Shannon, M.P. University of Texas, Austin Texas. Characterization and developmental analysis of the female-sterile mutant almondex of Drosophila melanogaster.

A combined genetic and embryological investigation (Shannon, 1972a, 1972b) indicates that the mutant almondex (amx) (1-27.7±) belongs to a small class of sex-linked recessive D. melanogaster mutants characterized by a peculiar type of female-sterility. Mutant females of this type produce eggs bearing a cytoplasmic defect that causes the death, usually during embryogenesis, of all offspring that do not inherit a wild-type allele from the father. Generally, in matings of mutant females to mutant males, all progeny die; in matings of mutant females to non-mutant males, all regular (XY) male progeny die, but all or most female (heterozygous) progeny and occasional non-disjunctional non-mutant (XO) male progeny survive. An hypothesis to explain these results (Lynch, 1919) is that the wild-type allele can function in the zygote to repair the cytoplasmic defect, thus making possible survival of females and exceptional males. Heretofore, only three mutants exhibiting this partial sterility pattern - deep orange, fused, and rudimentary - have been carefully studied (Carlson, 1971; Counce, 1956a,b,c; Fausto-Sterling, 1971a,b; Hildreth and Lucchesi, 1967; Lucchesi, 1968; Lynch, 1919; Merrell, 1947; Nørby, 1970).

Fig. 1. Heterozygous (amx/+) female offspring from the mating amx ♀ x + ♂ showing non-lethal maternal effect. Note thoracic defects and absence of left hindleg.

The cytoplasmic defect in the eggs produced by amx females is so severe that most offspring die regardless of the presence of a wild-type allele in the genotype. Matings of amx females to amx males are lethal to all embryos, as would be expected from Lynch's hypothesis. Matings of amx females to non-amx males are lethal to all ordinary (amx/Y) male embryos and to most female (amx/+) embryos as well. Offspring that escape death during embryogenesis often die after puparium formation, and most of those that survive to the adult stage have thoracic defects, including crippled or missing legs, and defects of the abdominal sternites (Fig. 1).

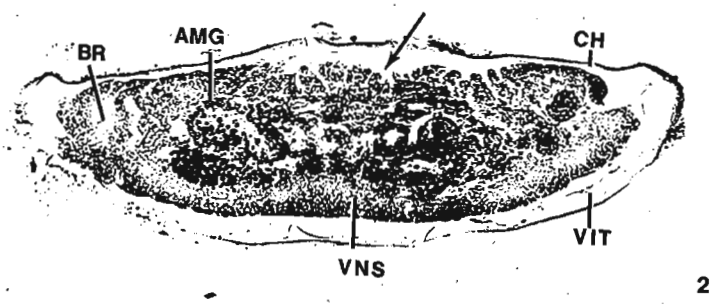

Fig. 2. Sagittal section of an 18-hour-old embryo from the mating amx ♀ x amx ♂, showing characteristic pattern of damage. Anterior end at left. Arrow indicates abnormal segmentation. AMG - anterior midgut; BR - brain; CH - chorion; VIT - vitelline membrane; VNS - ventral nervous system.

Almondex females have apparently normal genitalia, and egg yield is good. Fertility is enhanced by a temperature of 27°C (the amx female is essentially sterile at lower temperatures), by moderate crowding of the culture bottles, and apparently by increasing age of the mutant female.

Embryos produced by matings of amx females to amx males undergo advanced but abnormal development. Morphological abnormalities are first noticeable shortly after the time of maximal germ band extension. The principal anomalies involve derivatives of the ectoderm and somatic mesoderm. The main features of the pattern of damage (Fig. 2) include incomplete differentiation of the hypoderm, abnormal segmentation, exposure and often hypertrophy of the nervous system (which does not condense), tracheal defects, breakdown of the foregut, and lack of somatic musculature. Endodermal abnormalities are relatively minor. Both dorsal closure and head involution are impaired.

Matings of amx females to non-amx males produce a similar pattern of damage in male embryos, but in female embryos there is an amelioration of development.

