Uso razionale dei fitofarmaci e relativi meccanismi di azione

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Campobasso, Hotel San Giorgio, 04 aprile 2022

Artropodi

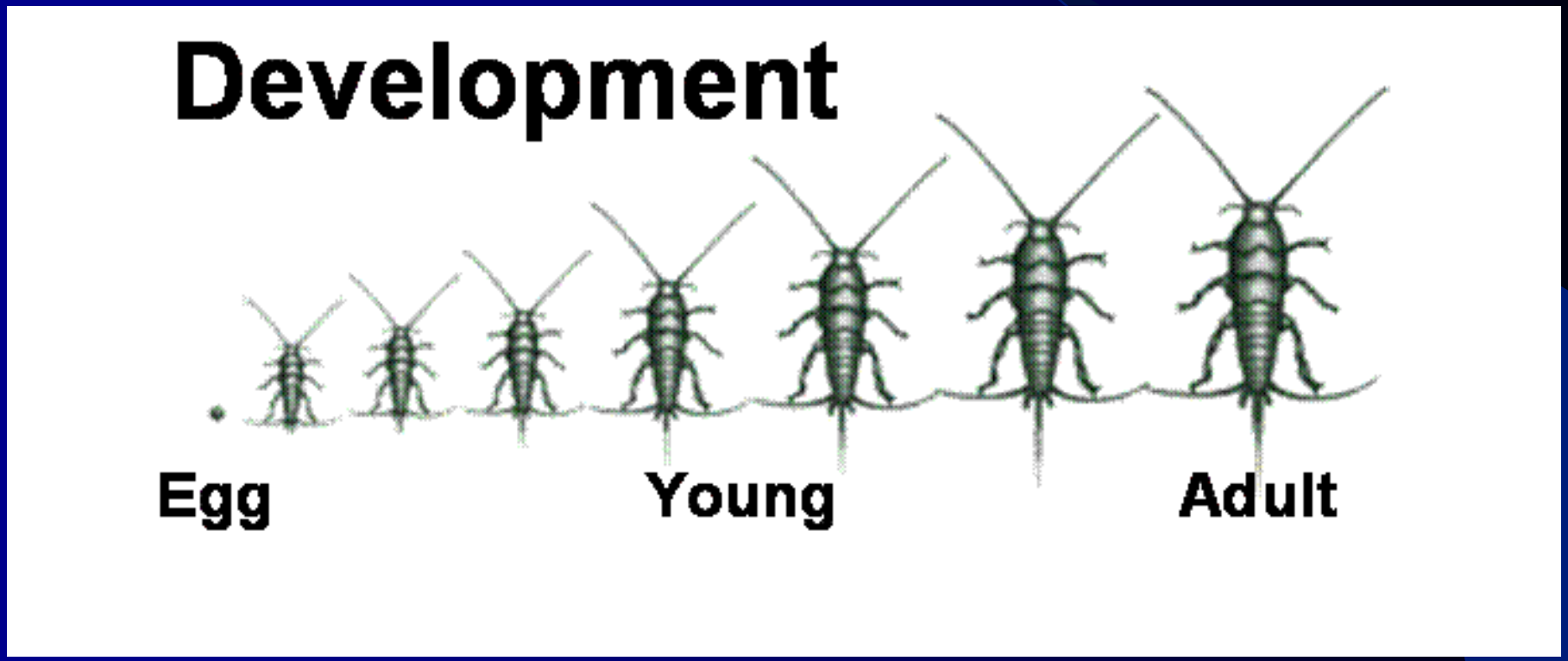
Insetti

Acari

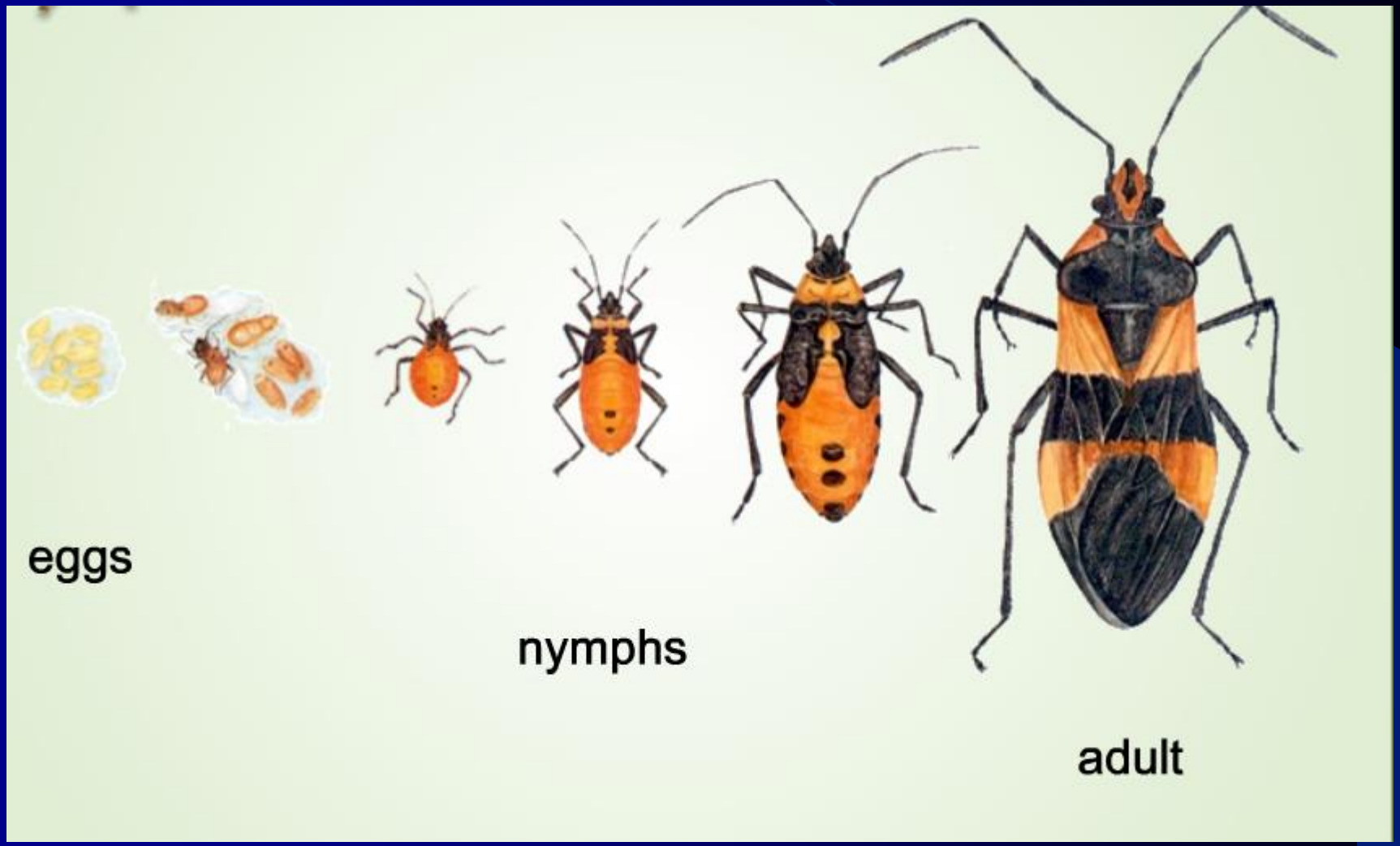
Basi dell'uso razionale dei fitofarmaci

1. Biologia del fitofago da controllare
2. Monitoraggio
3. Campionamento
4. Soglie economiche di tolleranza, intervento, danno
5. Obiettivo: riduzione del numero degli interventi fitosanitari

