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Intersterility as a consequence of Tedion selections in *Tetranychus urticae* Koch

In the passed two years we have been confronted with some curious phenomena which arose from a simple study into the inheritance of resistance to Tedion. First of all I want to offer you the facts and thereafter I will give an abstract of my experimental manipulations.

In 1961 Mr MELTZER (*) gathered spidermite stocks frim several elder-bushes (*Sambucus niger* L) in a limited area in the dunes of the Netherlands. These stocks were cultivated as one colony in the laboratory under environmental constancy. The effective population size of the colony never dropped under an estimated 2,000 individuals.

It appeared that with respect to Tedion the colony as a whole reacted susceptible. From this colony series of simultaneous selections resulted in a Tedion resistant strain. This strain and the susceptible mother-colony were used for genetic research.

Genetic experiments with ovicides are especially attractive because of the possibility of toxicological determinations of haploid offsprings. Gametic analysis seems very simple in realization. However, this analysis could not be executed in my experiments. It appeared that hybrid eggs from the reciprocal crossings between the resistant strain (R) and the susceptible colony (S) were resistant to Tedion, but a sudden unexpected complication interfered with further experimentation. The unfertilized hybrids of both reciprocal crossings, which were supposed to produce the material for the mentioned gametic tests, appeared to produce a great percentage of sterile eggs, up to 60%.

Since there is no phenotypical difference between sterility and susceptibility to Tedion I can not offer you the promised data of the

(*) I am very much obliged to Mr MELTZER (Agro-biological Laboratory « Boekesteyn » at's Graveland) for placing the material at my disposal.

gametic analysis. I had to follow a roundabout route to attain my end.

There are two points which call our attention:

1) Since resistance to Tedion and sterility deal with the hatchability of eggs, one wonders if there is any causal relationship between both phenomena.

2) What is the explanation of the occurence of sterility factors in the original colony?

As you undoubtly know backcrossing procedures can give information about problems concerning relationship. Can we, for instance, dismantle Tedion resistance from the sterility factors by backcrossing the hybrids with susceptible males under simultaneous selection pressure of Tedion?

It should be borne in mind that with respect to this character it is attractive to perform these backcrosses in two different ways. Since hatchability is in picture it is quite conceivable that maternal components are involved. Therefore we started with hybrids from the crossing $S \times R$ ($\varphi \times \sigma$) and $R \times S$ respectively.

In the first series the hatchability turned to normal after 4 backcrosses, as could be stated from samples after each backcrossing. The percentages of unhatched eggs in the B_5, B_6 and B_7 of this series were equal to the percentages of the intrapopulational hatchability of S and R respectively.

During the backcross procedures the impression was created that one mendelian factor is responsible for the tolerance to the selection pressure, since escorting selections with Tedion caused about 50 % kill in repetition. This was confirmed later by other experiments.

After the seventh backcrossing in this series a strain was established in which homozygosity for resistance can be taken for granted. This strain showed good genetic affinity to the S-colony. So the sterility factors were apparently be ruled out by substitution. By means of the described route of backcrossing I finally acquired suitable material for further experiments. It is important to mention that the data obtained from gametic analysis were in accordance with the existence of one mendelian dominant. The progeny of the hybrids of crosses between the backcross-strain and the susceptible colony consisted of two classes of eggs (susceptible and resistant) in a 50-50 ratio.

The second series showed a more complex course. In this series,

it should be recalled that the mothers of the hybrids were from the resistant strain. It appeared that the sterility-factors could not be substituted within a span of seven backcrossings. The percentage of unhatched eggs dropped to 18 % in the B_2 and remained on that level during the further course. The relation resistance-intersterility could not be dismantled in this series. In the first series starting from the cross S × R, resistance could be separated from the sterility factors. What indicates the difference between both series?

Several explanations can be offered, but the most plausible inference is the presence of an extrachromosomal factor. Of course the stated phenomena cannot be explained by this factor alone, because of the normal percentages of unhatched eggs, which we find in the R-strain and the S-colony. The situation is apparently more complicated. In fact it is necessary to assume at least two chromosomal factors which are interacting with this cytoplasmic component.

