid	Dacthal (Chlorthl-dimethyl)
4	O	Action like indole acetic acid (synthetic auxins)	Phenoxy-carboxylic-acid	2,4-D
			Benzoic acid	Banvel (Dicamba)
			Pyridine carboxylic acid	Lontrel (Clopyralid)
			Cyclopropylpyrimidine	Method (Aminocyclopyrachlor)
5	C1	Inhibition of photosynthesis at photosystem II	Triazine	AAtrex (Atrazine)
			Triazinone	Sencor (Metribuzin)
6	C3	Inhibition of photosynthesis at photosystem II	Nitrile	Pardner (Bromoxynil)
			Benzothiadiazinone	Basagran (Bentazon)
7	C2	Inhibition of photosynthesis at photosystem II	Urea	Linuron

Table 11. Classification of herbicides according to mode of actions

(continued)

WSSA Group	HRAC Group	Mode of action	**Chemical family** (Group)	**Example of commercial product** (Active Ingredient)
8	N	Inhibition of lipid synthesis — not ACCase inhibition	Thiocarbamate	Eptam (EPTC)
			Benzofuran	Nortron (Ethofumesate)
9	G	Inhibition of EPSP synthase	Glycine	Roundup (Glyphosate)
10	H	Inhibition of glutamine synthetase	Phosphinic acid	Liberty (Glufosinate)
13	F4	Bleaching: Inhibition of DOXP synthase	Isoxazolidinone	Command ME (Clomazone)
14	E	Inhibition of protoporphyrinogen oxidase (PPO)	Diphenylether	Reflex (Fomesafen)
			Phenylpyrazole	Blackhawk (in part) (Pyraflufen-ethyl)
			N-phenylphthalimide	Valtera (Flumioxazin)
			Triazolinone	Authority (Sulfentrazone)
15	K	Inhibition of very long chain fatty acid synthesis	Chloroacetamide	Dual (Metolachlor)
			Acetamide	Devrinol (Napropamide)
			Oxyacetamide	Define (Flufenacet)
19	P	Inhibition of auxin transport	Phthalamate Semicarbazone	Distinct (in part) (Diflufenzopyr-Na)
21	L	Inhibition of cell wall (cellulose) synthesis	Benzamide	Gallery (Isoxaben)
29			Fluoroalkyltriazine	Allion (Indaziflam)
22	D	Photosystem-I-electron diversion	Bipyridylium	Reglone (Diquat)
27	F2	Bleaching: Inhibition of 4-hydroxyphenyl-pyruvate-dioxygenase (4-HPPD)	Triketone	Callisto (Mesotrione)
			Isoxazole	Converge (Isoxaflutole)
			Pyrazolone	Armezon (Topramezone)

Overall, these classification systems are very pertinent for growers or anyone involved in agriculture, but there could be instances where a pest exhibits multiple resistances across different groups listed in a table. In this case, the value of this classification system is diminished. This is why it is important to emphasize that these classification systems are not aimed to be used as a resistance-risk rating but as a tool used by the grower or Crop Advisor on the best choice of the different pesticides according to their MoA. This allows tank mixtures and rotations of active ingredients to work in harmony.

4. Discuss an IPM framework that includes multiple effective sites/ mechanisms of action or tools to delay resistance development.

- Consider all chemical control options before planting, in-crop and after harvest.
- Read and carefully follow pesticide label instructions, such as application rates, timing and equipment recommendations.
- Be well-acquainted with the weeds, insects or pathogens present in the grower's fields and nearby non-crop areas to tailor a control program according to pest densities and economic thresholds.
- Consider options for minimizing insecticide or fungicide use by planting early-maturing or pest-tolerant varieties.
- Use any pesticide products at their full, recommended doses. Using reduced doses quickly select populations with average levels of tolerance. On the other hand, doses that are too high may impose excessive selection pressures.
- Proper and well-maintained equipment should be used to apply any pesticides. Recommended dilutions, spray pressures and optimal environmental conditions should be respected to obtain optimal coverage.
- Adhere to label guidelines for alternations or sequences of different classes of pesticides with different MoAs as part of an Integrated Pest Management (IPM) strategy.
- In the event that multiple applications per year or growing season are required, alternate products of different MoA classes.
- Pesticide mixtures may offer a short-term solution to delay resistance development, but it is imperative that each component of a mixture belongs to a different pesticide MoA class, and that each component is used preferably at its full rate.
- The sole use of pesticide mixtures or pesticide rotations is not acceptable to delay resistance. There must be a diversification plan that also incorporates mechanical, cultural and biological practices (see next section).
- As much as possible, select pesticides and other pest management tools that preserve beneficial organisms.
- When there is pest control failure, do not re-apply the same pesticide but change the class (group) of pesticide to one having a different MoA and to which there is no known cross-resistance in the neighbouring area.
- Regular monitoring for incidence of resistance in large agricultural areas and assessing levels of control obtained after any applications is central to an effective IPM framework.

