Hara, T. and H.Kurokawa. University of Tsukuba, Sakura-mura, Japan. Analysis by a partial chromosome substitution for interspecific difference in male 6th sternite bristles between D.auraria and D.biauraria.

Male flies of D.auraria have about 14 bristles on the 6th sternites, while those of D.biauraria have generally none. Backcross experiments were made by using mutant markers of D.auraria chromosomes. It was found that both X- and A-chromosomes were obviously responsible for the bristle manifestation (Hara & Kurokawa 1983).

A chromosome assay was performed by using a wild strain of D.biauraria and mutant strain of D.auraria being homozygous for y(X), cn(A), cu(B). The A-chromosome had a major effect which manifested 8.6 bristles on an average and the X-chromosome had a moderate one being conformable to 4.1 bristles. The effect of the B-chromosome was a little, corresponding to 1.1 at the most. Interactions between the effects of different chromosomes were negligibly small in all combinations.

References: Hara, T. & H.Kurokawa 1983, Jpn.J.Genet. 58:497-504.

Harshman, L.G. University of California, Davis, USNA. The incidence of ovarian dysgenesis in Drosophila simulans.

The P-M syndrome of Drosophila melanogaster is a set of progeny aberrations that can be induced by crossing males (P) that are recently isolated from the field with lab-stock females (M). The reciprocal cross does not show the response (Kidwell 1977), which typically includes male recombination, increased mutation rate, and a failure of the germline to develop (ovarian dysgenesis in females). In Drosophila simulans it has been observed that crosses within and between strains collected in France during the 1970's produced a pattern of ovarian deterioration and transmission ratio distortion which indicated the presence of a system like P-M (Periquet 1981).

This note describes a study designed to characterize the pattern of ovarian dysgenesis in Drosophila simulans by crossing lab stocks with recently isolated lines. The lab stocks used were fa rb, y w , f^2 nt pm st e, and A. The markers fa, pm, rb, st, w, and y were described by Sturtevant (1929), and A, f^2, and e are known to have been in culture for at least fifteen years. The f^2 nt pm st e stock was constructed in the last few years. Little else is known about the history of the strains but presumably they are comparable to old stocks in Drosophila melanogaster. Ten samples of recently isolated lines were collected from July to September 1982, no more than nine months before the experiment. They include isofemale lines from nine California populations, which ranged from San Diego 350 miles north to Patterson, and isofemale lines from Belmont, Massachusetts. A total of 32 isofemale lines, from two to five per population, were employed in the study.

The crosses were made by confining two males with a virgin female in a vial of standard cornmeal medium. The ovarian dysgenesis phenotype of Drosophila melanogaster is temperature-dependent (Engels & Preston 1979), consequently all simulans crosses were initiated and maintained at 28°C. After eclosion adult progeny were transferred to fresh media for two to four days. Thereafter, approximately twenty females were dissected from each cross and scored for the presence of an ovary or ovaries that were so deteriorated that no normal eggs were present.

In the course of the survey female progeny from 190 matings were examined. In crosses between males from recently isolated lines and lab stock females 32 of 1891 offspring (1.69%) had one or two deteriorated ovaries. In the reciprocal cross 41 of 1531 offspring (2.68%) had a dysgenic phenotype. None of the lab stocks showed a strong propensity to produce ovarian deterioration, and none of the populations sampled had particularly reactive isofemale lines. It is possible that these laboratory strains or isofemale lines from the regions of collection are neutral and unreactive. However, it appears that Drosophila simulans does not have a direct analog to the P-M system of Drosophila melanogaster.

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References: Engels & Preston 1979, Genetics 92:161; Kidwell et al. 1977, Genetics 86:813; Periquet 1981, Heredity 46:255; Sturtevant 1929, Carnegie Institute of Washington Publ. No. 399.