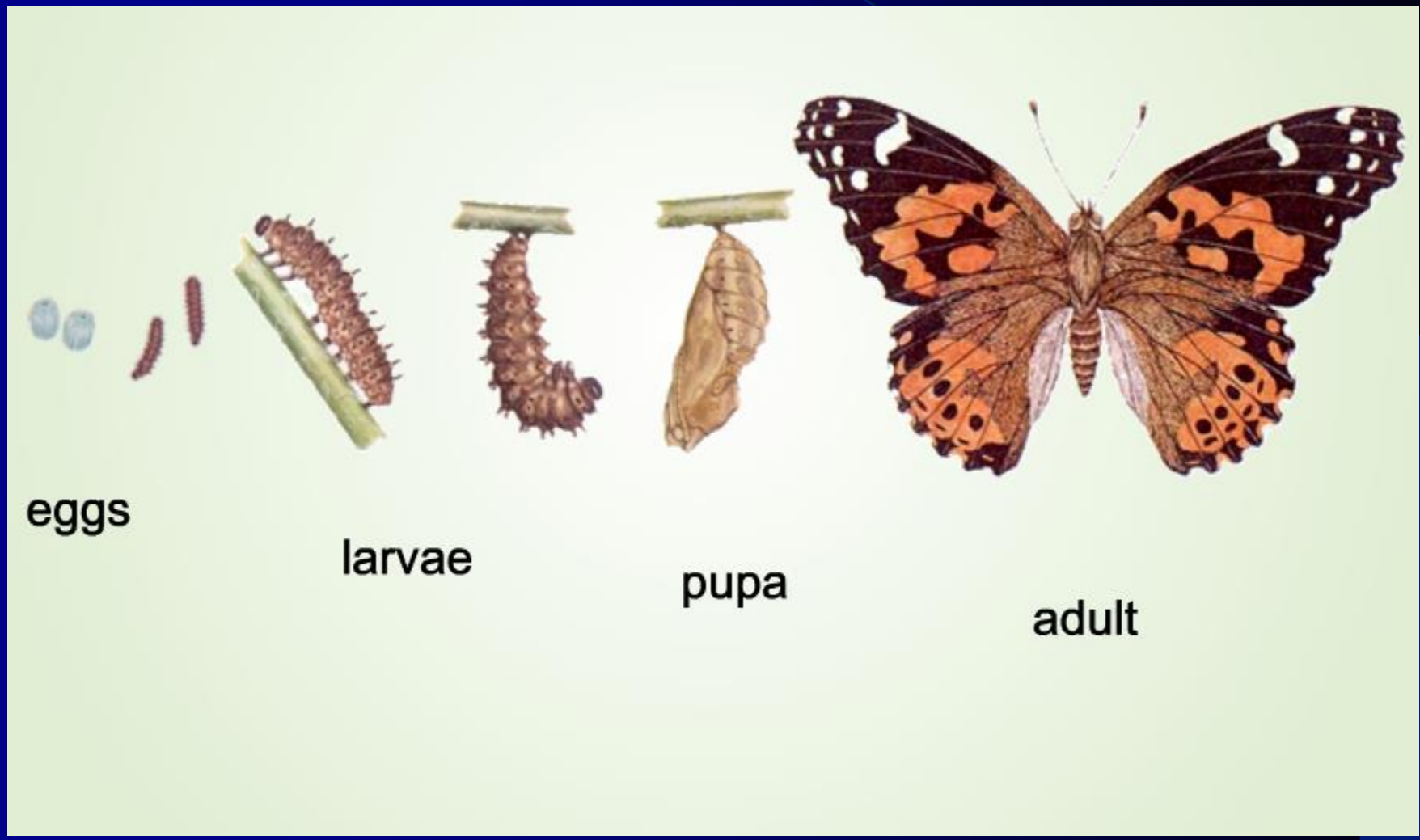
Ametaboli

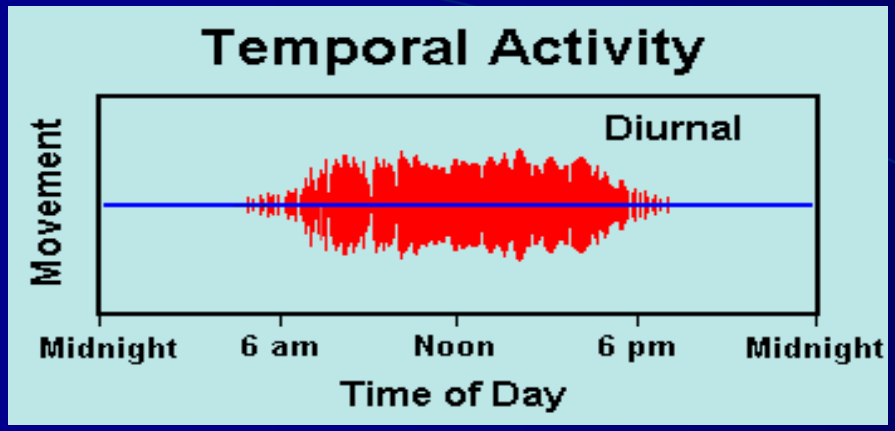


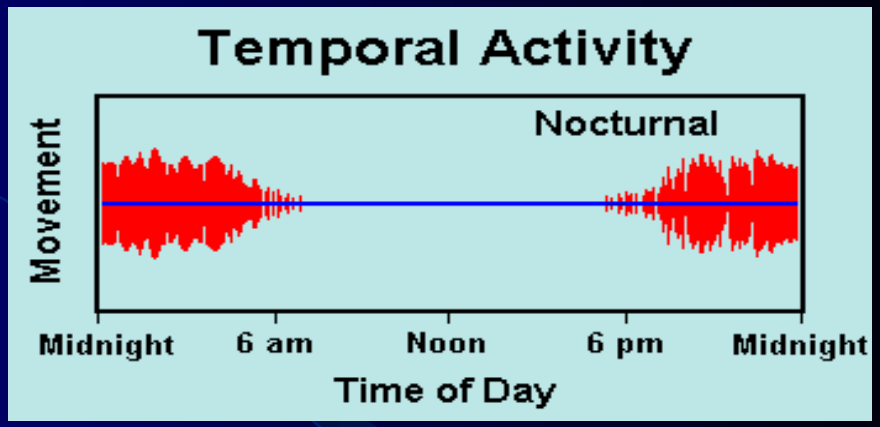
Eterometaboli



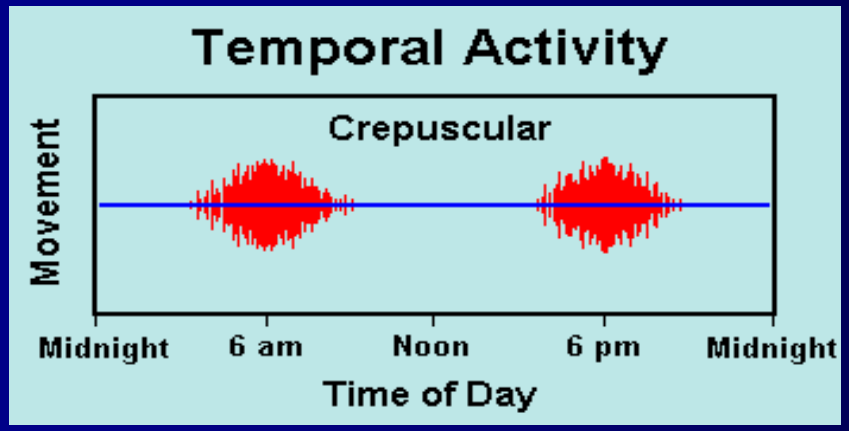
Olometaboli

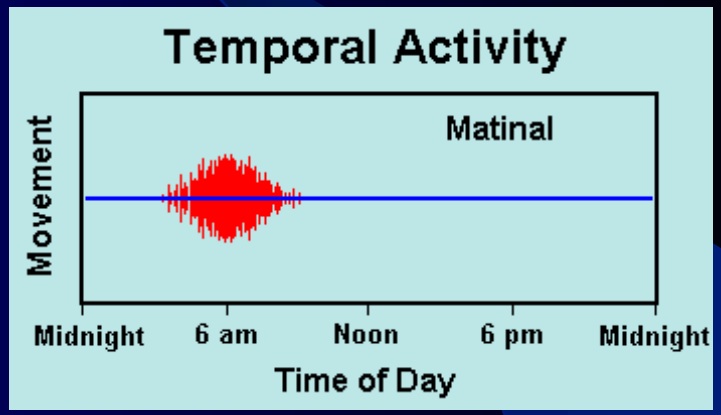


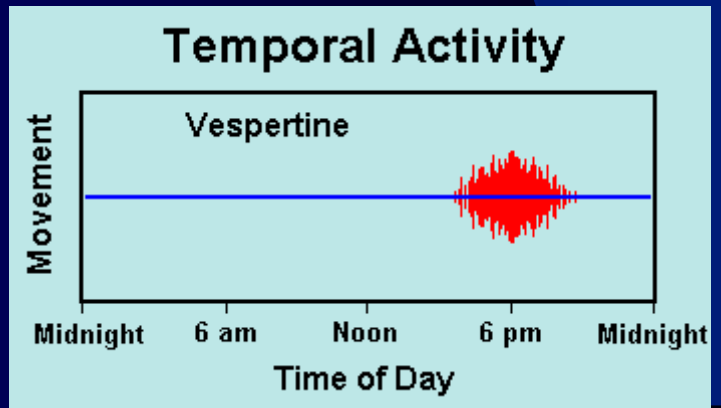




Bioritmi circadiani







Alcune attività in determinate specie possono essere regolate dalle fasi lunari (muta, emergenza durante le fasi senza luce) con cicli di ca. 28 giorni, o dall'alternanza delle stagioni (migrazioni) con cicli di circa 12 mesi

Scelta razionale dei mezzi chimici in agricoltura integrata

1. Fitofagi da controllare (caratter. morfo-funzionali e biologiche in rapporto alla pianta)
2. Principi attivi disponibili e loro modalità di penetrazione e maccanismi di azione
3. Autorizzazione all'uso per la coltura da proteggere e per l'avversità
4. Fitotossicità e miscibilità
5. Stadi del fitofago da controllare
6. Fase fenologica della pianta
7. Intervallo di sicurezza
8. Limite di tolleranza (ppm), ADI (DGA), Concentrazione autorizzabile per alimento (CAA)
9. Formulazione e dose di impiego
10. Attrezzature e macchine in dotazione per la distribuzione (sicurezza, efficacia, deriva)
11. Tossicità
12. Persistenza agronomica o fitosanitaria
13. Grado di selettività per le specie utili e altri effetti collaterali indesiderabili
14. Destino ambientale nei comparti acqua, aria, suolo (e vegetazione)
15. Prezzo

Mode of Action Classification

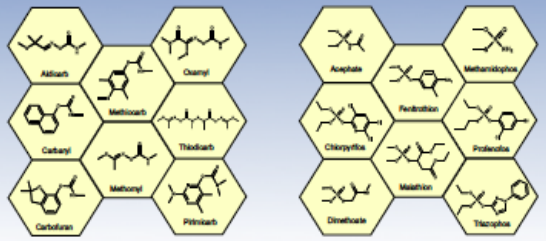
IRAC

Insecticide Resistance Action Committee

The Key to Resistance Management

- Successive generations of a pest should not be treated with compounds from the same MoA Group.
- Not all of the current groupings are based on knowledge of a shared target protein. For further information, please refer to the IRAC Mode of Action Classification document.
- The color scheme used here associates modes of action into broad categories based on the physiological functions affected, as an aid to understanding symptomology, speed of action and other properties of the insecticides, and not for any resistance management purpose. Rotations for resistance management should be based only on the numbered mode of action groups.

