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DATA SHEET ON PESTICIDES

No. 68

DISULFOTON



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CLASSIFICATION:

Primary use: Insecticide, acaricide, miticide

Secondary use: None

Chemical group: Organophosphorus compound

Date issued: March 1988

1.0 GENERAL INFORMATION

1.1 COMMON NAME: disulfoton (BSI, E-ISO, F-ISO, ESA), ethylthiodemeton (JMAF), M-74 (USSR)

1.1.1 Identity

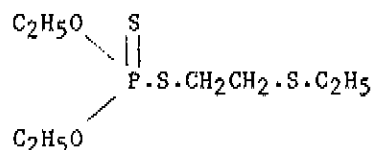
IUPAC: O,O-diethyl S-2-ethylthioethyl phosphorodithioate

CAS: O,O-diethyl S-[2-(ethylthio)ethyl] phosphorodithioate

CAS Reg. No.: 298-04-4

Molecular formula: C₈H₁₉O₂PS₃

Relative molecular mass: 274.4

Structural formula:

Synonyms: Bay S276, Bay 19639, Bayer 19639, Dimaz, Disipton, Disulfaton, Di-syston G, Disyston^R, Di-syston, Disystox, dithiosystox, ENT-23347, ethyl thiometon, ethylthiometon, Frumin, Frumin AL^R, Frumin G, Glebofos, M 74, Solvigran, Solvirex^R, thiodemeton, TwinSpan, Vuagt 1-4, Vuagt 1964.

1.2 SYNOPSIS: Disulfoton is a systemic insecticide and acaricide used as a seed coating and for soil application to protect from insect attack. Control for up to seven weeks may be obtained. Disulfoton is metabolized to potent inhibitors of cholinesterases, and has an extremely high acute toxicity in mammals.

1.3 SELECTED PROPERTIES

1.3.1 Physical characteristics: The pure product is a colourless oil with a sulfurous odour, m.p. > -25 °C, b.p. 128 °C at 1.33 hPa, density d₄²⁰ 1.14. The technical product is a yellowish oil.

1.3.2 Solubility: In water 15 mg/L at 20 °C. Soluble in most organic solvents.

1.3.3 Stability: It is relatively stable under normal storage conditions. Hydrolysis occurs in alkaline media.

1.3.4 Vapour pressure: 24 mPa at 20 °C.

1.4 AGRICULTURE, HORTICULTURE AND FORESTRY

1.4.1 Common formulations: Granules (20-150 g/kg), emulsifiable concentrates (850 g/kg). Also in combination with Dasanit^R, Ethimeton and Ekanon in spray concentrates, granular (20-100 g/kg) and emulsifiable concentrates.

1.4.2 Susceptible pests: Used against aphids and mites on corn, sorghum, cotton, vegetables, fruits, nuts, ornamentals, small grains and other field crops; also used against leafhoppers, fleabeetles, lacebugs, leafrollers, whiteflies, mealybugs, leafminers and Mexican bean beetles.

1.4.3 Use pattern: Applied at 560 to 3 400 g per hectare on crops. Applied to soil by drilling, side dressing or broadcasting; applied prior to planting, prior to emergence or post-emergence. Work into the soil and water thoroughly. May be used as a seed dressing and on fruit trees by spreading from drip line to trunk of tree.

1.4.4 Unintended effects: High dosages may injure seeds. Some leafburn in alfalfa has occurred, garden lily bulbs have been injured. Plant injury may be enhanced when used with some pre-emergence herbicides. Disulfoton may be toxic to insect pest predators and bees.

1.5 PUBLIC HEALTH USE: No recommended use.

1.6 HOUSEHOLD USE: No recommended use.

2.0 TOXICOLOGY AND RISKS

2.1 TOXICOLOGY - MAMMALS

2.1.1 Absorption route: Disulfoton may be absorbed from the gastrointestinal tract, through the intact skin or by inhalation of fine dusts.

2.1.2 Mode of action: Acetylcholinesterase inhibition by some of the metabolites of disulfoton.

2.1.3 Excretion products: Excretion is rapid, half of an oral dose of O-ethyl-¹⁴C-labelled disulfoton was recovered in the excreta within six hours in male rats, 32 hours in female rats. For either sex urinary excretion accounted for 80% of the dose, expired air 9%. For both sexes the major urinary metabolites diethylphosphate and diethylthiophosphate accounted for >90% of the urinary excretion. These metabolites were probably formed by hydrolysis of the products of oxidative metabolism, namely: the sulfone and sulfoxide of disulfoton, and the sulfone and sulfoxide of the oxygen analogue of disulfoton. Minor proportions of the latter three metabolites were identified in rat urine.