- Avoid using a product to which resistance has developed until susceptibility returns. This is a valid approach if other alternative chemical classes are available to provide effective pest control.
- Maintain detailed field/greenhouse records and use of fungicides, insecticides or herbicides every growing season.
- Crop Advisors should be familiar and up to date on current recommendations on IPM programs for pathogens, insects and weeds.

COMPETENCY AREA 2

Resistance Management

1. Develop a resistance management plan:

a) Assessment/scouting pre- and post-treatment;

b) Identification;

c) Control methods:

   i) Biological

   ii) Chemical

   iii) Cultural

   iv) Mechanical;

d) Sanitation;

e) Reporting, evaluation and follow-up.

a) Assessment/scouting pre- and post-treatment

Timely pest scouting (or monitoring) helps establish a management program tailored to the specific needs of a field. Scouting allows you to identify (1) pest species in the field; (2) their location within the field; and (3) to evaluate the severity of the infestation.

Scout early and often: In general, pests become more difficult to control as they get larger or more mature. Scouting should start early and be done regularly throughout the growing season in order to catch weeds or insects when they are young or at the larvae stage. Insecticides or herbicides are often most efficient when used on young pests. For example, harder to control weeds such as Canada fleabane or waterhemp must be sprayed before they reach four inches tall in order for the herbicides to be most effective.

Scouting procedure:

Different procedures exist for scouting depending on crops and type of pests. The Ontario Certified Crop Advisor Study Guide, Section 3 on Integrated Pest Management, gives a detailed description on how to perform scouting according to pests (pp. 121-128). Other websites are also useful such as the Integrated Weed Management (IWM) Resource Center (http://integratedweedmanagement.org) and the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA) scouting guidelines for common potato insects (http://www.omafra.gov.on.ca/english/crops/facts/potato_insects.htm#cpb) or insects in orchards (http://www.omafra.gov.on.ca/IPM/english/apples/ipm-basics/how-to-scout.html).

For **weeds**, fields should be monitored before planting:

- To ensure effective elimination of problem weeds before plants become too large to control with chemicals.
- To assess the need for alternative pre-plant weed management strategies such as tillage.
- To make sure that crop seeds are planted into weed-free fields.

Scout for **insects** and **diseases** in both Bt and non-Bt (refuge) crops looking for signs of suspicious feeding activity by the target pests. Scouting in both Bt and non-Bt fields is a crucial part of a resistance management plan. According to scientists across North America, European corn borer (ECB) and corn rootworm (CRW) could develop resistance to Bt crops under conditions of continued use. Scouting is also important to monitor for pests and diseases not controlled by Bt, such as wireworm, white grubs, seed corn maggot, seed corn beetle, flea beetles, aphids, mites, stalk rots and leaf diseases.

Scouting post-treatment is equally as important as the pre-treatment monitoring to determine if the pesticide application was efficient and to decide if other treatments will be required. The same scouting procedure should be done post-treatment and records should be compared with the ones recorded in pre-treatment. Never assume your pesticide application has provided adequate control; this is why post-treatment scouting is so important in a resistance management plan.

Scouting for resistance:

In general, scout for pest species that are known to have developed pesticide resistance in Ontario. This is also true if there are reported resistance cases in the surrounding provinces or just south of the border. For weeds, refer to Table 1 for a current list of herbicide-resistant weeds in Ontario. To stay up to date, please visit The International Survey of Herbicide Resistant Weeds website (http://www.weedscience.org/default.aspx). This site is a collaborative effort between weed scientists in over 80 countries and includes reports of any new cases of weed resistance. This site can be searched by countries: by clicking "Canada" users can quickly find detailed information about new resistant weeds reported in the country and in Ontario. The website was created to maintain scientific accuracy in the reporting of herbicide-resistant weeds globally and is funded by HRAC and CropLife International.

