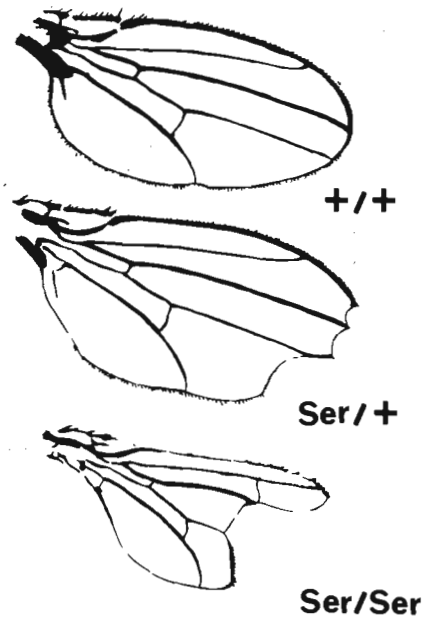
Belt, A.L. University of Sheffield, England. A non-lethal allele of Serrate?

TM3 Ser (Lewis 1960 D.I.S. 34, Tinderholt 1960 D.I.S. 34) carrying the dominant visible and recessive lethal mutant, Serrate, was used as a third chromosome cross-over suppressor to maintain heterozygosity of the melanotic tumor gene tu-C4 for a selection experiment. The tu-C4 chromosome carried the Stubble mutant to prevent survival of tu-C4 homozygotes. Among the progeny were found flies which were described as 'extreme Serrate'. They were characterised by the absence of the dominant marker Stubble and by an apparent extreme expression of Serrate so that the wings were very much reduced. The phenotype of extreme Ser, heterozygous Ser and $Ser^+$ are shown in the figure. The development time of the extreme Ser flies was longer than that of normal heterozygotes and they were tumorous. They were isolated from the selection lines and crossed with each other. Stubble did not reappear in the $F_1$ or subsequent generations and the same extreme Ser expression was seen in all the flies. When they were crossed with wild type, all the $F_1$ progeny showed the normal heterozygous expression of Ser. In the $F_2$, extreme Ser, heterozygous Ser and wild type, segregated out.




The possibility of survival of TM3 Ser homozygotes was ruled out because of the tumorous nature of the flies and because they showed no evidence of the recessive mutants also carried by that chromosome. This was confirmed by a salivary-gland chromosome analysis of the extreme Ser flies (carried out by Dr. M. Ashburner, University of Cambridge) which revealed that the Ser chromosome was completely wildtype in sequence.

From this evidence it was concluded that Ser had been transferred from the TM3 Ser chromosome to the tu-C4 chromosome (probably as a result of the double cross-over), and at the same time the lethal effect of the mutant gene had been alleviated. As a result, Ser now segregates as a dominant visible, non-lethal mutant. The most likely explanation for this is that during the cross-over events the dominant visible effects of Ser were separated from a very closely linked recessive lethal mutant. Alternatively, the Ser mutant may have undergone further mutational change to a non-lethal allele which has retained its visible effects.

It is obvious from these findings that the complex inversion system present in TM3 cannot be reliably used as a complete balancer for chromosome three, since cross-over events producing viable gametic products occur at an appreciable frequency. Neither can the recessive lethal mutant Ser be used successfully to maintain heterozygosity of the third chromosome.

Godbole, N.N., R.M. Kothari and V.G. Vaidya. University of Poona, India. An observation on the uric acid content in the excretion of D. melanogaster larvae.

During our studies on the nature of the excretory products in D. melanogaster larvae, a striking variation was observed in the amount of uric acid in the excreta.

Ten larvae of Oregon K strain of D. melanogaster were reared in each of the 25 glass vials each containing 6 g ± 50 mg of the standard cornmeal-agar medium. The vials were placed at 22 ± 1°C during the development of the larvae. After pupation, the medium from each vial was homogenized separately in 100 ml distilled water. Few drops of concentrated KOH solution were added to facilitate maximum extraction of uric acid. Uric acid was quantitatively estimated for each vial by Brown's reaction (Brown, 1945) using Klett and Summerson's photoelectric colorimeter at 660 mμ.

It was observed that the uric acid content of the extracts varied between nil and 330 μg. Similar results were obtained by rearing larvae on six different media. This indicates that composition of the medium plays no role in the observed pattern of uric acid excretion. An alternate pathway resulting from an error in uric acid metabolism is suspected in some flies from our culture.

Reference: Brown, H. 1945 The determination of uric acid in human blood. J. Biol. Chem. 158: 601-608.