Mutation Notes - Drosophila melanogaster

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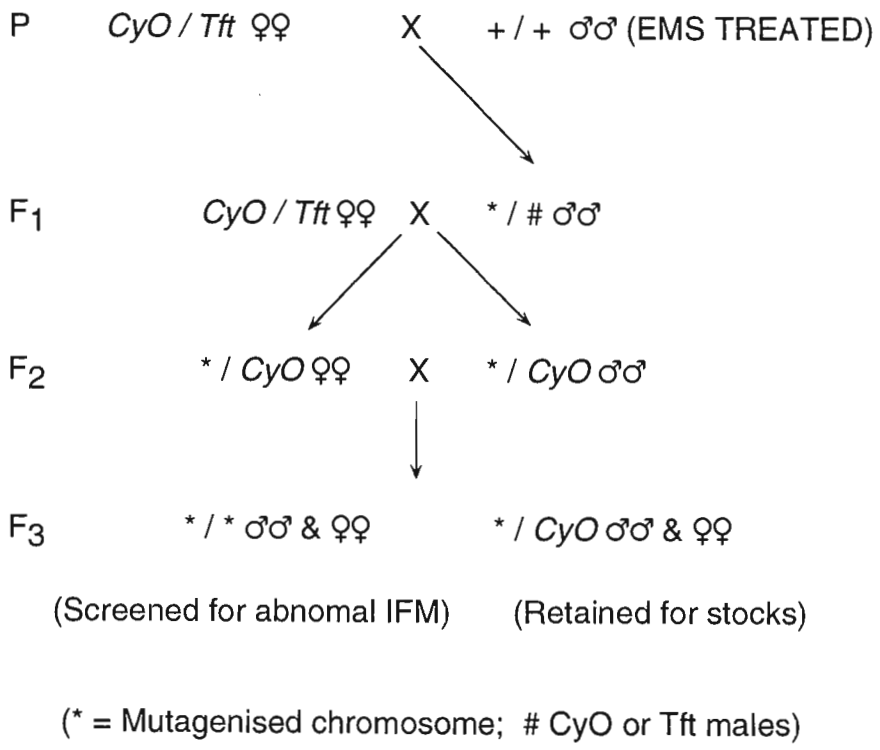
Isolation of an allele of yellow body mutation in *Drosophila melanogaster* by an unusual pattern of inheritance.

Chemical mutagenesis is commonly used in *Drosophila* genetics to induce point mutations and chromosomal aberrations (Ashburner, 1989). One of the main areas of our research activity is to induce mutations in general and then to select viable recessive mutations confined to second chromosome which affects the Indirect Flight Muscle (IFM) development in *Drosophila melanogaster* by using ethyl methanesulphonate (EMS). The protocol used for induction and detection of mutations on the second chromosome is as follows:

In this proto-col, 3500 lines of the flies were set up to screen for mutations. All the males and females of the F2 progeny were analyzed. Surprisingly, in one line, out of 25 males and 30 females, only one male had a yellow body phenotype along with the *CyO/Tft* markers. This was not an expected result. Perusal of the literature, since 1916, when Morgan and Bridges reported for the first time the yellow body mutant, many alleles of yellow body mutant, localized on X-chromosome have been reported (Lindsley, 1992). In the present study, the mutagenized X-chromosome is not retained in the males of F1 progeny. Then, the question is, how come a yellow body mutant appeared in the F2 generation? The possibilities are it might have arisen by spontaneous mutation or by some means yellow body colour gene was inserted to autosomes and then activated or by any other unknown mechanisms.



To understand the inheritance pattern and to preserve the mutant chromosome, this yellow body mutant was crossed to three females of a strain of *D. melanogaster* having normal X-chromosome with *CyO/Tft* markers on chromosome 2. This was done because the virgin females of the above strain were readily available since these are being used for routine experiments. From this cross, one can usually expect that all the F1 individuals should possess wild type body colour with markers, if the yellow body colour is recessively inherited. Interestingly, the phenotype of the observed progenies were different from the expected results. In the F1 generation, out of 31 flies scored, 23 (11 males and 12 females) had the yellow body phenotype and 8 (4 males and 4 females) had wild type phenotype. These yellow body mutant flies along with the markers bred true and are being maintained as stocks. This indicates that the yellow body colour gene is not behaving as recessive or dominant genes, but it behaves as unusual dominant-like gene and it appears in the F1 progenies in 3:1 ratio. The puzzling question now is how a single copy of the X-chromosome present in the yellow body male could give two copies of the yellow body gene or X-chromosome to generate yellow body females? This is possible only if the female parents have at least one copy of yellow body mutant chromosome or by contamination. However, our data on the ratio and progenies scored showed no such indications. Moreover, the strain

having normal X-chromosome with *CyO/Tft* markers on second chromosome is being used for routine experiments and is a pure line.

