and the negatively phototactic go to the left; the most sluggish flies remain in the central chamber ("start"). The strain here studied is a "wild" one, called "Namur", and known as polymorphic for some enzyme variants. The tests have been done at the same time for both methods at the same temperature, with three repetitions for each temperature, according to a "latin square" design. Males and females were tested separately.

The Fig. 1 shows the results obtained using the Kekic maze. The Fig. 2 shows a three-dimensional representation of the final distribution of the flies in the different tubes after the tests done according to Benzer. In both methods, the phototactical behavior of the males and of the females differ significantly (with a probability of 0.0005, as shown by a $X^2$ test).

The influence of temperature is evident: at 30°C, the dispersion of the flies in the different test tubes or chambers is much more marked (the differences seem to be highly significant: probability of 0.0005 in the $X^2$ test).

The simplest explanation of it seems to be that the flies are more active at 30°C than at a lower temperature.

Perhaps a selection procedure for phototactism should give better results if the flies are tested at 30°C rather than 25°C.

References: Benzer, S. 1967, Proc.Nat.Acad.Sci. 58:1112; Kekic, V. 1981, DIS 56:178.

Duttagupta, A. and S.Banerjee. University of Calcutta, India. In vivo synchronization by Aphidicolin and Ricin in Drosophila.

Larval salivary glands of Drosophila contain an asynchronous cell population. They are in array of a replicating types, covering the whole of the S-phase. In our previous publication (Achary et al. 1981), we reported the usefulness of 5'-Flurodeoxyuridine in in vivo synchronization. In this report we present the results of our similar experiments with Aphidicolin and Ricin.

Aphidicolin is a tetracyclic deterpene tetraol, obtained from a fungus (Cephalosporium aphidicola). It is a specific inhibitor of DNA polymerase α with no effect on DNA polymerase β and γ (Ikegami et al. 1978). It binds to all eukaryotic DNA polymerase α reversibly (Huberman 1981). Ricin (Ricinus communis) a highly toxic plant protein, is also a potent inhibitor of DNA polymerase α (Bhattacharyya et al. 1979).

Early third instar giant female larvae of Drosophila melanogaster were fed on 1 ml (1M) sucrose containing 24 µg/ml Aphidicolin for 24, 48, 72, 96, 120 and 168 hrs and RIcin (1 mg/ml) was fed for 48 hrs only. Autoradiograms were prepared from the larval salivary gland. The frequency of labelling patterns was scored according to the classification of Chatterjee & Mukherjee (1975).

It can be observed from Table 1 that there was a net increase of 3C-3D types of nuclei (mid part of the S-phase), which reached its peak at 48 hrs, where 77% synchronized cells could be obtained. This then gradually declined as the feeding progressed. The frequency of DD-1C-2C (early patterns) remain more or less unchanged. Similarly Ricin produced about

Table 1. Frequency percent within the labelled nuclei.

| Patterns | Aphidicolin (24 µg/ml) | | | | | | Ricin(1mg/ml) |
|---|---|---|---|---|---|---|---|
| | 24 hr | 48 hr | 72 hr | 96 hr | 120 hr | 168 hr | 48 hr |
| DD | 3.07 | - | 0.68 | 0.50 | - | 1.23 | - |
| 1C | - | - | - | 0.50 | - | 1.23 | - |
| 2C | 1.53 | 0.61 | 1.37 | 2.50 | 2.05 | 2.46 | - |
| 3C | 16.92 | 27.60 | 28.96 | 16.00 | 23.28 | 27.77 | 29.26 |
| 3D | 49.23 (66.15) | 49.68 (77.28) | 46.19 (75.15) | 47.50 (63.50) | 45.20 (68.48) | 27.15 (54.92) | 45.12 (74.38) |
| 2D | 6.15 | 8.58 | 4.82 | 4.00 | 6.84 | 1.85 | 13.41 |
| 1D | 23.07 | 12.88 | 15.17 | 26.00 | 21.91 | 38.27 | 12.19 |
| CHL | - | 0.61 | 2.75 | 3.00 | 0.68 | - | - |

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

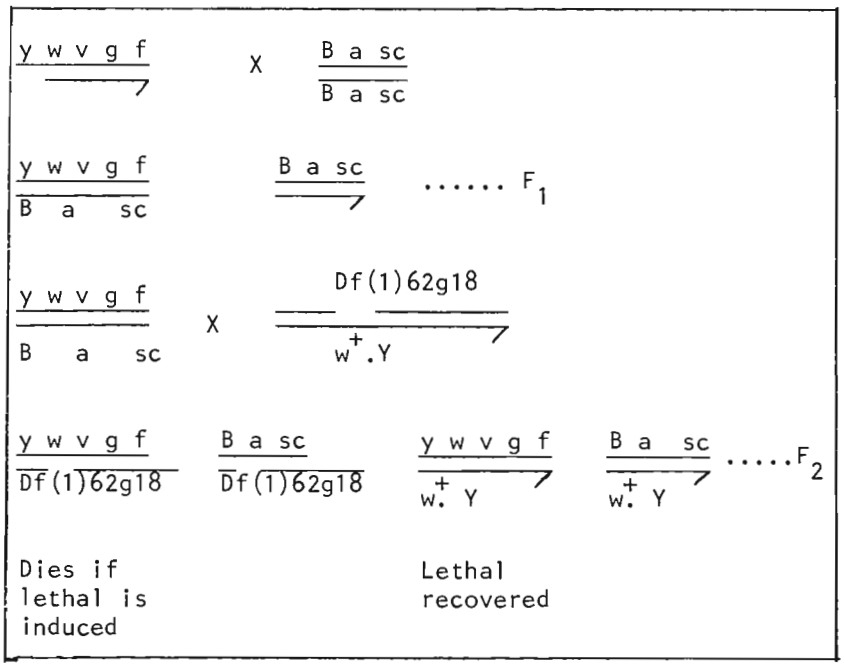


Fig. 1. Screening protocol for isolation of recessive lethals in the region 3A1-4. Males with chromosome markers y w v g f were fed with 0.025M EMS according to the method of Lewis & Bacher (1968).

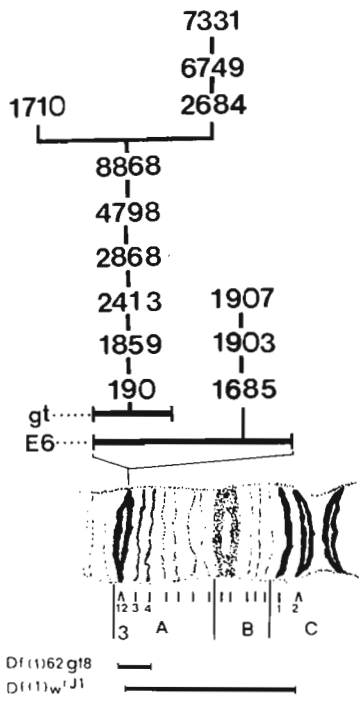


Fig.2