2.1.4 Toxicity, single dose: As for many other pesticides, the nature of the solvent or carrier used to aid administration of the compound may affect the magnitude of the observed LD₅₀ values.

Oral LD₅₀:

Rat	(M)	6.2 - 12.5	mg/kg b.w.
Rat	(F)	1.9 - 4.2	mg/kg b.w.
Mouse	(M)	5.8 - 27.0	mg/kg b.w.
Mouse	(F)	2.7 - 27.0	mg/kg b.w.
Guinea pig	(M)	8.9	mg/kg b.w.
Guinea pig	(F)	12.7	mg/kg b.w.

Dermal LD₅₀:

24 hour exposure	Rat	(M)	15.9 - 25	mg/kg b.w.
24 hour exposure	Rat	(F)	3.6 - 6	mg/kg b.w.
4 hour exposure	Rat		41	mg/kg b.w.

Intraperitoneal LD₅₀:

Rat	(M)	7.5	mg/kg b.w.
Rat	(F)	2.1 - 3.1	mg/kg b.w.
Mouse	(M)	6.7	mg/kg b.w.
Mouse	(M,F)	14.0	mg/kg b.w.

Inhalation LC₅₀:

1 hour	Rat	(M)	290	mg/m ³
1 hour	Rat	(F)	63	mg/m ³
4 hour	Rat	(M,F)	15 - 60	mg/m ³

Most susceptible species: Among the mammals tested, the rat and mouse appear equally to be the most susceptible to disulfoton poisoning, and in both species the female is more susceptible than the male.

2.1.5 Toxicity, repeated doses: Tolerance to continued exposure of sub-lethal amounts of disulfoton has been demonstrated following gavage, dietary exposure or intraperitoneal injection. Although the cholinergic symptoms (tremors, fasciculations, excessive salivation) may disappear as the duration of exposure

increases, the acetylcholinesterase activity remains depressed. (See 2.1.7 for biochemical interpretation.) The rate and extent of adaptation are dependent on the route of administration, the magnitude of the dose and the animal species, or even the strain.

Female rats receiving disulfoton during 30 days 1.0 mg/kg/day intraperitoneally fully recovered from symptoms of cholinergic poisoning and weight loss which had been evident for the first seven days of administration. Although symptomless, the brain and serum cholinesterase activities in these rats, and in rats receiving 0.25 mg/kg/day (intraperitoneally), remained low throughout the 30 day period.

Histopathological changes in the optic nerve and retina were observed in all dogs receiving a gelatin capsule dose of 0.5, 1.0 or 1.5 mg/kg/day, five days/week for two years. Retinal lesions observed were mild.

2.1.6

Dietary Studies

Short term: No mortality or effect on body weight gain, haematology, urinalysis, clinical chemistry or gross and microscopic tissue and organ analyses were observed in studies on rats fed on a diet of ≤ 5 ppm disulfoton for 90 days, in mice fed on a diet containing 10 ppm for 16 weeks, nor in mongrel dogs on a diet containing 10 ppm for 12 weeks. Cholinesterase activity was depressed in brain tissue, measured in rats and mice only, generally more severely in females than males and in erythrocytes and in plasma of all three species. In dogs plasma cholinesterase activity rapidly returned to normal after cessation of dosing, but erythrocyte cholinesterase remained inhibited for more than four weeks. The no observed effect level (NOEL) for cholinesterase inhibition was determined as 1 ppm for all three species.

Dietary administration of disulfoton at a dose of 7.5 ppm to male albino rats for 30 days resulted in 4/71 deaths, decreased weight gain and inhibition of cholinesterase activity in the brain, stomach and diaphragm. Cholinesterase activity of the stomach and diaphragm regain pre-treatment values after 19 days but the brain cholinesterase activity remained depressed. Dietary administration of disulfoton at a dose of 7.5 ppm to female Holtzman rats for 62 days produced no overt signs of toxicity. At a dose of 20 ppm, diarrhoea, excessive urination and tremors were seen initially. The severity of the symptoms decreased with duration of exposure but some tremors, fasciculations and reduced weight gain were apparent at 62 days.

