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Eradication of Histopathological and biochemical activities in testes and reproductive organs on Exposure of organophosphate, [ethion] in albino mice.

PROF.SUVARNARAJESH Y KRISHNAPUR M.sc.Ph.D, B.Ed.D.C.A,
Asst professor dept. of Zoology Kittel science college Dharwad Karnataka south India 580024
Email s_krishnapur2002@yahoo.co.in cell no +91 9035120787

PROF. BASAPPA .B.KALIWAL M.SC.Ph.D.POST DOC .UK
Vice chancellor of university Davangari Karnataka south India 580024
b_kaliwal@yahoo.com cell no +91 9448029309

Abstracts Ethion an organophosphate was orally administrated at16mg/ was significantly Increased testis at the dose of 20, 30, significantly increase and decrease in the epididymis, cowpe's gland and liver weight. Whereas adrenal hyphalamohypophysial - Testicular axis albino mice for 5,10.20 &30 days control mice take similar quantities of olive oil. And was noted the mice were sacrificed by cervical dislocation after 24 hours' terminal exposure of ethion. in the number of spermatocyte and spermatids in 20 days and number of spermatid 10 days treated mice, the testis of the mice treated with Ethion for long duration spermatogial cell and sperms, when compared with controls, in the mice treated 20 and 30 days showed significantly decreases in the weight of the prostate gland, however increases in significantly in 30 days however weight of Cowpers's gland decreased in significantly in 30 days treated mice. There was significantly increased testis at the dose 20, 30, significantly increased and decrease in the epididymis, whereas adrenal and -Testicular axis **hypothalamo-hypophyseal**

Keywords: Ethion spermatogenic cells: Male mice: Histology: Biochemical constituents

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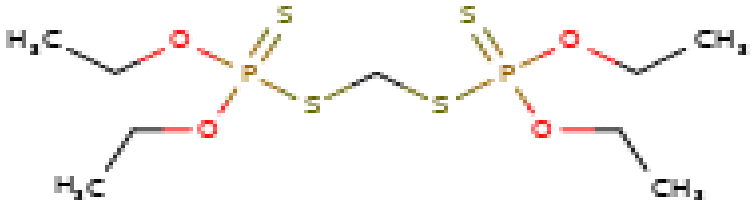
1. Introduction Materials and methods Chemical Technical grade ethion (purity 98%) was obtained from Rallies India Ltd., Mumbai (India) had been used for the experiments the dose were given orally in olive oil for 5,10,and 30d below their acute LD50 level of intoxication According to their body weight is 62mg/kg weight

Chemical formula $C_9H_{22}O_4P_2S_4$



Ethion.

12L; 12D at room temperature 260 the experiment was conducted with five groups, each charring 10 animals. Ethion was administered orally in olive oil vehicle at effect dose of for 5, 10,20and 30 days to respective groups, olive oil treated mice served ethion. The parent treatment. The Testis. Epididymis, vasadeferentia seminal vesicle prostate glands, cowpea's' gland coagulators glands were dissected out. The 100g body weight. Histological studies the testes were fixed in Bouins fluid embedded in paraffin and sectioned at 5μm thickness and stained in hematoxyin-eosin. Was assessed, from each testis ten good sections were randomly selected from each group, the histological observations made with an ocular and stage micrometre. The histometric data are the diameter of seminiferous tubules

Biochemical studies freshly removed testis, liver and kidney tissues were (1950).and DNA Schneider (1957) cholesterol peters and vanstyke (1946) Statistical 5) Results Testes and accessory reproductive weight Testes and prostate gland treated for days 5.10.and 20 days

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Table 1 Temporal effect of ethion on weight of testes and number and diameter of spermatogonia. And spermatocyte and spermatids albino mice.

Duration of Treatment (days)	Testes weight Mg/100g body weight	Diameter (μm) of spermatogenic cells		
		Spermatogonia	Spermatocytes	Spermatids
Control	610.40±1.22	5.40±0.24	6.00±0.44	6.01±0.31
5	577.40±0.60*	3.60±0.24	4.40±0.40	4.00±0.31
10	594.80±0.74*	3.20±0.37	4.20±0.20	3.80±0.20*
20	609.80±1.48	3.40±0.30	4.20±0.20	3.60±0.24*
30	573.00±1.21*	4.00±0.31	3.40±0.50	4.01±0.30

*Significant P 0.05compared with control.

