



Our *Drosophila* cultures were kept at 25°C. At lower temperatures body weight increases. The described relationship with ADH activity does not hold in this case. ADH activity per mg is then even somewhat reduced with increasing body weight.

References: Bijlsma-Meeles, E. and W. van Delden 1974, *Nature* (Lond.) 247:369; Ursprung, H., H. Sofer and N. Burroughs 1970, *Wilhelm Roux' Archiv* 164:201; van Delden, W., A. Kamping and H. van Dijk 1975, *Experientia* 31:418.

Vasudev, V. and N.B. Krishnamurthy. University of Mysore, India. Effect of Dithane M-45 on rate of development and viability in *D. melanogaster*.

Rate of development and viability are the two parameters by which toxicity of a chemical is measured. Such parameters were used to test the effect of Dithane M-45 on *D. melanogaster* (Oregon-K). Eggs of the same age ( $\pm 4$  hours) were collected following the procedure of Delcour (1969). 35 eggs were then placed into each 3" x 1" vial containing chemical-supplemented media and normal medium and permitted to develop at a constant temperature of  $23 \pm 1^\circ\text{C}$ . Concentrations of 2, 5, 10, 15, 20, 25 and 30 mg of the chemical were thoroughly mixed in 100 ml wheat cream agar medium. The normal medium was used as control. The flies were scored each day from the time of emergence up to the end of eclosion. The pattern of emergence of flies in the control and in different concentrations of Dithane M-45 is depicted in Fig. 1 (see following page). It is clear from this graph that in the control the emergence of flies started on day 9 with a peak on day 11 and terminated on day 17. In contrast to this, the rate of development is prolonged in different concentrations of the chemical, thus affecting the time of emergence. In the lowest concentration (2 mg/100 ml food medium) eclosion commenced on day 11 and ended on day 22 with a peak on day 14. On the other hand, in the highest concentration (30 mg/100 ml) emergence began on day 19 and terminated on day 29. Here the peak of emergence was confined to day 25. The effect of Dithane M-45 on viability was measured by the number of flies emerged in each group. Thus the number of flies obtained in the control is 93.57%, while in the lowest concentration it is 82.14%; in the highest, 3.57%. From these results it is clear that Dithane M-45 has a significant toxic effect at higher concentrations.

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Reference: Delcour, J. 1969, DIS 44:133-134.

Vasudev, V. and N.B. Krishnamurthy. University of Mysore, India. Effect of aspirin on *D. melanogaster*. II. Non-induction of sex-linked recessive lethals.

(1978) that aspirin has a pronounced effect on the rate of development and viability in *D. melanogaster*. So far, there are no mutagenic reports of this drug. Hence, the authors tested the mutagenic property of this drug by scoring sex-linked recessive lethals in *D. melanogaster*.

Oregon-K and M-5 of *D. melanogaster* formed the materials for the present study. Aspirin was fed to *D. melanogaster* larvae in concentrations of 300 and 350 mg per 100 ml of food

Acetyl salicylic acid, marketed under the name "Aspirin", is well known for its antipyretic, analgesic and anti-inflammatory activity. It has been convincingly shown that aspirin produces drastic changes in experimental animals and plants. It is reported by Vasudev et al.



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*.

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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>/1 substrate is known to cause significant prolongation in the rate of development.