Duttagupta, A. and A.DuttaRoy. University of Calcutta, India. Reassessment of breakpoints of a deficiency on 2L of Drosophila melanogaster.

The deficiency M(2)-z^B was originally isolated by Bridges. The breakpoints of the deficiency has been mentioned as 24E2-F1; 25A1(2) (Lindsley & Grell 1968). In our cytological preparation (stock obtained from Mid-America Drosophila Stock Center at Bowling Green, Ohio USNA), we found that the distal breakpoints as mentioned is alright; we however observed that the proximal breakpoint is between 24F7-8 and 25A1(2). The band 25A1(2) remains intact in this deficiency (Figure 1).

Figure 1.

The deficiency M(2)-z^B noncomplements three alleles, namely dumpy (dp), dwarf-24F(dw-24F) and M(2)-z. Broderick & Roberts (1982) assigned M(2)-z at 25A1(2) by making use of a series of duplications. Since different Minute mutations instead of being additive show complementary genetic effects among each other (Schultz 1929), the failure of M(2)-z to complement DfM(2)-z^B awaits a proper explanation.

We recently isolated 45 mutations in DfM(2)-z^B locus (38 lethals and 7 new Minutes). None of these mutations can kill M(2)-z in heterozygous condition (DuttaRoy et al. 1984). We therefore can assume that (i) the deletion in M(2)-z^B may involve some interband between 24F7-8 to 25A1(2). The same interband may have a lesion in M(2)-z that does not allow the recovery of the trans-heterozygote M(2)-z/M(2)-z^B, (ii) M(2)-z might be acting as a polarity mutation or (iii) M(2)-z^{B+} and M(2)-z^+ region may involve together in the synthesis of a molecule with a dimeric structure. Therefore, mutation in both the loci seriously impedes some essential function of the fly and leads to lethality.

References: Broderick, D.J. & P.A.Roberts 1982, Genetics 102:71; DuttaRoy, A., P.K. Manna & A.K.Duttagupta 1984, J.Biosci. (in press); Schultz, J. 1929, Genetics 14:366.

Duttagupta, A. and A.K.Ghosh. University of Calcutta, India. Effect of cadmium chloride on the polytene chromosome of Drosophila salivary gland.

It is well known that different heavy metal ions stabilize or labilize the ordered conformation of DNA molecules in vitro. They may either promote the reversible unwinding and rewinding of multiple stranded helix. We have studied the effect of $CdCl_2$ (a heavy metal salt) on the polytene chromosome of Drosophila.

Second instar larvae of Drosophila ananassae were fed with $CdCl_2$ salt (1×10^{-6} molar dissolved in sucrose solution) for 48 hrs. The larval salivary gland chromosomes were then prepared by squas technique. In case of control, larvae were fed with only sucrose solution from 2nd instar for 48 hrs.

Out of 103 treated nuclei observed, 95 chromosome arms showed asynapsis, of which 19 were X-chromosomal and 76 autosomal arms. In case of control of the 103 nuclei observed only 31 chromosomes show such asynapsis, of which 9 were X-chromosomal and 22 autosomal arms (see Table).

	Total number of nuclei observed	Number of asynapsed chromosomes	X-chromosome	Autosome	% of asynapsis
Control	103	31	9	22	6.00
Treated	103	95	19	76	18.40

The data, therefore, reveals that in treated nuclei chromosomes asynapsis is 3 times more than that of control nuclei. The high percentage of asynapsis in treated nuclei is probably due to some ionic disturbance.

Duttagupta, A., A.Kar and A.DuttaRoy. University of Calcutta, India. A deficiency Minute mutation that acts as an enhancer of position-effect variegation.

