

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.

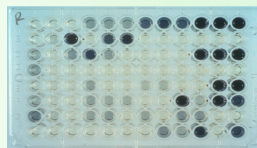
First reports of insecticide resistance in *M. persicae* date to 1955. Four major resistance mechanisms presented here in short have been detected to date. Altogether, they particularly confer resistance of *M. persicae* to carbamates, organophosphates (OP's), pyrethroids and neonicotinoids. Whereas 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

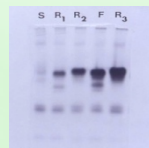
- esterases are soluble enzymes hydrolysing ester bonds
- carboxylesterases (E4 and EF4) sequester or degrade esters of organophosphate and carbamate insecticides before they reach their target site
- overproduction of named carboxylesterases causes resistance of *M. persicae* to organophosphates, carbamates, 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



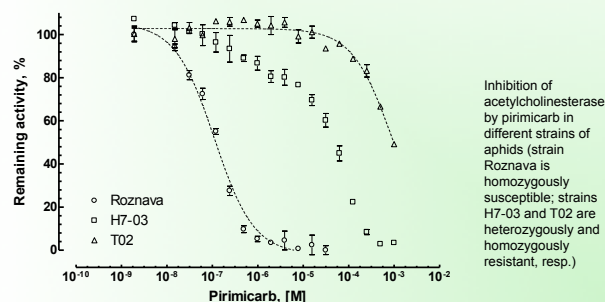
Electrophoresis

References

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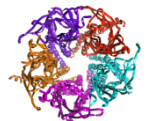
2. MACE (modified acetylcholinesterase)

- 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



3. nAChR target-site resistance

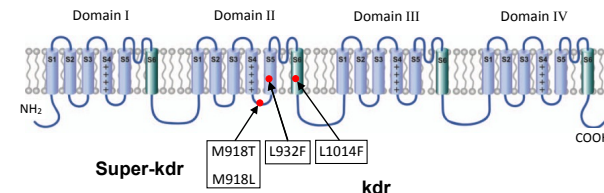
- a single point mutation, R81T in the *M. persicae* β 1-subunit (loop D) of the nAChR confers neonicotinoid resistance
- the R81T mutation confers a loss of direct electrostatic interactions of the electronegative pharmacophore with the basic arginine residue at this key position within loop D



Species	Amino Acid Number of <i>Myzus persicae</i> β 1 Subunit											
	77	78	79	80	81	82	83	84				
<i>Homo sapiens</i> β 2	N	V	W	L	T	Q	E	W				
<i>Gallus gallus</i> β 2	N	V	W	L	T	Q	E	W				
<i>Rattus norvegicus</i> β 2	N	V	W	L	T	Q	E	W				
<i>Drosophila melanogaster</i> β 1	C	V	W	L	R	L	V	W				
<i>Anopheles gambiae</i> β 1	N	V	W	L	R	L	V	W				
<i>Bemisia tabaci</i> β 1	N	V	W	L	R	L	V	W				
<i>Locusta migratoria</i> β 1	N	V	W	L	R	L	V	W				
<i>Heliothis virescens</i> β 1	N	V	W	L	R	L	V	W				
<i>Ctenocephalides felis</i> β 1	N	V	W	L	R	L	V	W				
<i>Myzus persicae</i> 4106A β 1	N	V	W	L	R	L	V	W				
<i>Myzus persicae</i> 5191A β 1	N	V	W	L	R	L	V	W				
<i>Myzus persicae</i> FRC β 1	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



- 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 (IPM)

IRAC Group	Mode of action	Subgroup	Chemical class
1	Acetylcholinesterase inhibitors	A	Carbamates
		B	Organophosphates
3	Sodium channel modulators	A	Pyrethroids
4	nAChR agonists	A	Neonicotinoids
		C	Sulfoxaflor
9	Effectors of chordotonal organs	D	Flupyradifurone
23	Inhibitors of acetyl-CoA carboxylase	B	Pymetrozine
		C	Fonicamid
28	Ryanodine receptor modulators	None	Spirotetramat
			Cyantraniliprole