

Introduction and biological background

Green peach aphid *Myzus persicae* (Sulzer) is a cosmopolitan and polyphagous pest. Primary hosts are predominantly *Prunus persica* (including var. nectarina), while secondary hosts include plants in 40 different plant families as well as economically important crops. In addition to direct plant damage, *M. persicae* is a highly efficient vector of over 100 different plant viruses.

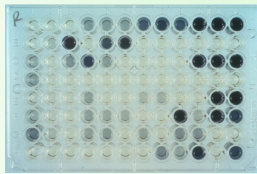
First reports of insecticide resistance in *M. persicae* date to 1955. Three major resistance mechanisms presented here in short have been detected to date. Altogether, they particularly confer resistance of *M. persicae* to carbamates, organophosphates (OP's) and pyrethroids. Whereas no validated field resistance reports are known for MoA groups 4A, 9 and 23. Combined use of resistance detection techniques against field populations provides farmers with information on possible problems with certain insecticides and helps in better management strategies for *M. persicae* control.

1. Enhanced expression of esterases

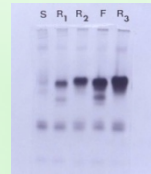
- esterases are soluble enzymes hydrolysing ester bonds
- carboxylesterases (E4 and EF4) sequester or degrade esters of organophosphate and carbamate insecticides before they reach their target site
- overproduction of named carboxylesterases causes resistance of *M. persicae* to organophosphates, carbamates and some pyrethroids
- detection is done by artificial model substrates or by ELISA
- simple handling and quick determination are further advantages



Homogenizer



ELISA detection of E4



Electrophoresis

References

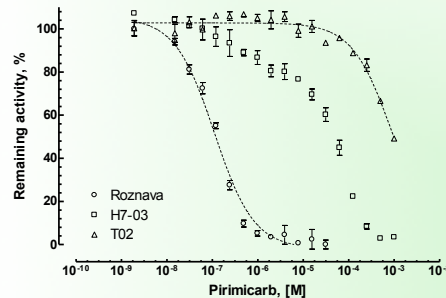
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Myzus persicae resistance around the globe



2. MACE (modified acetylcholinesterase)

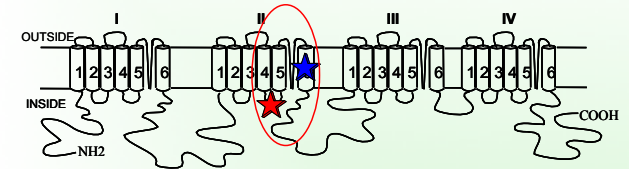
- carbamates and OP's act by inhibiting acetylcholinesterase (AChE)
- substitution of a serine at position 431 by a phenylalanine in AChE-2 leads to target site resistance to dimethylcarbamates, such as pirimicarb
- the resistance mechanism is genetically dominant
- resistant aphids are identified with microplate AChE inhibition assays



Inhibition of acetylcholinesterase by pirimicarb in different strains of aphids (strain Roznava is homozygously susceptible; strains H7-03 and T02 are heterozygously and homozygously resistant, resp.)

3. kdr (knock-down resistance)

- pyrethroid insecticides cause knock-down resistance ("kdr" or "super kdr"), conferred by changes in a voltage-gated sodium channel protein



★ Leu 1014 to Phe (kdr mutation)

★ Met 918 to Thr (super-kdr mutation)

- voltage-gated sodium channel in the central nervous system has 4 transmembrane domains with 6 subunits each
- substitution of leucine to phenylalanine results in kdr genotypes, a mutation found in many pyrethroid resistant pest species
- kdr resistant individuals usually also show high levels of E4 esterase (which contributes to pyrethroid resistance)
- overall effects in *M. persicae* is a loss in fitness

Resistance Management Guidelines

- compounds should be used according to the label recommendations
- rotating compounds from different mode of action groups is strongly recommended
- non-chemical control measures should be incorporated (integrated pest management)

MoA Group	Primary Site of Action	Chemical Sub-group or Exemplifying active Ingredient
1	Acetylcholinesterase (AChE) inhibitors (Nerve Action)	1A Carbamates 1B Organophosphates
3	Sodium channel modulators (Nerve Action)	3A Pyrethroids, Pyrethrins
4	Nicotinic acetylcholine receptor (nAChR) agonists (Nerve Action)	4A Neonicotinoids 4C Sulfoxaflor
9	Selective homopteran feeding blockers	9B Pymetrozine 9C Flonicamid
23	Inhibitors of acetyl CoA carboxylase. (Lipid synthesis, growth regulation)	Spirotetramat
UN	Compounds – unknown/uncertain MoA	Pyrifluquinazon