In the event of control failure:

Following post-treatment scouting, an observed failure of a pesticide application to control a problem pest does not necessarily mean that resistance is present. While genetic resistance must be confirmed through laboratory testing, there are other signs that may indicate potential resistance:

- Spreading area of a single pest species.
- Failure of a pesticide application to control a pest that was usually effective in the past, especially if other surrounding pest species are successfully controlled.
- Single pest species left standing (i.e, unaffected or injured pests that have resumed growth or normal behaviour and are standing beside dead ones).

b) Identification

To identify resistance, a detailed review of the field history needs to be conducted. The questions listed on page 28 (Process for identifying pest resistance) will help the Crop Advisor and grower decide if they are facing a case of pest resistance, and if samples should be sent to an appropriate lab for further screening and confirmation.

c) Control methods

A combination of control tactics to manage and prevent any further cases of resistance will achieve more acceptable and sustainable results than the use of a single approach. In a resistance management program, the different approaches will encompass chemical, cultural, mechanical and biological methods in an integrated fashion to control the problematic pest. It is critical to not rely excessively on one method in particular over the others. An overall approach of the following tools will support a reduction in selection pressure and eliminate the dominance of resistant pests.

i) Biological:

Biological control simply means the control of pests and diseases using natural enemies. Three groups of beneficial organisms can be distinguished:

Predators: predatory mites, bugs, beetles and gallmidges

Parasites: parasitic wasps and parasitic flies

Micro-organisms: nematodes, fungi, bacteria and viruses

It is essential to consider the following points when using beneficials:

- Become familiar with the biology of your pest and its natural enemies.
- The earlier beneficials are introduced, the better the effect. Begin management in a period where the incidence of pest infestation is still relatively low. By doing this, the use of pesticides can be limited and natural enemies will have a greater chance of establishment in the crop.
- Quality of the material is crucial. Buy the material from a recognized supplier who guarantees its quality and quantity.
- Follow storage temperature and use before expiry date.
- Assist natural enemies to enter the crop by creating optimal conditions for which they can thrive.
- Make use of refuge plants when possible. They can be good sources of predators and parasites.
- Be mindful of the fact that any action done on the crop (e.g., harvesting, pruning, spraying, etc.) should not extensively reduce the population of beneficials or other natural enemies.
- In particular to weeds: when possible, introduce post-harvest grazing of remaining weeds to reduce overall seedbank.

ii) Chemical:

Any pesticide application falls under chemical control. When used within a context where resistance management is the goal, pesticides should be relied upon as a corrective measure and seen as part of a multi-approach solution and not as the only solution to rely on. It is recommended to keep the following in mind when using pesticides in a resistance management plan:

- Always give priority to a selective pesticide when choosing a chemical for spraying. In general, selective pesticides are designed to be non-toxic or very slightly toxic to beneficials present in the environment. They usually do not affect their development and/or reproduction.

- Whenever possible, choose an application technique that will not greatly affect any beneficial populations. Even if a pesticide is not safe for a beneficial population, the manner chosen to apply this pesticide could greatly reduce any negative effect it could have on the population – for example, by using a drip system or planting seeds treated with a pesticide coating or restricting application to young plants, etc.
- Where practical, use spot treatments, barrier treatments or banded treatments to better target pest populations or the zone where pest control is required.
- Select a pesticide with a short persistence. Some chemicals can be very toxic at the time of application, but their effect does not last long in the environment. Not long after, it is possible for beneficials or natural enemies from surrounding areas to come back and re-populate the treated field. In contrast, pesticides with long persistence in the field tend to inhibit the development or thriving of beneficials and natural enemies over a long period of time.
- If the use of a pesticide with a long persistence is required, avoid its application just prior to the use of a biological control.
- Keep in mind that pesticides or their vapours may drift from the area of application into another field under biological control.

iii) Cultural:

Cultural control means the use of agricultural practices that have a positive effect on crop protection by disrupting the environment of the pest, and/or prevent its movement. Good cultural practices aim to increase the resistance or competiveness of the plant to pests, diseases and weeds. The following conditions will help to achieve this goal:

