The following information was generated from the
Hazardous Substances Databank (HSDB),
a database of the National Library of Medicine's TOXNET system
(http://toxnet.nlm.nih.gov) on December 4, 2003.

Query: The chemical name dimethoate was identified.
The following terms were added from ChemIDplus:
phosphamide
systoate
systemin
sinoratox
roxion
rogor p
rogor l
rogor
rebelate
racusan
phosphamid
perfekthion
lurgo
fostion mm
fosfotox r
fosfotox
fortion nm
dimevur
dimeton
dimethogen
CAS Registry Number: 60-51-5

1
NAME: DIMETHOATE

HSN: 1586

RN: 60-51-5

HUMAN HEALTH EFFECTS:

HUMAN TOXICITY EXCERPTS:
... SPLASH OF INSECTICIDE LIQ CONTAINING BOTH DIMETHOATE &

CARBARYL

INTO EYES NO SERIOUS DAMAGE DEVELOPED, ONLY TRANSIENT INJURY OF CORNEAL

EPITHELIUM, ACCOMPANIED BY MUCH SWELLING OF LIDS, BUT THIS ALL CLEARED

RAPIDLY. [Grant, W.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986. 347]**PEER REVIEWED**

The cytotoxic, cytostatic, and cytogenetic effects of 14 organophosphate pesticides on human lymphoid cells in vitro were studied. The compounds tested were azodrin, diazinon, dichlofenthion, dimethoate, dursban, ethion, fenthion, malathion, methyl parathion, parathion, phorate, phosdrin, R-1303, and viozene. Cultures of human lymphoid LAZ-007 cells were exposed to the test compounds at concentrations of 0.02, 0.2, 2.0, or 20 ug/ml for 48 hr with or without metabolic activation by rat liver microsomal S9 product. When dimethoate was admin at concentration of 20 ug/ml, the cell viability count was 53% of control & the 2 highest concn significantly increased frequency of sister chromatid exchanges. Significant enhancement (1% level) in the frequency of sister chromatid exchanges was caused by 20 ug/ml of metabolic activated dimethoate. [Sobti RC et al; Mutat Res 102 (1): 89-102 (1982)]**PEER REVIEWED**

Acute intoxication in 6 longshoremen exposed to Vantal (DDT and dimethoate) is described. A number of pathologic conditions were documented from gastritis to duodenal ulcer. Three subjects out of six showed alterations in EEG and EMG. [Biscaldi G et al; G Ital Med Lav 4 (4-5): 203-6 (1982)]**PEER REVIEWED**

SYMPTOMATOLOGY: 1. Nausea is often the first symptom, followed by vomiting, abdominal cramps, diarrhea, and excessive salivation (sialorrhea). Hypothermia has been reported ... at least once in man as an early sign. 2. Headache, giddiness, vertigo, and weakness. 3. Rhinorrhea and a sensation of tightness in the chest are common in inhalation exposures. 4. Blurring or dimness of vision, miosis (with fixed pinpoint pupils), tearing, ciliary muscle spasm, loss of accommodation, and ocular pain. None of these eye effects are diagnostically dependable except in primary ocular exposures. ... 5. Bradycardia or tachycardia. Varying degrees of AV heart block are described, as well as atrial arrhythmias. 6. Loss of muscle coordination, slurring of speech, fasciculations and twitching of muscles (particularly of the tongue and eyelids), and generalized profound weakness. 7. Mental confusion, disorientation, and drowsiness. 8. Difficulty in breathing, excessive secretion of saliva and of respiratory tract mucus, oronasal frothing, cyanosis, pulmonary rales

and rhonchi, and hypertension presumably due to asphyxia. 9. Random jerky movements, incontinence, convulsions and coma. 10. Death primarily due to respiratory arrest arising from failure of the respiratory muscles, intense bronchoconstriction, or all three. /Parathion/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. III-340]**PEER REVIEWED**

A 68 year old male attempted suicide by drinking three ounces of a concentrated Cygon 2-E (23.4% dimethoate). The individual was immediately brought to the hospital, and responded to standard treatment (ipecac, activated charcoal, 2-PAM, and atropine), and was transferred from the intensive care unit to general care 24 hr after the exposure. Within 8 hr of the transfer, he relapsed ... requiring 5 mg of atropine every 10 minutes for 24 hr, before starting on atropine drip (0.5-2.4 mg/kg/hr) for five weeks. The individual required a total atropine dose of 30 g, the largest amount reported to have been given to a human. Although serum acetylcholinesterase activities gradually increased they were not found to be helpful in determining when the atropine drip could be stopped. Control of hypersecretions served as the best monitoring parameter for titration of the atropine drip rate. The patient recovered completely with the exception of a detectable sensorineural hearing deficit, a slight, non-specific personality change, and minimal spastic rigidity thought to be secondary to several anoxic episodes. [LeBlanc FN et al; J Toxicol Clin Toxicol 24 (1): 69-76 (1986)]**PEER REVIEWED**

The symptoms of dimethoate poisoning are similar to those of poisoning by other organophosphorus insecticides, but the clinical picture evolves much more slowly. [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 1641]**PEER REVIEWED**

Dimethoate was administered orally 5 days/week. The number of subjects was 12 at 0.068 mg/kg/day for 28 days (duration of test), 9 at 0.202 mg/kg/day for 39 days, 8 at 0.434 mg/kg/ day for 57 days, 6 at 0.587 mg/kg/day for 45 days and 6 at 1.02 mg/kg/day for 14 days. Whole blood cholinesterase activity and erythrocyte cholinestrase activity were inhibited (as compared with pretreatment levels) only at dosages greater than or equal to 0.434 mg/kg/day. At 0.434 mg/kg, the downward trend in activity started at 20 days of treatment and continued to the end of the 57 day test period. At the two higher dosages, this effect was seen earlier and progressed more rapidly. No localized gastrointestinal effects or any other signs of toxicity were noted. [Edson EF et al; Br Med J 4 (2): 554 (1967) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate

(Final) p.23 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

Cholinesterase activity measurements for 542 California agricultural pesticide applicators under medical supervision during the first 9 mo of 1985 were analyzed. Medical records of applicators were used if the subject had been exposed for over 3 hr in a 30 day period to category I and II organophosphate and carbamate pesticides. Employers of all workers with cholinesterase activity depressions that fell to 70% or less of the workers's plasma or RBC baselines were contacted to obtain a list of pesticides handled in the 2 wk interval preceding the greatest reported cholinesterase activity depression. In evaluating pesticide exposure data, it was not possible to distinguish listed pesticides primarily or cumulatively responsible for the noted cholinesterase activity depressions from those not responsible for the cholinesterase activity depression, but coincidentally used during the same period. The pesticides associated with plasma or RBC cholinesterase activity depression to 70% of baseline or lower were listed. Dimethoate usage in California for 1985 was 361,400 lb. The frequency of dimethoate by % of baseline to depress cholinesterase activity was 0 for plasma (< 50% of baseline) and 4 and 2 for RBC (< 70% and 60% of baseline, respectively). Twenty six workers, 4.8% of the sample, had cholinesterase values at or below the California action limit value for removal from continued exposure to cholinesterase inhibiting pesticides. Eight of these 26 workers, 31.5%, had pesticide related illnesses. [Ames RG et al; Am J Ind Med 15 (2): 143-50 (1989)]**PEER REVIEWED**

Dimethoate ... is an indirect-acting anticholinesterase. [Sullivan, J.B. Jr., G.R. Krieger (eds.). Hazardous Materials Toxicology-Clinical Principles of Environmental Health. Baltimore, MD: Williams and Wilkins, 1992. 1020]**PEER REVIEWED**

Erythrocyte acetylcholinesterase is more sensitive to inhibition by dimethoate than plasma pseudocholinesterase ... . [Sullivan, J.B. Jr., G.R. Krieger (eds.). Hazardous Materials Toxicology-Clinical Principles of Environmental Health. Baltimore, MD: Williams and Wilkins, 1992. 1020]**PEER REVIEWED**

Symptoms and signs of acute dimethoate poisoning: 1. Mild - headache, dizziness, weakness, anxiety, miosis, impairment of visual acuity 2. Moderate - nausea, salivation, lacrimation, abdominal cramps, vomiting, sweating, slow pulse, muscle tremors 3. Severe - diarrhea, pinpoint and nonreactive pupils, respiratory difficulty, pulmonary edema, cyanosis, loss of sphincter control, convulsions, coma and death /from table/ [Zenz,

C., O.B. Dickerson, E.P. Horvath. Occupational Medicine. 3rd ed. St. Louis, MO., 1994 632]**PEER REVIEWED**

Results of mutagenicity tests: positive unscheduled DNA synthesis occurred with SV-40 transformed human fibroblast cell line VA-4 @ 100 & 1000 umol. /from table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.45 (1989)]**PEER REVIEWED**

... Described a case of attempted suicide of a 34-yr-old female who ingested 10 g dimethoate. Half an hour after admission to the hospital, the serum-dimethoate level was 2340 mg/l. Combined hemoperfusion and hemodialysis were applied and, after 18 hr, dimethoate was no longer detected in the serum. [WHO; Environ Health Criteria Number 90: Dimethoate p.51 (1989)]**PEER REVIEWED**

A severe case of poisoning after ingestion of approx 20 g dimethoate was reported ... On admission, the 52-yr-old man was comatose with unmeasurable pseudo-cholinesterase (< 200 U/l). He had been admitted 2 hr after ingestion and received, every 20 min, an injection of 20 mg atropine. Two hemoperfusions with activated charcoal and amberlite were performed, and atropine was given by infusion up to day 12. Twenty-five days after admission, he was discharged, fully recovered. [WHO; Environ Health Criteria Number 90: Dimethoate p.51 (1989)]**PEER REVIEWED**

... Presented a case of a patient who died on the 9th day after dimethoate poisoning with an atypical central neurological disorder. The neuropathological finding, which were similar to those observed in severe forms of Wernicke's encephalopathy, included severe hemorrhagic necrosis of the walls of the ventricles. The authors suggested that the increased level of acetylcholine in the brain had led to thiamine depletion in the regions of predilection of Wernicke's encephalopathy. [WHO; Environ Health Criteria Number 90: Dimethoate p.51 (1989)]**PEER REVIEWED**

... reported an increase in the frequency of breaks and stable chromosome aberrations in 2 patients who died after dimethoate intoxication. [WHO; Environ Health Criteria Number 90: Dimethoate p.51 (1989)]**PEER REVIEWED**

Thirty firemen, exposed to dimethoate in the air as a result of an accident in a dimethoate-manufacturing plant, developed symptoms of intoxication ... Peripheral blood lymphocytes from 20 of these workers were examined for the frequency of sister chromatid exchanges, 2 months after the accident. The frequencies were 9.2 + or - 0.2 for the exposed

and 8.5 + or - 0.2 for unexposed persons (P < 0.05). Dicentric chromosomes and a low frequency of chromatid breaks were found in 2 exposed workers. It is not certain to what the firemen were exposed besides dimethoate. [WHO; Environ Health Criteria Number 90: Dimethoate p.52 (1989)]**PEER REVIEWED**

The results of a number of oral studies in which human volunteers, without occupational exposure to organophosphates, were given dimethoate. ... From these studies it can be concluded that repeated doses of up to 0.2 mg/kg bw did not inhibit cholinesterase activity in the blood. [WHO; Environ Health Criteria Number 90: Dimethoate p.52 (1989)]**PEER REVIEWED**

A 28 year old farmer, who reportedly had worn protective rubber clothing and equipment, had sprayed olive trees with dimethoate the day before he was hospitalized. The man began to experience profound weakness, faintness, and somnolence, followed by attempts to vomit, chills, and profound prostration on the day he was hospitalized. His general condition was found to be serious; his pulse was weak, he exhibited pronounced myosis, vomited and sweated profusely, and had pronounced inhibition of cholinesterase activity. Treated with large doses of atropine (20 mg on day 2, 12 mg on day 3, and 5 mg until day 9), prednisone, analgesics, and penicillin, he recovered. [WHO; Environ Health Criteria Number 90: Dimethoate p.52 (1989)]**PEER REVIEWED**

A case of poisoning in a woman working in agriculture and exposed to dimethoate in the field, 2 days after spraying, was reported. ... Within 3-3.5 hr after beginning work, the woman noted an unpleasant odor recognized as dimethoate. She developed headache, dry cough, dyspnoea, nausea, vomiting and was admitted to hospital in a somnolent state with muscular fibrillations and an asthmatic component. After treatment with saline soln, glucose, caffeine, atropine, and insulin, the state of acute intoxication was overcome. Allergic symptoms were treated with dimedrol. [WHO; Environ Health Criteria Number 90: Dimethoate p.52 (1989)]**PEER REVIEWED**

Contact allergy due to dimethoate was reported in a 53-year-old female. She had a positive skin test ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.52 (1989)]**PEER REVIEWED**

