

Short communication

Nuclear-nuclear and nuclear-cytoplasmic interactions contribute to the reproductive incompatibility between two strains of the twospotted spider mite

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Introduction

When different strains of the twospotted spider mite (*Tetranychus urticae* Koch (Acari: Tetranychidae)) are crossed, hybrids are usually obtained, but high mortality of eggs laid by the hybrid females is often observed (Boudreaux, 1963; Helle & Pieterse, 1965; Overmeer & Van Zon, 1976; De Boer, 1981, 1985). Little is known about the genetic basis of this partial sterility of interstrain hybrids. The partial sterility could be caused by incompatibilities between nuclear genes contributed by the different parent strains, incompatibilities between nuclear genes contributed by one parent strain and cytoplasmic elements contributed by the other, or production of unbalanced gametes due to the parent strains differing with respect to one or more chromosomal rearrangements. In some cases, the mortality rate of eggs laid by the F₁ females depends on the direction of the original cross, giving evidence that nuclear-cytoplasmic interactions are involved in the partial sterility (Overmeer & Van Zon, 1976; De Boer, 1982). However, the lack of such differences between reciprocal crosses cannot be used to rule out nuclear-cytoplasmic interactions in other cases, because cytoplasm contributed by each parent strain might be equally incompatible with nuclear genes contributed by the other (De Boer, 1982). Direct evidence for the role of nuclear-nuclear interactions or chromosome rearrangements in causing the partial sterility of hybrids between

naturally occurring *T. urticae* populations is lacking. Investigation of the possible role of chromosome rearrangements in causing the partial sterility has been hampered by the extremely small size of the chromosomes of *T. urticae* (Pijnacker & Ferwerda, 1976).

One way to distinguish between the three possible causes of the partial sterility phenomenon is to cross strains differing with respect to one or more marker genes. If a marker locus is linked to a gene (or genes) involved in the partial sterility, then eggs that receive different marker alleles from the heterozygous F₁ females may differ in their probability of hatching. Thus, if surviving male (haploid) progeny of the F₁ females are examined, the ratio of the two genotypes at the marker locus could deviate from the expected ratio of 1:1 (*T. urticae* is haplodiploid, meaning that males are haploid and develop from unfertilized eggs). If an allele from one parent strain at a locus linked to the marker locus causes egg mortality by interacting with alleles at other nuclear loci contributed by the opposite parental strain, the distortion of genotypic ratios at the marker locus should be in the same direction in the reciprocal crosses. However, if the partial sterility allele causes egg mortality by interacting with the cytoplasm provided by the second strain, then distorted ratios should be observed only in one reciprocal cross. Finally, if chromosome rearrangements are the cause of the partial sterility, normal genotypic ratios at the marker locus

would most likely result. This is because balanced gametes produced by the hybrids should contain the marker alleles in a 1:1 ratio; thus, distorted ratios at the marker locus would be expected only if one of the unbalanced meiotic products were to give rise to viable males, an unlikely possibility considering the haploidy of males.

Here, I report that hybrids between two strains of *T. urticae*, one bearing the marker mutation *white-eye*, produced surviving male offspring in ratios strongly deviating from 1 white-eye: 1 wild-type, along with a considerable proportion of inviable eggs. The distorted ratios were in the same direction in both reciprocal crosses, and the data indicate that they were caused by differences in the hatching rates of eggs bearing the *white-eye* and wild-type alleles, rather than distorted segregation ratios at meiosis. Thus, the results imply that a gene contributed by one of the parent strains at a locus linked to the *white-eye* locus, or several such linked genes, caused egg mortality when combined with nuclear genes at other loci contributed by the opposite parental strain. In addition, the hatching rates of eggs laid by hybrid females differed substantially between the reciprocal crosses, indicating that nuclear-cytoplasmic as well as nuclear-nuclear interactions contributed to the observed partial sterility of hybrids.

