

## Effect of some Insecticides on Fat Metabolism and Blood Enzymes in Rats

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FIVE experiments were carried out on 187 male albino rats (Sprague Dawley Strain) to test the effect of acute toxicity of insecticides on: liver and plasma lipids and some blood-enzymes. Rats of treated groups received one or repeated half lethal doses of the following insecticides : DDT, Endrine, Dursban, Cytrolane and Lannate. 24hr after the administration of the last dose of insecticides animals were killed and liver and blood samples were collected for the determinations of : liver and serum lipid fractions, blood transaminases (GOT and GPT), alkaline phosphatase and cholinesterase activities.

The main results obtained are:

1. Significant increase in liver weight as percentage of body weight only in rats treated with DDT.
2. DDT, Endrin or Dursban administration produced significant rise in liver total lipids and triglyceride. The effect of Cytrolane was only on cholesterol fraction of the liver where was decreased. Lannate produced increase in liver total lipids due to the rise of cholesterol and triglyceride fractions.
3. DDT administration Lowered P/C ratio in serum due to the decrease in cholesterol fraction. Dursban poisoning resulted in significant increase in serum triglycerides and free fatty acids associated with the production of fatty liver. Cytrol and Lannate increased only serum phospholipid fraction.
4. All studied insecticides (except Endrin) produced significant increase in GOT and GPT activities. Endrin effect was only on GPT activity.
5. Alxaline phosphatase activity increased only in case of Dursban Lannate intoxication.
6. Cholinesterase activity significantly lowered only with Dursban, Cytrolane and Lannate poisoning.

Chlorinated-hydrocarbon, organophosphate and carbamate compounds are known to have potent effects upon the nervous system of a wide variety of organisms from primitive invertebrates to man. This action had been used to considerable advantage in pest control where these compounds constitute the major classes of insecticides.

Organophosphates are desirable for field application because they break down rapidly in the environment and do not persist in animal tissues (Stickel, 1974). Nevertheless, certain organophosphates are extremely toxic and have caused spread mortality among exposed laboratory and farm animals (Mills, 1973; Mehany, 1974; Stickel, 1974; Mendelssohn, 1977 and Zinkl *et al.*, 1978).

Animals poisoned with different compounds of insecticides showed inhibition of cholinesterases and alterations in some other hydrolysis which play an important role in lipid metabolism (Onikienko, 1963 ; Sam kacew and Radhay, 1973 and Peters *et al.*, 1973). These changes produced reduction in the capacity of the liver to form and secrete lipoproteins or to oxidise fatty acids thus causing accumulation of fat in the liver (Fiegelson *et al.*, 1961 ; Cecil *et al.* ; 1977). Depression in blood and brain cholinesterase activity was also accompanied by an increase in the activity of blood transaminases (Roe, 1969 and Uppal and Ahmad, 1977). Changes in the activity of blood transaminases and alkaline phosphatase associated with degenerative changes in the liver was revealed in animals poisoned with different compounds of insecticides (Hansell and Ecobichon , 1974 and Barros *et al.*, 1978).

The aim of the present work is to study the hepatic physiological response of rats to some insecticides. The results of the study may help in understanding the toxic effect of these substances to farm animals. The tested insecticides are : DDT and Endrin (organochlorines), Dursban and cytolane (organophosphorous) and Lannate (Carbamate).

### Material and Methods

This work was carried out in the Medical Physiology Laboratory, National Research Centre, Egypt, total of 187 male albino rats (Sprague Dawley Strain) bred in the animal house of the centre were used in this study.

Five experiments were conducted to study the effect of some organochlorine (DDT & Endrin), Organophospho (Dursban & Cytrolane) and carbamate (Lannate) insecticides on: fat metabolism (Changes in liver weight and in liver and serum lipids) and on blood serum enzymes. Rats of each experiment were divided into control and treated groups. Treated rats received one or repeated half lethal doses of one of the following insecticides as indicated in the Table 1

#### *Collection of samples and methods used for biochemical analysis*

##### *1. Blood analysis*

Rats were decapitated at 24 hr after the administration of the last dose of insecticide. About 10 ml of blood were collected, and left for 30 min under room temperature then centrifuged at 2000 r.p.m. for 10 min for separation of serum.

TABLE 1

Experiment	Treatment +	No. of animals	Dose mg/kg Body weight	Type of Administration
1	DDT	26	150	Oral, repeated (3 succ. days)
2	Endrin(19.2%)*	22	15	Oral, repeated (3 succ. days)
3	Dursban (85%)**	26	163	Intraperitoneal inj. 2 succ. days)
4	Cyrolane (25%***)	19	8.9	Oral, Single dose
5	Lannate (90%)	14	41	Oral, repeated (s succ. days)

+ The number of the control animals was, 28, 15, 18, 11 and 8 for the five experiments respectively.