- Optimize plant growth: a crop is more resistant to pest damage if factors such as fertilization, irrigation, pH, CEC, climate, etc. are optimal for growth. Plants are more prone to diseases or are less competitive when they are under stress. They become more vulnerable when growth factor is not optimal.
- When possible, choose crop varieties that are resistant (or more tolerant) to pest attacks.
- When using a susceptible crop, grow it preferably in a time of reduced infestation of a problematic pest.
- Altering the crop planting date, row spacing and harvest timing help to disrupt the weed's life cycle or the reproductive cycle of other pests. For example, increasing field crop density can make them more competitive against weeds; however, under some circumstance too high a crop density can promote thin and weak plants that are in turn susceptible to pests and diseases.
- Purchase of certified seeds, free of weed seeds.
- Use of cover crops to suppress weeds and encourage beneficial insects.
- In horticulture, harvesting at irregular intervals weakens plants and makes them more vulnerable to pests.
- Crop rotation is key to a good cultural approach. When crops susceptible to particular pests are rotated with crops affected by other pests, the overall level of infestation will remain relatively low.

iv) Physical (including mechanical):

The use of control methods based on physics (i.e., methods relying on energy and force) are among the most ancient methods that have been used to control pests. These include various alteration of energy (heat) or movement (traps, mulches) as well as the use of various machines and tools (mechanical control). Several physical methods can be employed to eliminate problematic pests. The choice will be made according to the crop, the environment, the geographical conditions and also the economical aspect of each method available. Mechanical control involves the use of any devices or machines to control pests or alter their environment. Traps, screens, barriers, fences and nets are all examples of devices used to prevent pest activity or remove pests from an area.

- Trap insects: By using sticky traps, trap plants, pheromone traps, etc., it is possible to trap winged insects and decrease the level of infestation and reproduction. The use of special nets also trap many insects such as aphids, butterflies, capsids, flies, thrips, etc.
- Solarization: This is an effective method to control weeds, insects, nematodes and other diseases. According to the University of California (Division of Agriculture and Natural Resources), soil solarization is a non-pesticidal method of controlling soil-borne pests by placing plastic sheets on moist soil during periods of high ambient temperature. The plastic sheets allow the sun's radiant energy to be trapped in the soil, heating the upper levels. Solarization during the hot summer months can increase soil temperature to levels that kill many disease-causing organisms (pathogens), nematodes, and weed seed and seedlings. This method leaves no toxic residues and can be easily used on a small or large scale. In general, strip coverage is more practical and economical than full coverage and works well against weeds. For nematodes or other pathogens, full coverage might be preferred in the event of a bad infestation.
- Removal of infested plant materials or weeds: manual removal of diseased plants or hand-weeding or ploughing/tillage help control newly emerged plants. Place them in a bag and destroy.
- Flaming or burning: These are control methods that eliminate pests or breeding sites. Overwintering bugs hiding in grasses or cereal stubbles can be eliminated by these methods prior to planting a new crop.
- Steaming or pasteurizing of soil or growing media in greenhouse production allows the elimination of soil-borne diseases and insects.
- Other mechanical practices include new emerging methods such as the use of seed destructors, robotic weed control, chaff carts, interrow precision weeding or hoes equipped with tools for intra-row weeding, etc.

d) Sanitation:

Crop sanitation aims at preventing or eliminating any sources or vectors of pests and diseases. Good sanitation reduces the population of harmful pests, which in turn decreases the need to use chemical pesticides heavily and increase the selection pressure. It also helps any biological control set in place to be more efficient.

Often the reason a problematic or resistant pest spreads into a neighbouring field is due to a lack of sanitation. The use of farm machinery or other equipment can be a constant source of contamination from one field to the next. Sometimes, unbeknownst to field workers, farm equipment can be a very efficient method of transport for weed seeds and other pathogens. Due to soiled equipment, pests can easily travel long distance and populate other fields.

