

Acaricide Mode of Action Classification:

A key to effective acaricide resistance management

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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.



Sequence of acaricides through season

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. Nethomyl), **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. 24 Cvclodiene Orcanochlorines (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).

<u>Group 6</u> Glutamate-gated chloride channel (GluCI) allosteric modulators

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

<u>Group 19</u> 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)

<u>Group 32</u> Nicotinic acetylcholine receptor (nAChR) allosteric modulators, Site II Allosterically activate nAChRs (at site II), causing hyperexcitation of the nervous system. Acetylcholine is the major excitatory neurotransmitter in the insect central nervous system. GS-omega/kappa HXTX-HV1a Peptide

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, Dicofol.

Targeted Physiology: Rotations for resistance management should be based only on the numbered mode of					
action groups.					
	Nerve & Muscle	Growth & Development	Respiration	Midgut	Unknown or Non-specific



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.

<u>Group 13</u> 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.

Pyrroles (Chlorfenapyr), Dinitrophenols (DNOC) and Sulfonamides (Sulfluramid).

<u>Group 20</u> 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). <u>Group 25 Mitochondrial complex II electron transport inhibitors</u> 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. Acaricdes that act on this system are usually slow acting. The target proteins are not always known.

Group 10 Mite growth inhibitors affecting CHS1

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

<u>Group 15</u> Inhibitors of chitin biosynthesis affecting CHS1 Incompletely defined mode of action leading to inhibition of chitin biosynthesis. Benzoylureas (e.g. Flucycloxuron, Flufenoxuron).

Group 23 Inhibitors of acetyl CoA carboxylase

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