\* Dissolved in corn oil.

\*\* Diluted in water (1 : 20).

\*\*\* Water soluble.

*Fresh serum was immediately used for enzyme determinations of*

1— Blood transaminases (glutamic oxaloacetic and glutamic pyruvic transaminases). The method of Reitman and Frankel (1957) was applied.

2— Cholinesterase activity. Bigg's *et al.* (1958) method was used.

3— Alkaline phosphatase (as described by King and Armstrong, 1943)

*A part of serum was stored at 20° until used for the determination of :*

1. Triglycerides. (The method of Van Handel and Zilversmith (1957) was applied.).
2. Cholesterol (estimated by the method of Morrison, (1964).
3. Phospholipids. (measured as described by Bloor *et al.*, 1922).
4. Free fatty acids (the method of Duncombe, 1963 was used).

*Liver analysis*

Immediately after decapitation, rats were dissected to collect livers. The liver was blotted between two filter papers and weighed.

Specimens from livers, were fixed in 10% formaldehyde saline, frozen sectioned and stained by the method of (Helmy, 1962 and Pearse, 1968) for fat deposition examinations.

The other part of the liver was stored at 20° until used for the determination of:

1. Total lipids (The method of Folch *et al.* 1957 was applied).
2. Triglycerides (as described by Ibrahim, 1981))
3. Phospholipids („ „ „ „ 1981)
4. Cholesterol („ „ „ „ 1981)

Statistical comparisons were done using the Stuent's test.



## Results

### 1. Effect of insecticide administration, on liver weight and liver lipid fractions

As shown in Table 2, rats suffering from DDT poisoning showed hepatomegalic effect as indicated by the significant increase in their liver weight as percentage body weight. Such effect was not observed when Endrin was administered to rats.

Administration of DDT or Endrin produced significant increase in liver total lipids and in triglyceride fractions. This increase was greater in the case of DDT poisoning than that produced by Endrin (Table 2).

Data in Table 1 also reveal the significant increase in total lipids, triglyceride and phospholipid fractions of animal's liver which received Dursban. The effect of Cytrolane was only on the liver cholesterol fraction, which significantly decreased in the livers of treated rats in comparison with the control ones (Table 2).

TABLE 2. Effect of administration of insecticides on liver lipid fractions.  
(mean  $\pm$  SE)

Experiment	Treatment	Liver weight (as % body weight)	Total lipids (gr/100grl)	Triglycerides (mg/gr. l)	Cholesterol (mg/gr l)	Phospholipids (mg/gr l.)
1	Control (10)	3.4 $\pm$ 0.2	4.5 $\pm$ 0.2	5.0 $\pm$ 0.8	$\pm$ 0.12	25 $\pm$ 1.1
	DDT(7)	5.0 $\pm$ 0.2*	7.3 $\pm$ 0.4*	13.0 $\pm$ 1.6*	3.3 $\pm$ 0.17	30 $\pm$ 2.9
2	Control (7)	3.3 $\pm$ 0.1	3.4 $\pm$ 0.1	3.1 $\pm$ 0.2	2.0 $\pm$ 0.18	21 $\pm$ 0.9
	Endrin(7)	3.4 $\pm$ 0.1	4.3 $\pm$ 0.3*	6.2 $\pm$ 1.3*	2.4 $\pm$ 0.15	23 $\pm$ 0.9
3	Control(10)	3.6 $\pm$ 0.2	3.0 $\pm$ 0.3	3.8 $\pm$ 0.4	3.0 $\pm$ 0.22	19 $\pm$ 1.3
	Dursban(8)	3.5 $\pm$ 0.2	7.6 $\pm$ 0.6*	12.3 $\pm$ 3.2*	2.8 $\pm$ 0.07	23 $\pm$ 1.2*
4	Control(6)	3.5 $\pm$ 0.2	4.2 $\pm$ 0.1	4.5 $\pm$ 0.9	3.1 $\pm$ 0.17	24 $\pm$ 0.8
	Cytrolane(12)	3.2 $\pm$ 0.2	5.1 $\pm$ 0.5	4.3 $\pm$ 0.7	2.2 $\pm$ 0.17*	25 $\pm$ 0.7
5	Control(4)	3.9 $\pm$ 0.1	3.1 $\pm$ 0.1	5.15 $\pm$ 1.2	3.2 $\pm$ 0.11	22 $\pm$ 2.5
	Lannate (8)	3.9 $\pm$ 0.3	4.4 $\pm$ 0.1*	5.5 $\pm$ 0.5	4.7 $\pm$ 0.09*	30 $\pm$ 2.2*

\* Significant at P/0.05

Lannate administration produced significant increase in the liver total lipids. This increase is due to the significant rise in cholesterol and phospholipid fractions (Table 2).

