

Toxic effect of sevin on Drosophila melanogaster.

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Abstract

The toxicity of Sevin, a systematic carbamate pesticide, has been evaluated in the *Drosophila melanogaster* by larval feeding method. Eggs of same age (± 4 hours) were cultured on the Sevin containing media for their rest of life. Toxicity of Sevin was analyzed using parameters like rate of development, viability, morphology, and fecundity.

Key words: Sevin, Rate of development, Viability, Morphology, Fecundity

Introduction

The man-made chemicals are deliberately added to the environment as they are being widely used to manage several categories of pests. Unfortunately, most of these are not strictly selective and are also toxic to non-target species, which includes man himself (Davies *et al.*, 1975; McEwen and Stephenson, 1979). The impact in non-targets may lead to toxic effect and mutations that ultimately influence the progeny by heredity. Carbaryl, a carbamate pesticide, causes behavioral and neurological (Sideroff and Santolucito, 1972; Dsi, 1974; Anger and Setzer, 1979; Branch and Jacqz, 1986), reproductive, and carcinogenic problems (Cantor, 1992; and Davis, 1993) in exposed systems. Doull (1975) stated that "It is essential that the formulated forms as well as their subsequent changed formulations be tested. Hence, the present work was done on *Drosophila melanogaster* using carbamate pesticide so as to assess toxicity results.

Materials and Method

Most widely used Carbaryl, that is Sevin (CAS No.63-25-2), was tested. The standard Oregon-K (O.K.) strain of *Drosophila melanogaster* has been used in the present studies. All the experiments were maintained at $25 \pm 1^{\circ}$ C and RH 60%. The same aged eggs (\pm 4 hours) were obtained by Delcour (1969) method after crossing males with the virgin females. The same eggs at a density of 50 eggs/vial were exposed to different doses (2.5, 5.0, 7.5, 10.0, 12.5 and 15 mg/100ml of medium) of Sevin separately for their rest of their life as most of the insects are exposed to pesticides during their larval stages in which stage extensive feeding is seen (Vogel, 1977). Five hundred eggs were allocated to each dose of Sevin, and toxicity on fruit fly was analyzed using parameters like rate of development, viability, morphology, and fecundity. The results of rate of development and morphological traits were expressed by Mean \pm S.E. A simple t-test was used to know the significance between control and treated groups. Similarly, ANOVA was used to calculate the statistical significance of treated series to control with respect to morphological characters and fecundity.

Results

The concentrations above 17 mg of Sevin were found to be too toxic. Most of the eggs did not hatch and larvae die soon after hatching. Hence, concentrations below 17 mg ranging from 2.5 mg to 15.0 mg were selected to know their toxic influence on *Drosophila*.

The mean developmental time of Drosophila in different concentrations of Sevin is shown in Table 1. It varied from 10.06 ± 0.23 days of control to 25.33 ± 0.27 days in 15.0 mg of Sevin food media. As the amount of chemical in food is increased, there is a delay in the developmental time. However, the concentrations 2.5 mg and 5.0 mg are ineffective to bring significant changes. But, the remaining concentrations (7.5, 10.0, 12.5 and 15.0 mg) produced a delay, which is statistically significant to control.

Table 1. Effect of Sevin on rate of development of *Drosophila melanogaster*.

Concentration (mg/100ml)	Rate of development (Days)
Control	10.06 ± 0.23
2.5	10.43 ± 0.37
5.0	11.65 ± 0.61
7.5	12.31 ± 0.25*
10.0	16.23 ± 0.44*
12.5	20.14 ± 0.53*
15.0	25.33 ± 0.27 *

Note: All values are Mean ± S.E *Control versus treated significant at 5% level by simple t-test.

Table 2. Effect of Sevin on Viability of Drosophila melanogaster.

Concentration (mg/100ml)	Viability (Percentage)
Control	93.01
2.5	90.54
5.0	76.23*
7.5	70.19*
10.0	67.45*
12.5	45.32*
15.0	20.54*

^{*}Significant to control (p<0.05) by ANOVA.

Survival value as a measure of lethality is expressed in Table 2. The viability has been reduced from 93.01% (control) to lowest of 20.54% (15.0 mg). Except for 2.5 mg of Sevin, other concentrations significantly affected the viability of *Drosophila*.

Table 3. Effect of Sevin on the morphology of Drosophila melanogaster.

Concentration (mg/100ml)	Pupa length (mm)	Body length of fly (mm)	Wing length (mm)
Control 2.5 5.0 7.5 10.0 12.5	2.96 ± 0.71 2.88 ± 0.84 2.45 ± 0.66* 2.34 ± 0.49* 2.22 ± 0.41* 2.16 ± 0.39*	2.83 ± 0.85 2.74 ± 0.54 2.31 ± 0.60* 2.12 ± 0.31* 1.98 ± 0.47* 1.88 ± 0.50*	2.09 ± 0.55 2.04 ± 0.64 2.02 ± 0.66 $1.92 \pm 0.45^*$ $1.88 \pm 0.57^*$ $1.78 \pm 0.46^*$
15.0	2.05 ± 0.82*	1.67 ± 0.39*	1.66 ± 0.89*

Note: All values are Mean ± S.E

^{*}Control versus treated significant at 5% level by simple t-test.

