Weiss, R.L. Albright College, Reading, Pennsylvania. The inhibition of fertilization in D. melanogaster under the influence of sodium tungstate.

D.S. Aaron (DIS, May 1977) recognized the very rapid marked onset of homosexual behavior in virgin male flies treated with sodium tungstate. The males mounted each other and engaged in pseudo-copulation. This experiment set out to test if the sodium tungstate effect was an aphrodisiac effect or an inducer of homosexual behavior. Glass vials with impregnated (1% sucrose with $1x10^{-4}M$ sodium tungstate) glass wool were used, following Aaron's procedure. Eight virgin females and a single virgin male of D. melanogaster were placed in the vial overnight. A control using only 1% sucrose in the glass wool was also prepared. The following day the male was terminated and the females were placed in individual vials. After 10 days the presence or absence of larvae in each vial was noted. In the control the average number of fertilized females was 3.33 of 8 females. In the sodium tungstate trials there was no fertilization in all seven trials performed. The sodium tungstate clearly shows an effect of inhibiting fertilization between male and female Drosophila.

Welter, R.J. and J.F. McDonald. Iowa State University, Ames, Iowa. A search for third-chromosome ADH active loci.*

McDonald and Ayala (1979) recently reported the presence of putative "regulatory elements" on chromosome III of D. melanogaster that result in the increased activity of alcohol dehydrogenase (ADH; structural locus: II, 50.1). However, at least two alternatives to the "regulatory" model could account for their observations.

One, a noncontiguous, independent locus codes for the synthesis of a unique, but funcionally related, alcohol dehydrogenase. For example, the octanol dehydrogenase (ODH; structural locus: III, 49.2), whose product ordinarily oxidizes only long-chain alcohols (Chambers et al. 1978) has mutated such that it now encodes a product that contributes (though less efficiently) to the oxidation of short-chain alcohols. Or two, the ADH structural locus itself has been transposed to chromosome III, which in combination with Adh^{+}, elevates total enzyme activity (but is not dosage effected).

To test these hypotheses, we felt that a "second-site" might be uncovered if it were in combination with a functionally inoperative, second-chromosome structural locus. Two third chromosomes (3F and 4F), previously isolated from natural populations and known to increase ADH activity levels (3F, McDonald and Ayala 1978; 4F, personal observation), were combined with a second chromosome bearing an ADH-negative allele (Adh^{n2}; for a complete description, see Schwartz and Sofer 1976; this chromosome, also marked with pr, cn, and b, was generously provided by W. Sofer). The test strains were constructed by routine manipulations: n2/n2; Sb/+ X Cy/+; 3F(4F)/3F(4F) and the progeny intercrossed to yield n2/n2; 3F(4F)/3F(4F).

Starch-gel electrophoresis of whole-fly homogenates (5-10 days post eclosion) was performed according to the techniques of Ayala et al. (1972). Total ADH activity was measured according to McDonald and Avise (1976).

No ADH activity was detected in either analysis. We therefore conclude that there are no structural loci coding for a functionally active ADH or ADH-like enzyme on these third chromosomes. These results are consistent with the existence of ADH "regulatory elements" of as yet undetermined function.

References: Ayala, F., J. Pwell, M. Tracey, C. Maurao and S. Perez-Salas 1972, Genetics 70:113-139; Chambers, G.K., J. McDonald, M. McElfresh and F.J. Ayala 1978, Biochem. Genet. 16: 757-767; McDonald and Avise 1976, Biochem. Genet. 14:347-355; McDonald, J. and F. Ayala 1978, Genetics 89:371-388; Schwartz, M. and W. Sofer 1976, Genetics 83:125-136.

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