### 2. Effect of insecticides on serum lipid fractions

As shown in Table 3, slight but not significant decrease in serum cholesterol, phospholipids fractions and phospholipid/cholesterol(P/C)ratio was observed in animals receiving DDT. Serum free fatty acids tended to increase.

Serum cholesterol of rats treated with Endrin, was the only serum lipid fraction which significantly increased in comparison with the control ones (Table 3). Endrin tended to increase serum phospholipid fraction but without affecting P/C ratio (Table 3).

The changes in lipid fraction of the serum due to Dursban toxication showed highly significant increase, almost to 3 times in the triglyceride and the double in free fatty acids (Table 3).

As indicated in Table 3, serum phospholipid was the only serum fraction which significantly increased due to cytolane toxication.

TABLE 3. Effect administration of insecticides on serum lipid fractions ( mean  $\pm$  SE)

Experiment	Treatment	Triglycerides (mg/100ml)	Cholesterol (mg/100 ml)	Phospholipids(p) (mg/100ml)	Free fatty (umol/100 ml)	P/C ratio
1	Control (10)	55 $\pm$ 4	68 $\pm$ 3	134 $\pm$ 20	85 $\pm$ 4	2.1 $\pm$ 0.4
	DDT (7)	59 $\pm$ 5	62 $\pm$ 3	120 $\pm$ 7	101 $\pm$ 10	1.9 $\pm$ 0.2
2	Control (7)	53 $\pm$ 0.7	112 $\pm$ 7	130 $\pm$ 10	97 $\pm$ 4	1.2 $\pm$ 0.2
	Endrin (7)	53 $\pm$ 101	146 $\pm$ 15*	159 $\pm$ 12	94 $\pm$ 4	1.1 $\pm$ 0.1
3	Control (10)	74 $\pm$ 8	91 $\pm$ 6	153 $\pm$ 11	99 $\pm$ 11	1.8 $\pm$ 0.2
	Durshan 8	209 $\pm$ 38*	96 $\pm$ 7	176 $\pm$ 6	187 $\pm$ 17*	1.9 $\pm$ 0.2
4	Control (c)	53 $\pm$ 3	103 $\pm$ 9	142 $\pm$ 13	96 $\pm$ 10	1.4 $\pm$ 0.1
	Cyrolane (12)	59 $\pm$ 8	108 $\pm$ 4	185 $\pm$ 12*	98 $\pm$ 8	1.7 $\pm$ 0.1
5	Control (4)	38 $\pm$ 5	84 $\pm$ 15	133 $\pm$ 25	125 $\pm$ 26	8.1 $\pm$ 0.5
	Lannate (8)	64 $\pm$ 10*	122 $\pm$ 14	350 $\pm$ 59*	271 $\pm$ 29*	3.1 $\pm$ 0.6

\* Significant at P/0.05

Data in Table 3 also reveal significant increase in serum triglycerides, serum phospholipids and in serum free fatty acids in animals receiving Lannate.

### 3. Effect of insecticides on serum enzymes (GOT, GPT, AL.P. and Ch. E.)

Administration of DDT to rats produced changes in the activity of serum enzymes. Statistical analysis of the obtained results showed significant increase in the activities of both glutamic-oxaloacetic (GOT) and glutamic pyruvic (GPT) transaminases occurred in serum of treated rats over that of control ones. The values for alkaline phosphatase activity in serum of treated rats was within the normal level (Table 4).

Statistical analysis of the results of cholinesterase activity, in terms of international units of acetylcholine hydrolyzed per ml serum per minute, revealed slight decrease in its activity in rats treated with DDT.

In case of Endrin poisoning, the activity of GOT was the only serum enzyme which changed by significant increase.

The activities of serum transaminases of rats treated with Dursban were significantly higher than in control animals. GOT and GPT activities were almost  $1\frac{1}{2}$  and 3 times that of control rats (Table 4). Alkaline phosphatase activity in serum of rats treated with Dursban showed significant rise in its activity in comparison with the control ones (Table 4). Great effect of Dursban on cholinesterase activity was observed. Five rats out of thirteen treated with Dursban had no active cholinesterase. In general, cholinesterase activity in serum of treated animals was highly significant low than in control rats (Table 4).

As in the case of Dursban the second organophosphorous compound Cytrolane produced also significant rise in GOT and GPT activities associated with decrease in treated rats. No changes were found in alkaline phosphatase activity (Table 4).

TABLE 4. Effect of administration of insecticides on serum enzymes (Mean  $\pm$  SE).