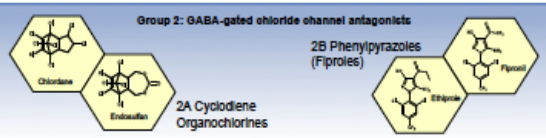
Group 1: Acetylcholinesterase (AChE) inhibitors (Only major representatives of the groups are shown)



1A Carbamates

1B Organophosphates

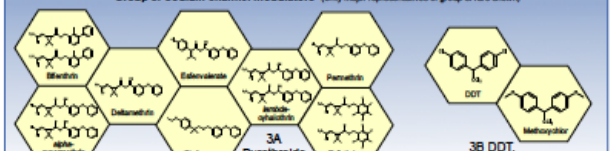
Group 2: GABA-gated chloride channel antagonists



2A Cyclodiene Organochlorines

2B Phenylpyrazoles (Fiproles)

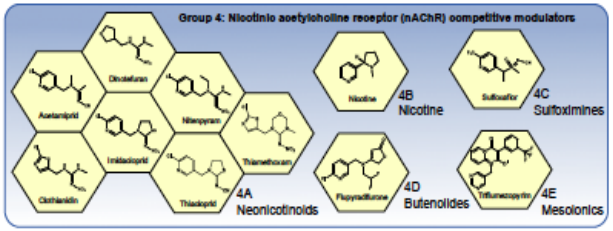
Group 3: Sodium channel modulators (Only major representatives of group 3A are shown)



3A Pyrethroids Pyrethrins

3B DDT, Methoxychlor

Group 4: Nicotinic acetylcholine receptor (nAChR) competitive modulators



4A Neonicotinoids

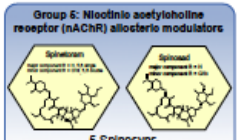
4B Nicotine

4C Sulfoximines

4D Butenolides

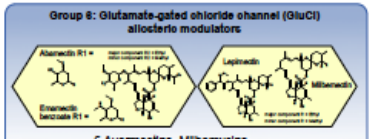
4E Mesoionics

Group 5: Nicotinic acetylcholine receptor (nAChR) allosteric modulators



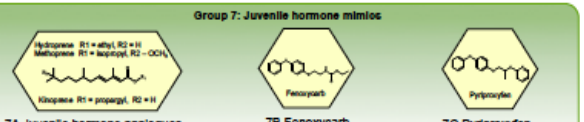
5 Spinosyns

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



6 Avermectins, Milbemycins

Group 7: Juvenile hormone mimics

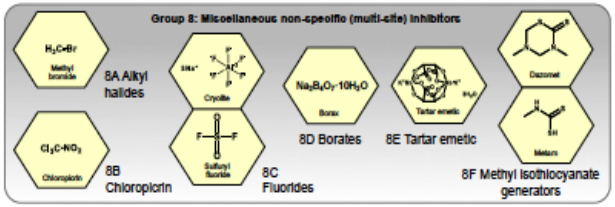


7A Juvenile hormone analogues

7B Fenoxycarb

7C Pyriproxyfen

Group 8: Miscellaneous non-specific (multi-site) inhibitors



8A Alkyl halides

8B Chloropicrin

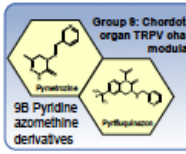
8C Fluorides

8D Borates

8E Tartar emetic

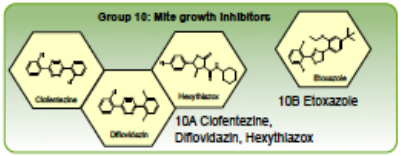
8F Methyl isothiocyanate generators

Group 9: Chordotonal organ TRPV channel modulators



9B Pyridine azomethine derivatives

Group 10: Mite growth inhibitors



10A Clofentezine, Diflovidazin, Hexythiazox

10B Etoxazole

Group 11: Microbial disruptors of insect midgut

{ Includes transgenic crops expressing *Bacillus thuringiensis* toxins (however, specific guidance for resistance management of transgenic crops is not based on rotation of modes of action) }

Different *B.t.* products that target different insect orders may be used together without compromising their resistance management.

Rotation between certain specific *B.t.* microbial products may provide resistance management benefits for some pests. Consult product-specific recommendations.

* Where there are differences among the specific receptors within the mid-guts of target insects, transgenic crops containing certain combinations of these proteins provide resistance management benefits.

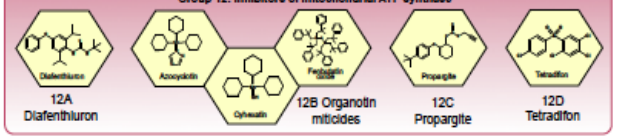
Bacillus thuringiensis and the insecticidal proteins produced: *B.t. israelensis*, *B.t. aizawai*, *B.t. kurstaki*, *B.t. tenebrionis*

Bt crop proteins *: Cry1Ab, Cry1Ac, Cry1Fa, Cry1A.105, Cry2Ab, Vip3A, mCry3A, Cry3Ab1, Cry3Bb1, Cry34Ab1/Cry35Ab1

11A *Bacillus thuringiensis*

11B *Bacillus sphaericus*

Group 12: Inhibitors of mitochondrial ATP synthase



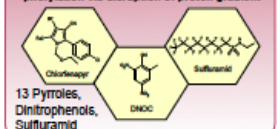
12A Diafenthiuron

12B Organotin miticides

12C Propargite

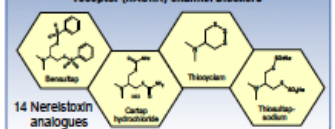
12D Tetradifon

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



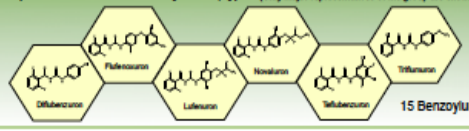
13 Pyrroles, Dinitrophenols, Sulfluramid

Group 14: Nicotinic acetylcholine receptor (nAChR) channel blockers



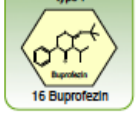
14 Nereistoxin analogues

Group 15: Inhibitors of chitin biosynthesis, type 0 (Only major representatives of the group are shown)



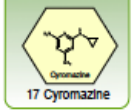
15 Benzoylureas

Group 16: Inhibitors of chitin biosynthesis, type 1



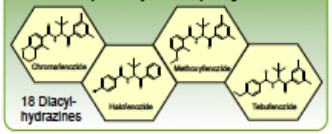
16 Buprofezin

Group 17: Moulting disruptors, Dipteran



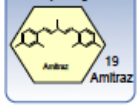
17 Cyromazine

Group 18: Ecdysone receptor agonists



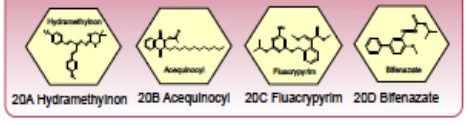
18 Diacylhydrazines

Group 19: Octopamine receptor agonists



19 Amitraz

Group 20: Mitochondrial complex III electron transport inhibitors



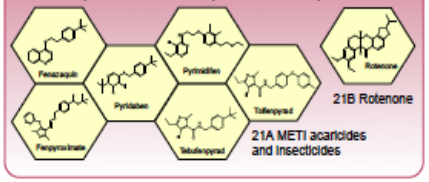
20A Hydramethylnon

20B Acequinocyl

20C Fluacrypyrim

20D Bifenazate

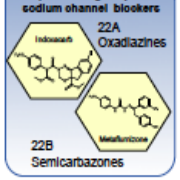
Group 21: Mitochondrial complex I electron transport inhibitors



21A METI acaricides and insecticides

21B Rotenone

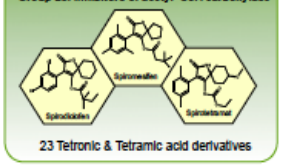
Group 22: Voltage-dependent sodium channel blockers



22A Oxadiazines

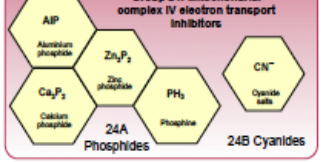
22B Semicarbazones

Group 23: Inhibitors of acetyl CoA carboxylase



23 Tetronic & Tetramic acid derivatives

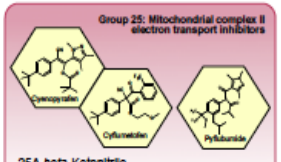
Group 24: Mitochondrial complex IV electron transport inhibitors



24A Phosphides

24B Cyanides

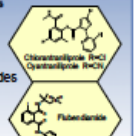
Group 25: Mitochondrial complex II electron transport inhibitors