A value represents the mean ± S.E.M of ten animals

Table 2

Duration Treatment (days)	Testes weight Mg/100g body weight	Number of spermatogenic cells		
		Spermatogonia	Spermatocytes	Spermatids
Control	610.40±1.22	25.00±0.20	54.00±0.24	60.00±0.31
5	577.40±0.60*	44.00±0.24	38.00±0.20	44.00±0.24
10	594.80±0.74*	38.00±0.20	42.00±0.20	48.00±0.20
20	609.80±1.48	26.00±0.24*	40.00±0.00	48.00±0.20
30	573.00±1.21*	30.00±0.31	28.00±0.20*	36.00±0.24

*Significant P 0.05compared with control.
A value represents the mean ± S.E.M of ten animals.

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Intoxicated mice were after the administration of ethion. There was significantly decrease in epididymis Cowper's gland mice, kidney., liver adrenal weight in 5,10and 20 days ethion treated mice significantly changes when compared to control (Table 3), body weight and adrenal glands were changed significantly in all groups,

Table 3

Mice	Prostate Gland (mg)	Cowper's Gland (mg)	Testis (g)	Epididymis (mg)	Liver (g)	Kidney (mg)	Adrenal (mg)
Control	9.35±1.131	81.2±0.32	17.78±0.14	11.46±0.20	1.97±0.01	993®0.92	1.00±0.00
5	82.2±0.69	70.8±0.46	15.85±0.56	10.68±0.10	1.72±0.01*	975±2.15	6.59±0.04*
10	72.3±0.63*	81.7±0.83	17.49±0.28	10.93±0.00	1.77±0.00*	540±1.96*	6.35±0.08*
20	103.2±0.31*	123.5±0.87*	18.71±0.33*	14.24±2.02	1.74±0.00*	504±1.24*	8.29±0.03*
30	132.2±0.57*	100.9±0.34*	18.47±0.16*	12.36±0.03*	1.63±0.02*	331±3.18*	10.1±0.04*

Significant P 0.05 compared with control.

A value represents the mean ± S.E.M of ten animals.

Histological studies Number and diameter of spermatogenic cells were no significant treated with ethion 10 and 20d were significantly increase however. Number and diameters of spermatogenic cells in 5days number and ethion. Histological observation of the testis showed normal

spermatogenesis with spermatogenic cells at different stages of development in control mice (Fig.1) histological examination of the testis of the mice treated with ethion for 5 days showed normal spermatogenesis with spermatogenic cells and interstitial tissue consists of Leydig cells (Fig.2) histological observations of the testis with increasing duration of exposure to ethion treated mice for 10,20,and 30 revealed inhibition of spermatocyte maturation resulting in less number of sperms in the lumen of seminiferous tubules ,interstitial cells were also affected (Fig 3-5)

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Figure 1 transverse section of testis

