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Soutullo, D.[1] and E.Costas.[2] 1-Universidad de Santiago, Espana. 2-Instituto Espanol de Oceanografia, La Coruna, Espana. A method to find the genetic origin of a mutational unstability phenomenon in Drosophila melanogaster.

The present method allows us to tell whether a mutational unstability phenomenon is due to the action of mendelian genes or to mutator polygeneic systems, or even whether it is caused by the presence of transponsable genetic elements.

The procedure consists of substituting every chromosome of the mutator strain--which might eventually be responsible for the unstability--for stable chromosomes marked with dominant markers while making sure that no crossing-over will take place between the different chromosomes (due to the presence of inversions in the marked chromosomes). Previously the 'dot' of the unstable strain has been substituted by means of balancers (it is not described in the Figure), and then it is tested whether the unstability remains. In this way we are able to avoid the taking into account of the IV chromosome in order to establish the possible conclusions of the analysis.

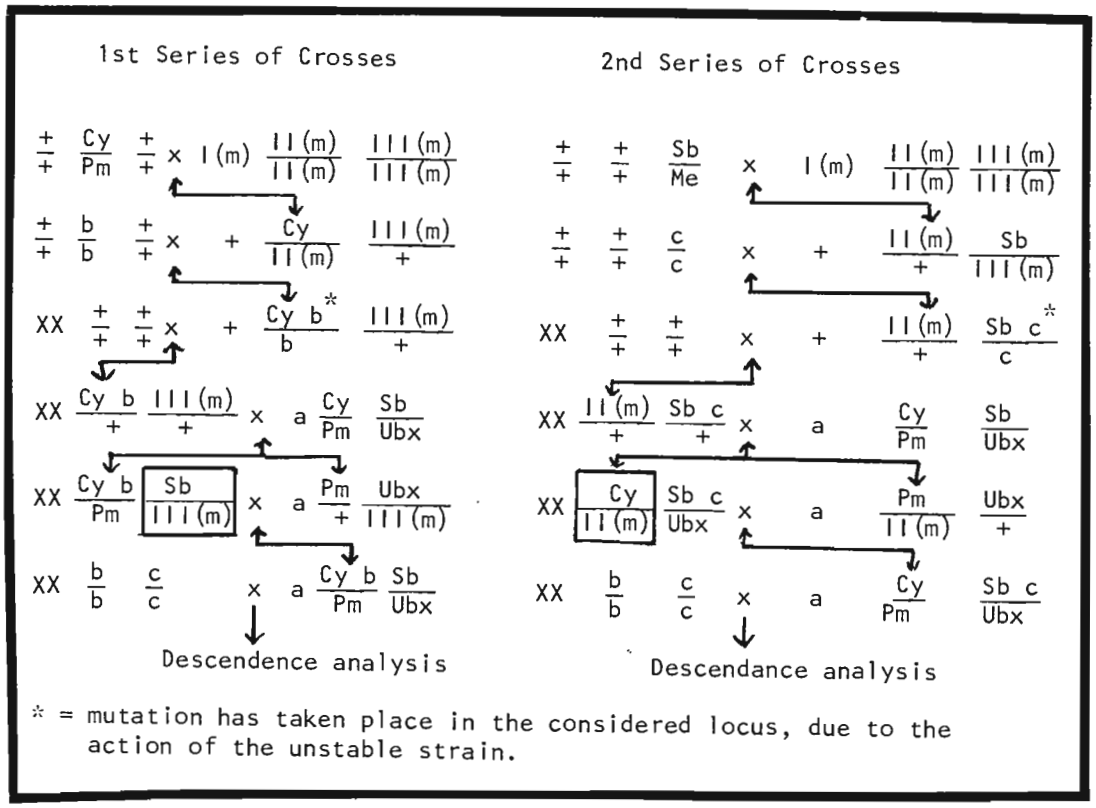
Two series of crosses were performed, one in which the unstability appears initially associated with the II chromosome and another one in which it appears associated with the III chromosome, testing at the end of each series if the unstability remained. This test is performed by checking the sensitive to mutagenic action loci in every major chromosome by means of adequate recessive markers (see Figure).