25A beta-Ketonitrile derivatives

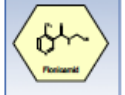
25B Carboxanilides

Group 28: Ryanodine receptor modulators

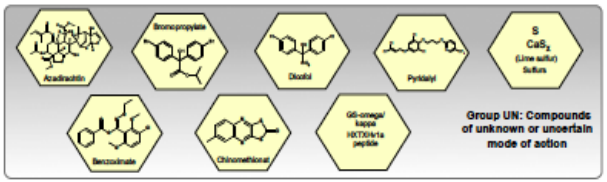


28 Diamides

29: Chordotonal organ modulators – undefined target site



29 Flonicamid



Group UN: Compounds of unknown or uncertain mode of action

Targeted Physiology

- Nerve & Muscle
- Growth & Development
- Respiration
- Midgut
- Unknown or Non-specific

Use of Groups and Sub-Groups:

- Alternations, sequences or rotations of compounds between MoA groups reduces selection for target site resistance.
- Applications are arranged into MoA spray windows defined by crop growth stage and pest biology.
- Several sprays of a compound may be possible within each spray window, but successive generations of a pest should not be treated with compounds from the same MoA group.
- Local expert advice should always be followed with regard to spray windows and timing.
- Actives in groups 8 (Miscellaneous non-specific multi-site inhibitors), 13 (Uncouplers) and UN are thought not to share a common target site and therefore may be freely rotated with each other unless there is reason to expect cross-resistance.
- Sub-groups represent distinct structural classes believed to have the same mode of action.
- Sub-groups provide differentiation between compounds that may bind at the same target site but are structurally different enough that risk of metabolic cross-resistance is lower than for close chemical analogs.
- Cross-resistance potential between sub-groups is higher than between groups, so rotation between sub-groups should be considered only when there are no alternatives, and only if cross-resistance does not exist, following consultation with local expert advice. These exceptions are not sustainable, and alternative options should be sought.
- Sub-group 3B: DDT is no longer used in agriculture and therefore this is only applicable for the control of insect vectors of human disease, such as mosquitoes, because of a lack of alternatives.
- Sub-group10A: Hexythiazox is grouped with clofentezine because they exhibit cross-resistance even though they are structurally distinct, and the target site for these compounds is unknown. Diflovidazin has been added to this group because it is a close analogue of clofentezine and is expected to have the same mode of action.

Poster Notes:

- Groups 26 and 27 are unassigned.
- The poster is for educational purposes only. Information presented is accurate to the best of our knowledge at the time of publication, but IRAC or its member companies cannot accept responsibility for how this information is used or interpreted. Advice should always be sought from local experts or advisors, and health and safety recommendations followed.
- Representative compounds are shown. Please visit www.irac-online.org for the complete IRAC classification.

CropLife

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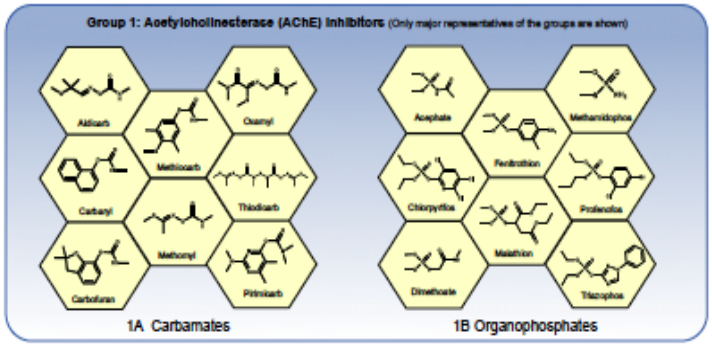
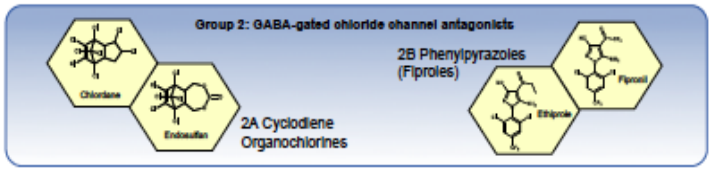
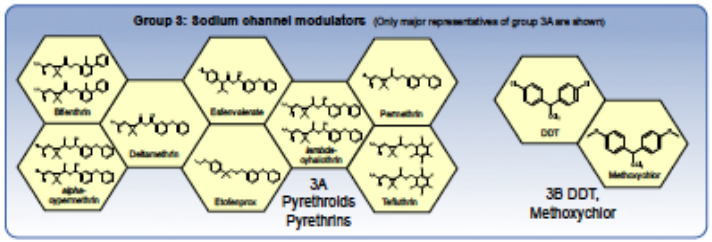
Poster Edition 6.1, April 2016. Based on the MoA Classification Version 8.1

Sito d'azione dei diversi insetticidi

1. Acetilcolinesterasi
2. Recettori GABA
3. Canali sodio
4. Recettori nicotinici dell'acetilcolina
5. Canali del cloro
6. Chitinosintetasi
7. Inibitori del trasporto di elettroni del complesso mitocondriale III
8. Bloccanti dei canali sodio voltaggio dipendenti
9. Acetil-CoA carbossilasi (glicolisi)
10. Recettori delle rianodine

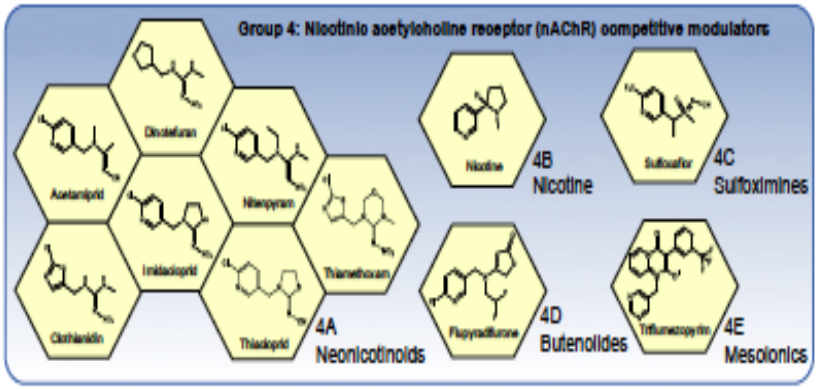


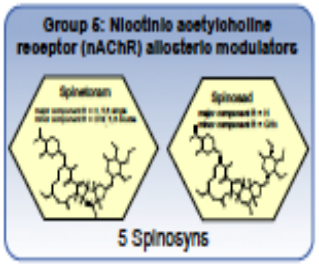


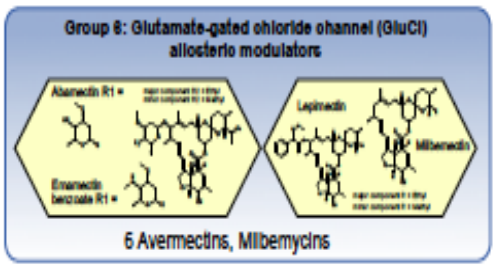


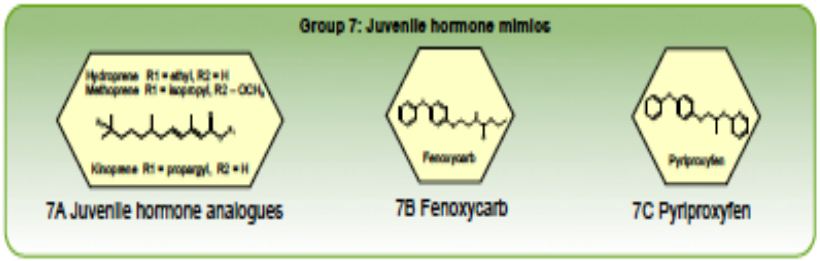
Nerve & Muscle | Growth & Development | Respiration | Midgut | Unknown or Non-Specific