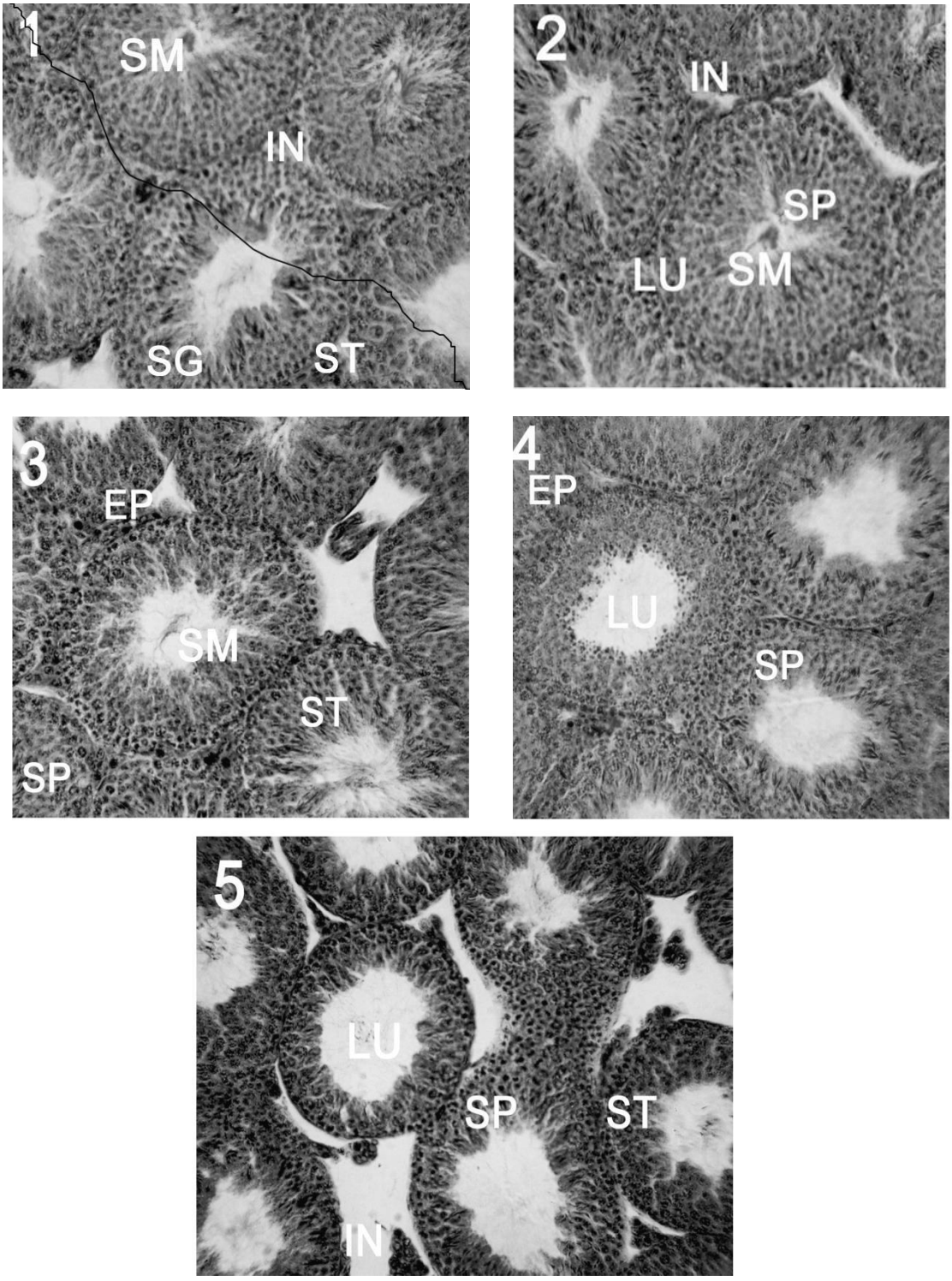

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Fig .1 testis of the mouse treated with olive oil (control) showing different stages of spermatogenesis.
Fig. 2 testis of mouse treated with ethion for 10 days showing disorganization of interstitial tissue and sperm in lumen.
Fig.4 testis of the mouse treated with ethion for 20days showing formation of giant cell resulting into reduced number of spermatogenic cells and lumen with loss of sperms.
Fig.5 testis of the mouse treated with ethion for 30 days showing formation of giant cells resulting in marking reduction in number of spermatogenic cells and sperms, interstitial cells were affected.

Abbreviations.ST, Seminiferous tubules; EP, epithelium; IN, intertubular tissue;
SG, spermatogonia; SY, spermatocytes; SP, spermatids; SM, sperm; LU, lumen (scale line indicates 30µm).

Biochemical changes in testis where as significant un changed DNA in testis, whereas liver and kidney all the biochemical constituents decrease significantly in 5 testis and liver kidney not changed significantly. In 5, 10, 20 and 30 days not changed significantly when compared with control (Table.4)

Duration Treatmen t (days)	µ/mg wet of testis			
	Protein	Glycogen	Cholesterol	DNA
Contro l 5	1.80±0.00 1.61±0.01*	1.80±0.00 1.61±0.00	2.93±0.01 4.15±0.02	1.41±0.00 1.37±0.03
10	1.50±0.04*	1.24±0.02*	3.19±0.02*	1.41±0.00
20	1.21±0.01*	1.22±0.00*	5.11±0.00*	1.55±0.01
30	1.11±0.02*	1.41±0.00*	6.18±0.02*	1.64±0.01

*Significant P 0.05compared with control.

