Pipkin, S.B. and N.E. Hewitt. Howard University, Washington, D.C. The influence of the X chromosome on specific activity of alcohol dehydrogenase of Drosophila.

The electrophoretic mobilities of Drosophila alcohol dehydrogenase (ADH) fast and slow isozyme variants, respectively, are correlated with the specific activities (SA) of these variants, measured spectrophotometrically. The assay mixture consisted of 0.05ml 2-butanol, 0.05ml NAD^+ (10 mg/ml), 0.2ml enzyme homogenate taken from 10 flies 4-6 days old, ground in 1ml 0.004 M K_2HPO_4 buffer and filtered; 0.8ml of the same buffer. SA is expressed as micromoles of NAD^+ reduced/1.1 ml/min/mg live weight.

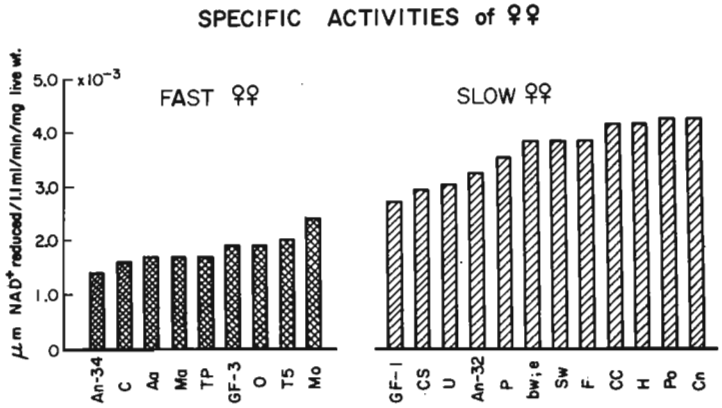

Fig. 1

Eight fast strains of diverse geographic origin have significantly lower SA than 12 slow ADH strains (Fig. 1). The range of SA of the fast strains is less than that of the slow strains. Fast ADH strains studied include those from Oahu, Hawaii; Canton, Ohio; Australia; Malvern, Arkansas; Austin, Texas; and Montgomery County, Great Falls, and Takoma Park, Maryland. Slow strains include those from Hikone, Japan; Ponape, Caroline Islands; Sweden (Swedish-b); Lima Peru; Urbana, Ill.; Florida; Cool Springs and Great Falls, Maryland; Austin and Corpus Christi, Texas; and the laboratory strains cinnabar and brown ebony. In both fast and slow strains males show higher SA than females, but the difference between the sexes is greater in the slow strains. Two experiments show that the X chromosome has a regulatory influence on the SA of ADH, the structural locus of which is located in the second chromosome (Grell et al., 1965). In the progeny of T5 fast triploid females with slow ADH males of four different strains, the 2X3A intersex progeny displayed SA similar to that of their respective 1X2A diploid male siblings and higher than that of 3X3A and 2X2A female siblings (Table 1). Triploid female progeny with 2X's derived from the T5 strain and one from the paternal strain showed SA not differing significantly in 3 of the 4 crosses, but the SA of the corresponding diploid (2X2A) female siblings varied significantly and was in each case higher than that of the 3X3A triploid siblings. The cause of this variability is in part due to the fact that diploid females included both those inheriting their two X's from the T5 triploid female parent and those inheriting one maternal X and one paternal X chromosome. In a second experiment the SA of hybrids between slow and fast strains was found to be intermediate between the SA of parental strains but sometimes differed from that expected on the hypothe-

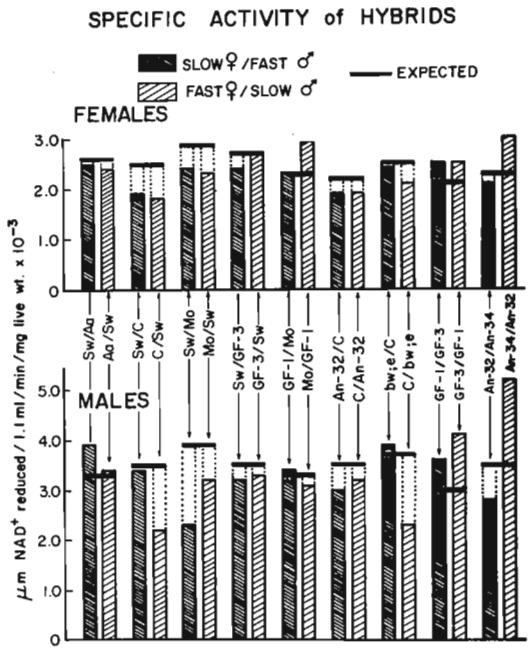

Fig. 2

Table 1

T5 FAST TRIPLOID ♀ PARENT

♂ PARENT ↓	PROGENY 3X3A ♀	2X3A intersex	2X2A ♀	1X2A ♂
T5 fast	1.47 ± .06	3.12 ± .20	1.91 ± .10	2.72 ± .13
ADH_{n_1}	0.9 ± .03	1.8 ± .21	1.1 ± .10	1.9
CC slow	2.46 ± .04	3.69 ± .23	2.96 ± .12	4.05 ± .19
H slow	2.03 ± .20	4.42 ± .45	4.36 ± .16	5.09 ± .35
Sw slow	2.12 ± .10	3.01 ± .37	2.47 ± .11	4.07 ± .49
Cn slow	2.11 ± .16	4.05 ± .06	2.91 ± .21	4.09 ± .24

