Cytogenetic Studies on the Effect of Insecticide Monocrotophos in Mouse Somatic and Germ Cells

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Received August 9, 2005; accepted August 23, 2005

Summary The ability of pure insecticide “Monocrotophos” to induce chromosomal abnormalities in mouse somatic and germ cells was investigated. Male swiss mice were treated by gavage with the doses 0.05, 0.1 and 0.2 mg/kg⁻¹ body wt. Samples were taken 6, 24 and 48 h after treatment.

The percentage of chromosomal aberrations increased by increasing the concentrations of the insecticide and reached its maximum 24 h following oral treatment in both somatic and germ cells. In splenic cells, it reached 24.8±0.73 and 17.8±0.86 ($p<0.01$) 24 h after treatment with highest and lowest doses of the insecticide respectively compared with 26.2±1.02 ($p<0.01$) 24 h after i.p. injection with Mitomycin C at 1 mg/kg⁻¹ body wt.

In primary spermatocytes at diakinesis metaphase I of meiosis, the percentage of chromosomal aberrations reached 21±0.71 ($p<0.01$) 24 h after treatment with the highest dose of Monocrotophos.

The frequency of sister chromatid exchanges (SCE's) in mouse bone-marrow cells was dose dependent and statistically significant after treatment with all doses of Monocrotophos.

The insecticide significantly increased chromosomal aberrations in mouse spleen, bone marrow and spermatocyte cells. Royal jell decreased the percentage of the induced chromosomal aberrations in somatic and germ cells after treated by gavage.

Key words Genotoxicity, Monocrotophos, Chromosomal aberrations, Mouse.

Monocrotophos possesses abroad insecticidal activity. It is used to control insect pests affecting tomatoes (Chalfant 1973), potato (Bodhade *et al.* 1983), Sweet corn (Janes 1973), Sugar can (Varma *et al.* 1978). Monocrotophos is toxic to insects which attack cotton (Singh *et al.* 1986).

It proved to be toxic to soybean looper larvae Ottens *et al.* 1984), the banana aphid (Suresh and Regupathy 1986), common earth worm (Bharathi and SubbaRao 1986), mites (Radke and Aherkar 1987) and adult beetles (Gupta *et al.* 1990).

Bhunya and Behera (1988) tested the mutagenicity of Monocrotophos in mouse cytogenetic test system. Cytogenetic assays like somatic chromosome aberrations, micronucleus test and sperm shape abnormalities with different doses (5, 2.5 and 1.25 mg/kg) and different exposure times (6, 24, 48 and 120 h) and different routes (i.p., p.o. and SC). Relative sensitivity of cytogenetic assays was found to be as: sperm shape abnormality>chromosome aberrations>micronucleus test. Monocrotophos was found to be mutagenic.

Monocrotophos is genotoxic in the chick *in vivo* test system (Jena and Bhunya 1992) and in both somatic and germ-line cells of Drosophila (Tripathy and Patnaik 1992).

Monocrotophos induced SCE's positively in Chinese hamster ovary (CHO) and rat tracheal epithelial (RTE) cells (Wang *et al.* 1987). Monocrotophos induced a significant increase in mean comet tail-length at all the doses when compared to control in Tilapia mossambica (Saleha *et al.* 2001).

Monocrotophos induced various types of chromosomal aberrations in meristematic cells of capsicum and chlorophyll mutations were recorded in M2 generation (Devadas *et al.* 1987).

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Materials and methods

Animals

Male Swiss mice aged 8:10 weeks and weighted 27 ± 2 gm obtained from a closed random-bred colony at the National Research Centre, were used.

Insecticide

Purified "Monocrotophos" (purity 100%) were kindly supplied by Prof. S.M.A.D. Zayed Department of Organic Chemistry, National Research Centre. The protective Royal Jell was obtained, and the tested concentration 1 mg/kg^{-1} body wt. was dissolved in distilled water.