The persistence of unstability would indicate that its origin is due to the presence of any type of transponsable genetic element, except if a double crossing-over between an unstable chromosome and its stable marked homologous one would have taken place. From the comparison between both series of crosses we observed the hypothetical double crossing-over to have taken place--if it existed--between homologous III chromosomes in the first case, whereas the double crossing-over would have taken place between homologous chromosomes in the second case (noted by squares in the Figure).

From the analysis of results we can get the following conclusions:

Case 1: Presence of mutation at the end of both series of crosses: The unstability would be due to transponsable genetic elements.

Case 2: Presence of mutation at the end of the first series of crosses and absence in the second series: The unstability would be caused by genes of the III chromosome in the unstable strain.



Case 3: Presence of mutation at the end of the second series of crosses and absence on the first one: The unstability would be due to genes of the II chromosome in the unstable strain. Case 4: Absence of mutation at the end of the both series of crosses: The unstability would not be caused by transponsable genetic elements, without being able to specificate which genes of the unstable strain are responsible for it.

NOTES: (1) The chromosomes coming from the unstable strain are indicated by the notation (m). (2) 'a', 'b' and 'c' indicate the recessive markers used in order to detect the presence of mutation.

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Stark, W.S., K.Srivastava, S.D.Carlson* and M.B.Garment.* University of Missouri, Columbia, USNA. *University of Wisconsin, Madison USNA. Characteristics of none, a mutant with no ocelli and narrow eyes.

Drosophila mutants with abnormalities of the visual system have been widely used for studies of the development and function of the visual system. Some years ago, one of us (WSS) obtained a mutant from Allen Shearn at The Johns Hopkins Univeristy. Shearn had names this mutant "no ocelli, narrow eyes (none)." In an effort to process this mutant for further electrophysiological studies of receptor function and microspectrophotometric studies of the photopigment, this mutant was made white eyed with w using standard Mendelian genetics. Now only the w;none stock is retained. The initially disappointing finding was that the compound eye had no electroretinogram (ERG), and thus, if there were receptor cells, they did not work (c.f. Stark et al. 1976). Further, the eye showed no deep pseudopupil or optical density changes, thus revealing no photopigment (c.f. Stark & Johnson).

Based on these physiological findings we sought ultrastructural evidence to explain this dysfunction. We processed flies for histology, transmission electron microscopy and scanning electron microscopy. The accompanying plate shows the external featuresof the head from scanning electron micrographs. (Calibration bars show 100 microns, top row, 5 microns, remainingrows.) The external morphology of the compound eye is in some disarray (right) when compared with a control fly (left). On higher magnification (second row) the fusion of corneal facets and displacement of corneal hairs can be observed; yet the mutant does have the characteristic corneal nipples, the fine granularity which functions as an impedance matching device and an anti-reflective coating. On close examination of the ocellar area (third and fourth rows) the normal fly's ocellar lenslets and the remnants of the mutant's lenselts can be observed.

We have preliminary observations of the compound eye from the High Voltage Electron Microscope (HVEM), an NIH Biotechnology Resource in Madison, WI (c.f. Stark & Carlson 1983). In spite of the external corneal disarray, Semper cells are present as are the pseudocones which the former secrete. A corneal lenslet with its underlying pseudocone make up the distal dioptric (optical) apparatus for one ommatidium. Proximally, the compound eye's peripheral retina is separated from the first synaptic neuropile (lamina ganglionaris) by a basement membrane. Between these dioptric and basement membrane areas there is a complete absence of photoreceptor cells. Most of the volume of the peripheral retina is occupied by pigmented glia based on cell morphology, electron density and types of organelles. In a survey of this metaplasia we found no recognizable specializations such as rhabdomeres (the microvillar organelles which house the visual pigment molecules). Also, beneath the basement membrane, glial elements and interneurons exist. Yet we have observed no organization such as the normal fly's optic cartridges which are formed by terminals of receptor axons onto discrete clusters of lamina monopolar interneurons. In conclusion, the finding of an all glial cell mass in the peripheral retina readily explains the lack of an ERG and the deep pseudopupil.

The morphological features of none's compound eye are not unlike those of Glued mutants (Harte & Kankel 1982). Possibly none is an allele of Glued. Unfortunately, the micrographs of Glued do not show the ocellar area.

We hope to further characterize the specific cellular and developmental deficits in this mutant. It would be particularly useful to section the ocelli of none flies and to compare these structures to those of normal flies. To our knowledge, ocellar ultrastructure in