1 **Acetylcholinesterase (AChE) inhibitors** Nerve action {Strong evidence that action at this protein is responsible for insecticidal effects}	**1A** Carbamates	Alanycarb, Aldicarb, Bendiocarb, Benfuracarb, Butocarboxim, Butoxycarboxim, Carbaryl, Carbofuran, Carbosulfan, Ethiofencarb, Fenobucarb, Formetanate, Furathiocarb, Isoprocarb, Methiocarb, Methomyl, Metolcarb, Oxamyl, Pirimicarb, Propoxur, Thiodicarb, Thiofanox, Triazamate,Trimethacarb, XMC, Xylylcarb
	1B Organophosphates	Acephate, Azamethiphos, Azinphos-ethyl, Azinphos-methyl, Cadusafos, Chlorethoxyfos, Chlorfenvinphos, Chlormephos, Chlorpyrifos, Chlorpyrifos-methyl, Coumaphos, Cyanophos, Demeton-S-methyl, Diazinon, Dichlorvos/ DDVP, Dicrotophos, Dimethoate, Dimethylvinphos, Disulfoton, EPN, Ethion, Ethoprophos, Famphur, Fenamiphos, Fenitrothion, Fenthion, Fosthiazate, Heptenophos, Imicyafos, Isofenphos, Isopropyl *O*-(methoxyaminothio-phosphoryl) salicylate, Isoxathion, Malathion, Mecarbam, Methamidophos, Methidathion, Mevinphos, Monocrotophos, Naled, Omethoate, Oxydemeton-methyl, Parathion, Parathion-methyl, Phenthoate, Phorate, Phosalone, Phosmet, Phosphamidon, Phoxim, Pirimiphos- methyl, Profenofos, Propetamphos, Prothiofos, Pyraclofos, Pyridaphenthion, Quinalphos, Sulfotep, Tebupirimfos, Temephos, Terbufos, Tetrachlorvinphos, Thiometon, Triazophos, Trichlorfon, Vamidothion
2 **GABA-gated chloride channel blockers** Nerve action {Strong evidence that action at this protein is responsible for insecticidal effects}	**2A** Cyclodiene Organochlorines	Chlordane, Endosulfan
	2B Phenylpyrazoles (Fiproles)	Ethiprole, Fipronil
3 **Sodium channel modulators** Nerve action {Strong evidence that action at this protein is responsible for insecticidal effects}	**3A** Pyrethroids Pyrethrins	Acrinathrin, Allethrin, d-*cis-trans* Allethrin, d-*trans* Allethrin, Bifenthrin, Bioallethrin, Bioallethrin S-cyclopentenyl isomer , Bioresmethrin, Cycloprothrin, Cyfluthrin, *beta*-Cyfluthrin, Cyhalothrin, *lambda*-Cyhalothrin, *gamma*-Cyhalothrin, Cypermethrin, *alpha*-Cypermethrin, *beta*-Cypermethrin, *theta*-cypermethrin, *zeta*-Cypermethrin, Cyphenothrin , (1*R*)-*trans*- isomers], Deltamethrin, Empenthrin (*EZ*)-(1*R*)- isomers], Esfenvalerate, Etofenprox, Fenpropathrin, Fenvalerate, Flucythrinate, Flumethrin, *tau*-Fluvalinate, Halfenprox, Imiprothrin, Kadethrin, Permethrin, Phenothrin [(1*R*)-*trans*- isomer], Prallethrin, Pyrethrins (pyrethrum), Resmethrin, Silafluofen, Tefluthrin, Tetramethrin, Tetramethrin [(1*R*)-isomers], Tralomethrin, Transfluthrin,
	3B DDT Methoxychlor	DDT Methoxychlor

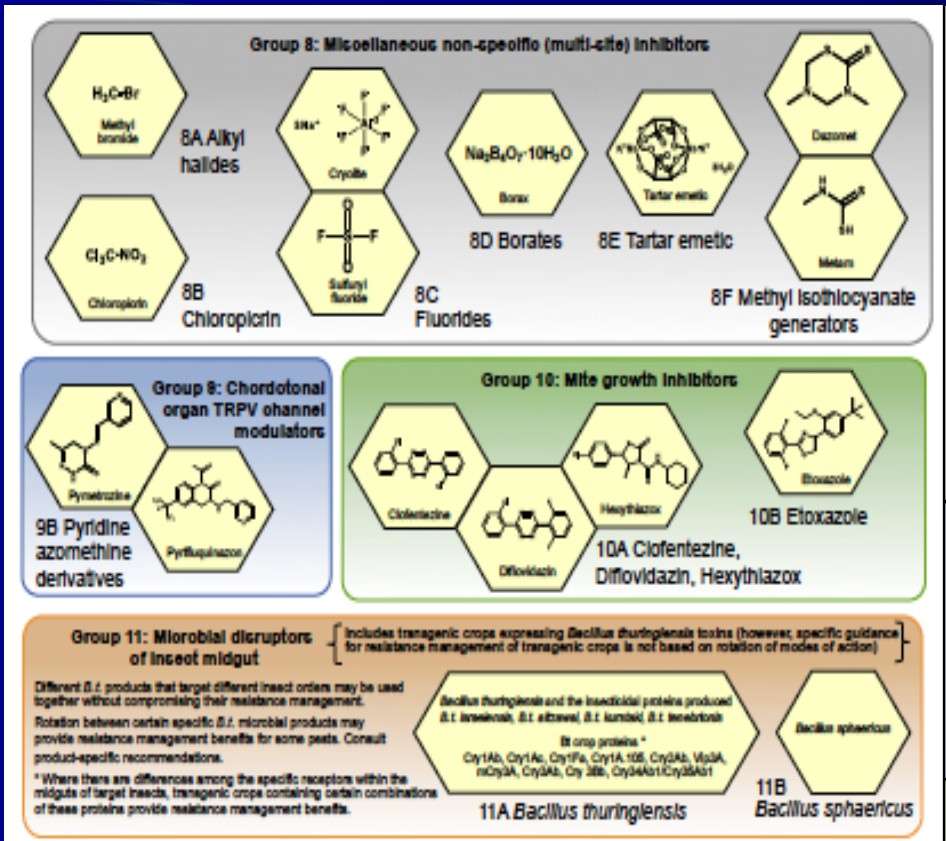




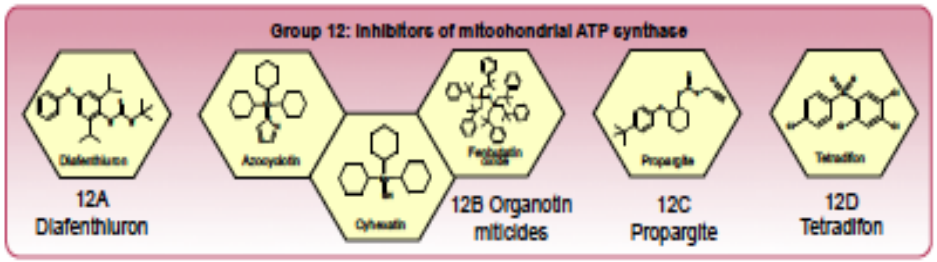


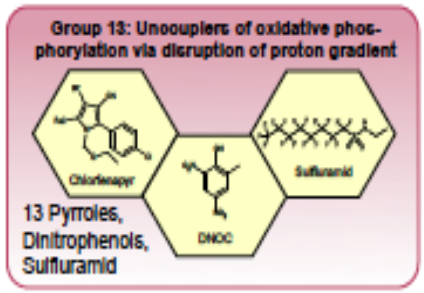


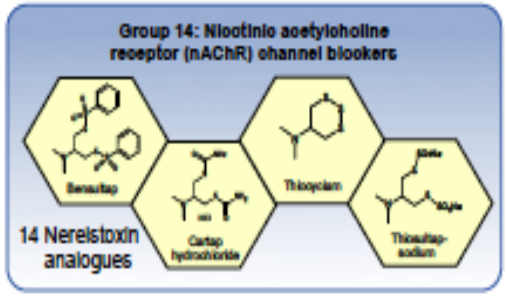
4 **Nicotinic acetylcholine receptor (nAChR) competitive modulators** Nerve action {Strong evidence that action at one or more of this class of protein is responsible for insecticidal effects}	**4A** Neonicotinoids	Acetamiprid, Clothianidin, Dinotefuran, Imidacloprid, Nitenpyram, Thiacloprid, Thiamethoxam,
	4B Nicotine	Nicotine
	4C Sulfoximines	Sulfoxaflor
	4D Butenolides	Flupyradifurone
	4E Mesoionics	Triflumezopyrim
5 **Nicotinic acetylcholine receptor (nAChR) allosteric modulators** Nerve action {Strong evidence that action at one or more of this class of protein is responsible for insecticidal effects}	Spinosyns	Spinetoram, Spinosad
6 **Glutamate-gated chloride channel (GluCl) allosteric modulators** Nerve and muscle action {Strong evidence that action at one or more of this class of protein is responsible for insecticidal effects}	Avermectins, Milbemycins	Abamectin, Emamectin benzoate, Lepimectin, Milbemectin
7 **Juvenile hormone mimics** Growth regulation {Target protein responsible for biological activity is unknown, or uncharacterized}	**7A** Juvenile hormone analogues	Hydroprene, Kinoprene, Methoprene
	7B Fenoxycarb	Fenoxycarb
	7C Pyriproxyfen	Pyriproxyfen



8 * Miscellaneous non-specific (multi-site) inhibitors	**8A** Alkyl halides	Methyl bromide and other alkyl halides
	8B Chloropicrin	Chloropicrin
	8C Fluorides	Cryolite (Sodium aluminum fluoride), Sulfuryl fluoride
	8D Borates	Borax, Boric acid, Disodium octaborate, Sodium borate, Sodium metaborate
	8E Tartar emetic	Tartar emetic
	8F Methyl isothiocyanate generators	Dazomet, Metam
9 Chordotonal organ TRPV channel modulators Nerve action {Strong evidence that action at one or more of this class of proteins is responsible for insecticidal effects }	**9B** Pyridine azomethine derivatives	Pymetrozine, Pyrifluquinazon
10 Mite growth inhibitors Growth regulation {Target protein responsible for biological activity is unknown, or uncharacterized}	**10A** Clofentezine Diflovidazin Hexythiazox	Clofentezine, Diflovidazin, Hexythiazox
	10B Etoxazole	Etoxazole
11 Microbial disruptors of insect midgut membranes (includes transgenic crops expressing *Bacillus thuringiensis* toxins, however specific guidance for resistance management of transgenic crops is not based on rotation of modes of action)	**11A** *Bacillus thuringiensis* and the insecticidal proteins they produce	*Bacillus thuringiensis* subsp. *israelensis* *Bacillus thuringiensis* subsp. *aizawai* *Bacillus thuringiensis* subsp. *kurstaki* *Bacillus thuringiensis* subsp. *tenebrionis* *B.t.* crop proteins: (* Please see footnote) Cry1Ab, Cry1Ac, Cry1Fa, Cry1A.105, Cry2Ab, Vip3A, mCry3A, Cry3Ab, Cry3Bb, Cry34Ab1/Cry35Ab1
	11B *Bacillus sphaericus*	*Bacillus sphaericus*