Doses and treatment

The doses used in the present study were 0.05, 0.1 and $0.2 \text{ mg Monocrotophos kg}^{-1}$ body wt. dissolved in (0.1 ml corn oil) was administered by gavage. The mice were killed 6, 24 and 48 h after treatment. Non-treated mice and the mice with the vehicle were used as controls, another group was intraperitoneal injected with $1 \text{ mg "Mitomycin C"}$ and used as positive control.

Mice were injected with colchicine 2–3 h prior to killing. The spleen was washed with RPMI medium and cut into small pieces. The cells were aspirated into a centrifuge tube and 0.07 M KCl was added. The cells were centrifuged at 1000 rpm for 10 min and fixed in methanol-acetic acid (3:1). After fixation, the cell suspension was dropped onto clean slides, that were stained with Giemsa in phosphate buffer pH 6.8 (Amer *et al.* 1993), 100 well spreaded metaphases were analyzed for each animal.

The testes were removed and meiotic chromosomal aberrations were observed according to the air-drying technique of Evans *et al.* (1964). Slides were coded and 100 well spread metaphases were analyzed for each animal, scoring different types of aberrations.

The method of SCE's was described by McFee *et al.* (1983). Bone marrow cells were fixed and stained with fluorescence plus Giemsa method of Perry and Wolff (1974). Five animals were taken for each treatment and control, 40 well spread metaphases per animal were examined.

The significance of the incidence of abnormalities in experimental versus control data was calculated using the *t*-test.

Results

Table 1 presents a detailed study of the effect of insecticide Monocrotophos on the induction of chromosomal aberrations in mouse spleen cells. The percentage of the induced aberrations increased by increasing the dose of Monocrotophos. It reached 17.8 ± 0.86 and 24.8 ± 0.73 ($p < 0.01$) 24 h after oral treatment by gavage with 0.05 and $0.2 \text{ mg/kg body wt.}$ respectively. The mean percentage of metaphases with chromosome aberrations was found to be statistically significant after excluding gaps. It reached 16.0 ± 0.71 ($p < 0.01$) 24 h with 0.1 mg/kg^{-1} body wt.

Monocrotophos induced both structure and numerical chromosomal aberrations which represented by gaps, fragment and/or breaks, deletions and Robertsonian translocations. Some metaphases with more than one type of aberrations were appeared (Fig. 1a).

Table 2 represented detailed study of the effect of the insecticide on the induction of chromosomal aberrations in the metaphase I of mice spermatocytes. The percentage of the induced chromosome aberrations was highly significant and dose dependent, it reached its maximum 24 h after treatment with the different concentrations of Monocrotophos. Its highest value was 13.6 ± 0.81 , 17.2 ± 1.16 and 21.0 ± 0.71 ($p < 0.01$) 24 h after treatment with 0.05, 0.1 and 0.2 mg/kg^{-1} body wt. of Monocrotophos respectively compared with 23.0 ± 0.71 ($p < 0.01$) 24 h after i.p. injection with Mitomycin C.

Table 1. Number and percentage of the different types of aberrations in mouse spleen cells after oral treatment by gavage with different doses of Monocrotophos

