74% synchronization within 48 hrs of Ricin feeding. Further work is in progress to obtain an increased frequency of in vivo synchronization, and to chase them in the later part of the same S-phase.

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References: Achary, P.M.R., K.Majumder, A.Duttagupta & A.S.Mukherjee 1981, Chromosoma 82:505-514; Battacharyya, P., I.Simet & S.Basu 1979, Proc.Natl.Acad.Sci. USA 76:2218-2221; Chatterjee, S.N. & A.S.Mukherjee 1975, Ind.J.Expt.Biol. 13:452-459; Ikegami, S., T.Taguchi, M.Ohashi, M.Oguro, H.Nagano & Y.Mano 1978, Nature 275:458-460.

Duttagupta, A., P.C.Das and P.K.Dutta. University of Calcutta, India. Genetic fine structure of Giant (gt) locus in Drosophila melanogaster.

The present paper concerns the genetic fine structure of the Giant (gt) locus (1.0-0.9) in Drosophila melanogaster, a locus responsible for an extra round of replication with concomitant increase in larval polyteny (Judd et al. 1972) and recently reported involvement in embryonic morphogenesis (Honisch & Campos-Ortega 1982). Recessive lethals were isolated in the region 3A1-4 according to the scheme outlined in Figure 1. Out of a total of 9055 chromosomes tested against Df(1)62g18 according to the scheme outlined in Figure 1, only 32 were found to be recessive lethals. The putative lethals were tested for allelism first against the mutant gt and then with two other alleles of gt, viz., gt^{x11} and gt^{E6}. In case, these recessive lethals were allelic to the mutant gt, then the heterozygous female class would be absent or its frequency would be low; such lethals were designated as an allele of gt. Allelism test against gt w^a showed that 10 out of the 32 lethals gave a very few or no survivors in heterozygous condition with gt. While test of allelism against alleles gt^{x11} and gt^{E6} revealed that the alleles were non-complementing all the 10 lethals that were allelic to gt and interestingly also non-complements certain lethals isolated against

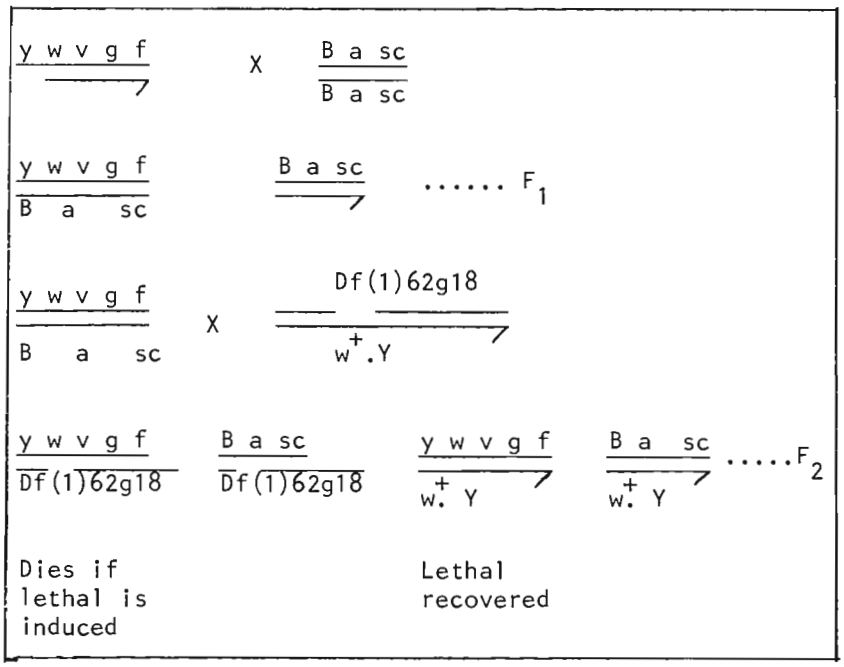

Fig. 1. Screening protocol for isolation of recessive lethals in the region 3A1-4. Males with chromosome markers y w v g f were fed with 0.025M EMS according to the method of Lewis & Bacher (1968).