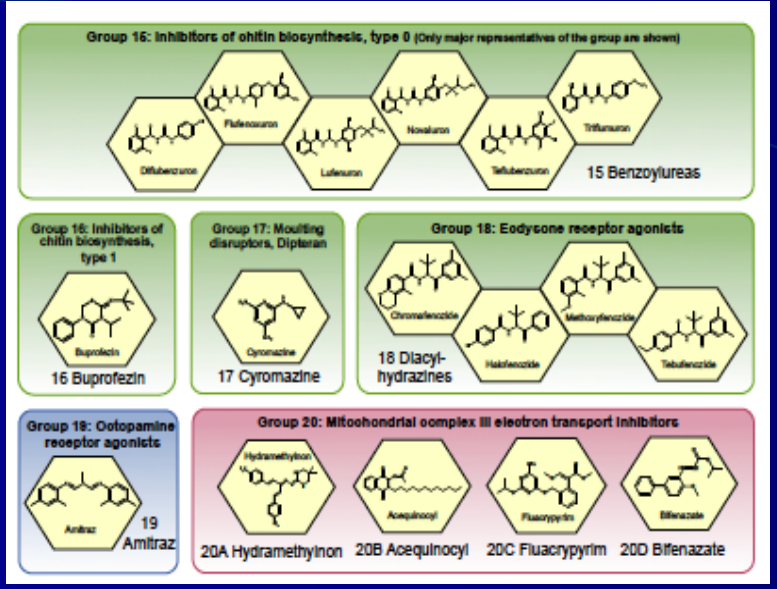






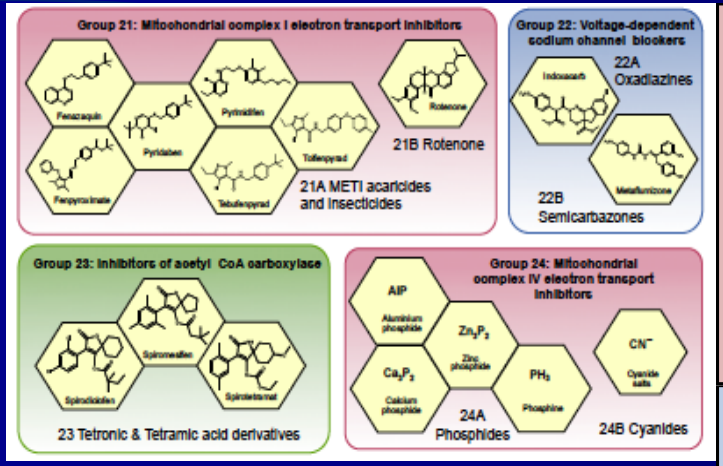
Nerve & Muscle | Growth & Development | Respiration | Midgut | Unknown or Non-Specific

12 **Inhibitors of mitochondrial ATP synthase** Energy metabolism {Compounds affect the function of this protein, but it is not clear that this is what leads to biological activity}	**12A** Diafenthiuron	Diafenthiuron
	12B Organotin miticides	Azocyclotin, Cyhexatin, Fenbutatin oxide
	12C Propargite	Propargite
	12D Tetradifon	Tetradifon
13 * **Uncouplers of oxidative phosphorylation via disruption of the proton gradient** Energy metabolism	Pyrroles Dinitrophenols Sulfluramid	Chlorfenapyr DNOC Sulfluramid
14 **Nicotinic acetylcholine receptor (nAChR) channel blockers** Nerve action {Compounds affect the function of this protein, but it is not clear that this is what leads to biological activity}	Nereistoxin analogues	Bensultap, Cartap hydrochloride, Thiocyclam, Thiosultap-sodium



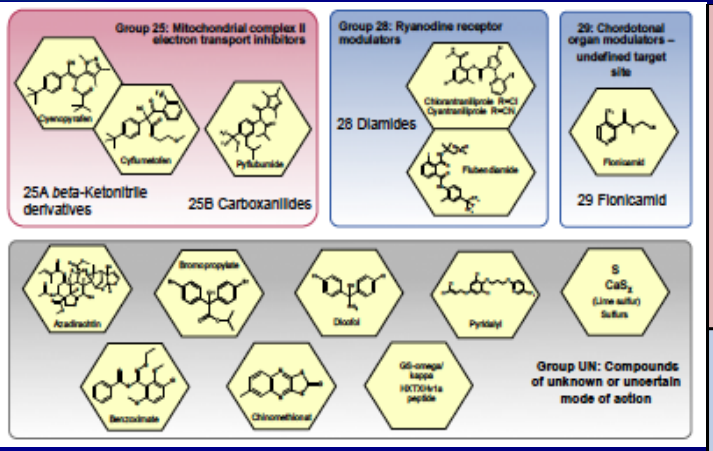
Nerve & Muscle | Growth & Development | Respiration | Midgut | Unknown or Non-Specific

15 **Inhibitors of chitin biosynthesis, type 0** Growth regulation {Target protein responsible for biological activity is unknown, or uncharacterized}	Benzoylureas	Bistrifluron, Chlorfluazuron, Diflubenzuron, Flucycloxuron, Flufenoxuron, Hexaflumuron, Lufenuron, Novaluron, Noviflumuron, Teflubenzuron, Triflumuron
16 **Inhibitors of chitin biosynthesis, type 1** Growth regulation {Target protein responsible for biological activity is unknown, or uncharacterized}	Buprofezin	Buprofezin
17 **Moulting disruptors, Dipteran** Growth regulation {Target protein responsible for biological activity is unknown, or uncharacterized}	Cyromazine	Cyromazine
18 **Ecdysone receptor agonists** Growth regulation {Strong evidence that action at this protein is responsible for insecticidal effects}	Diacylhydrazines	Chromafenozide, Halofenozide, Methoxyfenozide, Tebufenozide
19 **Octopamine receptor agonists** Nerve action {Good evidence that action at one or more of this class of protein is responsible for insecticidal effects}	Amitraz	Amitraz
20 **Mitochondrial complex III electron transport inhibitors** Energy metabolism {Good evidence that action at this protein complex is responsible for insecticidal effects}	**20A** Hydramethylnon	Hydramethylnon
	20B Acequinocyl	Acequinocyl
	20C Fluacrypyrim	Fluacrypyrim
	20D Bifenazate	Bifenazate



Nerve & Muscle | Growth & Development | Respiration | Midgut | Unknown or Non-Specific

21 **Mitochondrial complex I electron transport inhibitors** Energy metabolism {Good evidence that action at this protein complex is responsible for insecticidal effects}	**21A** METI acaricides and insecticides	Fenazaquin, Fenpyroximate, Pyridaben, Pyrimidifen, Tebufenpyrad, Tolfenpyrad
	21B Rotenone	Rotenone (Derris)
22 **Voltage-dependent sodium channel blockers** Nerve action {Good evidence that action at this protein complex is responsible for insecticidal effects}	**22A** Oxadiazines	Indoxacarb
	22B Semicarbazones	Metaflumizone
23 **Inhibitors of acetyl CoA carboxylase** Lipid synthesis, growth regulation {Good evidence that action at this protein is responsible for insecticidal effects}	Tetronic and Tetramic acid derivatives	Spirodiclofen, Spiromesifen, Spirotetramat
24 **Mitochondrial complex IV electron transport inhibitors** Energy metabolism {Good evidence that action at this protein complex is responsible for insecticidal effects}	**24A** Phosphides	Aluminium phosphide, Calcium phosphide, Phosphine, Zinc phosphide
	24B Cyanides	Calcium cyanide, Potassium cyanide, Sodium cyanide



Nerve & Muscle | Growth & Development | Respiration | Midgut | Unknown or Non-Specific

25 **Mitochondrial complex II electron transport inhibitors** Energy metabolism {Good evidence that action at this protein complex is responsible for insecticidal effects}	**25A** *Beta*-ketonitrile derivatives	Cyenopyrafen, Cyflumetofen
	25B Carboxanilides	Pyflubumide
28 **Ryanodine receptor modulators** Nerve and muscle action {Strong evidence that action at this protein complex is responsible for insecticidal effects}	Diamides	Chlorantraniliprole, Cyantraniliprole, Flubendiamide
29 **Chordotonal organ Modulators - undefined target site** Nerve action (Modulation of chordotonal organ function has been clearly demonstrated, but the specific target protein(s) responsible for biological activity are distinct from Group 9 and remain undefined.)	Flonicamid	Flonicamid
UN * **Compounds of unknown or uncertain MoA** {Target protein responsible for biological activity is unknown, or uncharacterized}	Azadirachtin	Azadirachtin
	Benzoximate	Benzoximate
	Bromopropylate	Bromopropylate
	Chinomethionat	Chinomethionat
	Dicofol	Dicofol
	GS-omega/kappa HXTX-Hv1a peptide	GS-omega/kappa HXTX-Hv1a peptide
	Lime sulfur	Lime sulfur
	Pyridalyl	Pyridalyl
	Sulfur	Sulfur

Insecticide Mode of Action Classification

Insecticide Resistance Action Committee www.irac-online.org

CropLife INTERNATIONAL

Introduction

Insecticide Resistance Action Committee [IRAC] promotes the use of a Mode of Action (MoA) classification of insecticides as the basis for effective and sustainable insecticide resistance management (IRM). Insecticides 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 insecticides or acaricides in IRM programs. Effective IRM of this type preserves the utility and diversity of available insecticides and acaricides.