Long term: In a two year dietary study in rats at 1, 4 and 16 ppm the no observed adverse effect level (NOAEL) for cholinesterase depression in both sexes was observed to be 1.0 ppm while body weight decrease was reported at 16 ppm in both sexes. No other significant adverse effects were observed at any dose level.

Beagle dogs received a diet of 0.5 or 1.0 ppm for two years. An additional dose group was fed on a diet of 2.0 ppm, for 69 weeks, increased to 5.0 ppm from week 70-72, and increased again to 8.0 ppm from week 73-104. No adverse effect on mortality, behaviour, growth, ophthalmological, clinical chemistry or haematology parameters, or urinalysis were recorded. Plasma and erythrocyte cholinesterase activity were depressed in a group fed on a diet of 2.0 ppm. The effect was accentuated by increased magnitude and duration of dosing. Brain cholinesterase activity was inhibited by 8.0 ppm diet at week 104. NOAEL for beagle dogs is 1.0 ppm.

2.1.7 Supplementary studies of toxicity

Carcinogenicity: No evidence of carcinogenicity was observed in a two year rat dietary study up to and including 16 ppm, the highest dose tested.

Teratogenicity: No teratogenic effects were observed in rats treated by gavage with 0.1, 0.3 or 1.0 mg/kg/day, embryotoxicity was observed at the highest dose and the NOAEL for cholinesterase activity in the dams was 0.1 mg/kg/day.

In rabbits, doses of 1.5 mg/kg/day and above were found to be maternally toxic, causing clinical signs of poisoning and death in some animals. No teratogenic, fetotoxic nor embryotoxic effects were observed at any dose level, up to and including 1.5 mg/kg/day.

Reproduction: In a rat dietary study at 1, 3 and 9 ppm the NOAEL was reported to be 1 ppm for reproductive effects including reduced litter size, pup weight and pup viability at 3 ppm and above. Maternal toxicity, weight loss and clinical signs of cholinesterase depression, were observed in the high dose group.

In another study dietary administration of disulfoton at doses of 2, 5 and 10 ppm had no adverse effect on rat reproduction parameters. At 10 ppm an increased mortality at weaning was observed in the first generation, and some fatty changes in the liver, (males especially) of the third generation. Erythrocyte cholinesterase activity was decreased in all treatment groups.

Inhalation: In a three week study with rats the NOAEL was reported to be 0.02 mg/m³ (6 hours/day) for both sexes for signs of toxicity due to cholinesterase depression at higher doses.

Mutagenicity: Disulfoton was not mutagenic in metabolically activated or unactivated systems with Saccharomyces cerevisiae D3 and D7, nor in metabolically activated systems with several Salmonella typhimurium strains, Escherichia coli strains or Bacillus subtilis strains. However, other workers report that disulfoton was mutagenic without metabolic activation in several S. typhimurium and E. coli strains. Disulfoton did not induce sister chromatid exchange in vitro with three mammalian cell lines. In a mouse micronucleus test, and also in a dominant lethal test in mice, mutagenic potential was not demonstrated.

Neurotoxicity: No clinical nor histopathologic evidence of delayed neurotoxicity was observed in a study with hens.

Following oral administration of 0.5-1.5 mg/kg/day to dogs for two years (Section 2.1.5) no degeneration of the optic nerves was observed.

Miscellaneous effects: Induction of microsomal multisubstrate oxygenases and particularly cytochrome P-450 have been demonstrated in the liver of mice. Barbiturate sleeping times were reduced in mice receiving 1/2 LD₅₀ doses for 10 days. Phenobarbital pre-treatment protected rats or mice against the lethal effects of oral or intraperitoneal administration of LD₅₀ doses of disulfoton.

2.2 TOXICOLOGY - MAN

2.2.1 Absorption: Disulfoton may be absorbed from the gastrointestinal tract, through the intact skin and by inhalation of fine dusts or mist.