I suggest that either an inherent structural defect in the presumptive ectoderm or some

metabolic defect to which the ectoderm is particularly susceptible may underlie the morphological defects found in the progeny from amx females.

References: Carlson, P.S. 1971 Genet. Res. Camb. 17:53-81; Counce, S.J. 1956a, Z. indukt. Abstamm. u. Vererb. 87:443-461; _____ 1956b, Z. indukt. Abstamm. u. Vererb. 87:462-481; _____ 1956c, Z. indukt. Abstamm. u. Vererb. 87:482-492; Fausto-Sterling, A. 1971a Develop. Biol. 26:452-463; _____ 1971b, J. Exp. Zool. 178:343-350; Hildreth, P.E. and J.C. Lucchesi, 1967 Develop. Biol. 15:536-552; Lucchesi, J.C. 1968 Genetics 59:37-44; Lynch, C. J. 1919 Genetics 4:501-533; Merrell, D.J. 1947 Am. Naturalist 81:399-400; Nørby, S. 1970 Hereditas 66:205-214; Shannon, M.P. 1972a (in press) Genetica; _____ 1972 (in preparation).

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Induction of mutations in D. melanogaster with O-Sulphobenzoic imide (saccharin).

Artificial sweeteners are of two types, namely cyclamates and saccharins. The cyclamates used in food stuffs and drinks belong to sodium and calcium salts of cyclamic acid. The cyclamates have been banned in various countries because of their genetic effects. The work on genetic effects of saccharin are scanty. Sram and Weidenhofferova (1969) have carried out experiments on D. melanogaster and found the chemical sodium saccharin to be not mutagenic while Sax and Sax (1968) have found sodium saccharin to cause chromosome breakage in onion root tip cells. The present work is undertaken with a view to find out whether or not saccharin (Madhurin marketed by M/s. Merck Sarabhai) is mutagenic in Drosophila melanogaster. 8.33% of solution of Madhurin (in 0.4% NaCl) was used so as to give a survival above 85% and 12.5% solution of Madhurin was used as to give a survival of 70%. 0.2 micro c.c. of the above solution was injected in the vicinity of the last two abdominal segments with the aid of a Agla micro-meter syringe. The flies were cultured on the usual standard Drosophila corn meal medium.

Sex linked recessive lethals and translocations were screened to study for any induced genetic damage. Six broods of three days interval were used.

Treated males were crossed individually with 3 virgin females of Y sc^{S1} In-49 sc^{8};bw;st stock. The F_1 females were mated individually with Y sc^{S1} In-49 sc^{8} males while the males were mated with bw;st females to score for sex linked recessive lethals and translocations respectively in F_2 generation. The results are presented in Table 1:

Brood	Sex linked recessive lethals									Translocations								
	Control			8.33% madhurin			12.5% madhurin			Control			8.33% madhurin			12.5% madhurin		
	T	l	%	T	l	%	T	l	%	T	t	%	T	t	%	T	t	%
A Brood	1366	3	0.219	227	-	-	269	-	-	1516	-	-	334	-	-	265	-	-
B Brood	1716	8	0.466	208	1	0.48	256	-	-	1496	-	-	217	-	-	249	-	-
C Brood	1894	3	0.26	209	-	-	233	-	-	1668	-	-	235	-	-	213	-	-
D Brood	1599	7	0.43	234	1	0.42	253	1	0.23	1539	-	-	212	-	-	235	-	-
E Brood	1015	0	-	215	-	-	251	-	-	1321	-	-	240	-	-	236	-	-
F Brood	1073	3	0.27	218	-	-	167	-	-	1367	-	-	154	-	-	256	-	-

T = Total number of X chromosomes or F_1 sons scored
l = lethals recorded
t = translocations recorded

The chi-square test has been done to compare the following groups: (1) control versus 12.5% madhurin; (2) control versus 8.33% madhurin. The results of statistical analysis are presented in Table 2:

Chi-square values for the differences in sex linked recessive lethals for the groups compared.

Group	Brood A	Brood B	Brood C	Brood D	Brood E	Brood F
Control vs 8.33%	-	0.0032	-	0.000064	-	-
Control vs 12.5%	-	-	-	0.1233	-	-