Female greenhouse workers working with dimethoate were reported to have a high percentage of specific leukocyte agglomeration, a raised index of lymphoblastic transformations, and antibodies against dimethoate ... With increasing duration of work, progressive sensitization towards the

pesticide was observed. [WHO; Environ Health Criteria Number 90: Dimethoate p.54 (1989)]**PEER REVIEWED**

On the basis of epidemiological observations and of dermal testing of workers with a 1-2% soln of dimethoate ... concluded that the index of sensitization was very low for dimethoate and that general intoxication might occur, but rarely contact eczema. [WHO; Environ Health Criteria Number 90: Dimethoate p.54 (1989)]**PEER REVIEWED**

For 21 days one adult ingested dimethoate at the rate of 18 mg/day (about 0.26 mg/kg) and another ingested 9 mg/day; neither showed any cholinesterase inhibition. For 4 weeks 20 adults ingested 2.5 mg/day (about 0.04 mg/kg), again with no toxic effect and no inhibition of cholinesterase... actual doses of 7, 21, 42, 63, and 84 mg were administered 5 days per week so that the average intakes were 5, 15, 30, 45, and 60 mg/day, respectively for the men and women in different groups. Twelve persons who received 5 mg/ day for 28 days and 9 persons who received 10 mg/day for 39 days showed no significant change in plasma or erythrocyte cholinesterase activity. Eight persons who received 30 mg/day began to show a decrease in cholinesterase activity by day 20 and the depression lasted until the end of the test on day 57. Depression of cholinesterase occurred earlier and to a somewhat greater degree in groups of six persons, each of whom received 45 and 60 mg/day, respectively. None of the volunteers experienced any clinical effect from the dimethoate. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

After he had worked for 2 weeks picking hops previously treated with dimethoate, a 16 year-old boy developed weakness, nausea, headache, and severe depression. The depression was characterized by psychomotor retardation, inability to concentrate, suicidal thoughts, guilt, loss of interest, and anxiety. Laboratory study revealed (in addition to moderate inhibition of cholinesterase activity consistent with the patient's history of exposure): indirect bilirubin, 3.08 mg%; SGOT, 85 units; SGPT, 60 units; and cadmium, 2+. Recovery was said to coincide with the normalization of cholinesterase activity in 2.5 months. Apparently, no treatment for poisoning by an organic phosphorus compound was needed or administered; no effort was made to account for the reportedly increased cadmium level; and no diagnostic study was directed toward the common causes of hepatitis in young people. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

A suicide accomplished by drinking a large amount of dimethoate illustrated the possibility of relapse. The 51 year old man who drank the compound was taken promptly to a hospital where his stomach was washed out at 2:00 p.m. Vomiting, apparently his greatest difficulty during the afternoon, stopped at 7:00 p.m. Next morning his general condition appeared quite good. He retained food but had several loose bowel movements. He suddenly collapsed at 12:15 p.m. while having lunch. When a physician arrived, the patient was unconscious, his pupils were pinpoint, and coarse ronchi and rales were present in his chest. Emergency treatment was started, but he died at 12:30 p.m. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

Objectives: To estimate the frequency of the intermediate syndrome in organophosphorus-poisoned patients, and examine its relationship to cholinesterase inhibition and electromyographic findings. Muscle biopsies were available in some patients. Design: A 3 yr prospective study. Setting: University teaching hospital intensive care unit. Patients: Consecutive patients with acute organophosphorus poisoning (n = 19). Measurements and Main Results: We determined the frequency of the intermediate syndrome in poisonings with various organophosphates, duration of (acetyl) cholinesterase inhibition and metabolite excretion, evolution of alterations on repetitive nerve stimulation, type and frequency of muscle lesions. A total of eight of 19 patients developed an intermediate syndrome. In some patients, short relapses of muscarinic symptoms superimposed on the intermediate syndrome. Agents such as methylparathion, fenthion, and dimethoate carry a high risk, but we also noted a prolonged intermediate syndrome in an ethyl-parathion-poisoned patient. Prolonged and severe cholinesterase inhibition occurred during the intermediate syndrome in all patients, and metabolite excretion was prolonged. As the intermediate syndrome evolved, repetitive nerve stimulation initially demonstrated decrement, then increment, and finally, normal responses. Necrotic fibers were noted in muscle biopsies, but these fibers were two sparse to explain severe muscle weakness and were similar in patients with and without the intermediate syndrome. No patients developed delayed neuropathy. Conclusions: The intermediate syndrome is not rare. Although it is more likely to occur with some organophosphates, it is not confined to a few distinct compounds. This syndrome coincides with prolonged cholinesterase inhibition, and is not due to muscle fiber necrosis. When viewed together, the clinical and electromyographic features are best explained by combined pre- and postsynaptic dysfunction of neuromuscular transmission. The intermediate syndrome is not related to an incipient delayed neuropathy. [De Bleecker J et al; Critical Care

Medicine 21 (11): 1706-11 (1993)]**PEER REVIEWED**

Cardiac complications and sudden death can occur after the initial clinical manifestations of toxicity have abated and the patient seems to have recovered from the acute respiratory and neurological symptoms. Clinical studies of cardiac complications associated with organophosphate poisoning are discussed. These have included persons who were accidentally exposed to or deliberately ingested malathion, methyl-parathion, and dimethoate in suicide attempts. Electrocardiographic abnormalities including intraventricular conduction disturbances prolonged Q/T intervals and ST/T changes occurred in 18.5 to 80% of the patients. The electrocardiographic changes generally correlated with the severity of intoxication. Arrhythmias and ventricular fibrillation were conmon clinical occurrences and frequently appeared without warning 1 to 15 days following exposure. Studies of cardiac abnormalities in experimental organophosphate poisoning in laboratory animals have shown that organophosphates induce electrocardiographic changes similar to those seen clinically in poisoning patient. Focal lesions occurring primarily in the left ventricular region were frequently seen. These were more pronounced in animals that also had neurological lesions. The pathophysiology of organophosphate induced cardiotoxicity was considered. Treating cardiac complications associated with organophosphate poisoning was discussed. The primary focus is on overcoming arrhythmias in the acute stage of poisoning utilizing atropine and oxygenation. Electrocardiographicr monitoring is recommended for early detection and treatment of late arrhythmias. [Roth A et al; Chest 103 (2): 576-82 (1993)]**PEER REVIEWED**

HUMAN TOXICITY VALUES:

On the basis of a number of cases, the oral lethal dose for human beings was estimated to be of the order of 50-500 mg/kg bw ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.51 (1989)]**PEER REVIEWED**

SKIN, EYE AND RESPIRATORY IRRITATIONS:

May cause eye irritation. [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

MEDICAL SURVEILLANCE:

Remove from exposure ... /individuals in whom/ cholinesterase in red cells or plasma falls to 50% of normal. May return to work when cholinesterase is again above 75% of normal. [ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 187]**PEER REVIEWED**

In all cases of clinical poisoning with dimethoate and other organophosphorus insecticides, it is essential to maintain general surveillance and cholinesterase and cardiac monitoring for at least 4 days, and longer if necessary, and to adapt general supportive and specific therapy in accordance with the finding. [WHO; Environ Health Criteria Number 90: Dimethoate p.54 (1989)]**PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE:

Hand sprayers involved in spraying dimethoate using two different spraying techniques on crops inside a greenhouse were exposed to dimethoate through respiratory exposure at concns of 0.059 and 0.001 mg/h, and through dermal exposure at concns of 346 and 10.5 mg/h; machine operators were exposed to dimethoate via respiratory and dermal exposure at concns of 0.034 and 0.0007 mg/h, and 29.6 and 1.5 mg/h, respectively(1). In a similar study(3), spraymen were dermally exposed to dimethoate at a concn range of 175.8-8322.1 ug/sq cm/day while respiratory exposure ranged 5.1-19.9 ug/day(3). Tractor operators involved in air blast spraying of citrus trees were dermally exposed to 0.05-23 ug/cm2/hr dimethoate while sitting in either an open tractor, in a cab with open windows, or a tractor with an open-cage canopy(2). Tractor operators were dermally exposed to dimethoate at concns of 0.01-0.81 ug/cm2/hr while sitting in a cab with closed windows(2). Workers are potentially exposed to dimethoate via dermal contact with readily dislodged foliar residues(3). After dimethoate was applied to orange trees at 1.4 kg/ha, potential worker exposure was measured to be 0.65, 0.2, 0.18, 0.029, 0.013 ug/sq cm after 3, 10, 17, 30, and 60 days, respectively(3). After dimethoate was applied to lemon trees at 1.4 kg/ha, potential worker exposure was measured to be 0.16, 0.034, 0.025, 0.008 ug/sq cm after 3, 10, 18, and 45 days, respectively(3). Based on several monitoring studies, the general population may be exposed to dimethoate via consumption of contaminated foods(SRC). [(1) Adamis Z et al; Int Arch Occup Environ Health 56: 299-305 (1985) (2) Carman GE et al; Arch Environ Contam Toxicol 11: 651-9 (1982) (3) Copplestone JF et al; Bull World Health Organ 54: 217-23 (1976) (3) Iwata Y et al; J Agric Food Chem 27: 1141-5 (1979)]**PEER REVIEWED**

Dimethoate can be absorbed by man through the unprotected skin, when inhaled as vapors or airborne droplets, or by ingestion. [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 1641]**PEER REVIEWED**

AVERAGE DAILY INTAKE:

The Average daily intake (AVDI) of dimethoate in 8 population groups in

1982-1984 was determined according to the FDA's monitoring program for chemical contaminants in the U.S. food supply (Total Diet Study or Market Basket Study)(1). In 6-11 month old infants, the AVDI was 7 ng/kg-body weight-per day. In 2 yr old toddlers, the AVDI was 6.4 ng/kg-body weight-per day(1). In 14-16 year old females, the AVDI was 2.9 ng/kg-body weight-per day(1). In 14-16 year old males, the AVDI was 2.5 ng/kg-body weight-per day(1). In 25-30 year old females, the AVDI was 10.1 ng/kg-body weight-per day(1). In 25-30 year old males, the AVDI was 7.7 ng/kg-body weight-per day(1). In 60-65 year old females, the AVDI was 7.9 ng/kg-body weight-per day(1). In 60-65 year old males, the AVDI was 7.8 ng/kg-body weight-per day(1). The AVDI of dimethoate in adult males age 16 -19 as determined by the Total Diet Study was 0.001 and 0.003 ng/kg-body weight-per day for FY80 and FY81/82, respectively(2). The AVDI of dimethoate in toddlers as determined by the Total Diet Study was 0.001 ng/kg-body weight-per day for FY81/82(3). The AVDI estimated from FDA's 1990 Total Diet Study was 0.0081, 0.001, and 0.0023 ug/kg/day in 6-11 month old infants, 14-16 yr old males, and 60-65 yr old females, respectively(4). [(1) Gunderson EL; J Assoc Off Anal Chem 71: 1200-9 (1988) (2) Gartrell MJ et al; J Assoc Anal Chem 69:146-61 (1986) (3) Gartrell MJ et al; J Assoc Anal Chem 69: 123-45 (1986) (4) Winter CK; Rev Environ Contam Toxicol 127: 23-67 (1992)]**PEER REVIEWED**

EMERGENCY MEDICAL TREATMENT:

EMERGENCY MEDICAL TREATMENT:

<p>The following Overview, *** DIMETHOATE ***, is relevant for this HSDB record chemical.

LIFE SUPPORT:

- This overview assumes that basic life support measures have been instituted.

CLINICAL EFFECTS:

0.2.1 SUMMARY OF EXPOSURE

0.2.1.1 ACUTE EXPOSURE

A. The following are general effects due to anticholinesterase activity of the organophosphate class of compounds. Not all of these effects may be documented for dimethoate, but could potentially occur in individual cases.

B. MUSCARINIC EFFECTS (PARASYMPATHETIC) - Bradycardia, hypotension, bronchospasm, bronchorrhea, salivation, lacrimation, diaphoresis, urinary incontinence, vomiting, diarrhea, miosis.

C. NICOTINIC EFFECTS (SYMPATHETIC/MOTOR) - Tachycardia, hypertension, fasciculations, muscle cramps, mydriasis, weakness, respiratory paralysis.

D. CENTRAL EFFECTS - CNS depression, agitation, confusion, restlessness, anxiety, headache, psychosis, delirium, coma, seizures; may be slowly reversible or irreversible.

E. CHILDREN may exhibit different predominant signs/symptoms than adults: CNS depression, stupor, flaccidity, dyspnea, and coma are the most common; classic muscarinic signs may not develop.

F. ONSET may appear within a few minutes or up to 12 hours (rarely longer) after exposure. Cholinergic crisis from lipophilic compounds may be delayed from 2 to 5 days, and can recur up to several weeks after apparent improvement for some compounds (fenthion, fenitrothion, chlorpyrifos). Lethal effects occurred 24 hours after apparent recovery in one dimethoate case; mechanism may be a combination of high lipid solubility and requirement for metabolic activation.

G. Hydrocarbon diluents and/or impurities in formulated pesticides can enhance or contribute to toxicity.

0.2.3 VITAL SIGNS

0.2.3.1 ACUTE EXPOSURE

A. Vital sign changes can include bradycardia or tachycardia, hypotension or hypertension, tachypnea, respiratory paralysis, and fever.

