Introduction and Background

The agrochemical industry has developed a broad range of very effective insecticides for the control of sucking insect pests such as aphids, whiteflies and hoppers. Unfortunately, as a consequence of the use of these insecticides, many species have developed resistance. The green peach aphid (Myzus persicae), and the sweet potato whitefly (Bemisia tabaci) are important examples of sucking pests that have developed resistance to a wide range of chemical classes.

In recent years the industry has worked especially hard to develop new types of insecticides with novel modes of action, but this process is becoming ever harder and more costly. It is therefore vital that effective insecticide resistance management (IRM) strategies are implemented, to ensure that resistance does not develop to these new compounds, or to older chemistries that are still effective.

In order to help prevent or delay the incidence of resistance, IRAC promotes the use of a Mode of Action (MoA) classification of insecticides in effective and sustainable IRM strategies. Available insecticides are allocated to specific groups, based on their target site, as described below. By using sequences or alternations of insecticides from different MoA classes, resistance is less likely to occur. Available at IRAC website www.irac-online.org, this IRAC MoA classification list provides farmers, growers, advisors, extension staff, consultants and crop protection professionals with a guide to the selection of insecticides in IRM programs.

Nerve and Muscle Targets

Most current insecticides act on nerve and muscle targets. These insecticides 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. Chlordrin, Chlorpyrifos).

**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 Cyclo Sheridan Organochlorines (e.g. Endosulfan).

2B Phenylpyrazoles (e.g. Fipronil).

**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 Pyrethrins, Pyrethroids (e.g. Cypermethrin, Lambda-Cyhalothrin).

**Group 4** Nicotinic acetylcholine receptor (nAChR) agonists

Mimic the agonist action of acetylcholine at nAChRs, causing hyperexcitation. Acetylcholine is the major excitatory neurotransmitter in the insect central nervous system.

4A Neonicotinoids (e.g. Acetamiprid, Imidacloprid, Thiamethoxam).

4C Sulfonflox, 4D Fluorpyriduron.

**Group 5** Cholinergic organophosphates

Incompletely defined mode of action causing selective inhibition of aphid and whitefly feeding.

5B Pyridylace derivatives (e.g. Pymetrozine, Pyrinfluquinazon).

**Group 6** Voltage-dependent sodium channel blockers

Block sodium channels, causing nervous system shutdown and paralysis. Sodium channels are involved in the propagation of action potentials along nerve axons.

22A Indoxacarb (limited spectrum – a few selected whitefly e.g. white apple leafhopper).

**Group 7** Ryanodine receptor modulators

Activate muscle ryanodine receptors, leading to contraction and paralysis. Ryanodine receptors mediate calcium release into the cytoplasm from intracellular stores.

28 Diamides (e.g. Cyantraniliprole).

**Group 8** Cholinergic organophosphates – undefined target site

Disrupt the function of cholinergic stretch receptor organs, which are critical for the senses of hearing, gravity, balance, acceleration, proprioception and kinesthesia. This disrupts feeding and other behaviours in target insects.

29 Florfenicol.

**Group 9** 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.

30 GS-omega/kappa HX7/HT7/1Peptide

Growth and Development Targets

Insect development is controlled by the balance of two principal hormones: juvenile hormone and ecdysone. Insect growth regulators act by mimicking one of these hormones by or directly affecting cuticle formation/deposition or lipid biosynthesis. Insecticides that act on individual targets in this group are generally slow to moderately slow acting.

**Group 7** Juvenile hormone mimics

Applied in the pre-metamorphic instar, these compounds disrupt and prevent metamorphosis.

7A Kinoprene. 7C Pyriprolene

**Group 15** Inhibitors of chitin biosynthesis affecting CHS!

Incompletely defined mode of action leading to inhibition of chitin biosynthesis.

15 Benzoylureas (e.g. Nuvolaron, Bifluturon)

**Group 16** Inhibitors of chitin biosynthesis, Type 1

Incompletely defined mode of action leading to inhibition of chitin biosynthesis in a number of insects, including whiteflies.

16 Buprofezin

**Group 23** Inhibitors of acetyl CoA carboxylase

Inhibition of acetyl CoA carboxylase, part of the first step in lipid synthesis, leading to insect death.

23 Tetronic and Tetramic acid derivatives (e.g. Spiromesifen, Spirelometram)

Respiration Targets

Mitochondrial respiration produces ATP, the molecule that energizes all vital cellular processes. In mitochondria, an electron transport chain uses the energy released by oxidation to charge a proton gradient battery that drives ATP synthesis. Several insecticides are known to interfere with mitochondrial respiration by the inhibition of electron transport and/or oxidative phosphorylation. Insecticides that act on individual targets in this group are generally fast to moderately fast acting.

**Group 12** Inhibitors of mitochondrial ATP synthase

Inhibit the enzyme that synthesizes ATP.

12A Diflubenzuron

**Group 21** Mitochondrial complex I electron transport inhibitors

Inhibit electron transport complex I, preventing the utilization of energy by cells.

21A Tolfenpyrad, Pyracidrin

Effective IRM strategies: Sequences or alternations of MoA

Effective insecticide resistance management (IRM) strategies seek to minimise the selection of resistance to any one type of insecticide. In practice, alternations, sequences or rotations of compounds from different MoA groups provide sustainable and effective IRM.

Example:

- **MoA w**
- **MoA x**
- **MoA y**
- **MoA Z**
- **MoA w**
- **MoA x**

Sequence of insecticides 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 sucking pest species of concern. Local expert advice should always be followed with regard to spray windows and timing. Several sprays may be possible within each spray window, but it is generally essential 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; where this is known to occur, the above advice should be modified accordingly.

What MoA works for which pest group?

The table below lists which mode of action groups of those mentioned on the poster principally provide control of aphids, whiteflies and hoppers. However, the availability of individual modes of action may regionally differ due to registration status.