Treatment	Mice killed after (h)	Chromosomal aberrations No.	% of metaphases with:—					% of chromosomal aberrations			
			Gaps	Fragment and/or Break	Gaps+ Frag.	Deletions	Del.+ Frag.	Robertsonian translocation	Polyploidy	Including gaps	Excluding gaps
I. Control (non treated)		22	1.2	1.8	0.4	—	0.4	—	0.6	4.4±0.51	3.2±0.58
II. Corn oil (0.1 ml)	6	22	1.0	2.6	0.4	—	—	—	0.4	4.4±0.75	3.4±0.75
	24	21	1.8	2.0	0.2	—	—	—	0.2	4.2±0.86	2.4±0.51
	48	18	1.2	1.0	0.4	0.8	0.2	—	—	3.6±0.81	2.4±0.75
III. Positive control 1 mg Mitomycin C kg ⁻¹ body wt.	24	131	7.0	8.2	2.2	4.0	1.8	1.4	1.6	26.2±1.02**	19.2±0.86**
IV. Monocrotophos:	48	108	6.8	6.2	1.8	3.2	1.4	1.0	1.2	21.6±1.08**	14.8±0.58**
a) 0.05 mg kg ⁻¹ body wt.	6	75	3.6	4.2	1.6	2.4	1.2	0.8	1.2	15.0±0.71**	11.4±0.68**
	24	89	4.6	3.8	2.2	3.0	1.4	1.2	1.6	17.8±0.86**	13.2±0.86**
	48	57	3.2	2.4	1.4	2.0	0.6	0.8	1.0	11.4±0.81**	8.2±1.16**
b) 0.1 mg kg ⁻¹ body wt.	6	92	5	4.4	2.0	2.4	1.6	1.2	1.8	18.4±0.93**	13.4±0.93**
	24	110	6	5.2	2.2	3.0	1.8	1.6	2.2	22.0±0.71**	16.0±0.71**
	48	77	3	3.4	2.4	2.8	1.2	1.2	1.4	15.4±0.93**	12.4±1.03**
c) 0.2 mg kg ⁻¹ body wt.	6	115	6.0	6.6	2.6	3.0	1.6	1.4	1.8	23.0±0.71**	17.0±0.71**
	24	124	6.4	7.0	2.8	3.6	1.4	1.6	2.0	24.8±0.73**	18.4±1.08**
	48	92	5.4	4.0	2.0	2.6	1.2	1.2	2.0	18.4±0.68**	13.0±0.71**
V. Royal Jell 1 mg/kg ⁻¹ body wt.	24	17	1.4	1.0	0.4	0.2	0.4	—	—	3.4±0.32	2.0±0.40
VI. Protection Royal Jell 1 mg/kg ⁻¹ body wt. added with 0.2 mg monocrotophos kg ⁻¹ body wt.	6	96	5.2	6.0	2.4	2.0	1.4	1.0	1.2	19.2±1.07**	14.0±0.71**
	24	105	5.8	6.2	2.0	2.2	1.0	1.2	1.6	20.0±0.71**	14.2±0.66**
	48	67	4.0	4.0	1.0	1.2	1.0	1.0	1.2	13.4±0.68**	9.4±0.93**

* Significant at 0.05 level. ** Significant at 0.01 level (t-test).

Monocrotophos induced abnormal chromosome associations including autosomal, X-y univalent, fragments and/or breaks and chain IV (Fig. 1b, c).

Monocrotophos induced a significant and dose dependent increase in SCE's frequency in mouse bone-marrow cells. The frequency of SCE's/cell was found to be 14.62 ± 0.93 ($p < 0.01$) after oral treatment by gavage with the highest dose (0.2 mg/kg^{-1} body wt.) compared with 3.71 ± 0.51 and 16.38 ± 1.28 for non treated and mitomycin C respectively (Table 3, and Fig. 1d).

It was observed that the percentage of abnormalities in spleen, spermatocyte and bone marrow cells in mice treated with Monocrotophos decreased after post-treatment with the protective Royal Jell (Tables 1, 2, 3).

Discussion

The *in vivo* chromosome aberrations assay is one of the important bioassays for monitoring the genotoxicity of environmental chemical (AdHoc Committee 1972, Dulout *et al.* 1983) and it may be more useful in predicting possible health risks. For this reasons, the *in vivo* test was used to study the genotoxic effect of the organophosphorous insecticide Monocrotophos.

Fig. 1. Metaphases from mouse treated with different doses of "Monocrotophos" showing: a) chromatid gap in mouse spleen cells. b) A diakinesis-metaphase I cell with X-y & autosomal univalent and c) break in mouse spermatocyte. d) Metaphase from mouse bone-marrow cells showing SCE's induced after treatment with "Monocrotophos" insecticide.