A value represents the mean ± S.E.M of ten animals

2. Discussion Spermatogenesis (Clermont, 1972). Spermatogenesis involves inter play of sex steroid and pituitary gonadotropins (Sharpe, 1987). Paired testicular mass, a valuable index of reproductive toxicity in mal animals (Amman, 1982), decreased with increasing during of pesticide effect and decrease in testicular mass was consistent with elimination of germ cells (Chapin and study weight of testis decreased significantly with increasing duration of pesticide treatment in mice, similar effects have been and zineb (Lucier et al., 1977).it has been observed that carbonyl induce sperm abnormalities,

reduced number of in mice and rats (Kitagawa et al., 1977). It has been showed that the treatment with a carbamate insecticide carbaryl affects Spermatogenic cells and cause Leydig cells and (Shrivastava and Shrivastava, metabolites like ETU and carbon disulfide (CS_2) (O'Neil and Marshal, 1984), Ethion carbamate mancozeb and affect the vaginal cyclicity

Effect of ethion on testis and accessory organs revealed on the male reproductive system of mice namely the anti-spermatogenic and anti-androgenic effects. The anti-spermatogenic advice effect is reflected by the decreased of prostate and Cowper's glands the accessory male duct and glands are morphologically dependent upon the production of androgens (Williams-Ashman and Reddy, 1972). With prolong exposure of ethion results in decreased testicular weight, decreased number and diameters of spermatogenic cells. And decreased testosterone and increased cortisone similar results were reported in rats treated with organ chlorine. Insecticides like linane and dicofol. Lindane induced oxidative stress and decreased oxidant enzymes (sujatha et,. 2001: Jadramkunti and Kaliwal, 2002). It has been reported that male rats treated with organophosorus pesticides quinolphos showed direct effect on acetyl cholinesterase (Ache.) Resulting in alterionsin the master gland, gonadotropin following administration of carbamate (Kitagaw et al..1977), Goldman et al,. 1994, 1997; Shrivastava and Shrivastava, 1998).Goldman et al,. (1990) have been reported that the insecticide. Chlorodimeforrm may destroys endocrinology homeostasis by suppressing GnRH release, it has also reported that toxic agents may act directly on the gonadotropins to alter gonadotropins synthesis and secretion or indirectly by altering the master gland cells response to GnRH or gonadal steroid both action will result in in serum FSH and LH level (Dickarson et al.,) it has also reported Observed that members of pesticides such as endosulfon and its metabolite dithiocarbamate can interfere it cateclamine neurotransmitter, can by inhibiting the activity of dopamine and β-hydroxylase (BβH), this is an enzyme that converts dopamine to epinephrine and then stimulates the release of GnRH. This mechanism plays an important regulatory and modulatory role in brain hypothalamic control of master gland luteinizing hormone (LH) release (kalra and kala, 1983). In rats man et al,. 1994). This could be expected to affect gonadal steroidogenisis and spermatogenesis with ethion with prolonged exposure of ethion might have interfered with testis function directly on the testis as number of pesticides shows testicular toxicity. By doing it must hormonal feedback and stop timely produce gonadal steroid which are essential for to increasing duration of ethion resulted in kidneys and adrenal, similar findings it has been suggested that administration of ethion with a high and chronic exposure (360days) causes sign of poisoning. And hemorrhage and adrenal was slight enlarged and congested.

