

Basden, E.B. Institute of Animal Genetics, Edinburgh, Scotland. Some early Morgan specimens.

In the Royal Scottish Museum, Edinburgh, are 50 specimens, entered under No. 55, 1 to 12, for July 22, 1922 as follows:

"Series of specimens of *Drosophila melanogaster* Mg., of the original varieties bred (bred from original sets used erased) by Professor W.H. Morgan in his experiments on heredity. Presented by J.H. Ashworth, Zoological Department, University of Edinburgh. Spineless bithorax 16 specimens (9♂7♀); Plexus brown speck 6(3♂3♀); Lm.5. (15 on label) Haplo IV. 4(2♂2♀); Spineless glass. III. 2(2♀); Ebony 4. III. 3(1♂2♀); Dichaete hairless. 3(3♀); Black vestigial. 2(1♂1♀); Eosin miniature I 1(♂); Forked Bar I 4(1♂3♀); Yellow White I 3(1♂2♀); Vermilion I. 3(3♀); L.7.5/20 3(1♂2♀)".

The specimens are on micro-pins on papered cork mounts and are labelled in the same hand that made the above entry in the Museum's accession book. Empty pin-holes on all mounts suggest that more specimens were originally present. The number of specimens still there tallies with the numbers recorded above, to which I have added the sexes in brackets.

They agree with the usual descriptions of the respective mutants except:

plexus brown speck - speck is not apparant.

lm haplo-4 - no obvious abnormalities, no trident.

spineless glass - postscutellars not erect.

Dichaete Hairless - heads not deformed, veins not shortened.

vermilion I - eyes wild-type in colour.

Of L.7.5/20, which I cannot properly locate, 1♂1♀ are very pale, including eyes, veins and bristles, but 1♀ is darker.

It is surprising that so few "type" specimens of mutants are preserved for posterity. If others are known it is hoped they will be recorded.

Thanks are due to E.C. Pelham-Clinton for access to the specimens.

Mason, J.M. University of Washington, Seattle, Washington. A relationship between daughterless and the Y-chromosome.

daughterless (da) is a recessive on chromosome 2 in *D. melanogaster* which, when homozygous in female parents, results in the production of 100% male progeny regardless of the genotype of the male parent. The abnormal sex ratio is due

to differential zygote mortality (Bell, 1954). Sandler (1972) has shown that heterozygous da females produce homozygous da progeny whose viability depends upon the amount of sex chromosome heterochromatin either in the mother or in her offspring. In order to ask whether this relationship also applies to the viability of sons of homozygous da mothers, the following experiment was performed. Females with normal X-chromosomes and either da/da or +/+ were mated to males that were either X/Y; +/+ or  $\overline{XY}/0$ ; +/+. The results were:

mating	number of eggs	progeny		male survival
		female	male	
X/X;da/da x X/Y	2613	0	954	0.90
X/X;+/+ x X/Y	1034	465	415	
X/X;da/da x $\overline{XY}/0$	2299	0	685	0.66
X/X;+/+ x $\overline{XY}/0$	2029	898	922	

In the mating to X/Y males, the male survival is 0.90 where survival is defined as the ratio of adult male progeny to eggs in the da cross divided by that ratio in the control cross. In the mating to  $\overline{XY}/0$ , the male survival is 0.66. These data show that X/0 males produced by da/da mothers do indeed survive less well than X/Y males produced by such mothers.

Two additional points are worth noting. First, in this series of tests da females produced 5050 eggs while + females produced 4812 eggs under the same conditions. Second, if the male survival of 0.90 is different from 1.00 and due to da, then while only male progeny from homozygous da females survive, only a fraction of them do.

References: Bell, A.E. 1954, Genetics 39:958-959; Sandler, L. 1972, Genetics 70:261-274.