- 2.2.2 Dangerous doses: No published information available.
- 2.2.3 Observations in occupationally exposed workers: Between 1966 and June 1978, incidents of human exposure to disulfoton alone were reported in the United States of America: 48 involved agricultural, industrial or transportation accidents. The majority of the incidents arose from container damage, failure to decontaminate skin and clothing after exposure and failure to wear adequate protective equipment. Hospitalization was necessary in some cases, but there were no fatalities from exposure to disulfoton alone. The majority of urine samples collected daily for 25 weeks from 10 male formulators of disulfoton contained significant concentrations of the metabolites diethylphosphate, diethylthiophosphate and diethylphosphorothiolate. Plasma and erythrocyte cholinesterase activities were not, however, significantly reduced.
- 2.2.4 Observations on exposure of the general public: Several instances of contamination of people during aerial application were reported in the United States of America between 1966 and 1978, none requiring hospitalization.
- 2.2.5 Observations on volunteers: Five volunteers received an oral dose of 0.75 mg/day for 30 days without an adverse effect on plasma or erythrocyte cholinesterase.
- 2.2.6 Reported mishaps: In two separate incidents in the United States of America, drums or pesticide bags contaminated with disulfoton residues resulted in symptoms of severe poisoning and coma in three children playing with the containers. Hospitalization and atropine therapy gave slow but continuing recovery, the children were fully recovered after two or five days treatment. Alleged incidents of disulfoton ingestion were generally asymptomatic.

2.3 TOXICITY TO NON-MAMMALIAN SPECIES

- 2.3.1 Fish: Disulfoton is highly toxic to fish and crustaceans, however, there are no reported environmental killings of fish or other aquatic organisms. Disulfoton has no effect upon the hatchability of brine shrimp at 10 mg/L.

2.3.2 Birds

Oral LD₅₀:

Starlings	133.0 mg/kg b.w.
Mallards	6.54 mg/kg b.w.
Bobwhite quail (M)	31.0 mg/kg b.w.
Bobwhite quail (F)	28.0 mg/kg b.w.

Oral LC₅₀ (five days):

Bobwhite	715 ppm
Japanese quail	333 ppm
Ring-necked pheasant	634 ppm
Mallard	510 ppm

Dermal LD₅₀:

Mallard (M)	192 mg/kg b.w.
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2.3.3 Other species: Disulfoton is toxic to bees and terrestrial wildlife.

3.0 FOR REGULATORY AUTHORITIES: RECOMMENDATIONS ON REGULATION OF COMPOUND

3.1 RECOMMENDED RESTRICTIONS ON AVAILABILITY

(For definition of categories, see Introduction to Data Sheets).

Liquid formulations greater than 130 g/L and solid formulations greater than 500 g/kg, Category 1.

All other formulations, Category 2.

3.2 TRANSPORTATION AND STORAGE

All formulations: Should be transported or stored in clearly labelled rigid and leak-proof containers, and away from containers of food and drink. Storage should be under lock and key and secure from access by children and other unauthorized persons.

3.3 HANDLING

All formulations: Full protective clothing (see part 4) should be used by those handling the compound. Adequate washing facilities should be available close at hand. Eating, drinking and smoking should be prohibited during handling and before washing after handling.

3.4 DISPOSAL AND/OR DECONTAMINATION OF CONTAINER

Empty containers should not be re-used. After decontamination the container must be either burned or crushed and buried below topsoil. Care must be taken to avoid subsequent contamination of water sources.

3.5 SELECTION, TRAINING AND MEDICAL SUPERVISION OF WORKERS

All formulations: Pre-employment medical examination for workers is necessary. Workers suffering from active hepatic or renal diseases should be excluded from contact with disulfoton. Pre-employment and periodic blood cholinesterase tests for workers is desirable. Special account should be taken of the worker's mental ability to comprehend and follow instructions. Training of workers in techniques to avoid contact is essential.

3.6 ADDITIONAL REGULATIONS RECOMMENDED IF DISTRIBUTED BY AIRCRAFT

All formulations: Pilots and loaders should have special training in application methods and early symptoms of poisoning, and they must wear protective clothing. It is not advisable to employ flagmen, but if essential they must wear protective gloves, overalls, boots, a respirator and an impermeable hat and be located well away from the dropping zone.

3.7 LABELLING

All formulations

"DANGER - POISON"
(skull and cross-bones insignia)

Disulfoton is an organophosphorus compound which inhibits cholinesterases. It has an extremely high mammalian toxicity. Contact with the skin, inhalation of dust or spray, or swallowing may be fatal. Wear protective gloves, clean protective clothing, and a respirator of the organic-vapour type when handling this material. Bathe immediately after work. Ensure that containers are stored under lock and key. Empty containers must be decontaminated and disposed of in such a way as to prevent all possibility of accidental contact with them. Keep the material out of reach of children and well away from foodstuffs, animal feed and their containers. Do not tank mix with phosalone. In case of contact, immediately remove contaminated clothing and wash the skin thoroughly with soap and water; for eyes, flush with water for 15 minutes. If poisoning occurs, call a physician. Atropine sulfate is an effective drug if applied in time, repeated doses may be necessary. Artificial respiration also may be needed.