Table 2. Number and percentage of the different types of metaphases with chromosomal aberrations in mouse spermatocyte cells after oral treatment by gavage with different doses of Monocrotophos

Treatment	Mice killed after (h)	Number of abnormal metaphases	% of metaphases with:					Mean % of abnormal metaphase $\pm$ S.E.
			X-y univalent	Autosomal univalent	X-y+ A.U.	Fragment and/or break	Chain IV	
I. Control (non treated)		20	2.4	1.6	—	—	—	4.0 ± 0.71
II. Corn oil (0.1 ml)	6	21	1.6	2.0	0.6	—	—	4.2 ± 0.80
	24	20	2.0	1.4	0.4	0.2	—	4.0 ± 0.66
	48	17	1.8	1.2	0.4	—	—	3.4 ± 0.51
III. Positive control 1 mg	24	115	10.2	8.8	1.4	0.8	1.8	$23.0 \pm 0.71^{**}$
Mitomycin C kg^{-1} body wt.	48	86	8.6	6.0	1.0	0.4	1.2	$17.2 \pm 1.07^{**}$
IV. Monocrotophos:								
a) 0.05 mg kg^{-1} body wt.	6	60	6.0	5.2	0.2	0.2	0.4	$12.0 \pm 0.71^{**}$
	24	68	5.8	6.2	1.0	0.4	0.2	$13.6 \pm 0.81^{**}$
	48	46	4.4	4.0	0.6	0.2	—	$9.2 \pm 0.86^{**}$
b) 0.1 mg kg^{-1} body wt.	6	73	6.8	6.2	0.8	0.6	0.2	$14.6 \pm 0.75^{**}$
	24	86	7.4	5.0	1.0	0.4	0.4	$17.2 \pm 1.16^{**}$
	48	55	5.4	4.8	0.6	—	0.2	$11.0 \pm 0.71^{**}$
c) 0.2 mg kg^{-1} body wt.	6	92	8.0	7.0	1.4	1.0	1.0	$18.4 \pm 0.75^{**}$
	24	105	8.6	6.8	2.0	1.6	2.0	$21.0 \pm 0.71^{**}$
	48	61	5.4	5.0	0.8	0.4	0.6	$12.2 \pm 0.86^{**}$
V. Royal Jell 1 mg/kg^{-1} body wt.	24	16	2.0	1.2	—	—	—	3.2 ± 0.37
VI. Protection Royal Jell 1 mg/kg^{-1} body wt. added with 0.2 mg monocrotophos kg^{-1} body wt.	6	65	5.0	6.0	1.0	0.6	0.4	$13.0 \pm 0.71^{**}$
	24	87	6.2	7.4	2.0	0.8	1.0	$17.4 \pm 0.93^{**}$
	48	44	3.2	4.0	1.0	0.6	—	$8.8 \pm 0.58^{**}$

* Significant at 0.05 level. ** Significant at 0.01 level (*t*-test).

Table 3. Frequency of sister chromatid exchange (SCE's) in mouse bone-marrow cells induced after oral treatment by gavage with different doses of Monocrotophos

Treatment and doses	Mice No.	Metaphase No.	SEC's No.	SCE's/cell Mean $\pm$ S.E.
I. Control (non treated)	5	200	742	3.71 $\pm$ 0.51
II. Corn oil (0.1 ml)	5	200	826	4.13 $\pm$ 0.75
III. Positive control 1 mg Mitomycin C kg ⁻¹ body wt.	5	200	3277	16.38 $\pm$ 1.28**
IV. Monocrotophos:				
a) 0.05 mg kg ⁻¹ body wt.	5	200	1873	9.36 $\pm$ 0.26**
b) 0.1 mg kg ⁻¹ body wt.	5	200	2215	11.07 $\pm$ 0.71**
c) 0.2 mg kg ⁻¹ body wt.	5	200	2923	14.62 $\pm$ 0.93**
V. Royal Jell 1 mg/kg ⁻¹ body wt.	5	200	702	3.51 $\pm$ 0.37
VI. Protection Royal Jell 1 mg/kg ⁻¹ body wt. added with 0.2 mg monocrotophos kg ⁻¹ body wt.	5	200	2010	10.05 $\pm$ 0.66**

* Significant at 0.05 level. ** Significant at 0.01 level (*t*-test).

