Male recombination and mosaics induced in *Drosophila melanogaster* by feeding[1]

J.J. Hellack, J.N. Thompson, Jr, R.C. Woodruff[2] and B.N. Hisey

Department of Zoology, University of Oklahoma, Norman (Oklahoma 73019, USA), 14 September 1977

Summary. Contrary to earlier reports, outcrosses to a number of natural populations of *Drosophila melanogaster* have shown that low levels of male recombination can occur, often associated with chromosome breakage, mutator activity, sterility, and unique chromosome rearrangements. We have been able to induce low levels of male recombination and complex genetic mosaics by feeding an inbred laboratory control strain with homogenates of a strain that shows male recombination and of a laboratory wild type strain. This supports other evidence that the factor inducing male recombination, and presumably the associated chromosome breakage, may be an infective agent.

Genetic recombination in heterozygous males does not usually occur in laboratory stock crosses of *Drosophila melanogaster*[3], though several recent studies have shown that stocks outcrossed to wild populations often show low levels of male recombination[4-7]. This is commonly associated with other genetic events, including mutator activity, sterility, segregation distortion, and has been correlated with chromosome breakage during meiosis[8].

Even though the element(s) causing male recombination is heritable and can be mapped[6], the fact that recombination can be induced in males by injecting homogenates of male recombination strains into flies not normally undergoing male recombination[9], suggests that the causative factor is infective.

In an effort to understand potential methods of transmission, we have considered the possibility that flies dying on the food might be a source of infection, an hypothesis that suggests an interesting ecological association between a eukaryote and a chromosome-breaking mutator factor. To test this hypothesis, we prepared homogenates of 2 male recombination strains (OK1 and OK1012) in pH 7.6 tris buffer and added them to a standard potato-base instant *Drosophila* medium. An homogenate of Canton-S (a wild type strain not normally undergoing male recombination, see the table) was also prepared. Canton-S adults were then placed in the treated cultures and eggs, larvae, and pupae were allowed to develop on the treated medium.

The occurrence of male recombination was tested using standard crosses[7] with the 2nd chromosome marker stock dumpy (dp, 13.0), black (b, 48.0), cinnabar (cn, 57.5), brown (bw, 104.5), and having the 3rd chromosome marker veinlet (ve, 0.2). Treated or untreated adult males were first mated to dp b cn bw; ve females, and heterozygous single male progeny were testcrossed to dp b cn bw; ve. A 2nd generation testcross of heterozygous males to the marker stock provided a large number of bottles of progeny for each of the experimental and control situations. Both recombination and mosaics were scored. The results are summarized in the table, and are similar to unpublished results of B. Slatko (U. Texas, personal communication).

Recombination was induced in both the males fed OK1 and OK1012 homogenates, and the males fed Canton-S homogenates, though there was more recombination in the former. The level of recombination in all of these was significantly higher than in unfed Canton-S controls but was much lower than that found in the established male recombination strains.

In addition, 3 complex mosaic individuals were found. In the first of these, the right side was dumpy with normal veins, while the left side was normal in shape but had the shortened veins characteristic of veinlet. There was no sex comb on the right side, and the right side appeared to be darker, perhaps an expression of black. The eyes were brown, but mottled. The second mosaic individual was normal except for one brown eye and one missing sex comb on the right side. The 3rd mosaic had brown eyes but normal veins. Its wings were long and narrow, but it had the sternopleural bristle arrangements characteristic of the short wing mutant dumpy. The body was not black, but was clearly much darker than other wild type flies. All of these mosaics involve recombination of the markers used in the experiment and were so complex in makeup that it is difficult to propose an explanation other than one related to somatic chromosome breakage or massive interference with mitosis.

Thus, feeding homogenates of male recombination strains (and even of normal laboratory strains) has induced male recombination and complex mosaics in normal laboratory strains not showing male recombination. Further testcrosses confirmed that, in some cases, male recombination persisted for one more generation. This supports the hypothesis that an infective factor, perhaps a virus or mycoplasma, is found in some natural populations of *Drosophila melanogaster* and, in outbred strains, causes genetic aberrations. In inbred lines, such as the laboratory Canton-S wild type, it may be genetically suppressed.

Number of male recombinants and mosaics recovered in feeding and control crosses

Source of homogenate	Replicate	Number of recombinant events*	Number of mosaics	Total flies assayed
OK1	1	2	0	8,321
	2	4	1	16,140
OK1012	1	4 (2)	1	8,735
Canton-S	1	2	1	13,861
Canton-S	2	3 (28)	0	19,025
unfed control: OK1		510	0	25,597
unfed control: Canton-S		1	0	53,952

OK1 and OK1012 are 2 strains showing male recombination, while Canton-S is a control strain which does not show male recombination. (Control data from Woodruff and Thompson, 1977).
*The size of clusters is indicated in the parenthesis following number of recombination events.

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2 Present address: Department of Biological Sciences, Bowling Green State University, Bowling Green (Ohio 43403, USA).

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