Moyer, S.E. and D. Burton. Northeastern University, Boston, Massachusetts. Incomplete dominance in hybrids from D. melanogaster adapted to DDT or NaCl.

Strains have been selected for adaptation to extreme environments, DDT or NaCl in the food. An ebony (e) strain is adapted to .60g DDT/ $100 \, \text{cc} \, H_20$ of a commercial 50% wettable powder. A vestigal (vg) strain is adapted to 8% NaCl. "Instant medium" (Carolina) is used by adding

H₂O or a specified solution of DDT or NaCl to an equal volume of dry medium. Each strain is intolerable to the maximal stress environment of the other strain but grows well on normal food. There is no obvious effect of DDT in surviving e flies. The vg flies are very small when subjected to 8% NaCl and the wings have become much longer than non-adapted vg at any temperature or salinity of food. Disposable culture containers, "cartons" and "cups" used for this work are described in Moyer and Yarbrough (1969).

In this study, virgin e and vg females were collected within the first two days from normal food cultures. After ageing for two or three days mating chambers (paper cartons containing only a small cup of food) were employed for three mating groups: 1. 24 e couples and 24 vg couples; 2. 10 e couples and 40 vg couples; 3. 40 e couples and 10 vg couples.

After 24 hours, the e and vg females were transferred separately (without males) to "egg collection chambers". Eggs were collected on an agar-acetic acid-alcohol mixture (Delcour, 1969) in a plastic "cup" cover with a thick yeast suspension on the surface. Egg laying caps were changed daily. Eggs and the agar from the cap were transferred to half pint paper cartons containing medium with the desired concentration of DDT or NaCl. Preliminary results in the table show the proportions of wild type progeny which resulted from e (DDT) x vg (NaCl) matings as detected from the first brood of eggs on normal food, in relation to females mated by males of their own strain. Of course, the proportion of heterogamic matings was influenced by competitive disadvantages of mutants and the proportions of parents of each strain.

In any case, the most striking consequence is the very low proportion of heterozygotes which survive on DDT in contrast to ebony progeny from DDT adapted parents. Furthermore the body size of these wild types was much smaller than e progeny.

Survival of progeny in various environments.

Daily Brood Separated e φφ (afte	Food r mating)	24 e	eny ο	-	10 e	eny o 99 † 8 99	් රීරී	40 e	eny o	් රීරී
· 1	Normal	96	221		14	70		138	87	
2	.35 DDT	115	5		26	7		222	0	
3	.45 DDT	109	11		10	14		99	1	
4	.30 DDT	150	25		23	12		246	16	
5	.40 DDT	152	5		30	13		213	21	
Separated vg çç (aft	er mating)									
1	Normal		165	244		130	283		205	106
2	6 NaCl		. 56	79		63	108		47	24
3	8 NaCl		. 8	4		2	3		1	1
4	5 NaCl		59	34		89	187		65	64
5	7 NaCl		28	31		14	20		21	23

Less clear, so far, is the viability of hybrids on NaCl compared to vg salt adapted progeny in the same cultures.

References: Delcour, J., 1969 DIS 44: 133; Moyer and Yarbrough, 1969 Am. Biol. Teacher 31: 593-596.

Schalet, A.* and K. Singer. University of Connecticut, Storrs, Connecticut. A revised map of genes in the proximal region of the X chromosome of Drosophila melanogaster.

Following the initial report that a Y chromosome (y Ymal No.2) with a duplication for the proximal region of the X chromosome covered at least 10 genes located between M(1)n and bb (Schalet, 1963), two earlier maps of this

region have been presented (Schalet and Finnerty, 1968; Chovnik, Finnerty, Schalet and Duck, 1969). The present map adds 12 new sites, indicated by , to the one in Chovnick et al. The sites connected by ___ have not been ordered with respect to their left-right positions.

To the corrections and additions given in DIS 44 and 45 add: 1) The $\underline{1}$ N5 earlier positioned to the left of $\underline{1}$ 152 is an allele of $\underline{1}$ 151. This error was probably \overline{d} ue to a stock erroneously labled \underline{D} f($\overline{1}$)mal⁸. Subsequent retesting of lethals between mal and lf with one another and with cytologically analyzed deficiencies including mal⁸ has produced the order

shown here. 2) The site indicated by a \cdot represents the visible phenotype displayed by qq heterozygous for the deficiencies T2-14a/LB23 as well as All8/Q539 and the two other appropriate combinations of these lethals. 3) Since 122 had been lost, it has been eliminated from the map because its relation to the new lethals \overline{b} etween 120 and su(f) could not be tested.

The following features of the region may be noted: 1) We have only listed here those mutants that have thus far proved to involve single functional units. We have additional alleles of some of these mutants that also appear to involve single functional units. 2) As reported earlier (Schalet, Lefevre and Singer, 1970), 1A7 and the mutants to the right are in salivary division 20. 3) With one possible exception, su(f) is placed immediately to the left of bb. In consideration of some interesting characteristics associated with this locus, that are being reported elsewhere, this position may not be fortuitous. These attributes permit the speculation that su(f) is an element in the protein synthesizing mechanism. 4) All 16 genetically analyzed deficiencies, with a right break-point apparently between su(f) and bb and a left break-point extending different lengths to the left, have proved to be δ fertile. In our own material and from the literature it appears that deficiencies extending from the right or left into the bb locus are usually δ sterile. We have tested 14 such deficiencies and 10 were δ sterile. Since these deficiencies were not carefully analyzed as to whether the bb locus was completely or only partially removed no further comment is warranted. However, we are aware that $\delta\delta$ with the bb deficient sc^4-sc^8 chromosome are fertile.

Above the main map we have listed a number of lethals obtained from Dr. Raphael Falk. The positions given are in accord with tests for allelism with the mutants listed below them and supported by analysis with our duplications and deficiencies. Lethal 3DES behaves as an allele of su(f) with respect to forked suppression but does not show the phenotype associated with the combination su(f)/su(f). It has not been tested with bb yet. The following comments apply to the complementation map presented by Lifschytz and Falk, Mutation Research 1969. 1) The entire map lies to the right of mal. Section number 1 lies between 1E1C1 and 1152. 2) The "hot spot", section 17, as well as the "hot spot", section 18, for X-ray induced breaks attributed to intercalary heterochromatin, is in salivary division 20, probably 20A1-2. We have observed these "not spots" in our own material. 3) We note the disagreement with their map in respect to the 4 right lethals listed above the map presented here. The relative positions and section numbers on their map are: X4 (27), 3DES (29), R9-10 (31-32), Q463 (34). We further note the crucial position occupied by Q463 in their analysis of recombination data for the proximal region (Lifschytz and Falk, 1970). That the alleles of Q463 and 3DES, 120 and su(f) respectively, are positioned 120 left and su(f) right, is supported by a large scale recombination experiment. This experiment, which obtained evidence that crossing over at the bb locus in a normal ordered chromosome could generate bb mutants, utilized 120 and su(f). All 9 fertile recombinants buteen 120 and su(f) proved to be recombinant for the flanking markers mal 1 and y^{+} of the sc V1 duplication.

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