Experiment	Treatment	GOT IU/L.	GPT I.U/L.	AL.P. KA.U./100ml	Ch.E. I.U./ml.min
1	Control (5)	79 $\pm$ 2	32 $\pm$ 1	26 $\pm$ 3	2.1 $\pm$ 0.1 (13)
	DDT (5)	144 $\pm$ 11*	76 $\pm$ 5*	37 $\pm$ 10	1.8 $\pm$ 0.2(14)
2	Control (4)	71 $\pm$ 5	39.5 $\pm$ 3	25 $\pm$ 1.7	2.3 $\pm$ 0.2(4)
	Endrin (5)	92 $\pm$ 6*	37.0 $\pm$ 2	25 $\pm$ 5.0	2.0 $\pm$ 0.1 (10)
3	Control (3)	79 $\pm$ 6	33 $\pm$ 2	33 $\pm$ 3	1.8 $\pm$ 0.4 (5)
	Dursban (5)	126 $\pm$ 16*	110 $\pm$ 27*	65 $\pm$ 7*	0.4 $\pm$ 0.1* (13)
4	Control (5)	52 $\pm$ 1	31 $\pm$ 2	18 $\pm$ 1.4	1.7 $\pm$ 0.1
	Cytrolane(7)	82 $\pm$ 5*	57 $\pm$ 4*	15 $\pm$ 1.3	0.3 $\pm$ 0.1*
5	Contso (4)	71 $\pm$ 5	40 $\pm$ 3	25 $\pm$ 2	2.3 $\pm$ 0.2
	Lannate (6)	124 $\pm$ 6*	88 $\pm$ 9*	58 $\pm$ 3*	1.4 $\pm$ 0.2*

GOT : glutamic — oxaloacetic transaminase      A.J.P. :alkaline phosphatase

GPT : pyruvic transaminase                      Ch.E. : cholinesterase.

( )Number of animals.

\* Significant at P 0.05



Similar to the effect of Dursban, the activities of transaminases(GOT and G.PT)and alkaline phosphatase in the serum of rats treated with Lannate were significantly higher than those of the control animals. Lannate poisoning also significantly lowered the activity of serum cholinesterase but to less degree than in case of Dursban treatment (Table 3).

### Discussion

A common concept is that chlorinated hydrocarbon pesticides are transported in blood dissolved in the plasma triglycerides (Hayes *et al.*, 1958). Also binding of these pesticides to B-lipoprotein has been suggested by Luckens (1969). Their lipid affinity is further suggested by the fact that chlorinated pesticides accumulate in adipose tissue (Hayes *et al.*, 1958).

Mitchell *et al.* (1977) found that the absolute concentrations of DDT on a total lipid basis varied from tissue to tissue, the values for liver were the highest.

In the present study, increase in liver weight as percentage of body weight and in liver total lipids of animals treated with DDT was observed. These results are in accordance with the findings of Cecil *et al.* (1974). They found that DDT or polychlorinated biphenyl (Archor, 1242) fed torats or japanese quail produced an increase in liver weight and liver lipids.

Under the present experimental conditions DDT decreased the P/C ratio in the serum associated with an increase in liver triglycerides. Duff *et al.* (1950 and 1951) and Moore and Williams (1964) suggested that lowering of the P/C ratio in the plasma parallels the degree of atherosclerosis. Kingsbary *et al.* (1969) reported that the main blood lipid changes which play a role in the production of atherosclerosis are increased cholesterol and triglycerides. On the other hand Greer and Kail (1974) found that DDT and polychlorinated biphenyl are statistically associated with cardiovascular disease risk factor. Soit could be suggested that DDT poisoning lead to cardial failure by inducing advanced degree of atherosclerosis.

It is clear from the present study, that in acute toxicity with Dursban fatty liver occurred (Fig. 1,2). The rise in hepatic total lipids was due to the increase in both triglyceride and phospholipid fractions.This was associated with an increase in serum triglyceride and FFA. So it could be suggested that acute dursban toxicity induced fatty liver by increased mobilization of FFA from adipose tissue. Carlson *et al.* (9163) found that 24 hr after surgical trauma in the dog the liver glycerides increased, this increase was directly proportional to the increase of FFA . The accumulation of fat in the liver under many diverse experimental conditions has frequently been used as an index of fat mobilization from adipose tissue (Barrett *et al.*, 1938 and Luis and Farber, 1952). It has become clear that the free fatty acids (FFA) of the plasma represent a major transport for mobilization of lipid from adipose tissue (Gordon, 1957 and Fredrickson and Gordon, 1958).

In case of Lannate poisoning, no fatty liver occurred (Fig. 3), however the total lipids increased. The rise was in phospholipid and in cholesterol fractions rather than in triglycerides. This confirms the suggestion that production of fatty liver is mainly due to the increase in triglyceride fraction.

