Lawlor, T.A. University of California, San Diego, La Jolla, California. Genetic and cytological localization of mei-9.

Alleles of the X-linked meiotic mutant mei-9 have been mapped on the basis of both the somatic effect (monitored by an increase in frequency of multiple wing hair (mwh) clones in mwh/+, Baker et al. 1978) and the meiotic effect (monitored by an increase in frequency of X and 4th chromosome nondisjunction, Baker and Carpenter 1972).

y mei-9^a/w^e ec rb females were crossed to +/Y ; mwh/mwh males; all sons were scored for the visible markers and a subset of sons were tested for the presence of mei-9^a by removing one wing and scoring the number of mwh clones present. Results are in Table 1.

Table 1. Number of mwh clones per wing among sons of y^+ w^e ec rb/y mei-9^a x +/Y ; mwh/mwh.

	Number of progeny scored from:							
	Parentals		Recombinants					
Total mwh clones	y	y^+ w^e ec rb	y^+	y w^e ec rb	y^+ w^e	y ec rb	y^+ w^e ec	y rb
0	1	14				6	3	8
1	2	5				4	3	7
2		4		1		2	8	6
3							4	2
4	1		1		1		2	
≥5	4		18		22		15	7
Total wings	8	23	19	1	23	12	35	30
Total ♂ progeny	1949	1660	19	14	58	38	36	31

Although mwh clones turned out to be difficult to score in phenotypically yellow wings, the results are clear: mei-9^a maps between echinus (1-5.5) and ruby (1-7.5; map positions from Lindsley and Grell 1968). If, as seems reasonable from the other crossover classes, wings with 0-2 clones are considered mei-9^+ whereas wings with 5 or more clones are considered mei-9, then mei-9^a maps 15/29 (from the ec rb^+ recombinants) to 21/28 (from the ec^+ rb recombinants) of the distance from ec to rb. The somatic effect of mei-9^a therefore maps in the 6.5-7.0 interval, in agreement with the earlier rough genetic mapping of the meiotic effect (Carpenter and Sandler 1974).

Deficiency/mei-9 females were tested for the mei-9 phenotype in two separate experiments: (1) wings of deficiency/mei-9^a ; mwh/+ females were scored for mwh clones; (2) deficiency/mei-9 ; spa^{pol} females were tested for meiotic nondisjunction in crosses to B/Y; C(4)RM, ci ey^R males. Deficiencies HC 224, HC 163, and RC 40 were obtained from Dr. George Lefevre; GA 56 was obtained from Dr. Mel Green; HC 163B was one of two lethal segregants from my HC 163 stock (there is no obvious deficiency in the other segregant). The deficiency stocks were examined cytologically to determine breakpoints and crossed to ec rb stocks to confirm deletion extents roughly. All four deficiencies uncover rb (4C6-8) and HC 244 uncovers ec (3F1-2). In somatic effect experiments, HC 244 (3D6-3E1); 4F7-8) uncovers mei-9^a (11.6 mwh clones/wing, 17 wings scored). HC 163B, which has breakpoints of 4B1-2 ; 4F1-2 and is therefore likely to be a contaminant RC 40 (4B1-2; 4F1), also uncovers mei-9^a (10.8 mwh clones/wing, 12 wings scored). HC 244/+, HC 163B/+, and +/mei-9^a control females had background frequencies of 2.5, 1.0, and 3.2 mwh clones/wing, respectively. Based on the genetic and deficiency experiments above, the somatic effect of mei-9^a must be located between 4B1-2 and 4C6-8; that is, between the distal breakpoint of HC 163B and the bands for rb.

Three deficiencies were tested for uncovering the mei-9^a meiotic phenotype. mei-9^a/mei-9^a control frequencies were 27.0% X nondisjunction (ND) and 15.2% 4th ND (3422 total progeny; all frequencies are gametic frequencies). Two deficiencies uncovered the meiotic effect. mei-9^a/HC 244 gave 31.7% X ND and 30.5% 4th ND (631 total progeny); mei-9^a/HC 163B gave 28.5% X ND and 23.4% 4th ND (4038 total progeny). Two independently isolated mei-9 alleles, mei-9^b and mei-9^{AT1} (the latter obtained from Dr. P. Dennis Smith), are also uncovered by HC 163B. However, mei-9^a/GA 56 (4C1-5; 4D1-2 - breakpoints approximate) did not expose mei-9^a, giving 0.4% X ND and 0% 4th ND (2330 total progeny). Background frequencies are ca. 0.1% X and 4th ND, and mei-9 is recessive with respect to nondisjunction (Baker and Carpenter 1972).