The following guidelines can assist any growers achieve a high level of sanitation on the farm:

The culture:

- Begin with a clean growing area. The soil or any other substrate like an orchard should not be infested heavily with a particular pest. This is why crop rotation and/or other control methods are strongly advised.
- Buy clean plant material: for crops or in horticulture, purchase seeds or seedlings that are free of weed seeds or other pests.
- Check the culture regularly throughout the whole growth cycle to detect any pest population that could be on the rise and turn into a bad infestation.
- Keep weeds under control. Not only because they affect crop yield but they can also be hosts for pests and diseases and may carry over an infestation from an old to a new culture.
- Avoid any damage to the main crop as much as possible. Damaged plants are an easy mean of entry for certain pest infestations.
- When appropriate, remove and discard old plant material. Do not accumulate heaps of waste or other possible sources of infestation.
- Organize work on the farm. Any movement of people or equipment should always be done from a clean area/field towards the infested areas of the crop.

The equipment:

Various types of farm equipment may come into contact with soil, seed and/or crop debris that are carriers of weeds, insects, fungi or any other pathogens.

COMMON FARM EQUIPMENT THAT CAN BE CONTAMINATED WITH PESTS

- **Tillage equipment (plows, cultivators, discs, rippers, harrows)**
- **Fertilizer and pesticide applicators**
- **Seeders (drills, planters)**
- **Tractors, grain trucks, pickups, cars and ATVs**
- **Swathers and combines**
- **Grain handling equipment (augers, dryers)**
- **Forage harvesting equipment**
- **Miscellaneous equipment used for soil sampling, trenching, clearing brush, etc.**

- Remove soil and plant debris from all equipment by scraping, brushing or knocking off clumps.
- Any residual soil and small debris can be cleaned from surfaces by pressure washing, steaming or compressed air.
- For pathogens and diseases, a disinfectant (diluted bleach) sprayed over clean surfaces can help to kill any remaining pests.
- In a similar fashion clean any clothing, boots, shoes or gloves. They can also be an important source of contamination.

e) Reporting, evaluation and follow-up

CCAs should report to their employers and the farmer all his/her findings and advise on the most appropriate resistance management plan to follow in order to minimize any additional risks. Confirmation of resistance should be treated with the utmost confidentiality. Once the management plan is underway, the CCA should follow up with the producer to ensure that the recommendations are followed and give the expected results. The advisor should also ensure that any modifications to local, regional or regulatory conditions that may affect the effectiveness of the plan be taken into consideration and alternative measures are implemented when deemed necessary, after consultation with the producer. The progress of the management plan should be evaluated during the following months and even years to ensure that the conditions that led to the development of the problem do not occur again. Evaluations should be documented and resistance management plans updated if the need arises. Resistance management should be a dynamic and constantly changing approach in order to be efficient and practical for the farmer.

2. Discuss the roles that local situations and needs play in the development of resistance management plans.

Pesticide registration

Governments may decide to put restrictions on the use of certain pesticides and this could have impacts on resistance management plans. The main impact is the loss or limitation in the availability of certain products. These decisions are generally taken at the federal level, as the PMRA is responsible for pest control product registration in Canada. Provincial, and sometimes regional or municipal authorities have put restrictions on the use of some pest control products. In general, these restrictions are based on concerns with the environment and/or human health. They can, however, have profound effects on resistance management by limiting the availability of certain products, which results in less choice in the arsenal of chemical tools available for pest management.

Buffer zones and vegetation strips

Buffer zone requirements on pesticides exist to limit negative impact on sensitive environments such as watercourses. Preservation of buffer zones may have an impact on resistance management in many ways. Untreated areas may serve as areas sheltering weeds or insect pests that would not be controlled and could potentially infest the field at a later stage. It has also been speculated that for some weeds, the presence of buffer zones may accelerate resistance development. While the buffer zone itself is not subject to direct pesticide application, there is a possibility that pesticide molecules reach it. Under optimal spraying conditions, the interface between the sprayed field and the unsprayed buffer zone sees a band where the pesticide amount gradually goes from the full dose to zero. Within that band, weeds, insects or diseases will be subjected to below recommended rates that would select for minor resistance genes and polygenic resistance (refer to Proficiency Area I — Genetics of Resistance). In cross-fertile organisms, survivors will combine these minor genes resulting in progeny with increased level of resistance that could eventually be resistant enough to survive full dose application in the field. In Ontario, it has been speculated that giant ragweed, a weed naturally abundant in ditches, has developed glyphosate resistance through exposure to sub-lethal doses of glyphosate. Individuals, carriers of low-level resistance mechanisms, exchanged genes resulting in fully resistant plants that eventually invaded fields, being able to survive a full glyphosate dose.

