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# Biochemical mechanism of malathion and parathion resistance in *Tetranchus urticae* Koch

The ability of four strains of *Tetranychus urticae* (Niagara normal, Blauvelt resistant, Leverkusen normal, Leverkusen resistant) to degrade $C^{14}$ — malathion and $H^3$ — parathion was investigated. Both resistant strains had a higher breakdown capacity for these organophosphates in vitro as well as in vivo, due to carboxyesteratic and phosphoroesteratic enzymes. Homogenates of resistant Blauvelt mites also degraded malaoxon faster than those of the three other strains. The malathion and malaoxon degradation is made responsible for the low cholinesterase inhibition in vivo after poisoning Blauvelt mites with malathion. In contrast to this type of protective mechanism the Leverkusen resistant strain developed a unique cholinesterase resistant against organophosphate inhibition (SMISSAERT, 1964; VOSS and MATSUMURA, 1964). When Niagara and Blauvelt homogenates were compared with regard to their esteratic activity against some other esters, it was found that « Blauvelt » could hydrolyze $\alpha$-naphthylbenzoate significantly faster than « Niagara ». The reverse was true for $\alpha$-naphthylacetate. A part of the enzymic activity against the former substrate may be related to the malathion carboxyesterase. Phosphatase and carboxyesterase, the former being insensitive to DFP, were similar in their properties. Since the resistance of the Blauvelt strain seems to be monofactorial (DITTRICH, 1963) it is tempting to conclude that both enzymes are controlled by one gene or a closely associated group of genes.

## RIASSUNTO

L'A. ha esaminato la capacità di degradare il $C^{14}$-malathion e l'$H^3$-parathion in 4 ceppi di *Tetranychus urticae* (Niagara normale, Blauvelt resistente, Leverkusen normale e Leverkusen resistente).

I due ceppi resistenti hanno dimostrato maggior potere di demolizione nei confronti di questi composti fosforganici, sia *in vitro* che *in vivo*, grazie alla pre-

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senza di enzimi carbossiesterasici e fosforoesterasici. Gli omogenati di Blauvelt resistenti degradano inoltre il malaoxon più rapidamente che non gli omogenati degli altri 3 ceppi. La degradazione del malathion e del malaoxon è dovuta alla bassa inibizione colinesterasica *in vivo* dei Blauvelt trattati con malathion.

In contrasto con questo tipo di meccanismo protettivo, i Leverkusen resistenti sviluppano una sola colinesterasi resistente all'inibizione organofosfatica. Raffrontando gli omogenati Niagara e Blauvelt, con riferimento alla loro attività esterasica, si rileva che i Blauvelt possono idrolizzare l'$\alpha$-naftilbenzoato con maggiore rapidità dei Niagara; l'opposto si verifica rispetto all'$\alpha$-naftilacetato.

La fosfatasi e la carbossiesterasi (la prima non sensibile al DFP) hanno dimostrato proprietà simili. Poiché la resistenza del ceppo Blauvelt sembra dovuta ad un solo fattore, l'A. ritiene che i due enzimi siano controllati da un solo gene o da un gruppo di geni strettamente associati fra loro.

## LITERATURE

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## DISCUSSION

GASSER: Is the homogenate of mites characteristic for the esterase activity? We know that in higher animals these are big differences between the different organs.

Voss: I agree with the latter fact but in mites we have to work with total homogenates. Concerning breakdown mechanisms however the use of total homogenates will be all-right, because up till now we are not interested in what organs the breakdown takes place.

SMISSAERT: You will find a difference in ChE *in vivo* inhibition whatever the mechanism of resistance will be!

Voss: I agree with you!