0.2.4 HEENT

0.2.4.1 ACUTE EXPOSURE

A. Miosis, lacrimation, blurred vision, and salivation are common; mydriasis may occur in severe poisonings. Opsoclonus has occurred rarely.

B. Acute glaucoma has been reported from dimethoate poisoning.

0.2.5 CARDIOVASCULAR

0.2.5.1 ACUTE EXPOSURE

A. Bradycardia, hypotension, and chest pain may occur. Tachycardia is also common. Dysrhythmias and conduction defects may occur in severe cases.

0.2.6 RESPIRATORY

0.2.6.1 ACUTE EXPOSURE

A. Dyspnea, rales, bronchorrhea, or tachypnea may occur; with pulmonary edema in severe cases.

B. Bronchospasm may occur in previously sensitized asthmatics or as a muscarinic effect.

C. Acute respiratory insufficiency is the main cause of death in acute poisonings.

0.2.7 NEUROLOGIC

0.2.7.1 ACUTE EXPOSURE

A. Headache, dizziness, muscle spasms and profound weakness are common. Altered level of consciousness, seizures and coma may occur. Seizures may be more common in children.

B. Muscle weakness, fatigability, and fasciculations are common findings and may be delayed in onset by several days. Paralysis may supervene.

C. Peripheral neuropathy (mixed sensory-motor type) may be delayed in onset by 6 to 21 days. Recovery may be slow or incomplete.

0.2.8 GASTROINTESTINAL

0.2.8.1 ACUTE EXPOSURE

A. Vomiting, diarrhea, fecal incontinence and abdominal pain may occur.

B. Acute pancreatitis has been reported from dimethoate poisoning.

0.2.10 GENITOURINARY

0.2.10.1 ACUTE EXPOSURE

A. Increased urinary frequency or, in severe cases, urinary incontinence has occurred.

B. Acute renal failure is a rare complication of severe poisoning.

0.2.11 ACID-BASE

0.2.11.1 ACUTE EXPOSURE

A. Metabolic acidosis has occurred in severe cases.

0.2.13 HEMATOLOGIC

0.2.13.1 ACUTE EXPOSURE

A. Alteration in prothrombin time and/or tendency to bleeding may occur.

0.2.14 DERMATOLOGIC

0.2.14.1 ACUTE EXPOSURE

A. Sweating is a consistent but not universal sign.

B. Dermal sensitization may occur.

0.2.16 ENDOCRINE

0.2.16.1 ACUTE EXPOSURE

A. Hyperglycemia and glycosuria with or without ketosis may occur in severe poisoning.

0.2.17 METABOLISM

0.2.17.1 ACUTE EXPOSURE

A. The hallmark of dimethoate poisoning is inhibition of plasma pseudocholinesterase or erythrocyte acetylcholinesterase, or both.

0.2.18 PSYCHIATRIC

0.2.18.1 ACUTE EXPOSURE

A. Decreased vigilance, hallucinations, defects in expressive language and cognitive function, impaired memory, depression, anxiety or irritability and psychosis have been reported, more commonly in persons having other clinical signs of organophosphate poisoning.

0.2.19 IMMUNOLOGIC

0.2.19.1 ACUTE EXPOSURE

A. Dermal sensitization may occur.

0.2.20 REPRODUCTIVE HAZARDS

A. Sporadic reports of human birth defects related to organophosphates have not been fully verified.

B. One case of spontaneous delivery after acute dimethoate poisoning has been reported.

C. Dimethoate was teratogenic in rats and cats and embryotoxic in mice.

0.2.21 CARCINOGENICITY

0.2.21.2 HUMAN OVERVIEW

A. At the time of this review, no studies were found on the possible carcinogenic activity of dimethoate in humans.

0.2.21.3 ANIMAL OVERVIEW

A. Dimethoate has been carcinogenic in rats by the oral route but was not carcinogenic in mice.

0.2.22 GENOTOXICITY

A. Dimethoate has induced DNA damage, unscheduled DNA synthesis, mutations, chromosome aberrations, sister chromatid exchanges, and other genotoxic events at the chromosomal level in a variety of short-term test systems in vitro or in vivo (RTECS, 1991).

B. There are some equivocal studies suggesting increased chromosome aberrations from occupational exposure. No increase in chromosome aberration frequency was found in lymphocytes of two women after acute suicidal dimethoate ingestion. Inheritance of an amplified CHE gene or inducibility of the gene on chromosome 3 was suggested in a chronically exposed family (Van Bao et al, 1974; Prody et al, 1989).

0.2.23 OTHER

0.2.23.1 ACUTE EXPOSURE

A. Delayed toxicity can occur from acute exposure to dimethoate.

LABORATORY:

A. Dimethoate can be collected from ambient air and quantitatively analyzed.

B. Determine plasma and red blood cell cholinesterase activities. While there may be poor correlation between cholinesterase values and clinical effects, depression in excess of 50% activity is generally associated with severe symptoms. Correlation between cholinesterase levels and clinical effects in milder poisonings may be poor.

C. Monitor pancreatic ezymes, urine output, renal function tests, urinalysis, and EKG in significant poisonings.

D. Monitor arterial blood gases in patients with significant respiratory symptoms following exposure.

TREATMENT OVERVIEW:

0.4.2 ORAL EXPOSURE

A. INDUCING EMESIS IS CONTRAINDICATED - because of possible early onset of respiratory depression and seizures.

B. ACTIVATED CHARCOAL: Administer charcoal as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old.

C. GASTRIC LAVAGE: Consider after ingestion of a potentially life-threatening amount of poison if it can be performed soon after ingestion (generally within 1 hour). Protect airway by placement in Trendelenburg and left lateral decubitus position or by endotracheal

intubation. Control any seizures first.

1. CONTRAINDICATIONS: Loss of airway protective reflexes or decreased level of consciousness in unintubated patients; following ingestion of corrosives; hydrocarbons (high aspiration potential); patients at risk of hemorrhage or gastrointestinal perforation; and trivial or non-toxic ingestion.

D. Suction oral secretions until atropinization.

E. ATROPINE THERAPY - If symptomatic from organophosphate poisoning, administer IV atropine until atropinization is achieved (See details in Treatment Section). ADULT - 2 to 5 mg every 10 to 15 minutes; CHILD - 0.05 mg/kg every 10 to 15 minutes. Atropinization may be required for hours to days depending on severity.

F. PRALIDOXIME (Protopam, 2-PAM): Treat moderate to severe poisoning (fasciculations, muscle weakness, respiratory depression, coma, seizures) with 2-PAM in addition to atropine; most effective if given within 48 hours. May require administration for several days.

