Endrin Intoxication in Normal and Irradiated Rats
II. Lipid, Phospholipid and Cholesterol Content

Meena Kaushal and P. K. Gupta*
Department of Biophysics
Punjab University
Chandigarh, India
Correspondence address
Dr. P. K. Gupta, Scientist
Industrial Toxicology Research
Centre, Lucknow, INDIA

The persistence of chlorinated hydrocarbon insecticides like DDT, dieldrin, endrin etc., has aroused considerable interest on the mammalian biochemical functions. The toxicological significance of additional changes, if any, of various biochemical parameters, while often suspected, has not been established. Dieldrin and DDT have been shown to induce gluconeogenesis, increased plasma free fatty acids and enlargement of liver in DDT, dieldrin and endosulfan treated animals (SCHWABLE, 1964; BHATIA et al., 1972, GUPTA, 1974, and GUPTA, 1976). Our recent studies report glycogen pattern of rat tissues after endrin (GUPTA and KAUSHAL, 1976). This investigation is a continuation of our previous experiments and focuses on the effects of endrin on lipids, phospholipids and cholesterol content of normal and irradiated rats.

MATERIALS AND METHODS

One hundred twenty female albino rats weighing between 150-200 g were divided into four equal groups. Rats were fed on commercial diet ad libitum except that before sacrifice they were fasted for 12 hr in such a way that the same 12 hr-fasting period was used for all the animals.

The first group served as controls and received propylene glycol, the second group received endrin 10 mg/kg in propylene glycol intraperitoneally. Rats in the third group served as irradiated controls while rats in the 4th group received 10 mg/kg endrin half an hr after irradiation. For irradiation the animals were exposed at the abdominal region to a single dose of gamma rays emitted from Co^{60} source at the rate of 150 r/min for 6 min., total dose being 900 rads. Six animals from each group were sacrificed and tissue samples were collected at predetermined time intervals.

Total lipid content in various samples was estimated by the method of FOLCH et al. (1957), phospholipids by the method of SCHNEIDER (1945) and cholesterol by the method of ZLATKIS et al. (1953).

* Present address: Scientist-in-charge
Pharmacology & Toxicology
(referred Chemicals)
Industrial Toxicology Reserach Centre,
P.O. Box No. 80, Lucknow, India

Bulletin of Environmental Contamination & Toxicology,
Vol. 18, No. 3 © 1977 by Springer-Verlag New York Inc.

Table 1. Total Lipid Content (μg/g) of 12 hr-fasted Rats.

Hr		Control	Endrin (10 mg/kg ip)	P	Radiation (900 R)	P	Radiation (900R) + Endrin (10 mg/kg ip)	P
2	Liver	9.67±0.27	8.25±0.45	NS	8.70±0.20	NS	9.96±0.14	NS
	Kidney	7.19±0.21	7.15±0.16	NS	7.25±0.41	NS	8.28±0.20	NS
6	Liver	8.50±0.46	7.50±0.76	NS	4.04±0.27	≤0.001	3.75±0.15	≤0.001
	Kidney	6.46±0.18	7.52±0.31	NS	6.03±0.21	≤0.05	4.75±0.16	≤0.05
12	Liver	8.04±0.17	5.61±0.41	≤0.001	4.27±0.14	≤0.001	2.60±0.12	≤0.001
	Kidney	6.90±0.74	7.10±0.14	NS	6.17±0.39	NS	3.06±0.21	≤0.001
24	Liver	8.75±0.13	3.97±0.14	≤0.001	6.52±0.21	≤0.01	1.85±0.09	≤0.001
	Kidney	6.87±0.16	6.10±0.41	NS	6.20±0.18	≤0.05	4.00±1.17	≤0.001
48	Liver	10.00±0.21	6.70±0.19	≤0.001	4.14±0.16	≤0.001	4.25±0.31	≤0.001
	Kidney	6.52±0.20	4.02±0.23	≤0.001	6.64±0.47	NS	6.10±0.21	NS

± S.E. of 4 to 6 animals at each time interval.

Table 2. Phospholipids (mg/g) of 12 hr-fasted rats.

Hr		Control	Endrin (10 mg/kg ip)	P	Radiation (900 R)	P	Radiation (900R) + Endrin (10 mg/kg ip)	P
2	Liver	1.90±0.14	1.40±0.16	≤0.51	1.03±0.22	≤0.01	0.86±0.12	≤0.001
	Kidney	1.21±0.21	0.84±0.16	≤0.01	0.81±0.21	≤0.01	0.41±0.42	≤0.001
6	Liver	2.42±0.16	0.76±0.09	≤0.001	0.92±0.12	≤0.001	0.71±0.14	≤0.001
	Kidney	1.09±0.16	0.67±0.29	≤0.001	0.69±0.22	≤0.001	0.37±0.21	≤0.001
12	Liver	2.00±0.22	0.49±0.16	≤0.001	0.67±0.29	≤0.001	0.52±0.10	≤0.001
	Kidney	1.00±0.11	0.41±0.14	≤0.001	0.47±0.17	≤0.001	0.28±-.21	≤0.001
48	Liver	1.94±0.23	0.46±0.20	≤0.001	0.25±0.17	≤0.001	0.09±0.14	≤0.001
	Kidney	1.19±0.14	0.57±0.12	≤0.001	0.38±0.10	≤0.001	0.16±0.12	≤0.001

± S.E. of 4 to 6 animals at each time interval.

Table 3. Cholesterol Content mg/kg tissues wieght of 12 hr-fasted Rats.

