

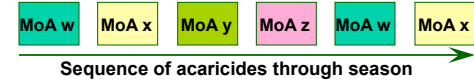
Acaricide Mode of Action Classification: A key to effective acaricide resistance management

Introduction

IRAC promotes the use of a Mode of Action (MoA) classification of insecticides and acaricides as the basis for effective and sustainable resistance management. Acaricides are allocated to specific groups based on their target site. Reviewed and re-issued periodically, the IRAC MoA classification list provides farmers, growers, advisors, extension staff, consultants and crop protection professionals with a guide to the selection of acaricides and insecticides in resistance management programs. Effective Resistance management of this type preserves the utility and diversity of available acaricides. A selection of relevant MoA groups is shown below.

Effective IRM strategies: Sequences or alternations of MoA

All effective pesticide resistance management strategies seek to minimise the selection of resistance to any one type of pesticide. In practice, alternations, sequences or rotations of compounds from different MoA groups provide sustainable and effective resistance management for acarine pests. This ensures that selection from compounds in the same MoA group is minimised, and resistance is less likely to evolve.



Applications are often arranged into MoA spray windows or blocks that are defined by the stage of crop development and the biology of the pest species of concern. Local expert advice should always be followed with regard to spray windows and timings. Several sprays may be possible within each spray window but it is generally essential to ensure that successive generations of the pest are not treated with compounds from the same MoA group. Metabolic resistance mechanisms may give cross-resistance between MoA groups, and where this is known to occur, the above advice must be modified accordingly. IRAC also provides general recommendations for resistance management tactics regarding specific MoA groups.

Nerve and Muscle Targets

Several current acaricides act on nerve and muscle targets. Acaricides that act on individual targets in this system are generally fast acting.

Group 1 Acetylcholinesterase (AChE) inhibitors

Inhibit AChE, causing hyperexcitation. AChE is the enzyme that terminates the action of the excitatory neurotransmitter acetylcholine at nerve synapses.
1A Carbamates (e.g. Methomyl), **1B** Organophosphates (e.g. Pirimiphos-methyl).

Group 2 GABA-gated chloride channel antagonists

Block the GABA-activated chloride channel, causing hyperexcitation and convulsions. GABA is the major inhibitory neurotransmitter in insects.
2A Cycloodiene Organochlorines (e.g. Endosulfan).

Group 3 Sodium channel modulators

Keep sodium channels open, causing hyperexcitation and, in some cases, nerve block. Sodium channels are involved in the propagation of action potentials along nerve axons.
3A Pyrethroids, Pyrethrins (e.g. Bifenthrin, Halfenprox).

Group 6 Glutamate-gated chloride channel (GluCl) allosteric modulators

Allosterically activate glutamate-gated chloride channels, causing paralysis. Glutamate is an important inhibitory neurotransmitter in insects.
Avermectins, Milbemycins (e.g. Abamectin, Milbemectin).

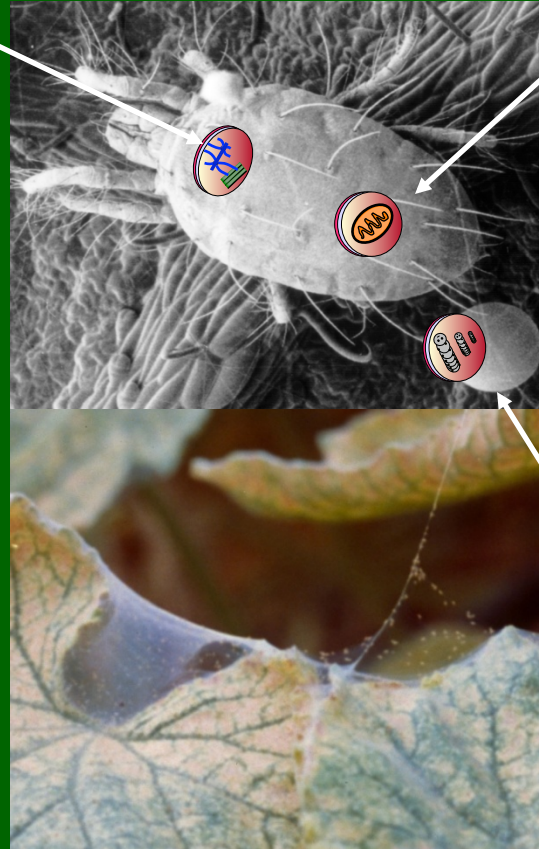
Group 19 Octopamine receptor agonists

Activate octopamine receptors, leading to hyperexcitation. Octopamine is the insect equivalent of adrenaline, the fight-or-flight neurohormone.
Formamidines (e.g. Amitraz)

Acaricides for which the mode of action is unknown

These compounds are not classified because there is not sufficient information available on their mode of action.

Benzoximate, Bromopropylate, Chinomethionat, Dicofof.



Respiration Targets

The mitochondrial respiration process produces ATP, which energizes all vital cellular processes. In mitochondria, an electron transport chain uses the energy released by oxidation to drive ATP synthesis. Several acaricides are known to interfere with mitochondrial respiration by the inhibition of electron transport and/or oxidative phosphorylation, and are generally fast to medium-fast acting.

Group 12 Inhibitors of mitochondrial ATP synthase

Inhibit the enzyme that synthesizes ATP.
12A Diafenthiuron, **12B** Organotin miticides (e.g. Azocyclotin, Fenbutatin oxide), **12C** Propargite.

Group 13 Uncouplers of oxidative phosphorylation via disruption of the proton gradient

Protonophores that short-circuit the mitochondrial proton gradient so that ATP can not be synthesized.
Pyroles (Chlorfenapyr), **Dinitrophenols** (DNOC) and **Sulfonamides** (Sulfuramid).

Group 20 Mitochondrial complex III electron transport inhibitors

Inhibit electron transport complex III, preventing the utilization of energy by cells.
20B Acequinocyl, **20C** Fluacrypyrim, **20D** Bifenazate.

Group 21 Mitochondrial complex I electron transport inhibitors

Inhibit electron transport complex I, preventing the utilization of energy by cells.
21A METI acaricides (e.g. Fenazaquin, Pyridaben, Tebufenpyrad).

Group 25 Mitochondrial complex II electron transport inhibitors

Inhibit electron transport complex II, preventing the utilization of energy by cells.
25A beta-Ketonitriles (Cyenopyrafen, Cyflumetofen), **25B** Carboxanilides (Pyflubumide).

Growth and Development Targets

Insect and mite growth regulators act by mimicking growth hormones, by directly affecting cuticle formation, or lipid biosynthesis. Acaricides that act on this system are usually slow acting. The target proteins are not always known.

Group 10 Mite growth inhibitors,

Incompletely defined mode of action leading to growth inhibition.
10A Clofentazine, **Hexythiazox**, **10B** Etoxazole.

Group 15 Inhibitors of chitin biosynthesis, type 0

Incompletely defined mode of action leading to inhibition of chitin biosynthesis.
Benzoylureas (e.g. Flucycloxuron, Flufenoxuron).

Group 23 Inhibitors of lipid synthesis

Inhibit acetyl coenzyme A carboxylase, part of the first step in lipid biosynthesis.
Tetronic & Tetramic acid derivatives (e.g. Spirodiclofen).