after gamma-irradiation. Pre-treatment of the D-32 males with caffeine does not modify this picture. However, pre-treatment with actinomycin-D (other things being equal) significantly enhances the occurring of the chromosome yellow mutations and reduces the yield of the gene ones. It would appear that the transformation of the pre-mutational lesions into chromosomal damages and less effective repair of the latter have both simultaneously taken place. These taken together (our own data and the well-known fact that actinomycin-D is bound with GC-repeats of DNA) argue that the chromosome changes, except for VVg, are initiated by the damages of the repetitive DNA sequences surrounding and/or interspersing the functional genetic unit. Therefore, as it follows from our own experimental data with neutrons (see Table), the quality (mode and perhaps density) of initial lesions predetermines the occurrence of rearrangements also. Thus both factors (i.e., the feature of the initial lesions determined by the radiation quality and the nature of the primarily damaged DNA sequences) appear to be decisive in the processing of the radiation-induced chromosome changes of all kinds.

However, the modifying effect of some other variables studied prove to be new and unpredictable. In particular, the combined irradiation by neutrons and gamma-rays (consecutive or simultaneous in the case of <sup>252</sup>Cf) increases the yield of VVg, but not a chromosome alteration. On the other hand, in the gamma-irradiated c(3)G post-meiotic male germ cells (repair-deficient mutant as proposed by Watson 1972), there was a very marked decrease in the frequency of the VVg in comparison with that in the gamma-irradiated wild-type germ cells.

One obvious question that needs to be answered is whether these variations in the specific-locus radiomutability are conditioned by the variables as such or are due to the feature of the locus studied. To answer this question, the spectrum and frequency of visibles for another loci will need to be studied under the same experimental conditions. Such genetical and cytogenetical analysis of the white, black and cinnabar mutations scored simultaneously with yellow ones is in progress now.

Acknowledgement: I am grateful to Dr. G.M. Obaturov, Head of the Department of Medical Physics, for his help and encouragement. My thanks also to my colleagues: Mrs. M.V. Alexandrova, Research Assistant (Cytogenetics); Mrs. N.I. Kosigina, Curator of Stocks; Obninsk 249020: Research Institute of Medical Radiology, Laboratory of Neutron Radiology, Komarova st., 4, USSR.

References: Alexandrov, I.D. 1984, Mutation Res. 127:123-127; Watson, W.A.F. 1972, Mutation Res. 14:299-307.

Andrade, C.A.C. and A.P. Gupta. Instituto de Biologia da UFRJ, Rio de Janeiro, Brazil. Studies on bristle number in hybrids between strains of **D.capricorni** from Brazil.

Drosophila capricorni is related to the "willistoni" species group but distinguishable from each other in externally visible characters (Dobzhansky 1951). It usually prefers cooler and humid regions. In Brasil, it extends from northern part to the southern most state. It has rarely been found in the summer while

it occurs in abundance during the winter season (pers. comm. from Prof. A.R. Cordeiro), and was confirmed partly by our data.

The object of the present research work was to examine the variance of bristle number in parental, F<sub>1</sub>'s and F<sub>2</sub> classes. For this purpose, several strains of D.capricorni were collected in July 1978 and February 1979 from Itatiaia (Resende, RJ), and were maintained in the laboratory as isofemale lines. A total of 13 strains (six from July 1978 and seven from the year 1979; these lines were maintained at 18°C for 540 and 980 days, respectively, before the commencement of the investigation) were utilized for the research work. Crosses in various combinations between strains (within and between the year of collection) were made to yield  $F_1$  and  $F_2$  classes. A total of 60 parental and 60  $F_1$ 's; 60 parental and 60  $F_2$  classes were analyzed at each of the two temperatures: 18° and 25°C. The parental and  $F_1$ 's were placed simultaneously at each temperature, using 50 eggs for each of the five or more replicates. Similar procedure was followed for the parental and  $F_2$  classes. The bristle number on 4th and 5th sternites, and the left and right esternopleurals were counted on the same individual in both sexes for each of the parental, F<sub>1</sub> and F<sub>2</sub> classes. In general, 8-10 males and 8-10 females from each of the three to six replicates for each class were examined. (However, there were cases where the sample size was very small which did not alter the final results.) The variance of the difference in bristle number between 4th and 5th sternites, and between left-right esternopleurals was computed. It was observed that the F<sub>1</sub>'s or F<sub>2</sub>'s had either equal, greater or smaller value of the variance when compared with their parental classes. However, in general, no significant difference in variance between parental and F1's, and between parental and F3's was observed for each sex at each temperature. Thus, these results indicated that the F1's and F3's were developmentally as buffered (stable/homeostatic) as their parental classes for the number of bristles examined even though the strains utilized in this experiment were maintained for the two different periods.

