

medium. The procedure for scoring sex-linked recessive lethals is described in detail by Abrahamson and Lewis (1971). In the present experiments two-day-old treated males were used to test for the induction of sex-linked recessive lethals.

Table 1. Frequency of sex-linked recessive lethals induced by aspirin in *D. melanogaster*.

| Concentration | No. of chromosomes tested | No. of lethals produced | % lethals |
|---------------|---------------------------|-------------------------|-----------|
| Control       | 895                       | 1                       | 0.11      |
| 300 mg/100 ml | 850                       | 2                       | 0.24      |
| 350 mg/100 ml | 615                       | 2                       | 0.33      |

Table 1 incorporates the data on the frequencies of sex-linked recessive lethals in controls as well as in the chemical-treated series. From this it is clear that both the concentrations tested were unable to induce a significant percentage of lethals compared to controls. By this, it can be concluded that these concentrations of aspirin are non-mutagenic to *D. melanogaster*. Consistent with this non-mutagenic nature of the drug, Maner et al. (1970) have reported that aspirin is unable

to induce chromosomal aberrations in human leukocytes. In contrast to these results, Jarvik and Kato (1968a,b) and Loughman (1971) in human leukocytes and Sen et al. (1975) in *Allium cepa* have shown significant chromosomal aberrations from aspirin and concluded it to be mutagenic. In the light of these highly contradicting results, more investigations on other animals and plants are necessary even though it is non-mutagenic in *D. melanogaster*.

Acknowledgements: The authors are grateful to Dr. M.R. Rajasekarasetty for his constant encouragement and valuable suggestions, and to the UGC for financial assistance.

References: Abrahamson, S. and E.B. Lewis 1971, in: Chemical Mutagens (A. Hollaender, ed.), Plenum Press, New York, pp. 464-469; Jarvik, L.F. and T. Kato 1968a, *Lancet* 1:250; \_\_\_\_\_ and \_\_\_\_\_ 1968b, *Science* 162:621; Loughman, W.D. 1971, *Science* 171:829; Maner et al. 1970, *Science* 169:829; Sen, P., O.S. Naik and K.N. Misra 1975, *Cur. Sci.* 44:713-714; Vasudev, V., N.B. Krishnamurthy and H.A. Ranganath 1978, *Inter. Symp. Environ. Agents & Biol. Effects* 59.

Vasudev, V. and N.B. Krishnamurthy. University of Mysore, India. Preliminary studies on the effects of cadmium chloride on *D. melanogaster*.

Cadmium pollution is increasing day by day due to its extensive use in industries and its existence as an impurity in zinc products. Cadmium has been demonstrated to induce drastic effects in experimental animals (Gunn and Gould 1970; Fowler et al. 1975; Tiggler et al. 1976;

Kumaraswamy and Rajasekarasetty 1976). Further, the disease "Ouchi-Ouchi" has been shown to be due to cadmium poisoning (Lucas 1975). Lucas (1975) has pointed out that no conclusive evidence links cadmium as a mutagen, carcinogen or teratogen for man. An attempt is made by the authors to investigate the effects of cadmium on the somatic and genetic systems of *Drosophila* and the preliminary results are presented.

*D. melanogaster* (Oregon-K) formed the material for the present study. Cadmium in the form of cadmium chloride was fed to larvae in concentrations of 0.05, 0.1, 0.5, 1.0 and 5.0 mg per 100 ml food medium. Normal medium was used as control. The eggs were collected following the procedure of Delcour (1969) and 35 eggs per vial were placed in each of the above concentrations. Flies were counted from the first day of eclosion to the last day of emergence. From the data, the rate of development and viability were estimated.

Fig. 1 (see following page) presents the pattern of emergence in different concentrations and in control. It is clear from this graph that the pattern of emergence is very much altered by the chemical. Developmental time is a fairly good indicator of various somatic effects caused by the chemical in the test substrate (Luning 1966). Hence, mean developmental time in control and in different concentrations of cadmium chloride has been estimated and presented in Table 1. Perusal of this table indicates that the rate of development is prolonged even at the lowest concentration tested. Prolongation of the mean developmental time becomes significant as compared to control ( $P < 0.05$ ). This is in line with the findings of Sorsa and Pfeifer (1973), wherein more than 1.25 mg CdCl<sub>2</sub>/l substrate is known to cause significant prolongation in the rate of development.