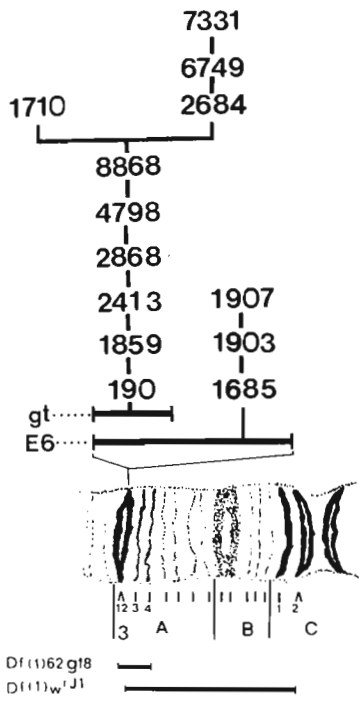

Fig.2

Df(1)62g18, but were complementing to gt. Inter se complementation analysis of the 10 lethals that were allelic to gt, revealed the presence of two complementation groups. Our finding has been summarized in Figure 2.

Inclusion of the mutant gt in the band 3A1 by Judd et al. (1972) was based on the complementation of this locus with $Df(1)w^{rJ1}$ and its non-complementation with three other deficiencies, Df(1)65j26, Df(1)X12 and Df(1)62g18, and in all of which the chromomere 3A1 was missing or reduced. The ten mutants that were found to have reduced the viability of the ℓ/gt heterozygote, partially or completely indicate that these lethals impaired seriously the functional unit of the gt allele in heterozygous condition, the survival of the ℓ/+ flies but not of the ℓ/gt class, indicates the presence of a decisive factor that acts in the trans heterozygotes, i.e., in the ℓ/gt, in trans dominant fashion. The isolation of 3 more lethals viz., 1685, 1903 and 1907 which complement gt but greatly reduces the viability in combination of gt^{x11} and gt^{E6}, points towards a difference between the alleles gt^{x11}, gt^{E6} and gt. This leads to the assumption that the functional areas of the alleles gt and E6 differ slightly. It therefore seems probably that the ten lethals which non complement gt, lie in that part (probably distal) of band 3A1 which is not covered by Df(1)62g18 and where gt locus has been mapped. This observation is specially significant in view of the findings by Judd et al. (1972) that the chromomere 3A1 is not deleted in $Df(1)w^{rJ1}$, but is reduced or missing in Df(1)62g18. The three other lethals viz., 1685, 1903 and 1907 seems to be larger lesions that probably span the whole chromomere 3A1 or further right to the proximal region of 3A1 and represents the true extent of this gt locus.

Further work on allelism test of these lethals against two other alleles of gt viz., gt^{13z} and gt^{Q292}, are in progress.

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References: Honisch, S. & J.A.Campos-Ortega 1982, DIS 58:76-77; Judd, B.H., M.W.Shen, & T.C.Kaufman 1972, Genetics 71:139-156; Lewis, E.B. & F.Bacher 1968, DIS 43:193.

Duttagupta, A. and A.DuttaRoy. University of Calcutta, India. Induction of a new Minute mutation in the second chromosome of Drosophila melanogaster.

During the course of saturation of a deficiency Minute mutation [$M(2)\text{-}z^{B}$; 24E1-2; 24F7-8)], we recovered some flies with Minute phenotype. Their genetical behaviour showed that these new Minutes failed to complement the deficiency, but complement M(2)-z mutation (for details see our report in this volume). When these Minute mutants are crossed with our seven complementing lethal alleles (DuttaRoy et al. 1984), it was observed that all these 7 groups kill this Minute as trans heterozygote. Analysis of polytene chromosome revealed that this new Minute allele bear a deletion for 24E region only (Fig. 1). A lethal allele of dumpy ($dp^{1\text{-}DG83}$) non-complemented this new Minute. This Minute, therefore, unlike others in this region is deleted for section 24E only.

Figure 1.

Reference: DuttaRoy, A., P.K.Manna & A.K.Duttagupta 1984, J.Biosci. (in press).