Community

For producers, and to some extent their advisors, knowledge of any specific situations at the community level can have a major influence in the decision to implement certain practices or not. For example, the confirmation of resistance in neighbouring properties and operations would more likely modify how a producer would tackle resistance management compared to a situation where resistance is not present in the area. In the former, producers are more likely to be receptive to adopt proactive measures as the local situation brings a constant reminder of the potential impact of resistance.

3. Discuss the effects of resistance BMPs on stewardship and production issues involving the following:

a) Soil conservation stewardship practices;

b) Surface water and groundwater quality protection practices;

c) Species at risk;

d) Reduction/mitigation of off-target impacts to pollinators and beneficial organisms.

a) Soil conservation stewardship practices

Soil conservation practices are used on the farm to diminish soil degradation and build organic matter. The main practices used are crop rotation, mulching, cover cropping and reduced tillage.

Crop rotation, mulching and cover cropping all increase soil organic matter, soil structure, rooting depth and pest biodiversity for insects and soil microorganisms. These are all advantages that are very much in line with the use of resistance BMPs to favour a healthy crop while encouraging biodiversity to avoid single pest infestation and therefore an excessive use of pesticides.

Reduced tillage is a conservation practice that is also very much promoted to decrease soil erosion and compaction. Tillage, be it primary tillage such as the use of the moldboard plough, or secondary tillage as done by disks or tine cultivators, has many roles and benefits including weed control. Reducing tillage, while beneficial for soils, precludes one of its benefits, namely its impact on weed populations. Attempts to encourage soil conservation either by promoting direct seeding in undisturbed soil or by reduced tillage techniques that leave 30 percent or more residue have been acceptable to growers only with the availability of highly effective herbicides. The important reduction in the use of tillage from the system means a replacement approach must be found to control weeds and this has always been done by herbicide application. To be effective, most conservation tillage systems will require the use of burndown herbicides, most often glyphosate, plus the application of in-crop selective products. Whereas these tools allow great weed control while enhancing soil conservation, they will, if used improperly, select for resistance in targeted weed species. As cases of resistance to herbicides have steadily increased in the past decades, consideration has been given to alternative methods. In order to reduce the reliance on herbicides, and diminish the selection pressure they impose, some agronomists have proposed a return to some form of tillage to control resistant weeds or to prevent their further selection. This has been met with a great measure of skepticism by members of the soil science community, but this is the reality we are facing. It is therefore paramount for crop advisors to explore any avenues that will allow reduction of selection pressure without compromising the gains of conservation tillage. Combining some form of reduced tillage that leaves sufficient residues on the soil with the planting of cover crops to provide soil coverage and reduce weed development is an example of practices that should reduce herbicide reliance and promote soil conservation. More details can be found at: http://www.cast-science.org/download.cfm?PublicationID=274097&File=1e30c0f2e3b1b907dc442c724c53e2e5f1f5TR or at http://www.cast-science.org/download.cfm?PublicationID=52723&File=7F1320FC85FB762965336E0A16948F7C.cfusion.

b) Surface water and groundwater quality protection practices

Pesticides can reach ground and surface water through various routes:

- **Surface water runoff** carries pesticide molecules off site in water. Soluble pesticides can be carried as they are dissolved in water and insoluble pesticides will be moved as suspension if they are adsorbed on soil particles. Once in the water runoff, these pesticides can rapidly reach and contaminate ponds, lakes and rivers.
- **Erosion:** heavy rains or excess irrigation can cause the movement of soil particles from the application site. If the pesticide molecule is adsorbed to the soil particles, the pesticide is also being moved off site and potentially to watercourses.
- **Leaching:** the movement of pesticides into the soil profile is governed by multiple factors:
    - Water solubility of the pesticide
    - Amount of rainfall or irrigation
    - Soil texture
    - Organic matter content
    - Adsorption potential of the pesticide molecule
    - Amount of pesticide applied

A pesticide molecule with a high water solubility and low adsorption potential that is applied at a high rate on a soil with a coarse texture and low organic matter has great potential to reach the ground water, especially under high rainfall conditions. Conversely, a pesticide that is highly adsorbed to soil colloids and that is applied at low rates has lesser chance of leaching especially on soils with high organic matter and/or fine texture. Degradation of the pesticide in the soil, especially by microorganisms also diminishes the potential for leaching into the water table.

