Major mechanisms of insecticide resistance in green peach aphid *Myzus persicae* Sulzer

Insecticide Resistance Action Committee

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Introduction and biological background

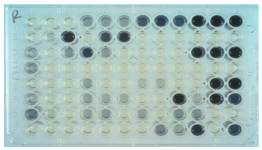
Green peach aphid *Myzus persicae* (Sulzer) is a cosmopolitan and polyphagous pest. Primary hosts are predominantly *Prunus persica* (including var. nectarina), while secondary hosts include plants in 40 different plant families as well as economically important crops. In addition to direct plant damage,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

First reports of insecticide resistance in *M. persicae* date to 1955. Five 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), pyrethroids and neonicotinoids. Whereas no validated field resistance reports are known for MoA groups 9, 23 and 28. 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.

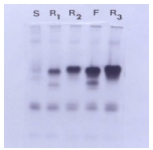
1. Enhanced expression of esterases

- 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, but less to 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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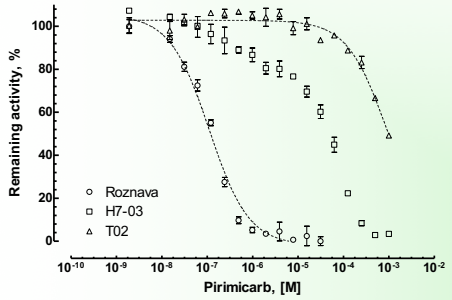
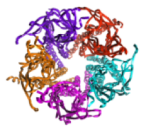
2. MACE (modified acetylcholinesterase)

- carbamates and OP´s act by inhibiting acetylcholinesterase (AChE)
- substitution of a serine at position 431 by a phenylalanine in ACE2 leads to target site resistance to dimethylcarbamates, e.g. 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. nAChR target-site resistance

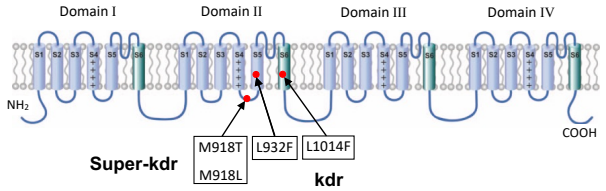


- a single point mutation, R81T in the *M. persicae* ß1-subunit of the nAChR confers resistance to IRAC MoA group 4 insecticides
- the R81T mutation confers a loss of direct electrostatic interactions of the electronegative pharmacophore with the basic arginine residue at this key position within loop D

Species	Amino Acid Number of *Myzus persicae* ß1 Subunit							
	77	78	79	80	81	82	83	84
Homo sapiens ß2	N	V	W	L	T	Q	E	W
Gallus gallus ß2	N	V	W	L	T	Q	E	W
Rattus norvegicus ß2	N	V	W	L	T	Q	E	W
Drosophila melanogaster ß1	C	V	W	L	R	L	V	W
Anopheles gambiae ß1	N	V	W	L	R	L	V	W
Bemisia tabaci ß1	N	V	W	L	R	L	V	W
Locusta migratoria ß1	N	V	W	L	R	L	V	W
Heliothis virescens ß1	N	V	W	L	R	L	V	W
Ctenocephalides felis ß1	N	V	W	L	R	L	V	W
Myzus persicae 4106A ß1	N	V	W	L	R	L	V	W
Myzus persicae 5191A ß1	N	V	W	L	R	L	V	W
***Myzus persicae* FRC ß1**	N	V	W	L	T	L	V	W

4. kdr (knock-down resistance)

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



- the mutation M918L is the main driver of high level pyrethroid resistance

5. Elevated levels of cytochrome P450

- CYP6CY3, has been shown to metabolise some neonicotinoids and nicotine. However, current knowledge suggests its expression is too low to compromise neonicotinoid field efficacy at recommended rates.

6. 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 (IPM)

IRAC main group	Mode of action	Sub-group	Chemical class or exemplifying active
1	Acetylcholinesterase inhibitors	A	Carbamates
		B	Organophosphates
3	Sodium channel modulators	A	Pyrethroids
4	nAChR competitive modulators	A	Neonicotinoids
		C	Sulfoxaflor
		D	Flupyradifurone
9	Chordotonal organ TRPV channel modulators	B	Pymetrozine
		D	Afidopyropen
23	Inhibitors of acetyl-CoA carboxylase	None	Spirotetramat
28	Ryanodine receptor modulators	None	Diamides
29	Chordotonal organ modulators - undefined target site	None	Flonicamid

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