Interestingly, subsequent crosses to Canton-S strain and yellow allele 1 of *D. melanogaster* (obtained from our Drosophila Stock Centre; Mysore) have shown the sex-linked pattern of inheritance. Therefore, this is an unusual pattern of inheritance observed only for one generation.

The phenotypic characteristics of the yellow body mutant isolated in the present study are as follows: Adult body colour is lighter than yellow allele 1 type. Hairs and bristles are brown with yellow tips. Wing veins and hairs are yellow. Larval setae is yellow to brown. Larval mouth parts are golden brown and mouth hooks are dark brown. The viability of this mutant is excellent. Thus, the yellow body mutant isolated in the present study is recessive sex-linked and allelic to yellow 1 type and we have named this mutant as y^{RU} allele.

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References: Ashburner, M., 1989, In: Drosophila. *A Laboratory Handbook.* CHS Press, London; Lindsley, D.L., and G.G. Zimm 1992, In: *The Genome of* Drosophila melanogaster. Academic Press Inc., USA.

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New FM7 versions from Strasbourg:

- FM7d: FM7, y[31d] sc[8] B, fertile.
- FM7e: FM7, y[31d] sc[8] oc ptg B, female sterile.
- FM7e P[ftz-lac,ry+]: FM7, y[31d] sc[8] P[ftz-lac,ry+] oc ptg B, "ftz blue FM7e".
- FM7f: FM7, y[93j] sc[8] oc ptg B, female sterile, y[-] marker.
- FM7f P[ftz-lac,ry+]: FM7, y[93j] sc[8] P[ftz-lac,ry+] oc ptg B, "ftz blue FM7f".
- FM7g: FM7, y[31d] sc[8] w[a] oc ptg v[Of] B, female sterile.
- FM7g fa[n]: FM7, y[31d] sc[8] w[a] fa[n] oc ptg v[Of] B.
- FM7g ct[ns]: FM7, y[31d] sc[8] w[a] ct[ns] oc ptg v[Of] B.
- FM7h: FM7, y[31d] sc[8] w oc ptg B, female sterile.
- FM7h N[PlacW]: FM7, y[31d] sc[8] w N[PlacW] oc ptg B, lethal, "N blue FM7h".
- FM7i: FM7, y[93j] sc[8] w oc ptg B, female sterile, y[-] marker.
- FM7j: FM7, y[93j] sc[8] w, very good fertility, y[-] marker.

Comments: These chromosomes represent new useful versions of the effective FM7 balancer performed at Strasbourg. The previous sn[X2] female sterile marker from FM7c has been replaced advantageously by the female sterile oc[1] inversion because sn males often stick on the food medium. The different markers used were introduced from In(1)dl-49 into FM7 through the medium of the In(1)sc[8] In(1)dl-49 chromosome. The amorphic y[93j] allele was EMS-induced on FM7, y[31d]. The B[+] FM7j version was obtained after unequal crossing over within the tandem duplication of B. The P[ftz-lac,ry+] insertion on a FM7 chromosome was obtained by Hiromi and recombined here on a FM7e version. N[PlacW] is a PlacW enhancer trap induced N haplo-insufficient allele obtained in the Jan's lab on In(1)dl-49, w; it was introduced in the FM7h version. I remember that occasional spontaneous compound-X chromosomes occur with balancers of the X and a normal X chromosome.

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The "nw^{P2ry+}" mutation of *Drosophila melanogaster* proves to be an allele of *tapered.*

We earlier described a recessive, *P*-induced mutation in *D. melanogaster* (Scheel and Doane, 1994; Bien-Willner *et al.*, 1996) that, in homozygotes, produces "narrow-like" wings with pointed tips, reduces viability, causes complete behavioral male sterility, and decreases the fertility of females. This mutation was assigned to chromosome *2R* through genetic analysis and tentatively placed in region 54E by *in situ* hybridization of a biotinylated *P* element probe to