Many BMPs that prevent resistance may also have a positive impact on water quality protection:

- Overall reduction in pesticide use to diminish selection pressure also lessens the potential for runoff, erosion or leaching movement of pesticides to ground and surface water;
- Cultural practices that increase soil health and biodiversity such as reduced tillage and the use of cover crops will increase pesticide degradation through increased microbial degradation. These practices can also prevent movement of pesticides by runoff or erosion.

When considering alternative pesticides in a rotation, take care to favour the product that has the lowest environmental impact without sacrificing efficacy. Environmental Impact Quotient (EIQ) is a metric that allows the comparison of multiple products for their environmental effects. More information on EIQ can be found at https://nysipm.cornell.edu/eiq/.

c) Species at risk

BMPs for resistance management can have positive or negative impact on species at risk.

If resistance BMPs result in less pesticide use, then no negative impact on species at risk is expected. In cases when resistance BMPs require the replacement of a pesticide for which there is resistance by one that has a higher toxicity to species at risk, then a negative impact could possibly happen. As mentioned, a more toxic pesticide that does not last long in the environment can be an advantage over a less toxic one with a long window of degradation. The CCA should always evaluate the pros and cons of using a more or less toxic option depending on the grower's situation.

d) Reduction/mitigation of off-target impacts to pollinators and beneficial organisms

BMPs for resistance management can have positive or negative impact on pollinators and beneficial organisms.

If resistance BMPs result in less pesticide use, then no negative impact on pollinators and beneficial organisms is expected.

Negative impacts could be expected if resistance BMPs require the replacement of the pesticide for which there is resistance by one that has higher toxicity to pollinators and beneficial organisms. Again, a more toxic pesticide with a short residual effect can be an advantage over a less toxic one with a long window of degradation. It is imperative for the CCA, in consultation with the grower, to evaluate the pros and cons of using a more or less toxic option depending on the grower's situation.

In addition, cases may arise when a producer wants to maintain biodiversity in field margins, buffer zones, ditches, etc. While this is commendable and helps multiple species including pollinators and beneficial organisms, this method requires constant monitoring and evaluating. For example, a buffer zone may become infested by a species that has resistance and to leave it there would provide a point of origin for the pest infestation (e.g., insects, spores or seeds) to move to the adjacent field and spread further. These events should be evaluated on a case-by-case basis and the CCA should not hesitate to consider all facets of the problem when advising a producer.

PROFICIENCY AREA III
PROFESSIONAL COMMUNICATION AND SHARING INFORMATION

COMPETENCY AREA 1

Communication and Resistance Management

1. Discuss why it is critical and how to identify and report resistance issues.

The number of pesticide-resistant pest species worldwide has increased considerably in the past 20 years with new cases reported on a regular basis. Resistance is now a game changer for agriculture. It is a threat to the way farming is done and it needs to be addressed head-on by everyone involved in the agricultural sector. Reporting new cases of resistance is crucial. By doing so, governments, industries and farm owners become aware of the changes in their areas and/or provinces; best management practices can then be implemented more efficiently in order to address the problem as soon as possible and make sure it remains manageable. Early reporting of new cases allows regulatory authorities and funding agencies to alter their policies and funding priorities, hopefully allowing for more rapid solutions to the problem. Clear identification of resistance is critical for producers, the environment and society as it helps prevent excessive use of ineffective pesticides. In Proficiency Area 1 (Identifying Resistance), the process to identify and report resistance is described. The CCA has a key role in sharing any new information concerning resistance management since he/she is often the main point of contact between growers and other organizations (e.g., governments, public agencies and private organizations).