The meiotic and somatic effects therefore most likely co-map to salivary region 4B1-2; 4C1-5; this region is defined by the distal breakpoints of HC 163B (which does uncover mei-9 alleles) and the proximal breakpoint of GA 56 (which does not uncover mei-9^a). This salivary

location is in agreement with the genetic map position of 6.5-7.0 determined above.

Acknowledgements: Thanks to Dr. Adelaide T. C. Carpenter for helpful criticism and advice; thanks also to SJB, IE, KP, and KC for stimulating and productive support.

References: Baker et al. 1978, Genetics 90:531; Baker, B.S. and A.T.C. Carpenter 1972, Genetics 71:255; Carpenter, A.T.C. and L. Sandler 1974, Genetics 76:453; Lindsley, D.L. and E.H. Grell 1968, Carnegie Inst. of Wash. Publ. No. 627.

Lee, T.J. Chungang University, Seoul, Korea. Sexual isolation among four species in the Drosophila auraria complex.

The D. auraria complex was divided into four species, D. auraria, D. biauraria, D. triauraria and D. quadraria (Bock and Wheeler 1972). The sexual isolation among three species, D. auraria, D. biauraria, and D. triauraria, was significantly demonstrated (Kurokawa 1960; Lee 1970).

For the experiment of mating preference a usual male multiple choice method was used. Results of the tests are summarized in Table 1. It is noted that the sexual isolation showed a weak degree in all of the crosses except for one case. A higher sexual isolation was seen in the crosses with D. quadraria males than in the reverse cases with D. auraria males. This difference caused by the males may be partly attributed to the morphological difference between their genitalia. It can hardly be concluded from morphological, physiological and distributional studies (Lee 1974a, 1974b) that, of the members belonging to species D. auraria complex, D. quadraria would be the ancestral species.

Table 1. Sexual preference tests among four species.

Crosses ♀♀	♂♂	Homogamic (%)	Heterogamic (%)	Isolation index	Coefficient of joint isolation
A , D X	A	45	51	0.063	0.338
A , D X	D	71	17	0.614	
B , D X	B	69	50	0.160	0.088
B , D X	D	64	62	0.016	
C , D X	C	50	63	0.115	0.162
C , D X	D	78	51	0.209	

(A: D. auraria, B: D. biauraria, C: D. triauraria, D: D. quadraria)

References: Kurokawa, H. 1960, Jap. J. Gen. 35:161-166; Lee, T.J. 1974a, Rev. Tech. & Sci., Chungang Univ. 1:9-16; Lee, T.J. 1974b, Theses Collection, Chungang Univ. 19:63-73.

Leigh Brown, A. J. National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina. Molecular weights of seven Drosophila enzymes.

Recent work in our laboratory (Leigh-Brown and Langley 1979; Leigh-Brown and Voelker 1979) has involved the estimation of the native molecular weight of several Drosophila enzymes for which such data were not previously available (O'Brien and MacIntyre 1978). As our earlier report gave only the results, I present here the methods used and the data on which those estimates were made.

Determination of sedimentation constants ($s_{20,w}$) by sucrose density gradient sedimentation was carried out according to the procedure of Martin and Ames (1961). Gradients were made in 5 ml cellulose nitrate tubes by layering 1.15 ml of each of 20%, 15%, 10% and 5% solutions of sucrose in 0.05M Tris-HCL pH 7.5 with 1 mM dithiothreitol (Sigma). They were then stored at 4°C for 24 hours. Crude fly homogenate was prepared in the same buffer by homogenising 0.5 g cn bw; ri e flies, centrifuging in the homogenate for 20 minutes at 15,000 rpm, and filtering through glass wool. The extract was then passed through a 40%/80% ammonium sulphate precipitation step and was diluted until the protein concentration, measured by $O.D._{260}/O.D._{280}$, was less than 20 mg/ml. Rabbit muscle Ldh was added (800 units/ml) and 0.1 ml was layered on top of each gradient. Three such gradients were centrifuged for 15.5 hr at 39,000 rpm in a Beckman SW 51 rotor at 4°C. After the run, two-drop fractions were collected on ice and assayed. Rabbit muscle lactate dehydrogenase and D. melanogaster alcohol dehydrogenase were used as standards.