The data obtained by Murphy (1966) demonstrated that acute poisoning of rats by several organophosphorous insecticides increased the activity of hepatic alkalinephosphatase and tryrosine-transaminase activity. Menrath (1973) found that oral administration of organophosphorous insecticide caused liver damage in dogs, indirect evidence of liver damage was provided by an increase in serum enzymes. The results of the present investigation indicate that the activity of (GOT) and (GPT) increased significantly due to DDT poisoning. Natsyuk *et al.* (1974) also found that oral administration of dichloroethane to rabbits caused an increase in serum transaminases. Endrin administration, in the present study, produced significant increase only in GOT activity. Klevay (1971) reported that radiolabeled Endrin added to perfusates appeared in bile of livers of male donors 2-21 times as rapidly as in bile of livers of female donors. These findings explain the lesser toxicity of Endrin in male rats found in the present work, which is probably due to lesser tissue storage in males.

The results obtained in the present study concerning Dursban and Cyrotolane intoxication, are in accordance with the results of Wright *et al.* (1966). These authors found that the cattle poisoned by oral administration of organophosphorous insecticide had a serum of highly GOT and GPT activities. This increase was correlated well with the depression of cholinesterase activity.

It is clear that cholinesterase was inhibited by Dursban and Cyrotolane, which are organophosphorous and not by DDT, which is an organochlorine. This indicates that organophosphate pesticides are toxic because they inhibit acetylcholinesterase in the blood and in the nervous system and death occurs from paralysis (Ludke- *et al.*, 1975).

The carbamate insecticide Lannate, markedly increased the activity of serum GOT, GPT and alkaline-phosphatase. While the activity of serum cholinesterase was decreased. Sakai and Matsumura (1968) reported that organophosphate and carbamate insecticides owe their toxicity to effect upon the nervous system through their inhibitory properties against cholinesterases.

Carbamate insecticides are biologically active because of their structural complementarity to the active site of acetylcholinesterase (ACHE) and their consequent action as substrates with very low turnover. Carbamates behave as synthetic neurohormones that produce their toxic action by interrupting the normal action of (ACHE) so that acetylcholine accumulates at synaptic functions.



EFFECT OF SOME INSECTICIDES . .

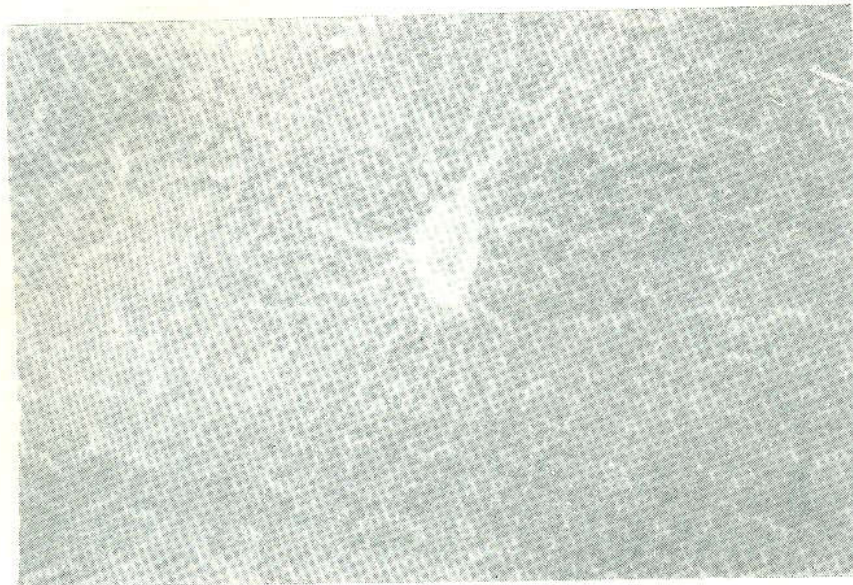


Fig 1. Microphotograph showing liver cells of control rat.

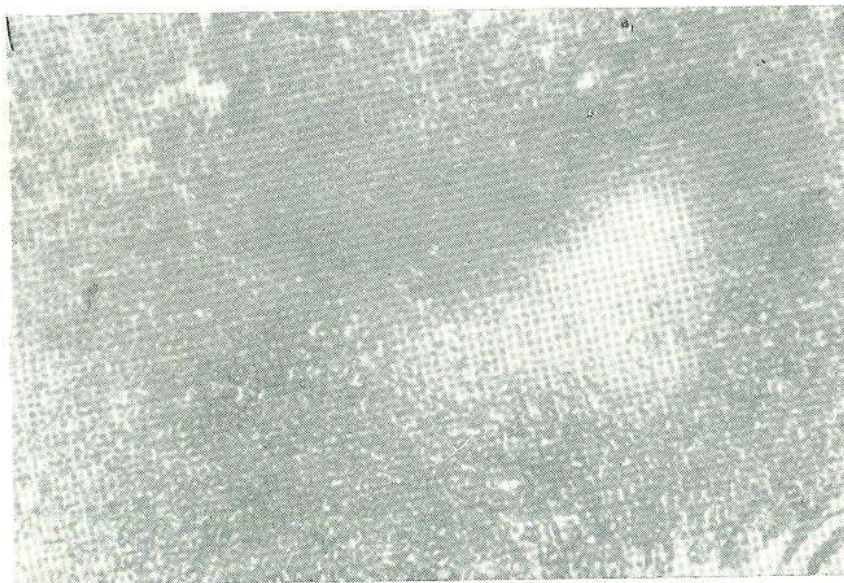


Fig 2. Microphotograph showing necrosis of midzonal type and fatty change at the periphery of the liver of rat treated with Dursban.