2. Identify the benefits of active networks, up-to-date information systems and other available communications tools.

Having access to active networks, up-to-date information systems, public websites or attending farmers' workshops conducted by the industry or government can be a great benefit to farmers and CCAs – this can never be emphasized enough. Identifying resistance in one's own field does not make someone a bad farmer. Pesticide resistance and its management is a complex system –

awareness is critical. The more that information is given through workshops and farm events or read online through websites hosted by OMAFRA, FRAC, WSSA, HRAC, IRAC or PMRA, etc., the sooner growers will realize that pesticide resistance isn't going away. It is definitely an aspect of farming that growers are going to have to deal with for a number of years to come. The main benefits for the CCA having access to a wide range of information are:

- Increase pesticide resistance awareness among the different stakeholders;
- Stay up to date on any situation concerning new cases of resistance;
- Learn about ways to manage or delay resistance;
- Become aware of the socio-economic aspects of resistance management; and
- Project a positive image of the agri-food sector.

3. Discuss the role of public agencies, non-profit and private organizations in advocating for resistance management approaches that are sustainable within an IPM framework.

The growing incidence of pests developing resistance is a critically important issue for all stakeholders in the agricultural sector including growers, landowners, CCAs, researchers, pesticide product regulatory authorities and the crop protection industry. Resistance threatens sustainable agricultural systems globally. Since pesticides provide effective and efficient crop protection, the longevity of their use must be preserved in order to avoid increases in food production costs that would eventually negatively impact consumers. Resistance is a costly problem; when it is identified in a growing area, other control measures need to be used that might be more expensive and less effective. All these repercussions are valid reasons and strong incentives to advocate resistance prevention and management approaches that are sustainable. Each stakeholder in the agriculture sector has a responsibility to share information about RM and increase awareness that it is a problem that will not go away on its own.

There are several reasons why many farmers have not yet adopted any pesticide resistance management plans. The main ones are:

- There is a lack of information;
- The management plans may look too complex;
- Their implementation is time consuming; and, most importantly,
- The economics is not favourable (i.e., RM seems too costly).

Most growers are worried about the short-term cost of implementing a resistance management plan especially if they are uncertain about the future benefits. All stakeholders need to cooperate to convey a consistent message about being proactive on the issue of delaying resistance in order to avoid the greater and more difficult problem of trying to eliminate it after it has appeared and spread. This latter alternative, corresponding to reactive management, comes at a greater social and financial cost than proactive management or prevention.

4. Discuss the importance of communicating integrated approaches that impact resistance with various stakeholders:

a) the public;
b) landowners;
c) producers;
d) news outlets.

a) the public

The general public has become more and more demanding, if not suspicious, of the agricultural sector over the last few years. This stems from concerns over pesticide usage, sustainability, and other environmental impacts of agriculture. By proactively addressing potential resistance issues and promoting sustainable management approaches, CCAs can contribute to the promotion of a responsible agricultural industry receptive to the general public possible misgivings.

b) landowners

In the case of leased lands, landowners may become apprehensive when resistance appears in their fields, as it can be a sign that the tenant may not be taking good care of the land. In the event where resistance problems become unmanageable, land value could decrease dramatically as it will be more difficult to farm or it becomes a source of contamination for neighbouring fields (e.g., equipment contamination, wind dispersal, etc.). Tenants, for instance, would be unlikely to bid high for land that is infested with a multiple-herbicide-resistant weed as it would require extra management costs. Promoting and communicating integrated pest management practices that prevent resistance development are beneficial for landowners as this will encourage maintaining healthy land values and sustainable and environmentally beneficial practices.

c) producers

When facing resistance, producers initially seek the simplest solution, which is often the use of an alternative pesticide product. While this may present a short-term benefit, it often leads to complications as multiple-resistance may develop in the long term. It is key to inform growers early on of the importance of integrated approaches in order to help sustain a productive agricultural sector.

d) news outlets

The media has an essential role in promoting the benefits of resistance management best practices and the proactive approach various stakeholders take in sustainably preventing and managing resistance. Various media exist nowadays that can be used to diffuse this type of information. Social media such as Twitter, Facebook and Instagram provide rapid response to enquiries and allow CCAs to obtain feedback on immediate issues. Social media can also help in directing users to sources that provide more permanent, precise and stable information such as dedicated websites and blogs. A great number of media specializing in agricultural issues can also channel information directed to producers, advisors, grower groups, government officials and industry partners. The media has the power to sway public opinion, hence to influence decision makers in their regulatory and funding decisions. This can ultimately affect the agricultural sector in a positive or negative manner.