1. WHO currently recommends an initial bolus of at least 30 milligrams/kilogram followed by an infusion of more than 8 milligrams/kilogram/hour.
2. ALTERNATIVE DOSE ADULT: 1 to 2 g 2-PAM in 100 milliliters of 0.9% saline infused over 15 to 30 min. Repeat this dose in 1 hr and then every 3 to 8 hr if muscle weakness or fasciculations persist, or begin continuous infusion of 500 mg/hr as a 2.5 percent solution.
3. ALTERNATIVE DOSE CHILD: 20 to 40 mg/kg (max 1 g/dose) infused over 30 minutes as a 5% solution in 0.9% saline. Repeat th1s dose in 1 hr and then every 3 to 8 hr if muscle weakness or fasciculations persist, or begin continuous infusion of 10 to 20 mg/kg/hour.

G. SEIZURES: Administer a benzodiazepine IV; DIAZEPAM (ADULT: 5 to 10 mg, repeat every 10 to 15 min as needed. CHILD: 0.2 to 0.5 mg/kg, repeat every 5 min as needed) or LORAZEPAM (ADULT: 2 to 4 mg; CHILD: 0.05 to 0.1 mg/kg).

1. Consider phenobarbital if seizures recur after diazepam 30 mg (adults) or 10 mg (children > 5 years).
2. Monitor for hypotension, dysrhythmias, respiratory depression, and need for endotracheal intubation. Evaluate for hypoglycemia, electrolyte disturbances,

hypoxia.

H. ACUTE LUNG INJURY: Maintain ventilation and oxygenation and evaluate with frequent arterial blood gas or pulse oximetry monitoring. Early use of PEEP and mechanical ventilation may be needed.

I. HYPERTENSION: Monitor vital signs regularly. For mild/moderate asymptomatic hypertension, pharmacologic treatment is generally not necessary. Sedation with benzodiazepines may be helpful in agitated patients with hypertension and tachycardia. For severe hypertension nitroprusside is preferred. Labetalol, nitroglycerin, and phentolamine are alternatives. See main treatment section for doses.

J. HYPOTENSION: Infuse 10 to 20 mL/kg isotonic fluid. If hypotension persists, administer dopamine (5 to 20 mcg/kg/min) or norepinephrine (ADULT: begin infusion at 0.5 to 1 mcg/min; CHILD: begin infusion at 0.1 mcg/kg/min); titrate to desired response.

K. CONTRAINDICATIONS - Succinylcholine and other cholinergic agents are contraindicated.

0.4.3 INHALATION EXPOSURE

A. INHALATION: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchospasm with inhaled beta2 agonist and oral or parenteral corticosteroids.

B. If respiratory tract irritation or respiratory depression is evident, monitor arterial blood gases, chest x-ray, and pulmonary function tests.

C. Carefully observe patients with inhalation exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary.

D. Suction oral secretions until atropinization.

E. Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

F. CONTRAINDICATIONS - Succinylcholine and other cholinergic agents are contraindicated.

0.4.4 EYE EXPOSURE

A. DECONTAMINATION: Irrigate exposed eyes with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or

photophobia persist, the patient should be seen in a health care facility.

B. Patients symptomatic following exposure should be observed in a controlled setting until all signs and symptoms have fully resolved.

C. SUCTION ORAL SECRETIONS - until atropinization.

D. Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

E. CONTRAINDICATIONS - Succinylcholine and other cholinergic agents are contraindicated.

0.4.5 DERMAL EXPOSURE

A. Systemic effects can occur from dermal exposure.

B. Remove contaminated clothing and jewelry; wash skin, hair and nails vigorously with repeated soap washings. Leather absorbs pesticides; all contaminated leather should be discarded. Rescue personnel and bystanders should avoid direct contact with contaminated skin, clothing, or other objects.

C. Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

D. Some chemicals can produce systemic poisoning by absorption through intact skin. Carefully observe patients with dermal exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary.

E. CONTRAINDICATIONS - Succinylcholine and other cholinergic agents are contraindicated.

RANGE OF TOXICITY:

A. Dimethoate has moderate toxicity relative to other organophosphates. Its acute toxicity is variable and depends strongly upon the kinetics of absorption and whether or not metabolic activation is required. Sudden absorption of a less toxic compound may have a more severe effect.

ANTIDOTE AND EMERGENCY TREATMENT:

Therapy for acute insecticide /including dimethoate/ poisoning: 1. Support respiration. Keep airways clear, Use artificial respiration with oxygen if cyanosis is indicated. Death from pesticide poisoning usually is due to respiratory failure. 2. Decontamination as indicated. Remove contaminated clothing. Wash skin, hair, and fingernails with soap and water. Sponge with alcohol. Cleanse eyes. If ingested, lavage stomach with 5% sodium bicarbonate if person is not vomiting. Protect first-aid and medical

personnel! 3. Draw 5 ml heparinized blood for cholinesterase determination. Save samples of first urine and first/early vomitus for possible laboratory analysis. 4. Consult insecticide label under "active ingredients' for specific chemicals involved. 5. When mixtures of organophosphates and chlorinated hydrocarbons are involved ... give specific treatment for organophosphates first and indicated support therapy and decontamination. [Zenz, C., O.B. Dickerson, E.P. Horvath. Occupational Medicine. 3rd ed. St. Louis, MO., 1994 632]**PEER REVIEWED**

Antidote for acute dimethoate poisoning: 1. Adults: After cyanosis is overcome, use atropine sulfate, 2-4 mg iv. Repeat doses at 5- to 10-min intervals until signs of atropinization appear. Maintain for 24 hr or longer if necessary. 2. Children: Atropine sulfate in proportion to body weight: approx 0.05 mg/kg. 3. Support atropine treatment with 2-PAM (pralidoxime chloride) ... Adult dose: 1 g, slowly, intravenously; Infants: 0.25 g, slowly intravenously ... contraindicated are morphine, aminophylline, theophylline, phenothiazine tranquilizers, and barbiturates. /from table/ [Zenz, C., O.B. Dickerson, E.P. Horvath. Occupational Medicine. 3rd ed. St. Louis, MO., 1994 632]**PEER REVIEWED**

Data on the effects of oxime reactivators in dimethoate poisoning are contradictory, some indicating that they may have a negative effect on cholinesterase inhibition ... It is therefore suggested, that if oxime reactivators are indicated, these should be used with caution and under close supervision. [WHO; Environ Health Criteria Number 90: Dimethoate p.54 (1989)]**PEER REVIEWED**

Hemoperfusion may be effective in the early stages of dimethoate poisoning ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.54 (1989)]**PEER REVIEWED**

ANIMAL TOXICITY STUDIES:

NON-HUMAN TOXICITY EXCERPTS:

DIMETHOATE IS PARTICULARLY TOXIC IN AVIAN SPECIES; HOWEVER, IT IS 20 FOLD

MORE TOXIC IN PHEASANTS BECAUSE THE OXYGEN ANALOG IS FORMED AND ACCUMULATES WHICH LEADS TO A SLOWER DEGRADATION TO NON-TOXIC PRODUCTS.