In rat the highest dose orally with Ethion showed medullary Congestion in their kidney (Suvarnarajesh.krishnapur and Kaliwal 2017) the prostate gland and adrenal, liver weight in duration dependent manner. Similar carbon disulfide decrease due to tress on hypothalamus it increase carpus lutum the ethion Suvarnarajesh krishnapur and Kaliwal (2016) effect of ethion Administration Schedule on Estrus Cycle and follicular

dynamics in Albino Mice ethion it. 1985 Proteins, present study revealed that were significantly not changed in low duration treatment. However, prolonged treatment with ethion caused significantly kidney all biochemical constituents inhibit testicular steroidogenisis enzymes. Testicular DNA.RNA and proteins and affects male reproduction (sujatha et al., 2001). Recently similar results have

Organophosphate pesticide poisoning is a common method of suicide in south India and a lead cause of death. Organophosphate inhibits acetyl cholinesterase leading to an accumulation of acetylcholine at cholinergic synapse in brain and at the neuromuscular junction. The resulting cholinergic stimulation and muscle weakness that occurs soon after poisoning rapidly utilize significant level of energy. This negative energy balances if not corrected can lead to muscle wasting. Muscle wasting in relation to over use of energy implicates a role for amino acids in gluconeogenesis effect of ethion on testis, it possible that the compared may interfere with physiological process involved in reproduction. Leading to infertility. The testis I a complex organ contain three important cells types (germs cells, sterols cells and Lyding cells) in endocrine parathyroid and autocuing mechanisms. A toxic insult of one element influent the function of the other elements if not in the low duration, certainly in high duration. DNA with ethion logical effect reported chronic exposure

3. Conclusion Ethion has injurious effect on public health especially on effect on reproductive organs and liver kidney etc. tissues but intensity of degree damage depends on exposure time dose level. As a result, strict law need to be formulated to prevent such oblivious use of ethion during applying in the agricultural sector and it should to be reflected in our health policy. In addition to the novel regulation, the pesticide Incident Monitoring System (PIMS) of Bangladesh have to be more watchful for appropriate implementation of the regulation. The worker should search out novel biological method for combating with insect and pest.

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5. Reference

1. World Health Organization. Environmental Health Criteria. Cypermethrin, Geneva: United Nations Environmental Programme, the International Labour Organization, and the World Health Organization. 82, 1989
2. Tao T Y, Wei L Z, Yang Y, Tao Z & Zhwo Y. (2008). Effects of alpha and theta cypermethrin insecticide on transient outward potassium current in rat hippocampal CA3 neurons. Pesticide Biochem Physiol, 90:1-7.
3. Bouwman H, Sereda B & Meinhardt H M. (2006). Simultaneous presence of DDT and pyrethroid residues in human breast milk from a malaria endemic area in South Africa. Environ Pollut. 144: 902-917.
4. Meister, R.T., 1992.
5. Pascual J A & Peris S J. (1992). Effects of forest spraying with two application rates of cypermethrin on food supply and on breeding success of the blue tit (Paruscaeruleus). Environ. Toxicol. Chem, 11:1271-1280.
6. Ray DE, Pyrethroid insecticides: mechanisms of toxicity, systemic poisoning syndromes, paresthesia, and therapy, In Handbook of Pesticide Toxicology, (Ed. Krieger R, Academic Press, USA) 2001, 1289–1303
7. Righi D & Palermo-Neto J. (2003). Behavioral effects of type II pyrethroidcyhalothrin in rats, Toxicol.Appl.Pharmacol. 191:167176.
8. Suvarnarajesh Y Krishnapur, B.B.Kaliwal (2017) dysfunction of liver and kidney induced by ethion on albino mice vol 8 issues 12 ISSN 2250-1959.
9. Ramadan A A, Bakry M N, Salam M A, Marei, Eldefrawi A T & Eldefrawi M E.(1988). Action of pyrethroidson GABAA receptor function, Pest. Biochem. Physiol, 32:97-105.
10. Toxicollett. 82: 933-940.
11. Rao, G V & Rao K S J. (1993). Inhibition of monoamine oxidase-A of rat brain by pyrethroids- an in vitro kinetic study, Molec. Cell. Biochem 124: 107-114.
12. Tample W A. & N.A. Smith, Cypermethrin. Environmental potential agency. 1996.
13. Amer S M, Ibra A & himel-Sherbeny KM. (1993). Induction of chromosomal aberrations and sister chromatid exchange in vivo and in vitro by the insecticide cypermethrin, J. Appl. Tox, 13: 341.
14. Singh A K, Tiwari M N, Upadhyay G, Patel D K, Singh D, Prakash O & Singh M P.(2012). Long term exposure to cypermethrin induces nigrostriatal dopaminergic neurodegeneration in adult rats: Postnatal exposure enhances the susceptibility during adulthood, Neurobiology of Aging, 33: 404-415.
15. Kamari V, Kaur B & Sandhu H S. (2002). Experimental cypermethrin toxicity in buffalo calves. Indian J Anim Sci, 72: 658-60.
16. Hussain S, Khan M Z, Khan A, Javed I & ASI M R. (2009). Toxico- pathological effects in rats induced by concurrent exposure to aflatoxin and cypermethrin, Toxicon, 53: 33-41.
17. Nagarjuna A & Doss P J. (2009). Acute oral Toxicity and Histopathological studies of Cypermethrin in rats, Indian J Anim Res, 43: 235- 240.
18. Righi D A & Palmermo-Neto J. (2003). Behavioral effects of type II pyrethroidcyhalothrin in rats, Toxicol.Appl.Pharmacol, 191:76167.