Materials and methods

In February of 1986, I crossed two strains of *T. urticae*. One ('Ann Arbor [AA] wild-type') was derived from mites collected in August, 1985, in Ann Arbor, Michigan, USA, on *Chelidonium majus*. The other ('North Carolina [NC] white-eye') was a stock bearing the *white-eye* mutation (see Helle, 1985, for description); this stock was derived from three mutant males discovered in the progeny of a single wild-type female of a strain originally collected in Chowan Co., North Carolina, USA, in July, 1984 (two different, non-allelic mutations giving the white-eye phenotype have been described in *T. urticae* [Helle, 1985]; it is not known which of these was present in the NC stock). The NC white-eye stock, when crossed to the parent NC wild-type stock, produced

females that gave rise to male progeny in the expected ratio of 1 wild-type: 1 white-eye (51.9% of 378 hatchlings were white-eye).

All mite strains were reared on detached lima bean leaves held on wet cotton at room temperature (22–27 °C), under continuous fluorescent lighting. For the crosses, I placed one quiescent deutonymph female (i.e., female in the midst of the final molt) from one strain on a lima bean leaf disc (15 mm diameter) along with three males of the opposite strain. Nine such crosses were performed in one direction and seven in the other. The females were allowed several days to emerge, mate, and lay eggs, and then were removed. Viability of the F₁ was not monitored closely, but appeared to be high. From the progeny of each cross, I took one or two sets of 1–3 quiescent deutonymph females; each set was placed on a separate leaf disc. These virgin females were allowed to lay ca. 20 eggs and then were removed. Hatching success and the proportion of white-eye mites among the haploid male hatchlings were monitored.

Results and discussion

F₁ females from both reciprocal crosses laid a considerable proportion of eggs that failed to hatch (Table 1; for comparison, hatching rates of eggs laid by fertilized females of both parent strains were 96%, N > 300 eggs in each case; although eggs laid by virgin females typically hatch at a lower rate than those laid by fertilized females, the difference is almost always slight [Overmeer & Van Zon, 1976; De Boer, 1979]). In addition, hatching success of eggs laid by F₁(AA wild-type ♀ X NC white-eye ♂) females was substantially lower than that of eggs laid by females from the reciprocal cross (Table 1; because there was significant heterogeneity of hatching rates among the progeny of females from different individual crosses within each reciprocal cross [chi-square tests, $P < 0.002$ in each case], I avoided pooling eggs from the different individual crosses for statistical comparison of hatching rates between reciprocal crosses). The difference in hatching rates between the reciprocal crosses indicates that a nuclear-cytoplasmic interaction contributed to the

Table 1. Results of crosses between the Ann Arbor wild-type and North-Carolina white-eye strains

Cross (♀ × ♂)	No. individual crosses	Mean no. F ₂ male eggs laid ^a	Mean percent hatching (S.D.)	Total no. hatchlings	Percent white-eye ^b
NC white-eye × AA wild-type	9	31.6	66.7 (25.5) ^c	190	58.4
AA wild-type × NC white-eye	7	38.1	24.5 (12.7)	68	67.6

^a I.e., per all F₁ females from the same individual cross.

^b The ratio of white-eye to wild-type hatchlings did not vary significantly among the progeny of females from different individual crosses within each reciprocal cross (chi-square tests, $P > 0.3$ for each).

^c Mean percent hatching differs significantly between reciprocal crosses, $P < 0.005$, Mann-Whitney U test ($U = 6.0$).

partial sterility of at least the F₁(AA wild-type ♀ × NC white-eye ♂) females.