Fig. 3. Microphot graph showing cellular necrosis, but without fatty changes, of the liver of rats treated with Lannate.



## References

- Barrett, H.M., Best, C.H.; and Ridout, J.H. (1938)A Study of the source of liver fat using deuterium as an indicator . *J. Physiol. (Lond.)* 93, 367.
- Barros, S.; Dem, B. and Saliba, A.M. (1978) Toxicity of the hexachlorocyclohexane in rats. *Toxicology*, 10, 271.
- Biggs, H.G., Shirleg, B.S. Asep, M.T. and Morrison, D.B. (1958) "A simple colorimetric method for measuring activities of cellular and plasma cholinesterase, *Amer. J. Clin. Path.* 30, 181.
- Bloor, W.R., Pelkan, K.F. and Allen, D.M. (1922) Determination of fatty acids and cholesterol in small amounts of blood plasma. *J. Biol. Chem.* III (1), 191.
- Bruce, J., H., Piekarski, J. and Nilsson, K. (1975) The effect of A PCB (2,4,2,4, tetrachlorobiphenyl) on lipidsynthesizing enzymes in rat liver microsomes,.. *Bull. Enviro. Contam. Toxicol.* 14, 415.
- Buchet, J.P., Lauwerys, R. and Roels, H. (1977) Long term exposure to organophosphorus pesticides and lipid metabolism in the rat . *Bull. Enviro. Contam. Toxicol.* 17 175.
- Carlson, L.A. and Liljedahl, S.O. (1963a) Lipid during 24 hr after trauma with special reference to the effect of Guanethidine. *Acta. Med. Scand.*, 173, 25.
- Carlson, L.A. and Liljedahl, S.O. (1963b) Lipid metabolism and trauma : II studies on the effect of Nicotinic Acid on norepinephrine induced fatty liver". *Acta. Med. Scand.* 173, 787
- Cecil, H.C., Susan, J.H. and Bitman, J. (1974) Effect of nonpersistent pesticides on liver weight lipids and vitamin A. of rats and Quail. *Bull. Enviro. Contam. Toxicol.* 11, 496.
- Duff, G.L. G.L. and Memillan., G.C. (1951) Pathology of arteriosclerosis. *Am. J. Med* 11, 92
- Duff, G.L., Payne, T.P.B. (1950) The effect of alloxan diabetes on experimental cholesterol atherosclerosis in the rabbit III. The mechanism of the inhibition of experimental cholesterol atherosclerosis in alloxan-diabetic rabbits. *J. Exp. Med.*, 92, 299.
- Duncombe, W.G. (1963) "The colorimetric micron determination of long, chain fatty acids. *Biochem. J.*, 88, 7.
- Fiegelson, E.B., Pfaff, W. W., Karmen A. and Steinberg, D. (1961) "Role of plasma free acids in development of fatty liver". *J. Clin. Invest.*, 40, 2171.
- Folch, J., Lees, M., and Stanley, G.H.S. (1957) A simple method for the isolation and purification of total lipids from animal tissue". *J. Biol. Chem.* 226, 497.
- Fredrickson, D.S., and Gordon, R.S.Jr. (1958) Transport of fatty acids. *Physiol. Rev.*, 38, 585.
- Gordon, R.S.Jr., Clarkes, A., and Gates, H. (1957) Unesterified fatty acids in human blood, plasma. II. The transport function of unesterified fatty acids. *J. Clin. Invest.*, 36, 810.
- Greer, D.E. and Keil J.E. (1974) "The effect of DDT and PCB on lipid metabolism in *E. Coli.* and *B. Fragilis.* *Bull. Enviro. Contam. Toxicol.* 12, 295.
- Hansell, M.M. and Ecobichon, D.J. (1974) Effect of chemically pure chlorobiphenyls on the morphology of rat liver". *Toxicol. Appl. Pharma.*, 28, 418.
- Hayes, W.J., Quinby, G.E., Walker, K.C., Elliott, J.S. and Upholt, W.M. (1958) *Arch. Environ. Hflh.*, 18, 398. Cited by : ( Q.F. Carlson, L.A., and Kolmodim-Hedman, B. 1972 Hyper — Lipoproteinemia in men exposed to chlorinated Hydrocarbon Pesticides". *Acta. Med. Scand.* 192, 29).
- Helmy, F.M. (1962) "Studies in comparative histochemistry" *Ph. D. Thesis.* Tulane University.