Nerve & Muscle Targets

Group 1 Acetylcholinesterase (AChE) inhibitors
- 1A: Carbamates (e.g. Thiodicarb),
- 1B: Organophosphates (e.g. Chlorpyrifos)

Group 2 GABA-gated chloride channel blockers
- 2A: Cyclodiene Organochlorines (e.g. Endosulfan)
- 2B: Phenylpyrazoles (e.g. Fipronil)

Group 3 Sodium channel modulators
- 3A: Pyrethrins, Pyrethroids (e.g. Cypermethrin)
- 3B: DDT, Methoxychlor

Group 4 Nicotinic acetylcholine receptor (nAChR) competitive modulators
- 4A: Neonicotinoids (e.g. Imidacloprid, Thiamethoxam)
- 4B: Nicotine
- 4C: Sulfoximines (e.g. Sulfoxaflor)
- 4D: Butenolides (e.g. Flupyradifurone)

Group 5 Nicotinic acetylcholine receptor (nAChR) allosteric modulators
- 5: Spinosyns (e.g. Spinosad, Spinetoram)

Group 6 Glutamate-gated chloride channel (GluCl) allosteric modulators
- 6: Avermectins, Milbemycins (e.g. Abamectin, Emamectin benzoate)

Group 9 Chordotonal organ TRPV channel modulators
- 9B: Pyridine azomethine derivatives (e.g. Pymetrozine, Pyrfluquinazon)

Group 14 Nicotinic acetylcholine receptor (nAChR) channel blockers
- 14: Nereistoxin analogs (e.g. Cartap hydrochloride)

Group 19 Octopamine receptor agonists
- 19: Amitraz

Group 22 Voltage dependent sodium channel blockers
- 22A: Oxadiazines (e.g. Indoxacarb)
- 22B: Semicarbazones (e.g. Metaflumizone)

Group 28 Ryanodine receptor modulators
- 28: Diamides (e.g. Chlorantraniliprole, Cyantraniliprole, Flubendiamide)

Group 29 Chordotonal organ modulators – undefined target site
- 29: Flonicamid

Midgut Targets

Group 11 Microbial disruptors of insect midgut membranes
- 11A: *Bacillus thuringiensis*
- 11B: *Bacillus sphaericus*

Miscellaneous non-specific (multi-site) inhibitors

Group 8 8A: Alkyl halides, 8B: Chloropicrin, 8C: Fluorides, 8D: Borates, 8E: Tartar emetic, 8F: Methyl isothiocyanate generators

Effective IRM strategies: MoA Sequences & alternations

All 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 for pest insects. 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, e.g. neonicotinoids (Group 4A).

MoA w | MoA x | MoA y | MoA z | MoA w | MoA x

Sequence of insecticides through season

MoA Sequences & alternations – Exceptions

IRAC recommends alternations, sequences or rotations of compounds from different MoA groups to provide a sustainable and effective approach to IRM. Three groups (8, 13 and UN) are exempt from the recommendations as they do not contain compounds acting at a common target site

Color Scheme Notes:

The color scheme used here associates modes of action into broad categories based on the physiological functions affected, as an aid to understanding symptomology, speed of action and other properties of the insecticides, and not for any resistance management purpose. **Rotations for resistance management should be based only on the numbered mode of action groups.** The cross-resistance potential between sub-groups is higher than that between different groups, so rotation between sub-groups should only be used where effective registered insecticides from other MoA groups are unavailable.

Respiration targets

Group 12 Inhibitors of mitochondrial ATP synthesis
- 12A: Diafenthiuron
- 12B: Organotin miticides (e.g. Cyhexatin)
- 12C: Propargite
- 12D: Tetradifon

Group 13 Uncouplers of oxidative phosphorylation via disruption of the proton gradient
- 13: Pyrroles (e.g. Chlorfenapyr), Dinitrophenols, (e.g.DNOC), Sulfuramid

Group 20 Mitochondrial complex III electron transport inhibitors
- 20A: Hydramethylnon
- 20B: Acequinocyl
- 20C: Fluacrypyrim
- 20D: Bifenazate

Group 21 Mitochondrial complex I electron transport inhibitors
- 21A: METI acaricides & insecticides (e.g. Pyridaben)
- 21B: Rotenone (Derris)

Group 24 Mitochondrial complex IV electron transport inhibitors
- 24A: Phosphides (e.g. Phosphine)
- 24B: Cyanides (e.g. Sodium cyanide)

Group 25 Mitochondrial complex II electron transport inhibitors
- 25A: *Beta*-ketonitrile derivatives (e.g. Cyenopyrafen, Cyflumetofen)
- 25B: Carboxanilides, (e.g. Pyflubumide)

Growth & Development targets

Group 7 Juvenile hormone mimics
- 7A: Juvenile hormone analogues (e.g. Methoprene)
- 7B: Fenoxycarb
- 7C: Pyriproxyfen

Group 10 Mite growth inhibitors
- 10A: Clofentezine, Diflovidazin, Hexythiazox
- 10B: Etoxazole

Group 15 Inhibitors of chitin biosynthesis, Type 0
- 15: Benzoylureas (e.g. Flufenoxuron, Novaluron)

Group 16 Inhibitors of chitin biosynthesis, type 1
- 16: Buprofezin

Group 17 Moulting disruptors, Dipteran
- 17: Cyromazine

Group 18 Ecdysone receptor agonists
- 18: Diacylhydrazines (e.g. Methoxyfenozide, Tebufenozide)

Group 23 Inhibitors of acetyl CoA carboxylase
- 23: Tetronic & Tetramic acid derivatives (e.g. Spirodiclofen)

Unknown

Group UN Compounds of unknown or uncertain mode of action (e.g. Azadiractin, Benzoximate, Bromopropylate, Chinomethionat, Dicofol, Lime sulfur, Pyridalyl, Sulfur)

This poster is for educational purposes only. Details are accurate to the best of our knowledge but IRAC and its member companies cannot accept responsibility for how this information is used or interpreted. Advice should always be sought from local experts or advisors and health and safety recommendations followed.

Designed & produced by the IRAC MoA Team, Dec. 2015, Ver. 4, Based on MoA Classification Ver. 8.0, Photograph courtesy of N. Armes
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Lepidoptera Insecticide Mode of Action Classification:

A key to effective insecticide resistance management

Insecticide Resistance Action Committee

www.irac-online.org

Introduction and background

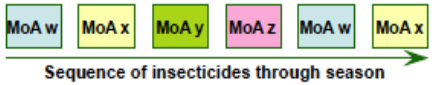
The agrochemical industry has developed a broad range of very effective insecticides for the control of lepidopteran pests. Unfortunately, as a consequence of the misuse or overuse of these insecticides, many species have developed resistance. Populations of *Plutella xylostella*, for example, have developed resistance to virtually every insecticide used against them. Additionally, there are numerous other species prone to resistance development. 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 the 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.

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:



Applications are often arranged into MoA spray windows or blocks that are defined by the stage of crop development and the biology of the Lepidopteran 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.

Nerve and Muscle Targets

Most current insecticides act on nerve and muscle targets. Insecticides that act on these targets 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, Thiodicarb) ***1B*** Organophosphates (e.g. 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 Cyclodiene 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, λ-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, Thiacloprid, Thiamethoxam)

Group 5 Nicotinic acetylcholine receptor (nAChR) allosteric modulators

Allosterically activate nAChRs, causing hyperexcitation of the nervous system. Acetylcholine is the major excitatory neurotransmitter in the insect central nervous system.

Spinosyns (e.g. Spinosad, Spinetoram)

Group 6 Chloride channel activators

Allosterically activate glutamate-gated chloride channels (GluCls), causing paralysis. Glutamate is an important inhibitory neurotransmitter in insects.

Avermectins, Milbemycins (e.g. Abamectin, Emamectin Benzoate, Lepimectin)

Group 14 Nicotinic acetylcholine receptor (nAChR) blockers

Block the nAChR ion channel, resulting in nervous system block and paralysis. Acetylcholine is the major excitatory neurotransmitter in the insect central nervous system.

Bensultap, Cartap

Group 22 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 ***22B*** Metaflumizone

Group 28 Ryanodine receptor modulators

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

Diamides (e.g. Chlorantraniliprole, Cyantraniliprole, Flubendiamide)

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 system are generally fast to moderately fast acting.

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.

Chlorfenapyr

Group 21 Mitochondrial complex I electron transport inhibitors

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

21A Tolfenpyrad

Midgut Targets

Lepidopteran-specific microbial toxins that are sprayed or expressed in transgenic crops.

Group 11 Microbial disruptors of insect midgut membranes

Protein toxins that bind to receptors on the midgut membrane and induce pore formation, resulting in ionic imbalance and septicemia.