In addition, we looked for the pattern for frequency for the phenotypic variance. For this purpose, the variance values for the difference in esternopleural bristle number were grouped into three class intervals: 0-1, 1-2, and >2, for parental,  $F_1$  and  $F_2$  classes for each sex at each temperature. At each temperature the major part of the variance values in both the sexes occurred in the central class (1-2). The frequency in the case of the females grouped in the class greater than two was larger when compared with those of the males at either temperature. On comparing the class interval >2 between temperatures, a slight increase at 18° was observed than at 25°C for each sex. However, no specific pattern on this aspect could be noticed in the case of the crosses made between the two periods of collection. Comparison of the parental with  $F_1$ 's yielded almost homogeneity for the frequencies in each of the three classes of variance. Overall,  $F_2$ 's grouped in the central class, 1-2, were in larger number when compared with those of  $F_1$ 's for each sex at 18°C. No consistent pattern for such a comparison was observed at 25°C.

Likewise, the values of the variance for the difference in bristle number between the two sternites were also classified, into three classes: 2-6, 6-10, and >10, in order to search for pattern. At 25°, the majority of the phenotypic variance for the parental,  $F_1$ 's and  $F_2$ 's occurred in the central class (6-10) for each sex. While at 18°, such a frequency was spread into two classes (2-6 and 6-10), indicating a reduced number of phenotypic variances in the class greater than 10 at this temperature. No concludable pattern was observed for the comparison of  $F_1$ 's or  $F_2$ 's with their parental classes. The other aspects are under investigation.

This work was supported by Research Grants from CNPq (Proc. 40.0312/79 and 40.2525/81) and FUJB (UFRJ) awarded to Prof. Anand Prakash Gupta. We thank Stavna Uchoa for typing the manuscript.

Reference: Dobzhansky, Th. 1951, in:Genetics and the Origin of Species, 3d ed rev, Columbia Univ, New York.

Andrews, K. and C. Chihara. University of San Francisco, California, USNA. An overproducer of a third instar urea-soluble cuticle protein in Drosophila melanogaster.

Third instar larvae of **Drosophila melanogaster** show a standard electrophoretic pattern for the urea-soluble cuticular proteins (Fristrom et al. 1978; Chihara et al. 1982) (Figure 1, lanes 1 and 4). The bands are designated from slowest (top) to fastest (bottom) as L<sub>3</sub>CP-1, 2, 2a, 3, 4, 5, 6, 7, 8, respectively.

L<sub>3</sub>CP-1, 2, 2a, 3, 4, 5, 6, 7, 8, respectively. The overproducer variant of L<sub>3</sub>CP-5 (named L<sub>3</sub>CP5<sup>op</sup>) was isolated from an Austin Species Center stock acquired from Kuala Lumpur, Malaysia. Several rounds of pair matings produced a stock which gave consistently dark bands at the position of protein 5, (125 samples). The intensity of the variant L<sub>3</sub>CP-5 was checked against wild type using a gel scanner with integrator. The density of the L<sub>3</sub>CP-5 band relative to the L<sub>3</sub>CP-4 band of both stocks was compared and the following ratios were obtained (Figure 1):

5°P: Mean density ratio of Bands 5:4 = 1.95(
$$\pm$$
0.65):1;n = 67 +/+: " " " = 0.97( $\pm$ 0.13):1;n = 7

The L<sub>3</sub>CP5<sup>op</sup> variant is at least partially dominant. When it was crossed to wild type the 5 band remained visibly darker than the wild type. This can also be seen in the crosses described below.

To make sure the darkened 5 band was due to overproduction of L<sub>3</sub>CP-5 and not to the introduction of a new protein with the same electrophoretic mobility, the L<sub>3</sub>CP5<sup>op</sup> variant was crossed with, and then backcrossed to, a recessive mutant known as omega. Omega is a putative processing gene whose product, when inactive, results in the non-processing of L<sub>3</sub>CP-5; as a result, L<sub>3</sub>CP-5 migrates much more slowly than wild type L<sub>3</sub>CP-5. This slow band is designated as the omega band (Figure 1, lanes 2 and 3).

Density comparisons between the  $L_3CP$ -omega band and  $L_3CP$ -4 were made as above (Fig. 2).

Omega/omega: Mean density ratio of omega:4 = 1.04:1, with a high value of 1.40 and a low of 0.84. This is comparable to the values for +/+, bands 5:4 shown above.

 $5^{op}$  omega/ $5^+$  omega: Mean density ratio of Omega:4 = 2.4:1, with a high of 3. and a low of 1.83 (Fig. 1, lane 5).

Wild type omega recombinant: Mean density ratio of Omega:4= 1.25:1, with a high of 1.67 and a low of 0.83.

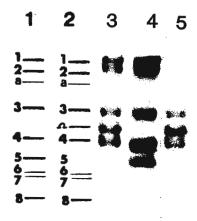


Figure 1. Lane 1: Schematic wild type banding pattern with numbers. Lane 2: Schematic omega homozygote pattern. Lane 3: Homozygous omega with wild type for gene 5. Lane 4: Typical wild pattern. Lane 5:  $L_3\text{CP5}^{\text{Op}}$  heterozygote with homozygous omega, note dark omega band.