I will not enter into an exact analysis of this interaction, but the following table can illustrate the complexity (table I):

TABLE I. - *Percentages of unhatched eggs in different F_2-progenies from crossings between the susceptible (S) colony and the resistant (R) strain.*

Mass crossing ♀ × ♂	F_2 diploid and haploid		F_2 haploid	
	eggs concerned	mortality in %	eggs concerned	mortality in %
S × S . . .	453	7	311	6
R × S . . .	566	26	516	31
S × R . . .	309	40	559	61
R × R . . .	412	8	547	8

Let us consider now our second question, the existence or maintenance of sterility factors in the S-colony. Since sterility is complex, as at least two chromosomal and one extrachromosomal factor are involved, the most obvious explanation is to assume that the resistance response resulted from a kind of intergroup selection in the beginning.

It has been stated by several authors (BOUDREAUX, 1963, HELLE and PIETERSE, 1965), that even adjacent populations can show genetic incompatibility. It is therefore not strange to assume that the popu-

lation in the laboratory was very heterogenous from the beginning. As the population reacted susceptible in the beginning, the frequency of the resistant mites would be low. One of the stocks gathered by Mr Meltzer had probably a resistance focus, and was not fully compatible with the other stocks. In the intergroup-competition, the R-group is supposed to drift away by a relatively greater loss by intersterility as compared with the other groups.

It seems an attractive hypothesis that the properties of the resistant strain are interknit wih a chromosomal alteration, like a translocation or an inversion. It would be also in agreement with the fact that the F_1 is normal, but after the meiosis (thus in the F_2) the incompatibility stands out. But it is hardly acceptable that this is established by one step, since in the sterility also an extrachromosomal component is integrated. I think that this complex sterility must have resulted from an intergroup variability in the original susceptible colony.

Moreover, it must be mentioned that this year a selection with Tedion in the S-colony had no result. More than 50,000 eggs were tested and all were killed. A number of 50,000 is more then 10 times the effective population size.

Upon summarizing these data, I think we are permitted to state that in a population of laboratory size the resistance could not have maintained itself during four years in the laboratory. This underlines the thought that the resistance response resulted from an intergroup selection.

SUMMARY

Starting with a colony of *T. urticae*, susceptible (S) to Tedion, a number of subsequent selections resulted in a strain R, highly resistant to tedion (resistance factor + 500 x).

In order to get information about the resistance to tedion crossing experiments were made between R and S. Tedion resistance proved to be a dominant character that could be transmitted by females and males both. Hybrid females produced eggs which were partly sterile. Therefore a simple genetic analysis was inadequate, since the unmated hybrid females from the crossing S♀ × R♂ produced eggs that were sterile up to 60 %. The eggs from unmated hybrid females from the reciprocal cross R♀ × S♂ remain unhatched up to 40 %.

Backcrossing the hybrid S♀ × R♂ under selection pressure removed the intersterility. However backcrossing R♀ × S♂ hybrids with S males did not remove the intersterility complex.

Genetic analysis with the first backcross-strain resulted in demonstration of a major factor responsible for the resistance to tedion.

RIASSUNTO

Partendo da una colonia di *T. urticae* sensibile al tedion (S), con successive selezioni è stato ottenuto un ceppo (R) altamente resistente al tedion (fattore di resistenza + 500 x).

È stato dimostrato, mediante incroci fra R ed S, che la resistenza al tedion è un carattere dominante e che può essere trasmesso sia dalle femmine che dai maschi. Le femmine ibride producono uova in parte sterili.

Una semplice analisi genetica risulta pertanto inadeguata dato che femmine vergini ottenute dall'incrocio S♀ × R♂ depongono il 60 % di uova sterili e femmine vergini derivanti da R♀ × S♂ il 40 %.

Il reincrocio degli ibridi S♀ × R♂ sotto pressione selettiva elimina l'intersterilità. Il reincrocio di ibridi R♀ × S♂ con maschi S non rimuove invece il complesso di intersterilità.

L'analisi genetica sul ceppo di primo reincrocio dimostra la presenza di un fattore principale responsabile della resistenza al tedion.

LITERATURE

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DISCUSSION

MELTZER: I should like to add that after collecting the S-strain in 1961 we selected a part of the stock with Tedion and a resistant strain was rapidly obtained. In 1962 and 1963 we repeated the selection several times starting from the original susceptible strain but we did not succeed in obtaining another resistant strain anymore.

OVERMEER: In 1963 I received your S-strain and the R-strain. A series of subsequent selections (shortly after I received the strains), resulted in another R-strain. Later on, repetitions of the selection program in the S-strain had no result. The old R-strain did not differ in properties from my new R-strain. It is my opinion that somewhere there must have been an infection of R animals into the S-colony. I don't believe that the result of my first selection program has anything to do with some mutation in the S-colony.