As represented in Table 3, the morphological traits, namely pupa length, body length of fly, and wing length are directly influenced by the nature of the food. The food with 5.0 mg and above concentration of Sevin significantly altered the pupa length and body length compared to control. However, 7.5 mg and higher are effective to make significant alterations in the wing length when compared to their respective control group. From this it is clear that the effect of pesticide on morphological traits is directly dependent on the amount itself, and there is a relation between dosage and effect produced.

Table 4 depicts the result on the fecundity. Control group laid a total of 6934 eggs with a value of 45.66 eggs by single female per day. The egg laying potency has been reduced to 3861 with a value of 19.92, which is lowest and is due to the high amount of 15.0 mg Sevin. However, the 2.5 mg of Sevin is not able to produce significant changes. while all the remaining have effectively brought significant fecundity changes. Furthermore, there is also dose dependent effect on fecundity by Sevin.

Table 4. Effect of Sevin on fecundity of Drosophila melanogaster.

Concentration (mg/100ml)	Lotal tecundity	Daily egg production / female
Control	6934	45.66
2.5	6899	43.86
5.0	6245	40.12*
7.5	6093	34.68*
10.0	5120	25.43*
12.5	4665	22.59*
15.0	3861	19.92*

^{*}Significant to control (p<0.05) by ANOVA.

Discussion

Toxic effect of the Sevin was analyzed by using parameter namely –rate of development, viability, morphological traits and fecundity in *Drosophila*. The present results confirmed that there is a difference between treated and control groups. The prolonged rate of development is due to chemical's effect. This is in support of Luning's (1966) view, where the lengthening of developmental time is a

meaningful and best indication of a chemical's effect on somatic cells of test system. Findings of Luning (1966), Sorsa and Pfeifer (1973b), Laamanen *et al.* (1976) with different chemicals are similar. In other systems, Huckabee and Griffith (1974) and Dial (1978) are of the same opinion. Sevin has effect on the survivality of *Drosophila*. There is a linear relationship between lethality and the amount of Sevin present in the food. Same type of dose response toxic effect has also been shown by Marton (1974), Laamanen *et al.* (1976), Sorsa and Pfeifer (1773a). Morphological changes are due to the differential response of genotype with varying environmental condition like food, temperature, and density. The reduced morphological characters are the result of chemical present in food. The effects on such traits are fair indicators of somatic variation, because of chemical. Same results are noticed by Robertson (1959), Chinnici *et al.* (1976), and Lalor *et al.* (1976). Due to toxic food there is lowering of egg laying by *Drosophila*. According to Lints (1971), fecundity is extremely sensitive to environmental factors. Similar observations are made by Georghiou (1965) and Lints and Lints (1971).

From the above results and discussion, we are of the opinion that Sevin is toxic and has potency to influence the somatic changes in *Drosophila*.

References: Anger, K.W., and J.V. Setzer 1979, J. Toxicol. Environ. Health 5: 793-808; Branch, R.A., and E. Jacqz 1986, Amer. J. Med. 80: 659-664; Cantor, K.P., 1992, Cancer Res. 52: 2447-2455; Chinnici, J.P., M.A. Booker, and G.C. Llewellyn 1976, J. Invertebrate pathology 27: 255-258; Davies, J.E., S.A. Poznanski, R.F. Smith, and V.H. Freed 1975, International dynamics of pesticide poisoning. In: *Environmental Dynamics of Pesticides*. (Haque, R., and V.H. Freed, eds.). Plenum press, New York. 275-287; Davis, J.R., 1993, Arch. Environ. Contam. Toxicol. 24: 87-92; Delcour, J., 1969, Dros. Inf. Serv. 44: 133-134; Dial, A.N., 1978, Terotology 17: 83-92; Doull, J.,

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1975, Factors influencing toxicology. In: Toxicology - the Basic Science of Poisons. (Casarett, L.J., and J. Doull, eds.). McMilan Publ. Co., New York. 133-147; Dsi, I., 1974, Toxiclol. Appl. Pharm. 27: 465-476; Georghiou, G.P., 1965, In: Carbamate Insecticides: Chemistry, Biochemistry and Toxicology. (Kuhr, R.J., and H.W. Dorough, eds.). CRC Press, USA.; Huckabee, J.W., and N.A. Griffith 1974, Trans. Amer. Fish. Soc. 103: 822-825; Laamanen, I., M. Sorsa, D. Bamford, V. Gripenberg, and T. Meretoja 1976, Mutation Res. 40: 185-190; Lalor, J.H., J.P. Chinnici, and G.C. Llewellyn 1976, Develop. Industrial Microbiol. 17: 442-449; Lints, F.A., 1971, Gerontologia 17: 33-51; Lints, F.A., and C.V. Lints 1971, Expl. Gerontol. 6: 417-426; Luning, K.G., 1966, Nature 209: 84-86; Marton, A., 1974, Boil. Abst. 57: 34078; McEwen, F.L., and R. Stephenson 1979, John Wiley and Sons, New York. 1-20; Robertson, F.W., 1959, Univ. Texas. Publ. 5914: 89-98; Sideroff, S.I., and J.A. Santolucito 1972, Physiol. Behav. 9(3): 459-462; Sorsa, M., and S. Pfeifer 1973a, Hereditas 74: 89-102; Sorsa, M., and S. Pfeifer 1973b, Hereditas 75: 273-277; Vogel, E.W., 1977, Identification of carcinogens by mutagen testing in *Drosophila*. In: *Origins of Human Cancer*. (Heatt, H.H., J.D. Watson, and J.A. Winsten, eds.). Book C., Cold Spring Harbor Laboratory, 1483-1497.