[White-Stevens, R. (ed.). Pesticides in the Environment: Volume 1, Part 1, Part 2. New York: Marcel Dekker, Inc., 1971. 169]**PEER REVIEWED**

IT IS NON-PHYTOTOXIC @ RECOMMENDED RATES EXCEPT TO A FEW CITRUS, FIG,
& NUT VARIETIES. ... NO IRRITATION OBSERVED AFTER APPLICATION OF 130 MG ACTIVE INGREDIENT (AS EMULSIFIABLE CONCENTRATE)/20 SQ CM SHAVED SKIN OF
RABBITS. [Worthing, C.R. and S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 8th ed. Thornton Heath, UK: The British Crop Protection Council, 1987. 298]**PEER REVIEWED**

ERYTHROCYTE CHOLINESTERASE OF RATS & DOGS IS MORE SUSCEPTIBLE TO
DIMETHOATE INHIBITION THAN PLASMA CHOLINESTERASE. [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984.,p. II-297]**PEER REVIEWED**

DOSES OF 40 MG/KG PRODUCED SEVERE TOXIC SYMPTOMS /IN CALVES/ & LEVELS
ABOVE 80 MG/KG WERE LETHAL. SHEEP WERE KILLED IN 10 TO 26 DAYS BY DAILY
ORAL DOSES OF 10 MG/KG; 2 MG/KG DAILY GAVE RISE TO CHRONIC POISONING,
CHARACTERIZED BY DEPRESSION, ANOREXIA, SALIVATION, & DIARRHEA, IN 100
TO 160 DAYS. [Clarke, M. L., D. G. Harvey and D. J. Humphreys. Veterinary Toxicology. 2nd ed. London: Bailliere Tindall, 1981. 150]**PEER REVIEWED**

CYGON 4E CONTAINING 47.3% DIMETHOATE & 52.7% OF UNKNOWN INGREDIENTS
WAS ADMIN ORALLY IN SINGLE DAILY DOSES TO CATS BETWEEN 14TH & 22ND DAY
OF PREGNANCY. 12 MG/KG WAS ASSOC WITH POLYDACTLY IN 8 OF 39 FETUSES
WHEREAS 3 & 6 MG/KG PRODUCED NO TERATOGENICITY OR EMBRYOTOXICITY.
[KHERA KS; J ENVIRON PATHOL TOXICOL 2 (6): 1283-8 (1979)]**PEER REVIEWED**

Mutagenic effects were studied in male CFLP mouse myelocytes in vivo 24 and 48 hours after ip treatment with 60 mg/kg dimethoate. The number of aberrant cells 48 hours after treatment was 51/200 (versus 6/200 in the controls): Chromatid type aberration in 35/200 (versus 4/200) in the controls. [Nehez M et al; Egeszsegtudomany 25 (3): 262-6 (1981)]**PEER REVIEWED**

Dimethoate and its nonalkylating O-demethyl derivative were tested for their ability to induce chromosomal alterations in bone marrow cells of mice after ip administration. A single dose of 60 mg/kg dimethoate increased the aberration rates above those of the controls. Considering the distribution of the several aberration types, the alkylating properties of dimethoate may be only in part responsible for its cytogenetic activity. [Nehez M et al; Regul Toxicol Pharmacol 3 (4): 349-54 (1983)]**PEER REVIEWED**

Commercial preparation of dimethoate was tested for its ability to induce complete and partial chromosome losses in Drosophila melanogaster males. The pesticide did not induce a significant increase in partial chromosome loss. [Woodruff RC et al; Environ Mutagen 5 (6): 835-46 (1983)]**PEER REVIEWED**

The effects of dimethoate were investigated in the mouse after acute (10 mg/kg, ip) or chronic treatment (0.6 ppm in drinking water, 5 days a week for 7 weeks). Dominant lethal mutations were scored for the chronic dose. Chromosome damage was also analyzed in bone marrow and spermatogonial cells at the same dose levels (from 12 to 48 hours after treatment). Methyl methanesulfonate (60 mg/kg, ip) was the positive control. Dimethoate did not show genotoxicity in any of the experiments. [Degraeve N et al; Mutat Res 119 (3-4): 331-7 (1983)]**PEER REVIEWED**

Studies on the carcinogenicity of the insecticide dimethoate in animals were reviewed. Examination of histological sections showed that /tumor incidences at all sites/ as well as malignant neoplasms, were increased in both low and high doses of dimethoate-treated male rats in the National Cancer Institute study. The malignant neoplasms were both carcinomas and sarcomas. Neoplasms of the endocrine organs, particularly carcinomas, were increased in male and female rats given dimethoate. These carcinomas, were observed in the adrenal, thyroid, and pituitary glands. Neoplasms were also increased in the liver of male and female rats and in the reproductive organs of female rats given dimethoate. Male and female rats treated with dimethoate developed monocytic leukemia. There also were toxic changes in rats. Male rats had atrophy of the testes, chronic renal disease, parathyroid hyperplasia, and polyarteritis. In addition, Wistar male and female rats given dimethoate by gavage or intramuscularly developed a significant increase in malignant neoplasms, mainly sarcomas, and granulocytic leukemia. Furthermore, AB male and female mice also had an increased incidence of malignant neoplasms and granulocytic leukemia after dermal applications of dimethoate. [Reuber MD; Environ Res 34 (2): 193-211 (1984)]**PEER REVIEWED**

Oral single and repeated doses of dimethoate affected the activities of serum glutamate oxaloacetate transaminase, glutamate pyruvate transaminase, acid phosphatase, and cholinesterase in rats. The change in these activities depended on the sex and duration of the exposure. None of these insecticides had any effect on the male or female serum bilirubin levels after 4-week exposure. [Enan EE; Int Pest Control 25 (2): 42-4 (1983)]**PEER REVIEWED**

The embryotoxicity of dimethoate (aqueous emulsion) applied externally to mallard (Anas platyrhynchos) eggs was investigated. Abnormalities were observed in bill, brain, joint, & growth in survivors (day 18). Gastroschisis was also noted. [Hoffman DJ, Albers PH; Arch Environ Contam Toxicol 13 (1): 15-27 (1984)]**PEER REVIEWED**

