group. This may indicate that a modifier gene increased the penetrance of the tumorous head trait as well as influencing or being responsible for the lethal effect. Because only a certain proportion of the lethal larvae developed melanotic tumors, it seems probable that the tumor formation is a secondary characteristic resulting from a ring gland defect.

Sokoloff, A. University of California at Berkeley. A possible maternal effect on quantitative characters in D. pseudoobscura.

In a previous report (DIS 33:162-165) comparison was made between measurements of length and width of wing and length of tibia derived from flies whose progenitors had been reared deliberately under different conditions. The statistical tool was the t-test, taking one character at a time in flies reared, say at 24°C, but whose mothers had been reared at 16° and 24°C and vice versa. It was reported that this test failed to show any maternal effect on the three characters mentioned. Recently, in analyzing some data demonstrating geographic variation in D. pseudoobscura, it became evident that differences between populations derived from different localities (some as close as six miles) cannot be demonstrated if one takes one character at a time but, if one takes two characters, significant differences are easily found when Fisher's Discriminant Function method is applied. In the light of this finding the problem of maternal influence on quantitative characters has been re-examined, applying this, more powerful, statistical method. The technical details can be found in the above reference. A summary of the history of the flies measured is given at the bottom of Table 1, and the results of the statistical analysis can be seen in Table 2. Although not all the possibilities were tested, the conclusions from this analysis are as follows:

(1) Flies reared in bottles differ significantly from those reared in vials at both temperatures (compare A-B and F-G variance ratios), even though attempts were made to avoid crowding.

(2) As expected, a period of starvation in the larval stages results in flies which are smaller than flies well fed for the whole larval stage (comparisons E-F). In turn, their progeny differ, even though they were reared under identical, non-crowded conditions (comparisons C-D).

(3) The temperature prevalent during the development of the progenitors appears to influence the body characters of the next generation when the latter flies are reared at the higher temperature (compare F-G), the bigger mothers producing generally bigger offspring but the reverse situation (B-D) appears to have no effect.

These experiments omitted some combinations, owing to time limitations. However, the results suggest that, in studies involving quantitative characters, the history of the preceding generation of D. pseudoobscura, particularly if the flies are reared at 24°C, must be known and specifically stated.

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Table 1. Means, variances and covariances of wing length (x) and tibia length (y) from D. pseudoobscura derived from Grand Canyon, Arizona. Upper block: flies reared at 16°C.; lower block: flies reared at 24°C. (for each sample N=50).

Sex		$\bar{m}_x$	s^2x	$\bar{m}y$	s^2y	cov_{xy}
	A	65.924	0.7998	23.612	0.2096	1,588.60
Females	B	63.692	1.181	23.100	0.2502	1,501.60
	C	63.308	1.080	22.896	0.1604	1,479.34
	D	63.372	1.022	23.052	0.1351	1,490.85
	A	59.184	0.9108	22.312	0.2010	1,347.54
Males	B	57.324	4.2365	21.808	0.7608	1,277.19
	C	57.208	0.7935	21.872	0.1824	1,276.99
	D	57.680	0.5878	21.988	0.1067	1,294.23

	E	56.984	0.9789	21.660	0.2322	1,259.71
Females	F	57.352	0.9012	21.484	0.1569	1,257.44
	G	57.832	1.398	21.788	0.1280	1,285.94
	H	58.036	0.8633	22.164	0.1629	1,312.74
	E	51.640	1.322	20.560	0.1845	1,083.61
Males	F	51.972	1.089	20.244	0.1849	1,073.74
	G	52.260	0.7465	20.672	0.1253	1,102.48
	H	52.400	1.037	20.764	0.2118	1,110.53

A = F_1 of single females reared in bottles at 16°C.
B = F_2 from A reared in vials at 16°C.
C = F_2 from E reared in vials at 16°C.
D = F_2 from F reared in vials at 16°C.
E = mass F_1 reared at 24°C.
F = F_1 of single females reared in bottles at 24°C.
G = F_2 from F reared in vials at 24°C.
H = F_2 from A reared in vials at 24°C.

Table 2. Results of the analysis of the data.