Hr		Control	Endrin (10mg/kg ip)	P	Radiation (900 R)	P	Radiation (900R) + Endrin (10 mg/kg ip)	P
2	Liver	8.67±0.19	7.64±0.74	NS	5.73±0.15	≤0.001	6.18±0.18	≤0.001
	Kidney	8.37±0.12	8.43±0.10	NS	5.59±0.12	≤0.001	4.85±0.19	≤0.001
6	Liver	7.51±0.19	8.56±0.28	≤0.05	5.23±0.10	≤0.001	11.55±0.21	≤0.001
	Kidney	6.26±0.46	6.46±0.10	NS	6.05±0.18	NS	6.18±0.21	NS
24	Liver	8.87±0.12	8.92±0.18	NS	5.32±0.10	≤0.001	4.12±0.22	≤0.001
	Kidney	7.76±0.23	8.90±0.15	NS	5.00±0.21	≤0.001	6.19±0.19	≤0.01
48	Liver	7.37±0.48	7.71±0.14	NS	5.46±0.10	≤0.001	6.40±0.11	≤0.01
	Kidney	7.61±0.11	8.40±0.24	NS	4.97±0.29	≤0.001	3.50±0.15	≤0.001

± S.E. of 4 to 6 animals at each time interval.

RESULT AND DISCUSSION

The total lipids of liver and kidney of normal and irradiated rats given endrin are summarized in Table 1. It is evident that endrin alone resulted in significant inhibition of total lipid content of liver during 12 to 48 hr of treatment. Maximum decrease was observed at 24 hr of treatment. In contrast, no significant change in total lipid content of kidney was observed during 24 hr of treatment except at 48 hr when significant decrease was observed.

Radiation alone caused significant decrease in total lipid content of liver during 6 to 48 hr of treatment, whereas no such change was observed in kidney. In irradiated rats given endrin the decrease in total lipids of liver and kidney was more marked compared to individual treatments. Decrease in total lipids observed in this study supports Nelson and TAN (1967) and KUZ (1968) who observed a similar decrease in total lipids. Such a decrease in the total lipids could be caused by a reduction in the fatty acid synthesizing enzymes of liver and kidney (BHATIA and VENKITASUBRAMANIAN, 1972). However, the possibility of other factors can not be ruled out. Furthermore, in irradiated rats given endrin decrease was earlier and more marked and thus radiation appears to add to and enhance this effect.

Mean values of phospholipid content of liver and kidney of rats subjected to different treatments are shown in Table 2. Endrin at a dose levels of 10 mg/kg produced significant decrease in phospholipids of liver and kidney during 2 to 48 hr of treatment. An almost similar trend was observed in rats exposed to radiation alone. In irradiated rats given endrin, the inhibition was more marked as compared to individual treatments. Inhibition of synthesis of phospholipids due to other related insecticides such as DDT has also been reported (KUZ, 1968) and has been attributed to distruption of membranes because of toxic effects of these chemicals. Since the decrease in phospholipid content of irradiated rats given endrin was more marked as compared to endrin alone, it seems probable that radiation sensitizes the permeability of mitochondia and thus interferes with the tri-carboxylic acid cycle, thereby causing more oxidation of fat phospholipids in thes organs.

The cholesterol content of liver and kidney of normal and irradiated rats given endrin is given in Table 3. Rats given endrin alone did not show any significant change in cholesterol content of liver and kidney whereas radiation alone resulted in significant decrease in cholesterol content of both the tissues throughout the entire period of study. In contrast, in irradiated rats given endrin, the cholesterol content of liver was significantly increased except at 2 hr when a decrease in cholesterol content was observed. The present findings are in agreement with KOHLI et al. (1975). who also failed to observe any change

in cholesterol content of liver of rats treated with dieldrin. This effect could be attributed to the hypertrophy of liver cells. Since we have also observed decreased amount of total lipid content, this further supports our findings. The increase in cholesterol content of liver and kidney of irradiated rats given endrin as observed during this investigation is very interesting and needs further investigation.

From this study it is concluded that exposure of rats to single dose of endrin or radiation leads to inhibition of total lipids and phospholipids with no change in cholesterol. Administration of endrin to irradiated rats added further to the effects of endrin and the changes were earlier and more marked in irradiated rats given endrin alone. This mechanism needs further investigation.

REFERENCES

BHATIA, S.C.; SHARMA, S.C., and VENKITASUBRAMANIAN, T.A.: Arch. Environ. Hlth. 14, 369 (1972).

BHATIA, S.C.; SHARMA, S.C., and VENKITASUBRAMANIAN, T.A.: Toxicol. Appl. Pharmacol. 24, 216 (1973).

FOLCH, J., SCHAFFER, FL.L., and KRIKS, P.K.: Arch. Biochem. and Biophys. 45, 319 (1953).

GUPTA, P.K: Acta, Pharmacol. et. Toxicol. 35, 191 (1974).

GUPTA, P.K.: J. Pharmacol. (1976/77) In Press.

KAUSHAL, M., and GUPTA, P.K., Chemosphere (1976/77) In Press.

KOHLI, K.K., BHATIA, S.C. and VENKITASUBRAMANIAN, T.A.; Environ. Physiol. Biochem. 5, 119 (1975).

KUZ, M.U.A.: Pharmacol. Toxicol. 5, 609 (1968).

NELSON, P., and TAN, W.C.: Biochem. Pharmacol. 16, 1813 (1967).

SCHEIDER, W.C.J.: Biol. Chem. 161, 203 (1945).

SCHWABLE, U.: Arch. Exp. Pathol. Pharmacol. 249, 195 (1964).

ZLATKIS, A., ZAK, B., and VOYLE, A.T.: J. Lab. Clin. Med. 41, 486 (1953).