3.8 RESIDUES IN FOOD

Maximum residue levels

Maximum residue levels have been recommended by the FAO/WHO Joint Meeting on Pesticide Residues.

4.0 PREVENTION OF POISONING IN MAN AND EMERGENCY AID

4.1 PRECAUTIONS IN USE

- 4.1.1 General: Disulfoton, an organophosphorus pesticide, is extremely toxic by the oral route. It is also readily absorbed through the intact skin, and by inhalation of dust or spray mist. Repeated exposure may have a cumulative effect on cholinesterase activity.
- 4.1.2 Manufacture and formulation - T.L.V.: 0.1 mg/m³. Closed systems and forced ventilation are required to reduce, as much as possible, the exposure of workers to the chemical.
- 4.1.3 Mixers and applicators: When opening the container and when mixing, protective impermeable boots, clean overalls, gloves and a respirator should be worn. Mixing, if not mechanical, should always be carried out with a paddle of appropriate length. When spraying tall crops or during aerial application, a respirator should

be worn, as well as an impermeable hat, overalls, boots, and gloves. The applicator should avoid working in spray mist and avoid contact with the mouth. Particular care is needed when equipment is being washed after use. All protective clothing should be washed immediately after use, including the insides of gloves. Splashes must be washed immediately from the skin, or eyes, with large quantities of water. Before eating, drinking or smoking, hands and any other exposed skin should be washed.

4.1.4 Other associated workers (including flagmen in aerial operations): Persons exposed to disulfoton and associated with its application should wear protective clothing and observe the precautions described above in 4.1.3 under "Mixers and applicators".

4.1.5 Other populations likely to be affected: With good agricultural practice, other persons should not be exposed to hazardous amounts of disulfoton.

4.2 ENTRY OF PERSONS INTO TREATED AREA

Unprotected persons should be kept out of treated areas for at least one day.

4.3 DECONTAMINATION OF SPILLAGE AND CONTAINERS

Re-use of containers should not be permitted. Residues in containers should be emptied in a diluted form into a deep pit, taking care to avoid contamination of ground waters. The empty container should be rinsed with 5% sodium hydroxide, which should be left in the container overnight. The rinse should be emptied into a deep pit, and the container crushed and buried, avoiding contamination of ground waters. Impermeable gloves should be worn during this work. Spillage of disulfoton and its formulations should be removed by washing with 5% sodium hydroxide solution and then rinsing with large quantities of water.

4.4 EMERGENCY AID

4.4.1 Early symptoms of poisoning: Early signs and symptoms of poisoning may include excessive sweating, headache, weakness, giddiness, nausea, vomiting, hypersalivation, stomach pains, blurred vision, slurred speech, and muscle twitching. Later there may be convulsions and coma in cases of severe poisoning.

4.4.2 Treatment before person is seen by a physician, if these signs and symptoms appear following exposure: The person should stop work immediately, remove contaminated clothing and wash the affected skin with soap and water, and flush the area with large quantities of water. If swallowed, and if the person is conscious, vomiting should be induced. In the event of respiratory difficulty ventilatory support should be given, bearing in mind that if mouth-to-mouth resuscitation is used, vomit may contain dangerous amounts of disulfoton.

5.0 FOR MEDICAL AND LABORATORY PERSONNEL

5.1 MEDICAL DIAGNOSIS AND TREATMENT IN CASES OF POISONING

- 5.1.1 General information: Disulfoton, an organophosphorus pesticide, is extremely toxic to mammals. It is readily absorbed from the gastrointestinal tract through the intact skin and from the lungs following inhalation of dust or spray mist. It is metabolized to the oxygen analogues of disulfoton which are potent inhibitors of cholinesterases. It does not accumulate in body tissues.
- 5.1.2 Symptoms and signs: Initial symptoms and signs of poisoning may include excessive sweating, headache, weakness, giddiness, nausea, hypersalivation, vomiting, stomach pains, blurred vision, slurred speech and muscle twitching. More advanced symptoms of poisoning may be convulsions, coma, loss of reflexes and loss of sphincter control.
- 5.1.3 Laboratory: The most important finding is reduction of activity of blood cholinesterases. Urinary levels of organic phosphorus containing metabolites may also be used as a measure of exposure. Neither method is specific for disulfoton.
- 5.1.4 Treatment: If the pesticide has been ingested, unless the patient is vomiting, rapid gastric lavage should be performed using 5% sodium bicarbonate. Care should be taken to avoid pulmonary complications from solvents which may be present in emulsifiable concentrate formulations. For skin contact, the skin should be washed with soap and water. If the compound has entered the eyes, they should be washed with large quantities of isotonic saline or water. Care must be taken by the victims attendants to avoid their own intoxication from contaminated clothing, skin or vomit.