μm NAD^+ reduced/1.1ml/min/mg live wt x 10^{-3}

sis of simple additive contribution to SA by the parental strains. The X chromosomes of different strains have a special effect on ADH activity of slow/fast hybrids. This is shown by the greater variability in SA of hybrid males (each carrying a single X of maternal origin) than of hybrid female progeny of reciprocal crosses of slow and fast strains (Fig. 2). Further, female hybrids of reciprocal crosses show SA differing less than those of male hybrid siblings from the SA values expected on the hypothesis of simple additive contribution to ADH activity of hybrids by the parental strains. In a cross of a mutant strain with no ADH activity (Grell's ADH_{nl}) with T5 triploid females, all progeny (3X3A, 2X2A, 2X3A and 1X2A) had about the same SA as expected on the hypothesis that each active ADH^F allele derived from the T5 triploid parent is coding for protein at the same rate as in corresponding control T5 triploid, intersex, and diploid forms, respectively (Table 1). In the progeny of ADH_{nl} males with T5 triploid females, the 2X3A intersexes and 1X2A diploid males have similar activities, about twice that found in the 3X3A and 2X2A females. This suggests that the ratio of X to autosomes is not only responsible for determining the sex type but also for determining a male or female like character of ADH activity. Supported by NSF Grant GB 8770.

Reference: Grell, E.H., K.B. Jacobson and J.B. Murphy, 1965 Sci. 149: 80-82.

Parkash, O. Institut für allgemeine Biologie, University of Vienna, Austria.

The behavioural changes produced by thymidine-induced temperature-sensitive lethal factors in D. melanogaster.

In some of the earlier publications, the author reported on the induction of absolute and incomplete (temperature-sensitive) lethals by feeding D. melanogaster on thymidine-containing culture medium. Further, the special significance of the temperature-sensitive lethals in general biological research was pointed out. The existence of the modifier genes, for example, was inferred from the analysis of one of these temperature-sensitive lethals (1969). This particular lethal manifested its lethal effect at 16°C, whereas at 26°C it behaved as a non-lethal. The experiment has been continued and some more such factors have been analysed in the mean time. Because of their general interest, it is thought worthwile to report on two of such factors in the present note.

One of these lethal factors $l^{ts}(1)$ 42.3±0.5 showed its effect in the direction opposite to that of the one reported earlier. This factor manifests its lethal effect at 26°C, whereas at 16°C it behaves as a non-lethal. This, incidently, is interesting as it indicates that thymidine is capable of inducing temperature-sensitive lethal mutations, some of which manifest their effect at lower while the others do so at higher temperatures.

The second one of the two factors, so far as the lethal effect is concerned, resembles the one reported earlier. This has been maintained in a balanced stock against y sc^{S1} In49 sc^{8} at 16°C. An attempt was made to obtain a pure stock of this l^{ts}. The cultures were kept at 26°C to obtain the normal-looking males carrying the lethal factor. The repeated attempts failed, as it turned out that the l^{ts} males were very sensitive to the ether anesthesia and the slightest amount was detrimental to them. The CO_2 anesthesia was less injurious; however it left these males so weak that they died an hour or so later. In a last attempt, the l^{ts} males were separated by employing a suction tube and no anesthesia at all. This method of separation succeeded but it appeared as if the males were infertile. Their mating behaviour was watched by the author and it was found that these males did not respond to the females (aversion to mating?). This abnormal behaviour was very conspicuous and this led the author to search for other physiological reasons. Consequently the males were carefully scrutinised but no macroscopic anomaly was detected. A dissection was then carried out and the testes examined under a microscope. This examination revealed a complete absence of sperm, the testes themselves being normal in size and appearance. The abnormal mating behaviour can, therefore, be traced back to the absence of the sperm. Here we have a case of genetically determined "aspermia" and the consequent abnormal mating behaviour and this may have its parallels in other animal domains. The attempts to localise this factor have, so far, been unsuccessful. Most probably, this is a compound lethal involving more than one locus.

This note points in favour of the study of the temperature-sensitive lethals. The work is being continued and will be reported elsewhere. Thanks are due to Prof. Mainx and Doz. Sperlich of the University of Vienna for their generous help.