Surprisingly, the proportion of white-eye mites among the hatchlings was substantially greater than 50% in both reciprocal crosses (Table 1; the deviation from 50% is statistically significant in each case, $P < 0.05$, chi-square test). To determine whether the biased genotypic ratios were caused by lower hatching rates of wild-type eggs than of white-eye eggs, I calculated Spearman rank correlation coefficients between the proportion of eggs hatching on each leaf disc and the proportion of hatchlings that were white-eye. Simple calculations show that if the biased ratios were caused by different hatching rates of white-eye and wild-type eggs, these correlations should be negative; in contrast, if the biased ratios were caused by more white-eye than wild-type eggs having been present originally (due perhaps to distorted segregation ratios at meiosis), no correlation between the variables should be observed. The correlations were -0.59 for the progeny of F₁(NC white-eye ♀ × AA wild-type ♂) females ($P < 0.05$, $N = 15$ discs) and -0.46 for the progeny of F₁(AA wild-type ♀ × NC white-eye ♂) females ($0.1 < P < 0.2$, $N = 12$ discs). Thus, the preponderance of white-eye hatchlings in both crosses was apparently due to lower hatching rates of wild-type eggs. Apparently, an allele contributed by the AA strain at a locus linked

to the *white-eye* locus caused egg mortality when combined with alleles at other nuclear loci from the NC strain (of course, it is possible that a group of linked loci were involved).

The hatching rates of white-eye and wild-type eggs can be estimated from the data in Table 1 by assuming that 50% of the eggs were white-eye and 50% were wild-type. For the NC ♀ × AA ♂ cross, this gives hatching rates of 0.78 and 0.56 for white-eye and wild-type, respectively; for the reciprocal cross, the corresponding values are 0.34 and 0.16. Thus, the partial sterility allele contributed by the AA strain at the locus linked to the *white-eye* locus caused a considerable proportion of the egg mortality in both reciprocal crosses. In fact, the effect of this allele is probably underestimated by the above figures, because crossing over between the partial sterility locus and the *white-eye* locus may have produced some white-eye eggs bearing the partial sterility allele from the AA strain, and some wild-type eggs bearing the alternative allele from the NC strain.

These results provide the first evidence that interactions between different sets of nuclear genes are sometimes responsible for the partial sterility of interstrain hybrids in *T. urticae*. Analysis of the partial sterility phenomenon using additional marker loci would be valuable. If enough markers are found to permit construction of a genetic map of *T. urticae*, this approach may eventually allow the mapping of factors causing the partial sterility phenomenon, as well as estimation of the magnitude of their effects.

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References

- Boer, R. de, 1979. Investigations concerning the development of reproductive incompatibilities between populations of the spider mite, *Tetranychus urticae*. Z. Angew. Entomol. 87: 113–121.
- Boer, R. de, 1981. Genetic affinities between spider mite *Tetranychus urticae* populations in a non-agricultural area II. Entomol. Exp. Appl. 30: 63–67.

- Boer, R. de, 1982. Nucleo-cytoplasmic interactions causing partial female sterility in the spider mite *Tetranychus urticae* Koch. (Acari: Tetranychidae). *Genetica* 58: 17–22.
- Boer, R. de, 1985. Reproductive barriers. In: W. Helle & M. W. Sabelis (eds), *World Crop Pests, Vol. 1A. Spider Mites: Their Biology, Natural Enemies and Control*. Elsevier, Amsterdam: 405 pp.
- Boudreaux, H. B., 1963. Biological aspects of some phytophagous mites. *Ann. Rev. Entomol.* 8: 137–154.
- Helle, W., 1985. Genetics. In: W. Helle & M. W. Sabelis (eds), *World Crop Pests, Vol. 1A. Spider Mites: Their Biology, Natural Enemies and Control*. Elsevier, Amsterdam: 405 pp.
- Helle, W. & A. H. Pieterse, 1965. Genetic affinities between adjacent populations of spider mites (*Tetranychus urticae* Koch). *Entomol. Exp. Appl.* 8: 305–308.
- Overmeer, W. P. J. & A. Q. Van Zon, 1976. Partial reproductive incompatibility between populations of spider mites (Acarina: Tetranychidae). *Entomol. Exp. Appl.* 20: 225–236.
- Pijnacker, L. P. & M. A. Ferwerda, 1976. Differential Giemsa staining of the holokinetic chromosomes of the two-spotted spider mite, *Tetranychus urticae* Koch (Acarina, Tetranychidae). *Experientia* 32: 158–160.