

Arking, Robert and Ralph Hillman. Temple University, Philadelphia, Pennsylvania. Analysis of the mode of action of the eyeless-Dominant allele.

Of the eight known loci on the fourth chromosome which produce an eyeless phenotype, only the ey^D locus is neither allelic nor hypomorphic when combined with any of the others (Hinton, Amer. Nat. 76:219, 1942). Its major phenotypic

effect is the gross reduction in the size of the eye (Patterson and Muller, Genetics 15:495, 1930) with secondary malformations of the antennae and head capsule.

Reciprocal pair matings have been performed utilizing the balanced lethal, sv^{de}/ey^D , and an inbred Oregon-R strain which has been maintained in this laboratory for thirty-five generations by brother - sister matings. The results of these crosses (Table 1) show the existence of three major classes of F_1 progeny: I. Those flies which possess a normal eye; II. Those flies which exhibit a severe decrease in the number of facets/eye; III. Those flies that never eclose and upon dissection can be seen to be either completely headless or else possess a very malformed and assymetrical head capsule.

Table 1. The number of facets/eye in the different classes of F_1 progeny

Class	$sv^{de}/ey^D \times +/+$			$+/+ \times sv^{de}/ey^D$		
	Range	Median	Mean \pm S.E.	Range	Median	Mean \pm S.E.
I	692 - 806	764	755 \pm 12.4	722 - 884	801.5	806.7 \pm 14.3
	668 - 779	721	718 \pm 8.9	736 - 791	749.5	756.8 \pm 5.3
II	0 - 364	0.0	35.6 \pm 20.5	0 - 232	0.0	22.8 \pm 6.1
	0 - 301	0.0	46.5 \pm 9.5	0 - 81	0.0	23.9 \pm 30.3
III	Lethal - Fails to Eclose			Lethal - Fails to Eclose		

In class II there is a highly non-parametric distribution of facets/eye. The Wilcoxon-White test shows that there is no significant difference in the frequency distribution of facets/eye between (a) the reciprocal crosses, or (b) males and females of the same mating. Although the scored flies had either two normal eyes or two abnormal eyes, those flies that were assymetrically abnormal exhibited no preference as to whether the most severely damaged eye would be on the right or on the left side of the head capsule. Similar genetic and developmental results are found when sv^{de}/ey^D flies are crossed to Canton-SA wild types and when $+/ey^D$ flies are crossed to flies from either Oregon-R or Canton-SA inbred stocks.

Most of the work thus far has involved the third, or lethal, class. Histological examination of the abnormally developing pupae has revealed the following syndrome: 1. normal differentiation and development of the imaginal legs, wings, hypoderm, etc. 2. the retention of the larval salivary glands, some in an apparently healthy condition, for periods of time ranging up to 156 hours post-pupation; 3. a partial to complete development of the imaginal salivary glands which occurred even in those organisms retaining the larval salivary glands; 4. a failure of the gonads to develop (cessation of development usually occurred at approximately the 0 - 4 hour pupal stage and by the 27 hour pupal stage at the latest.) 5. an abnormal eversion of the head capsule and its associated structures.

Similar histological evidence for the lethality of ey^D was obtained from the $F_1 \times F_1$ cross of $ey^D/+ \times ey^D/+$. Developmental data from this cross can be seen in Table 2. Although the histological picture in non-eclosed pupae is similar to that described above, the phenocritical phase actually begins in the late third larval instar. The non-pupating larvae do not undergo further larval molts, but remain in the third larval instar for periods of time ranging up to 120 hours. At this time they either die or undergo an abortive pupation. The effective lethal phase in the ey^D mutation therefore occurs in the beginning and early stages of metamorphosis.

Table 2. Developmental data of $ey^D/+ \times ey^D/+$ cross.