- Ibrahim, K.A. (1981). Effect of Some cotton warn insecticides used in Egypt on fat metabolis m in experimental animals. *M. Sc. Thesis, Fac. Agric., Cairo Univ.*
- King, and Armstrong (1934) King *et al.* (1937), (1942) (Q.F. Harold Varley (1967) "*Practical Clinical Biochemistry, 4th., ed, P. 453-454.*
- Kingsbury, K.J., Morgan, D.M., Stovold, R. and Brett, C.G. (1969) Polyunsaturated fatty acids and myocardial infarction". *Lancet*, 11, 1325.
- Klevay, Leslie, M. (1971) "Endrin excretion by the isolated perfused rat liver, A sexual difference. *Proc. Soc. Exp. Biol. Med.* 136, 878.
- Lewis, L. and Farber, R.K. (1952) Hormonal Factors which regulate the metabolization of depot fat to the liver. *Recent Progr. Hormone. Res.* 7, 399.
- Luckens, M.M. (1969) *Industr. Med. Surg.* 38, 56. (Q.F. Carlson, L.A. and Kolmodin Hedman, B. 1972) Hyper-Lipoproteinemia in Men exposed to chlorinated hydrocarbon pesticides. *Acta. Med. Scand.,* 192, 29.
- Ludke, J.L. Hill, E.F. and Dieter, M.P. (1975) "Cholinesterase (ChE) response and related mortality among birds fed Ch. inhibitors.
- Mehani, J.S., El-Habashi, A. and Ahmed, K. (1974) *The toxicity of phosvel Ain Shams Med. J.* 25, 345.
- Mendelssohn, H. (1977) *Biol. Conserv.* 11, 163. (C.F. White, D.H., King, K.A., Mitchella Ch. A. Hill, E.F. and Lamon, T.G. (1979) Parathion causes secondary poisoning in laughing gull breeding colony. "*Bull. Enviro. Contam. Toxicol.* 23, 281.
- Menrath, R.L., Sharad, A., Gray, K.W. and Gameron, C.W. (1973) Toxicity of bunamidine Z. Metabolic effects. *N. Z. Vet. J.,* 21, 212.
- Mills, J.A. (1973) *Proc. N. Z. Ecol. Soc.* 20, 65. (C.F. White, D.H., King, K.A. Mitcell, Ch. A., Hill, E.F., and Lomon, T.G. (1979) parathyon causes secondary poisoning in laughinghg gull breeding colony. *Bull. Enviro. Contam. Toxicol.* 23, 281.
- Mitchell, A.I., Plack, P.A. and Thomson, I.M.(1977) Relative concentrations of c14 DDT and of two polychlorinated biphenyls in the lipid of cod tissue after a single oral dose. *Arch. Enviro. Contam. Toxicol.* 6, 525.
- Moore, J.H. and Willams, D.L. (1964) The relationship between diet plasma lipid composition and aortic arteriosclerosis in rabbits. *Brit. J. Nutr.* 18, 431.
- Morrison, W.R. (1964) "A fast simple reliable method for micro determination of phosphorus in biological materials" *Analyt. Biochem.* 7, 218.
- Murphy, S.D. (1966) Response of adaptive rat liver enzymes to acute poisoning by insecticides. *Toxicol. Appl. Pharma.* 8, 266.
- Natsyuk, M.V., Lipkan, GIN. and Chernuka, F.S. (1974) Assessment of the severity of toxic hepatic lesions in acute poisoning with chlorinated hydrocarbons in relation to the activities of aminotransferase and sorbitol dehydrogenase. *Gig. Tr. Prof. Zabol.* 6, 53.
- Onikienko, F.A. (1963) Enzymic changes from early stage of intoxication with small doses of chloro-organic insecticides. *Sb.* 77, 80.
- Pearse, A.G.E. (1968) "Histochemistry, Theoretical and Applied, 3rd edn., I.J. and A. Cheerchill, Ltd (London).
- Peters, H.D.; Selhorst, P., Dinnendahl, V.; Helm, K.U. and Schonhofer, P.S. (1973) *Arch. Toxicol.*, 30, 199. (C.F. Buchet, J.P., Lauwerys, R. and Roels, H., 1977. Long term exposure to organophosphorus pesticides and lipid metabolism in the rat. *Bull. Enviro. Contam. Toxicol.* 17, 175.