Cytogenetic studies were performed on spleen, spermatocytes and bone marrow of the mouse, using oral treatment by gavage with the doses 0.05, 0.1 and 0.2 mg Monocrotophos kg⁻¹ body wt. The result indicated that the insecticide induced a high percentage of chromosome aberrations after all times of the treatment.

The percentage of the induced aberrations was dose dependent and it reached its maximum 24 h after the treatment with the different doses in somatic and germ cells. Monocrotophos induced a high percentage of chromosome aberrations in mouse spleen cell cultures and mouse bone-marrow (Ibrahim 1993). The same results obtained on Dicamba herbicide (Aly 1993), Dursban and Dichlorovos (Amer *et al.* 1998), Sevin (Amer *et al.* 2000b).

Bhunya and Jena (1993) observed that a significant induction of chromosome aberrations only after 24 h exposure with the highest dose (5 mg/kg⁻¹) of Monocrotophos in the chick *in vivo* system test.

Richardson *et al.* (1984) reported that, when assessing the data derived from *in vitro* cytogenetic assays, it is usual to regard gaps as a separate category of damage and accord all other types of damage equivalent weights.

The percentage of chromosome aberrations induced by Monocrotophos was found to be highly significant in mouse spleen cells after excluding number of metaphases with gaps. It reached 13.2 $\pm$ 0.86, 16.0 $\pm$ 0.71 and 18.4 $\pm$ 1.08 ($p<0.01$) 24 h after injection with 0.05, 0.1 and 0.2 mg Monocrotophos kg⁻¹ body wt. compared with 19.2 $\pm$ 0.86 ($p<0.01$) 24 h after intra-peritoneal injection with 1 mg Mitomycin C kg⁻¹ body wt. The same results were obtained on Trifluralin herbicide (Ibrahim 1998). Numerical aberrations show a low percentage in the form tetraploidy.

Also, chromosome aberrations were observed in the primary spermatocytes of mice treated with insecticide. The most common type of aberrations was the presence of univalents. The X-y univalents were more often sperated than autosomes. The phenomenon was observed also by Imai *et al.* (1981) and Hu and Zhu (1990). Reciprocal translocations were present in the form of chain IV in a low frequency. This indicates that Monocrotophos induced chromosome damage in germ cells.

The types of chromosome aberrations induced with Monocrotophos were also observed in primary spermatocytes of mice treated with other pesticides having different chemical structures, for example the organophosphorus insecticide pirimiphos-methyl (Aly and Fahmy 1995), the pyrethroid insecticide fenvalerate (Amer *et al.* 2003a); in mice fed faba beans treated with the organophosphorous insecticide DDVP (Amer *et al.* 2000a); in mice fed stored wheat grains treated with the organophosphorous insecticide malathion (Amer *et al.* 2002) and the fungicide benomyl

(Amer *et al.* 2003b).

Many carcinogens and/or mutagens are known to induces SCE's (Abe and Sasaki 1982, Fahmy 1999). The induced increase in SCE's frequency after treatment the bone-marrow cells with different doses of Monocrotophos was significant and dose-dependent, and the frequency of SCE's/cell reached 14.62 ± 0.93 after oral treatment with high dose of the insecticide.

The present results show that oral administration of Royal Jell 1 mg/kg^{-1} body wt. to mice treated with the highest dose of Monocrotophos 0.2 mg/kg^{-1} body wt. minimized the percentage of the induced chromosome aberrations and SCE's, the antimutagenic properties of Royal Jell have been demonstrated *in vivo*.

The positive results obtained from this study indicate that Monocrotophos is genotoxic in mouse somatic and germ cells. Also, the ability of Royal Jell to reduce the cytogenetic damage induced by the organophosphorous insecticide.

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