
In previous notes (DIS 46;54, C.R. Acad. Sc. Paris 272:2484, 1971 and 275:933, 1972) we reported the discovery of a female sterility consequent to the cross between two laboratory strains of Drosophila melanogaster; the first one bears the gene sepia (se), the other one is the well known wild type Oregon strain. Most of the eggs laid by the F₁ females from the cross se ♀ by Oregon ♂ fail to hatch, not going beyond the blastoderm stage. This sterility can be characterized by two peculiar physiological features. First, more eggs hatch when females grow older. Second, the sterility vanished nearly completely, albeit reversibly, when egg-laying females were put at 30°C. F₁ females from the reciprocal cross, Oregon ♀ by se ♂, and males from both crosses, are normally fertile.

We are reporting here the first chief points which have come out from the work on this phenomenon in our laboratory.

I - The three groups of strains:

Quite a number of strains, some being laboratory strains from "le laboratoire de Génétique des virus du C.N.R.S., Gif-s/Yvette, France" and others coming from flies caught in the wild, were assayed for their behaviour in the crosses with se and Oregon strains. They could be separated into three groups:

a) Inducer strains. They behave like the Oregon strain. They can intercross with this strain, without any sterility in the offspring, and sterile F₁ females arise when they are used as males in crosses with sepia females. All the strains (30) from the wild are inducer.

b) Reactive strains. They behave like the sepia strain. They breed with it, without bringing out any sterility, but produce sterile females when crossed with any inducer male.

c) Neutral strains. They breed freely either with the Oregon or the sepia strain. The tested laboratory strains are distributed in the three groups.

II - Genetic investigations:

The sterility is met again in the generations which follow the original cross, allowing genetic investigations to be carried on.

Sterile females arise when two factors, which have been called R and I (reactive and inducer factors) meet in the same fly. They are propagated in isolation, in the original strains. The two factors appear to be inherited in quite different ways.

A) Inheritance of the R factor. R is transmitted normally by maternal inheritance, and is independent of chromosomal segregations. An important complication arises, however, from the existence of a large variability in the R factor. A reactive female may be characterized by the mean hatching frequency of the eggs laid by its daughters by an inducer male. On this basis, it is observed that most of the reactive strains hold a mixture of "strong reactive" females whose daughters have but a very low fertility, and "weak reactive" females, which lead to a nearly normal fertility. Every kind of intermediate is apparently found.

The character responds to selection and "strong" as well as "weak" families were built artificially or found to have arisen spontaneously. Long lasting genetic stability seems however difficult, or perhaps impossible, to obtain, and spontaneous genetic drifts were frequently observed. These observations leave open the possibility that so called "neutral strains" are but very "weak reactive" strains, which may drift occasionally towards strongness.

Croses were made between "strong" and "weak" reactive families with the following results. The character is inherited chiefly from the maternal side. A slight, but significant deviation towards the paternal side is however regularly observed. This implies that in crosses within the reactive strains group at least, the R factor may be transmitted by males.

B) Inheritance of the I factor. From but preliminary investigations, it appears that the I factor, though in some circumstances it follows segregation of the chromosomes, does not behave like a regular Mendelian gene (or genes). Indeed, chromosomes from reactive strains appear liable to acquire the inducer potentiality, independently of any specific recombination.

Rules of transmission of the two factors allow the possibility of introducing both of them in the same strain. Thus, "autosterile" strains are built in which females are more or less sterile and, with any kind of males, breed daughters with the same kind of sterility. Males of these autosterile strains are inducer.

In our current working hypothesis, we assume that the death of the eggs arises from a complementation between inherited symbionts.