11A *Bacillus thuringiensis* ***11B*** *Bacillus sphaericus*

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 or by directly affecting cuticle formation/deposition or lipid biosynthesis. Insecticides that act on individual targets in this system are generally slowly to moderately slowly acting.

Group 7 Juvenile hormone mimics

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

7B Juvenile hormone analogues (e.g. Fenoxycarb)

Group 15 Inhibitors of chitin biosynthesis, Type 0

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

Benzoylureas (eg. Flufenoxuron, Lufenuron, Novaluron)

Group 18 Ecdysone receptor agonists

Mimic the moulting hormone, ecdysone, inducing a precocious molt.

Diacylhydrazines (e.g. Methoxyfenozide, Tebufenozide)

Unknown Several insecticides are known to affect less well-described target-sites or functions, or to act non-specifically on multiple targets.

Azadirachtin, Pyridalyl

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Aphids, Whiteflies and Hoppers - Insecticide Mode of Action Classification:

A key to effective insecticide resistance management

Insecticide Resistance Action Committee

www.irac-online.org

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 misuse or overuse 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.

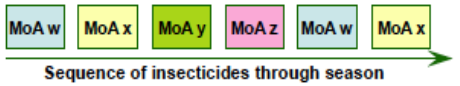
In recent years the industry has worked especially hard to develop new types of insecticides with novel modes of action, but this process is becoming ever harder and more costly. It is therefore vital that effective insecticide resistance management (IRM) strategies are implemented, to ensure that resistance 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 the 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.

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:



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.

Nerve and Muscle Targets

Most current insecticides act on nerve and muscle targets. Insecticides that act on these targets 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. 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 Cyclodiene 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, λ-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 Sulfoxaflor, *4D* Flupyradifurone

Group 9 Modulators of Chordotonal Organs

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

9B Pymetrozine

9C Flonicamid

Group 22 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 [1]

Group 28 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)

[1] limited spectrum – a few selected leafhoppers (e.g. white apple leafhopper)

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 or by directly affecting cuticle formation/deposition or lipid biosynthesis. Insecticides that act on individual targets in this system 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 Pyriproxyfen

Group 15 Inhibitors of chitin biosynthesis, Type 0

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

Benzoylureas (e.g. Novaluron, Bistrifluron)

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 (e.g. Buprofezin)

Group 23 Inhibitors of lipid synthesis

Inhibition of acetyl Coenzyme A carboxylase, part of the first step in lipid synthesis, leading to insect death. (e.g. Spiromesifen, Spirotetramat)

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 system are generally fast to moderately fast acting.

Group 12 Inhibitors of mitochondrial ATP synthase

Inhibit the enzyme that synthesizes ATP.

12A Diafenthiuron

Group 21 Mitochondrial complex I electron transport inhibitors

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

21A Tolfenpyrad, Pyridaben

Unknown

Several insecticides are known to affect less well-described target-sites or functions, or to act non-specifically on multiple targets. Pyrifluquinazon

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.



MoA Group	Aphids	Whiteflies	Hoppers
1A	X	X	X
1B	X	X	X
2A	X	X	X
2B			X
3A	X	X	X
4A	X	X	X
4C	X	X	X
4D	X	X	X
7A	X	X	
7C		X	
9B	X	X	X
9C	X	X	X
12A	X	X	
15		X	
16		X	X
21A		X	
22A			X
23	X	X	
28	X	X	X
UN	X	X	

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Insecticide Mode of Action Classification:
A Key to Effective Insecticide Resistance Management in Mosquitoes

IRAC
Insecticide Resistance Action Committee

www.irac-online.org

Introduction and background

Mosquitoes are vectors of many of the world's key human diseases, including malaria. The emergence of species resistant to insecticides widely used in vector control has the potential to impact severely on the control of these disease vectors. This may have a dramatic effect in Africa, as few affordable alternative insecticides are available for vector control. The extensive use and misuse of insecticides for agriculture and vector control has contributed to this problem. The lack of available suitable alternative insecticides for vector control has also been an issue, for example only pyrethroids are currently recommended by WHO for use on long lasting insecticide treated mosquito nets. Industry is now working in collaboration with the Innovative Vector Control Consortium (IVCC) to find new classes of insecticides with novel modes of action for use in public health. However the identification and approval process of a new active can to take up to 10 years and ~$200 million. It is therefore vital that effective insecticide resistance management (IRM) strategies are implemented to ensure that the efficacy of existing compounds can be maintained for as long as possible.

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 the IRAC website www.irac-online.org, this IRAC MoA classification list along with the IRAC Vector Manual provides NGOs, ministers, advisors, extension staff, consultants and public health professionals with a guide to the selection of insecticides and planning of IRM programs.

Effective IRM strategies

Sequences or alternations of MoA

All effective insecticide resistance management (IRM) strategies seek to minimise the selection of resistance to any one type of insecticide. It is recommended that alternations, mosaics or rotations of compounds from different MoA groups can provide sustainable and effective IRM for mosquitoes. This ensures that selection by compounds in the same MoA group is minimised, and resistance less likely to evolve. The practice of using an insecticide until resistance occurs becomes a limiting factor in public health and is rapidly eroding the number of suitable insecticides for vector control. The limitations of current public health interventions such as IRS and LNs mean that successive generations of the mosquito are exposed to compounds from the same MoA group. This makes IRM in public health more challenging than in agriculture. Therefore insecticide resistance monitoring is of vital importance, this can be done using bioassays (WHO[1] and/or CDC[2] standard test kits and procedures) and also biochemical/ molecular methods. This testing should ideally be conducted annually to monitor any changes in susceptibility that may occur and thus allow timely intervention of alternative vector control methods.

(Web sites: 1. www.who.int/whopes/resistance/en/ 2. www.cdc.gov/ncidod/wbt/resistance/assay/bottle/index.htm)

Nerve and Muscle Targets

Most current insecticides act on nerve and muscle targets. Insecticides that act on these targets are generally fast acting

Group 1 Acetylcholinesterase (AChE) inhibitors (Adults or Larvae)

Inhibit AChE, causing hyperexcitation. AChE is the enzyme that terminates the action of the excitatory neurotransmitter acetylcholine at nerve synapses.

1A Carbamates (e.g. propoxur & bendiocarb),
1B Organophosphates (e.g. Temephos, malathion, fenitrothion, pirimiphos-methyl)

Group 3 Sodium channel modulators (Adults only)

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. deltamethrin, permethrin, cypermethrin, alpha-cypermethrin, lambda-cyhalothrin, bifenthrin, etofenprox)
3B DDT

Group 5 Nicotinic acetylcholine receptor (nAChR) allosteric modulators (Larvae only)

Allosterically activate nAChRs, causing hyperexcitation of the nervous system. Acetylcholine is the major excitatory neurotransmitter in the insect central nervous system.

5 Spinosyns (e.g. spinosad,)

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 or directly perturbing cuticle formation/deposition or lipid biosynthesis. Insecticides that act on individual targets in this system 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 Juvenile hormone mimics (e.g. Methoprene, Hydroprene)
7C Pyriproxyfen

Group 15 Inhibitors of chitin biosynthesis Type 0

Incompletely defined MoA leading to inhibition of chitin biosynthesis

15 Benzoylureas (e.g. Diflubenzuron, Novaluron)

Midgut

Derived from bacteria, these toxins need to be ingested and disrupt the insect midgut membranes

Group 11 Microbial disruptors of insect midgut membranes
Bacillus thuringiensis var. israeliensis and Bacillus sphaericus

Adulticides WHOPES Approved Uses

MoA	Class	Insecticide or Product	IRS	ITN	LN
1A	Carbamate	Bendiocarb, Propoxur	✓	X	X
1B	Organophosphate	Malathion Fenitrothion Pirimiphos-methyl	✓	X	X
3A	Pyrethroid	Alphacypermethrin	✓	✓	✓*
		Deltamethrin	✓	✓	✓*
		Permethrin	X	✓	✓*
		Etofenprox	✓	✓	X
		Lambdacyhalothrin	✓	✓	✓
		Bifenthrin	✓	✓	X
		Cyfluthrin	✓	X	X
		Deltamethrin + PBO	X	X	✓
3B	Organochlorine	DDT	✓	X	X

* Indicates Full WHOPES approval as an LN
(NB: Those without * indicates Interim approval only.)

Larvicides WHOPES Approved

MoA	Class	Insecticide or Product
1B	Organophosphate	Temephos, Chlorpyriphos, Pirimiphos-methyl, Fenthion
5	Spinosyns	Spinosad
7A	Juvenile Hormone Mimics	Methoprene, Hydroprene
7C	Pyriproxifen	Pyriproxifen
15	Benzoylureas	Diflubenzuron, Novaluron
11	Bacterial Larvicide	*Bt var. israeliensis* and *Bacillus sphaericus*

Further reading:

Prevention and management of insecticide resistance in vectors and pests of public health importance www.irac-online.org

WHO (2006): Pesticides and their application. WHO/CDS/NTD/WHOPES/GCDPP 6th edition, 114pp www.who.int/whopes/en/

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CropLife INTERNATIONAL

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

Insecticide Resistance Action Committee

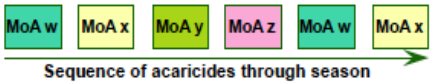
www.irac-online.org

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 Cyclodiene 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, Dicofol.

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

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 Clofentezine, 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).

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Grazie

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