		A	B	C	D
			66.88	138.36	131.08
Females at 16°C	A	---	51.17	74.39	72.41
				0.126	.0235
	B	62.72	---	2.243	.969
					.107
	C	90.23	2.72	---	2.069
	D	87.80	1.17	2.51	---

		A	B	C	D
			4.96	56.40	26.67
	A	---	14.08	47.50	32.66
Males				.0136	.0146
at 16°C	B	17.07	---	.7378	.7652
					.2765
	C	57.59	.895	---	3.3257
	D	39.62	.928	4.03	---

		E	F	G	H
			.825	.982	7.281
	E	---	5.746	6.267	17.066
				1.159	21.80
Females at 24°C	F	6.97	---	6.809	29.53
					2.831
	G	7.60	8.25	---	10.642
	H	20.69	35.81	12.91	---

		E	F	G	H
			2.34	.361	.652
	E	---	9.66	3.80	5.106
				3.71	5.01
Males at 24°C	F	11.72	---	12.18	14.16
					.0069
	G	4.61	14.77	---	.5253
	H	6.19	17.16	.6369	---

Note: Upper and lower entries in right triangle cells are sums of squares for $\frac{n^2}{2}D$ between and for D within, respectively $X10^3$. The cells in the left triangle show the variance ratios. (For $n_1 = 50$ and $n_2 = 50$ a variance ratio of 1.94 is significant at the 0.01 level, and a ratio of 1.60 is significant at the 0.05 level).

Glassman, E. and E. C. Keller, Jr. University of North Carolina. The maternal effect of ma-1^+: The effect of hypoxanthine; the effect of lxd.

The maroon-like (ma-1) eye color mutant lacks detectable amounts of xanthine dehydrogenase, pyridoxal oxidase (pyridoxal- pyridoxic acid), and the ma-1^+ complementation factor. When ma-1 flies are derived from female parents that carry an ma-1^+ gene, their eye colors are wild-type and xanthine dehydrogenase activity can be detected, especially in early development (Glassman and Mitchell, Genetics 44:547, 1959; Glassman and McLean, Proc. Nat. Acad. Sci. 48: 1712, 1962).

The maternally-affected eye color is observed only in adults that emerge in the early days of hatching. Those that emerge in older bottles have the usual mutant ma-1 eye color. This loss of the maternal effect is not due to the age of the mothers, and must be ascribed to changes in the food (Glassman and Mitchell, loc. cit.). Either a substance is being used up or an inhibitor is accumulating. We believe that the latter explanation is true, and that the purines which accumulate in the food are inhibiting the low amounts of xanthine dehydrogenase in the maternally-affected ma-1 flies so that little pterdine eye pigment is synthesized.

To test this we crossed y f:=; st females to m ma-1; st males and allowed eggs to be laid on three different media: a) the usual Drosophila medium (devised by E. Lewis), b) the usual medium containing 0.1% hypoxanthine, and c) the usual medium containing 0.1% uric acid.

In these experiments the maternal effect did not diminish on the control medium until 10 and 12 days (replicate experiments) had elapsed. On the other media, the maternal effect diminished by 2 and 4 days (replicate hypoxanthine experiments) and by 7 and 9 days (replicate uric acid experiments). Thus, hypoxanthine (and perhaps uric acid) accelerates the disappearance of the maternal effect. Whether these compounds accumulate to a sufficient extent in the food to account for the loss regularly observed, and whether the mechanism is through inhibition of the small amounts of xanthine dehydrogenase remains to be shown.

The mutant, lxd, lacks the ma-1^+ complementation activity as well as the pyridoxal oxidase activity. Since these are also lacking in ma-1, we tested the effect of lxd on the maternal effect as follows:

A. The cross y f:=; ru lxd by females x v f Bx^3 ma-1 males produced v f Bx^3 ma-1; ru lxd by/+ males all of whom (94 in the first three days) were maternally affected. Thus, homozygous lxd in the mother has no influence on the maternal effect in the progeny.

B. The cross, v f Bx^3 ma-1; ru lxd by/TSS x ru lxd by/D, produces three types of males which were classified and counted during the first three days of emergence. These were:

genotype	phenotype (number maternally affected)
v f Bx^3 ma-1; ru lxd by	Only 1 out of 41
v f Bx^3 ma-1; ru lxd by/TSS (or D)	56 out of 56
v f Bx^3 ma-1; TSS/D	22 out of 22

Thus, homozygous lxd in the progeny completely abolishes the maternal effect.

We interpret these results as follows: The maternal substance responsible for the maternal effect is the product only of the ma-1^+ locus. The lxd locus is not involved and thus lxd mothers can have progeny that are maternally affected. This is also true for the ry locus (Glassman and Mitchell, loc. cit.). However, for the maternal effect to be expressed in the progeny, enough product of the lxd^+ locus must be available for production of sufficient xanthine dehydrogenase for eye color synthesis to proceed normally. If lxd is in the progeny, then the maternal effect is abolished.

This interpretation is in accord with the idea that the lxd^+, ry^+, and ma-1^+ loci code for polypeptides (L, R, and M, respectively) that polymerize with each other. In this scheme, the maternal substance is, or is composed of, only the M subunit; the ma-1^+ complementation factor and the pyridoxal oxidase (these may be identical) are composed of M and L subunits;