19. Baligar, Ind.Health, 39235-243.
20. Suvarnarajesh Y Krishnapur and Kaliwal.B.B (2016) Effect of Ethion Administration Schedules on Estrus Cycle and Follicular dynamics in Albino Mice vol.4,Issue2,pp:202-210)
21. Harper, Aj. Rodwell, V.M., Meyer, P.A (1977) Review of physiological chemistry, 16th declare Medical Pub. Los. Actor., California. Hayes, W.J., Jr (1982) pesticides studied in man. Williams and Wilkins Col., Baltimore.
22. Jinna, R.R., Ozodinma, J.E, J.E and Desaiah, D(1989)Age related changes in rat brain ATPase during treatment with chlordecone.J.Toxicol. Environ.Hlth. 27,199-208
23. JMPR, http://www.who.int/pes/jmpr/2003.htm.
24. Kackar, R Srivastava, M.K., Raizada, In.Health, 104-11
25. King, J. (1965) in: practical clinical Enzymology, Edn. Z.Van, D.Norstrand, Co.London, p.83.
26. Kinter, W.B., Merkens, L.S., Janiki, R.H., Guarino, A.M.(1972) studies on the mechanism of toxicity of DDT and PCBS; Disruption of osmoregulation in marine fish, Environ.Hlth.pereps., 8,169-173)
27. Klassen, C.p. and Plaa, G.L.(1966)Relative effects of various chlorinated hydrocarbons on liver and kidney function in mice, Toxicol. And Appl.Pharmacol., 9.139
28. Barale, R.C., Seapoli, C.Meli, D., casini, M.Minunni, A Marrazzini, N.,Loprieno and Barrai,(1993) Cytogenetic effects of benzimidazoles in mouse bone marrow. Mutation res., 300, 15-28
29. Bhaynagar, V.K., Saigal, S., Khemani, L., D., Malviya, A.N. (1982) survey among workers in Pesticide factories, Toxicol. Lett, 10,129-132.
30. Brown, H.R. and Sharma, R.P (1976) Synatosomal adenosine triphospatease (ATPase) inhibition by organophates, Experentia, 32(12), 1540- 1542
31. Butler, A.M., Murray, M (1993) inhibition and inactivation of constitutive cytochromes P450 in rat liver by parathion. Mol. Pharmacol., 43,902-908.
32. Carrol, N.V., Langely, R.W.and Row, R.H (1956) Glycogen determination in liver and mode by use of anthrone reagent.J.Biol. Chem., 20,583-593.
33. Choudhary, N., sharma, M, Verma, P., Joshi,S.C (2003) Hepato and nephrotoxicity in rat exposed to endosulfan. J.Environ. Biol., 24,305-308.
34. Cid, M.G., Loria, D and Matos, E(1990) Genotoxicity of the pesticide propoxur and its nitroso derivative, Nopropoxur on human lymphocytes in vito.Mutation Res., 232, 45-48.
35. Vijverberg HP & van den Bercken J. (1990). Neurotoxicologically of pyrethroid insecticides, Crit Rev Toxicol, 21:105-26