$M(2)\text{-}z^{B}$ is a deletion spanning the polytene chromosome section 24E1-2; 24F7-8. We tested the effect of this deletion on brown-variegation (bw^{V1}). Level of Drosopterin pigment was measured following the methods of Reuter et al. (1983) by making bw^{V1} heterozygous with $M(2)\text{-}z^{B}$ chromosome. We have already reported the analysis of 38 lethal mutation in this region (DuttaRoy et al. 1984). Until now fourteen such lethal alleles have been tested with bw^{V1} (Fig. 1). Our analysis revealed that $DfM(2)\text{-}z^{B}$ act as a definite enhancer of brown-variegation where the quantity of pigments dropped down to less than half of the $bw^{V1}/+$ level. Some of the lethal alleles which behaved as point mutations also showed some reduction. The enhancement was not as pronounced as it was observed in case of deficiency $M(2)\text{-}z^{B}$. Work is in progress to see the effect of rest of the alleles.

Reference: Reuter, G. & J.Szidonya 1983, Chromosoma 88:277.

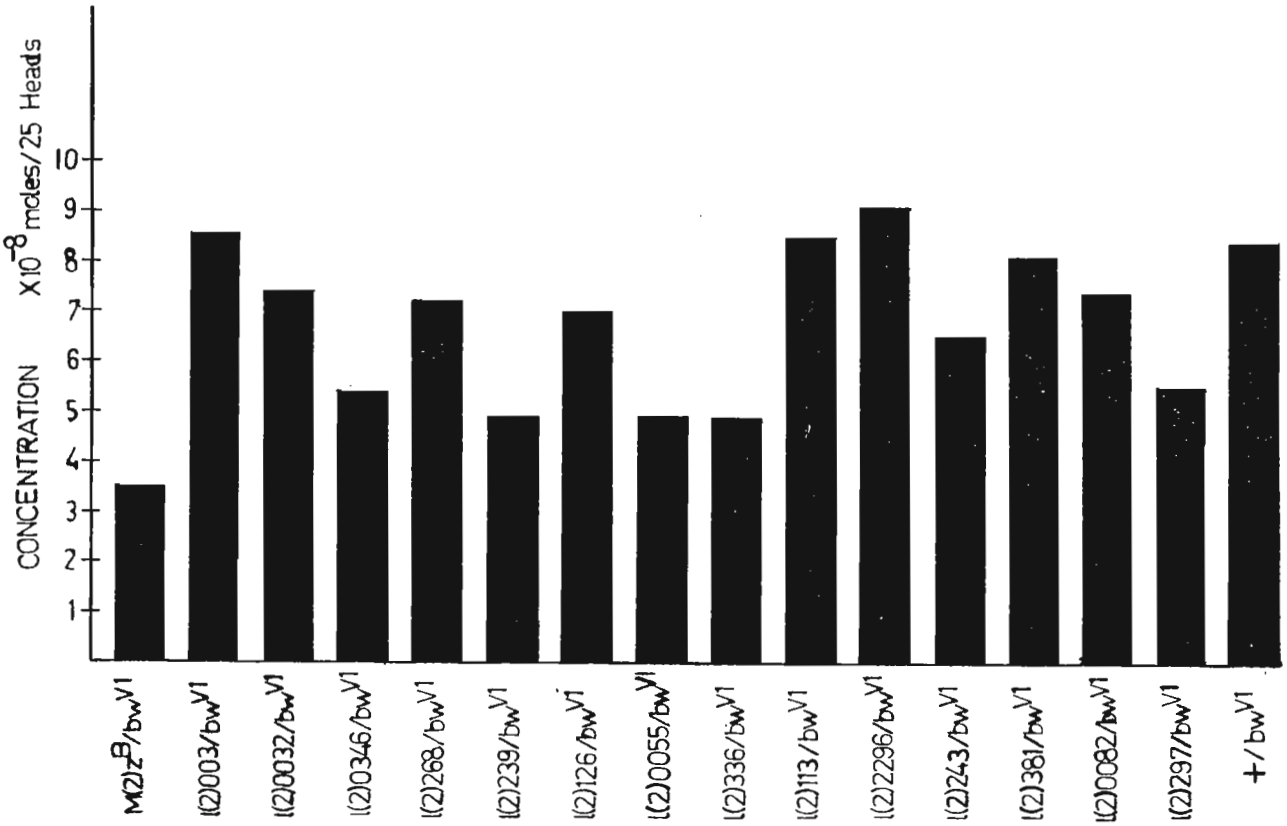

Figure 1.