Mutation Notes — Drosophila melanogaster

Saturation mutagenesis of region 82F.

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In an attempt to isolate mutations in the 82F gene responsible for the late puff there, an X ray and an ENU mutagenesis were run, starting with an isogenic *mwh red e* chromosome and testing against Df(3R)3-4 (82F3-4;82F10-11, my cytology, does remove the puff) for lethals and visibles in the F_2 . Out of 5756 fertile F_1 tests of X-rayed chromosomes (78% of males were fertile, 97% of females) there were 21 lethals = 3.6 x 10^{-3} for ca 9 bands; out of 3130 fertile F_1 tests of ENU-treated chromosomes (92% of males fertile, 95% of females) there were 14 lethals and one semi-lethal = 4.8 x 10^{-3} . These were then sorted into three regions by complementation testing against Df(3R)6-7 (82E3;82F3-7, not my cytology, does remove the puff) and Df(3R)110 (82C4;82F3-7, not my cytology, does not remove the puff). All mutations within a region were crossed to each other and also to all mutations in the adjacent region(s); 11 compementation groups resulted, with only one (l(3)82Fh) being at all complicated, and one deficiency was recovered: Df(3R)ME15, 81F3-6;82F5-7. All X-ray induced mutations had their cytology checked; if no aberration is indicated below, then the 82F region had no obvious cytological defect.

In group 1 (lethal over all four deficiencies):

l(3)82Fa: X ray alleles 1 and 2, ENU alleles 3 and 4. Larval-pupal lethals with inclusions; mitotic index low.

l(3)82Fb: ENU alleles 1 and 2. Both alleles are only semi-lethal; escapers have small heads and broad abdomens.

In group 2 (lethal over Df(3R)6-7, Df(3R)ME15, and Df(3R)3-4; viable over Df(3R)110):

l(3)82Fc: X ray alleles 1, 2, and 3, ENU allele 4. $In(3R)82Fc^{1}$, het;82F3-11; $In(3R)82Fc^{2}$, het;82F3-7. Pre-larval lethals.

l(3)82Fd: X ray alleles 1, 2, 4, 5, 6, 7, 8; ENU allele 3. $Tp(3;Y)82Fd^{l}$, 82F3-11;98F8-14; $T(2;3)82Fd^{l}$, 42E3-7;82F5-7; $Tp(3;3)82Fd^{l}$, 82F5-7;92D1+;92F3-5, new order 61A1-82F5|92D2-92F5|82F5-92D1|92F5-100. All alleles are eclosion lethals over deficiencies; flies assisted from their pupal cases are alive, and weaker combinations give significant levels of escapers who had been wingstuck. Alleles 2 and 3 have brown eyes over deficiencies and allele 1 has variegated brown eyes (since the parent chromosome carries red, this phene has not been assayed in the heteroallelic combinations); the rest have wild-type eyes. Allele 3 is homozygous viable and fertile; all other alleles are stronger, with roughly the order (strongest) 1, 2; 4, 7; 8; 5, 6 (weakest) based on relative viabilities over deficiencies and each other, although slightly different orders result from different comparisons, suggesting that the gene may be somewhat complex -- as indeed is already suggested by the variation in eye color effects. Allele 4 does not puff the 82F puff, suggesting that l(3)82Fd is the puff gene itself.

l(3)82Fe: X ray allele 1, ENU allele 2. $In(3)82Fe^{t}$, het;82F3-7. Pre-larval lethals.

In group 3 (lethal over Df(3R)3-4 only; viable over Df(3R)110, Df(3R)6-7 and Df(3R)ME15):

l(3)82Ff: one allele induced with ENU, early pupal lethal

l(3)82Fg: one allele induced by X rays, mid-pupal lethal

l(3)82Fh: alleles 1, 2, and 5 induced with X rays; alleles 3, 4, 6, 7, and 8 induced with ENU. $T(2;3)82Fh^2$, 57F3-11;82F7-11. Alleles 1 and 2 appear to be amorphic (from stages of death of hypomorphs over them) and are pre-larval lethals; the rest of the alleles are increasingly hypomorphic, with alleles 6, 7, and 8 complementing each other in all combinations. Lethal phases of hypomorphic

combinations range from pre-larval lethality through hanging up as wandering third instars to late pupal lethality.

l(3)82Fi: alleles 1 and 2 induced with X rays, allele 3 with ENU. $T(2;3)82Fi^2$, 57A10-B1;82F10-83A1. Pre-larval lethal.

1(3)82Fj: one allele induced with X rays, pre-pupal lethal. Ab(het;3R)82Fj¹, het;83A1+.

1(3)82Fk: one allele induced with ENU, leaky late pupal/eclosion lethal.

Other mutations recovered from X rays:

Df(3L)ru-22, 61F8;62A3-5. Detected because the Df(3R)3-4 chromosome used carried ru^{I} although this wasn't indicated on its label.

 $In(3LR)Sai^{l}$, 69D2-6;84E12-F3. Dominant outheld wings, recessive lethal allele of the *mirr* complementation group = $mirr^{Sail}$.

 Sai^2 , dominant outheld wings; no cytological defect, maps genetically to 3-37.9 relative to h and th. Recessive lethal allele of the *mirr* complementation group = $mirr^{Sai2}$.

 Sai^{l} , Sai^{2} , D^{l} , D^{3} , and $mirr^{DH-l}$ (homozygous viable hypomorphic mirr allele) fail to complement

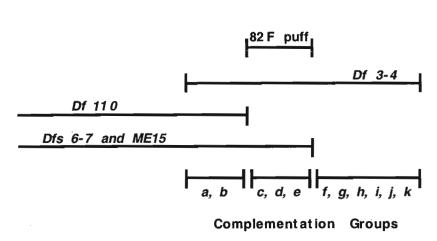


Figure 1.

each other; Sai^I is the strongest allele, then $D^I = Sai^2$, then D^3 . D^3 /mirr $^{DH-I}$ is nearly completely viable, though with mild head defects and missing bristles.

Other mutations recovered from ENU: saw several, kept only a scarlet (= st^{33}), again detected because the Df(3R)3-4 chromosome carried a st allele that wasn't indicated on its label.

Thirty-five mutations across 11 complementation groups = 3.2 hits per gene on average; although the distribution of numbers of hits per gene observed is very far off that expected from the Poisson distribution, that distribution predicts that the number of lethally- or visibly-mutable genes missed is 0.5.

New lethal mutations in the 97B1-10 to 97D13 region of the *Drosophila melanogaster* 3rd chromosome.

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In F2 EMS screens for mutations in the dPC2 gene, we recovered sixteen lethal mutations and one visible mutation over Df(3R)Tl-X and Df(3R)ro80b. Together, the deficiencies cover the 97B1-10 to 97D13 region and overlap in the 97D1-2 region (Anderson et al., 1985; Knibb et al., 1993). Nine lethal mutations and the visible mutation fail to complement both deficiencies and thus map to the 97D1-2 region that includes the dPC2 gene. These mutations are described elsewhere (D.T., A.R.K., and M.B., manuscript in preparation). Three (dt6, dt12, dt14) of the remaining 7 mutations recovered in our screens fail to complement Df(3R)Tl-X but complement Df(3R)ro80b and therefore are located beween 97B1-10 and 97D1 (Figure 1). The dt6, dt12, and dt14 mutations fail to complement one another and also fail to complement l(3)673, a previously identified lethal in the region (K. Anderson, unpublished). These mutations have recently been shown to be allelic to