Larvae Hatched	Non-pupating Larvae	Pupae	Non-eclosed Pupae	Total Adults	$+/+$	$ey^D/+$	Lethal
89	15	74	8	66	25	41	23

continued

The above syndrome suggests that the ecdyson/juvenile hormone balance in the ey^D flies has been upset. Whether the primary lesion affects the ecdysial glands, the corpora allata, the corpora cardiaca or other neuro-secretory elements is not known at this time. Neuro-secretory staining and implantation experiments are presently being performed in order to further study the mode of action of the ey^D locus. (The research reported above was supported by a NSF Predoctoral Cooperative Fellowship and in part by grant GM 10480 from the USPHS.)

Gethmann, Richard C. Oregon State University, Corvallis, Oregon. A reduced viability effect of a ring duplication, $Dp(1;f)65X^{c2}$.

In crossover tests of a reversed acrocentric, a reduced viability of one of the duplications used was observed. The reversed acrocentric used in the experiments was deficient for a large block of interstitial heterochromatin, and is

lethal in the absence of the heterochromatin. The missing heterochromatin was supplied by different duplications.

Since many of the exchanges within a reversed acrocentric result in lethal bridges, a reduction in the number of recovered female progeny would be expected. However, in parallel experiments, quite different sex ratios were recovered. An examination of the genotypes of the progeny from these crosses show that there is a marked reduction in the number of recovered males when the male zygotes receive $Dp(1;f)65X^{c2}$ (of crosses 1, 2, 3, and 4, also see Report of R. C. Gethmann, this DIS, for description of $Dp65X^{c2}$).

If the reversed acrocentric is heterozygous for $In(1)d149$, one would expect a reduction in exchanges, and hence, a higher sex ratio. This was the case, however, again the apparent lethal effect of the ring duplication was found (of crosses 5, 6, 7, and 8). Since lethal exchange classes are absent in a reversed metacentric, one would expect a 1:1 sex ratio from crossover tests with this type of a compound X chromosome. However, as can be seen from cross 9, there was a reduction in the number of recovered males. Again, these male zygotes received $Dp65X^{c2}$.

Finally, the duplication can be induced to segregate randomly if a Y chromosome is present as a pairing partner for the reversed acrocentric. An examination of the regular and exceptional progeny from this cross (cross 10), shows that in the regular progeny, there is a reduction of males (cross 10a), which is comparable to that found in crosses 1 or 2. However, in the exceptional progeny (cross 10b), the duplication is included in the female zygotes, rather than the males, and here, the female class is the one which is greatly reduced.

In conclusion, $Dp(1;f)65X^{c2}$ is lethal in a fraction of the zygotes, it appears that approximately 30% of the zygotes receiving the duplication do not survive to adulthood. (This work was supported by NSF grant GB-1864 to J. D. Mohler.)

Cross number	genotype of female progeny	number	genotype of male progeny	number	female to male ratio
1	RA/Dp60	471	$\overline{XY}/Dp65X^{c2}$	638	0.74
2	RA/Y, $su^+ - f$	549	X/Dp65X ^{c2}	765	0.72
3	RA/Y, $su^+ - f$	2862	X/Dp60	5921	0.49
4	RA/Y, $su^+ - f$	2870	X/Y, $su^+ - f$	5274	0.54
5	RA, d149/Dp60	831	$\overline{XY}/Dp65X^{c2}$	790	1.05
6	RA, d149/Y, $su^+ - f$	1704	X/Dp65X ^{c2}	1733	1.00
7	RA, d149/Y, $su^+ - f$	977	X/Dp60	1246	0.78
8	RA, d149/Y, $su^+ - f$	1680	X/Y, $su^+ - f$	2058	0.82
9	RM/Dp60	439	$\overline{XY}/Dp65X^{c2}$	333	1.32
10a	RA/Dp60	341	$\overline{XY}/Dp65X^{c2}$	469	0.73
10b	RA/Dp60/Dp65X ^{c2}	317	\overline{XY}	719	0.44