Persons without signs of respiratory insufficiency but with manifest peripheral symptoms, should be treated with 2-4 mg of atropine sulfate by intravenous injection followed by 1 000 mg pralidoxime chloride in split doses or 250 mg of toxogonin (adult dose) by slow intravenous injection. The additional therapy with pralidoxime or toxogonin is most effective if given within 24 hours of the onset of intoxication. However, treatment should continue for as long as benefits are observed. More atropine may be given as needed. Persons with severe intoxication, with respiratory difficulties, cyanosis, convulsions, or who are unconscious should immediately be given oxygen followed by atropine sulfate and then pralidoxime chloride. In such severe cases 4-6 mg of atropine sulfate should be given initially followed by repeated doses of 2 mg at 5-10 minute intervals until atropinization. Symptoms will reappear if tissue concentrations of disulfoton remain high when the effect of atropine wears off. Rales in the lung bases, myosis, nausea, or bradycardia may indicate inadequate atropinization; overdosage with atropine (pulse rate over 140, dry mouth, flushed face) is rarely serious, but under-dosage may be fatal. Diazepam may be given to control convulsions. The patient's condition including respiration, blood pressure, pulse rate, salivation and convulsions should be carefully observed as a guide to further administration of atropine.

The airways should be kept free and artificial resuscitation should be applied if required, preferably by mechanical means. If necessary, intubation should be performed.

Application of morphine, aminophylline, phenothiazines, reserpine and central nervous system stimulants is contraindicated. Return to work should be delayed until blood cholinesterase activity reaches at least 80% of the pre-exposure value.

5.1.5 Prognosis: If the acute toxic effect is survived, and if adequate artificial resuscitation (if needed) has been given, the chances of complete recovery are good. However, in very severe cases, particularly if artificial resuscitation has been inadequate, prolonged anoxia may give rise to permanent brain damage.

5.1.6 References of previously reported cases:

US EPA (1978) "Summary for Reported Incidents Involving Disulfoton".
Report No. 105, Office of Pesticide Programs, Environmental Protection Agency,
United States of America.

5.2 SURVEILLANCE TESTS

<u>Test</u>	<u>Normal level*</u>	<u>Action level*</u>	<u>Symptomatic level*</u>
Plasma cholinesterase	100%	50%	variable
Whole blood or erythrocyte cholinesterase	100%	70%	usually 40%

5.3 LABORATORY METHODS

5.3.1 Detection and assay of compound

Analysis of metabolites in urine may be performed by several methods:

Abbot, D.C., Crisp, S.S., Tarrant, V.R., Tattón, J.O'G. (1970), Pestic. Sci., 1, 10-13.

Bowman, M.C., Beroza, M. (1969,) J. Assoc. Offic. Anal. Chem., 52, 1231-1237.

Brokopp, C.D., Wyatt, J.L., Gabica, J. (1981), Bull. Environ. Contam. Toxicol., 26, 524-529.

Jensen, T.L. (1980), J. Assoc. Offic. Anal. Chem., 64, 869-872.

* Expressed as percentage of pre-exposure activity.

5.3.2 Other tests in case of poisoning:

Acetylcholinesterase activity in blood, provide the most useful diagnosis of poisoning.

Ellman, G.L., Courtney, D., Andres, V., Featherstone, R.M. (1961), Biochem. Pharmacol., 7, 88-95.

Fleischer, J., Woodson, S., Simet, L. (1956), Arch. Indust. Hyg., 14, (6), 510-520.

Michel, N.O., (1949), J. Lab. Clin. Med., 34, 1564-1568.

Wilhelm, D., Reiner, E. (1973), Bull. Wld. Health Org., 48, 235-238.

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10. WHO (1986), Environmental Health Criteria 63, Organophosphorus Insecticides: A General Introduction. UNEP/ILO/WHO, Geneva, Switzerland.
11. Worthing, C.R. (1987), The Pesticide Manual. A World Compendium, Eighth Edition, British Crop Protection Council, United Kingdom.