- Reitman, S. and Frankel, S. (1957) A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminases. *Am. J. Clin. Path.* 28, 56.
- Roe, R.T. (1969) Whole blood cholinesterase and serum enzymes level in cattale as indicators of exposure to organophosphorus insecticides. *Aust. Vet. J.*, 45, 411.
- Sakai, K. and Matsumura, F. (1968) Esterases of mouse brain active in hydrolyzing organophosphate and carbamate insecticides. *J. Agr. Food. Chem.*, 16, 803.
- Sam Kacew and Rad' ey, L.S. (1973) The influence of P P-DDT, heptachlor and endrin on hepatic and renal carbohydrate metabolism and cyclic AMP. AD only Cyclase system *Life Science*, 13, 1363.
- Stickel, W.H. (1974) Proc. Annu Conf. Western. Assoc. State. Game and Fish 484. (C.F. White, D.H., King, K.A.; Mitchell, Ch. A., Hill. E.F. and Lamon, T.G., 1979, Parathion causes secondary poisoning in a laughing gull breeding colony". *Bull, Enviro. Contam. Toxicol.* 23, 281.
- Uppal, R.P. and Ahmad, A. (1977) Blood cholinesterase and serum transaminases in malathion toxicity in buffalo calves. *Ind. J. Animal. Sci.*, 47, 636.
- Van Handel, E. and Zilversmith, D.B. (1957) Micromethod for the direct determination of serum triglyceride. *J. Lab. Clin. Med.*, 50, 152.
- Wright, F.C., Hunt, L.M. and Palmer, J.S. (1966) The biochemical effects of coumaphos and three oximes on certain enzymes and blood protein elements in cattle. *Am. J. Vet. Res.* 27, 177.
- Zinkl, J.G., Rathert, J. and Hudson, R.R. (1978) *J. Wildl. Mangage.* 42 406. (C.F. White, King, K.A. Mitchell Ch. A. Hill E.F. and Lamon, T.G. ,1979, parathion causes secondary poisoning in a Laughing gull breeding colony, *Bull, Environ. contam. Toxicol.*, 23. 281.

## تأثير بعض المبيدات على تهيئ الدهن وعلى انزيمات الدم في القطران

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اجريت خمس تجارب على ١٨٧ فأردكر من سلالة (سبراج داوى ) بهدف  
دراسة السمية الحادة للمبيدات على : الكبد ودهون وبعض انزيمات الدم .  
وقد اعطيت مجموعة القطران المعاملة نصف الجرعة السامة ( اومكراتها ) من  
٠ د ٠ د ٠ ت ، والاندرين ، الدورسبان والسيترولان أو اللانيت ٠ وبعد  
٢٤ ساعة من اعطاء القطران الجرعة الاخيرة من المبيد قتلت الحيوانات وأخذت  
عينات من كبدها ودمها لتقدير محتويات مكونات دهن الكبد والدم ونشاط  
انزيمات الدم ( ترانزاميتاز الجلوتاميك أو كسالواستييك والجلوتاميك  
بيروفيك والالكالين فوسفاتيز استيريز ) .

وتتلخص اهم النتائج فيما يلى : -

١ - زيادة معنوية فى وزن الكبد كنسبة مئوية من وزن الجسم فى  
القطران التى اعطيت ٠ د ٠ ت فقط .

٢ - زيادة معنوية فى الدهون الكلية والجلسريدات الثلاثة بكبد  
القطران التى تناولت ٠ د ٠ ت ، اندرين او الدورسبان ، اما السيترولان فكان  
تأثيره مقصورا على كولستيرول الكبد حيث زاد .

وقد ادى اللانيت الى زيادة الدهون الكلية نتيجة لزيادة الكولستيرول  
والجلسريدات الثلاثة .

٣ - انخفضت نسبة الفوسفوليبيدات الى الكولستيرول فى دم الحيوانات  
التي اعطيت ٠ د ٠ ت نتيجة لانخفاض الكولستيرول وقد ادى التسمم  
بالدورسبان الى زيادة معنوية فى الجلسريدات الثلاثيه والدهون البيرة بالدم .  
وكان ذلك مصحوبا بتكوين الكبد الدهنى . اما السيترولان واللانيت فقد  
تسببا فى زيادة فوسفوليبيدات الدم فقط .

٤ - تسببت جميع المبيدات التى درست ( ماعدا الاندرين ) فى  
احداث زيادة معنوية فى نشاط كل من انزيمى الجلوتاميك أو كسالواستييك  
والجلوتاميك بيروفيك ترانزاميتيز . الاندرين تسبب فى زيادة نشاط انزيم  
الجلوتاميك بيروفيك ترانزاميتيز فقط .

٥ - زاد نشاط انزيم الالكالين فوسفاتيز فى حالة التسمم بالدورسبان  
واللانيت فقط .

٦ - تسبب التسمم بالدورسبان ، بالسيترولان أو اللانيت باثبات  
نشاط انزيم الكولين استيريز بدرجات متفاوتة .