Male mice (Q strain) were given a single ip injection at the maximum tolerated dose of Luxan Tue-Taons (150 g dimethoate & 150 g fenitrothion/l). Luxan Tue-Taons (60 mg/kg) did not induce chromosome aberrations in bone marrow cells, spermatogonia or primary spermatocytes of the mice. No evidence of potential genetic effects was obtained in a dominant lethal mutation assay. [Degraeve N et al; Food Chem Toxicol 22 (8): 683-7 (1984)]**PEER REVIEWED**

Dimethoate was tested for toxicity and mutagenicity using Schizosaccharomyces pombe SP-198 ade 6-60-/rad 10-198/h-. The LD50 was 50 mM. The compound was not mutagenic at doses ranging from 1.3-131 mM. The addition of S-9 mix from a phenobarbital pretreated mouse had no appreciable effect on this result. [Gilot-Delhalle J et al; Mutat Res 117 (1-2): 139-48 (1983)]**PEER REVIEWED**

The induction of sister-chromatid exchanges (SCE) and mitotic delay by eight organophosphorus pesticides was investigated in cultured Chinese hamster cell line V79. Six pesticides, one of which was dimethoate, induced a significant increase of sister-chromatid exchange frequencies in a dose-dependent fashion. The six in decreasing order of sister-chromatid exchanges induction are methyl parathion, demeton, trichlorfon, dimethoate, malathion & methidathion. Cells were exposed to dimethoate for 29 hr at concn of 10, 20, 40, 80 ug/ml, respectively. All test cmpd caused a delay in cell cycle. [Chen HH et al; Mutat Res 88 (3): 307-16 (1981)]**PEER REVIEWED**

A study was conducted on the carcinogenic potential of dimethoate in Wistar rats of both sexes (40 rats/group) following oral or im administration, or in AB mice (19 mice/group) following dermal

application. Rats were treated twice weekly by gavage with 5, 15, or 30 mg/kg, or im with 15 mg/kg. Doses of 30 mg/kg oral and 15 mg/kg im resulted in statistically significant increases in the combined incidence of malignant tumors. Malignant tumors were found in 4/20 rats that had received 30 mg/kg oral. Tumors consisted of one liver sarcoma, one malignant reticulosis, and two sarcomas of the spleen. The im dose of 15 mg/kg body weight resulted in the production of one sarcoma of the spleen, one soft tissue sarcoma, one ovarian sarcoma, one reticulum cell sarcoma, one spleen sarcoma, and one liver sarcoma, for a combined incidence of 6/30. Incidences of tumors in the oral and im control groups were 0/36, 0/35, respectively. ... Five of 19 mice developed malignant tumors (4 leukoses and 1 breast carcinoma). Only fibroadenoma of the breast was reported in the control animals. Although incidence of malignant tumors at all sites was significant ... the absence of a positive response at any one site or type makes the evidence for carcinogenicity only weakly suggestive. [Gibel WKL et al; Arch Res 41 (4): 311-28 (1973) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.12 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

The organophosphorus insecticide dimethoate was tested for the induction of genetic damage in male germ cells of Drosophila melanogaster. Sex linked recessive lethals, sex-chromosome loss and non-disjunction induction were studied following different routes of administration: adult feeding, injection and larval feeding. The results /indicated/ that after injection, dimethoate induces a slight but significant increase in the frequency of point mutations. [Velazquez A et al; Mutat Res 172 (3): 237-43 (1986)]**PEER REVIEWED**

Genotoxicity of some organophosphorus pesticides in the Ames Salmonella assay was determined in a quantitative dose-dependent manner. ... Dimethoate (technical) was found to be a weak mutagen and caused base changes in DNA. [Vishwanath R, Jamil K; Indian J Exp Biol 24 (5): 305-8 (1986)]**PEER REVIEWED**

The 30 day estimated estimated minimum lethal dose for mallards of both sexes is 6.0 mg/kg/day. ... The 30 day estimated minimum lethal dose for 20 to 25 week old pheasants of both sexes appears to fit between 4.00 and 10.00 mg/kg/day. ... Acetylcholinesterase measurements were made on the brains of ... /the animals that died/ and survivors of the pheasant 30 day sMLD test. When compared with their estimated minimum lethal dose controls, survivors which were sacrificed on the day following final treatment showed 71.7% inhibition. When compared with their controls, mortalities showed 88.0% inhibition. [U.S. Department of the Interior,

Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

Signs of intoxication /in mallards & pheasant admin acute dosages of technical grade dimethoate/: Feathers drawn tightly to the body, mild tachypnea, ataxia, tenseness, fluffed feathers, imbalance, running and falling, tremors, clonic convulsions, and immobility, myasthenia. Signs appeared as soon as 13 min & ... /deaths/ occurred overnight after treatment. [U.S. Department of the Interior, Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

A bioassay of the carcinogenicity of technical-grade dimethoate was conducted using Osborne-Mendel rats and B6C3F1 mice. The test material was administered in feed to groups of 50 rats of each sex at either of two concentrations for 80 weeks, followed by 35 weeks of obervation. The "time- weighted average doses" for rats were 155 and 310 ppm for males and 192 and 384 ppm for females. All surviving rats were killed between 113 and 115 weeks. Dimethoate was administered in feed to groups of 50 male and 50 female mice at two concentrations. Female mice received diets containing 250 and 500 ppm of dimethoate for 80 weeks; male mice received the same dosage. However, high-dose males were returned to the control diet at 60 weeks, and low dose males at 69 weeks. All surviving mice were killed between 93 and 94 weeks. Tremors and hyperexcitability, both indications of dimethoate toxicity, were observed in the treated animals. Pathologic evaluation revealed no significant increase in tumors associated with dimethoate treatment in either species of animal, and it is concluded that there was no carcinogenic effect under the conditions of the experiment. [NCI; Toxicology and Carcinogenesis Studies of Dimethoate p.V Report No. 4 (1977) NIH Pub No. 77-804]**PEER REVIEWED**

Dimethoate (as cygon 4E, 47.3% dimethoate, 52.7% unspecified ingredients) was tested for teratogenicity in groups of 20 female Wistar rats. Three, 6, 12, or 24 mg cygon 4E in corn oil/kg bw/day was administered by gavage on days 6-15 of gestation. The control group received corn oil only. The two highest doses produced a significant increase in the incidence of wavy ribs. No significant fetal toxicity was observed. [Khera KS et al; Bull Environ Contam Toxicol 22 (4-5): 522-9 (1979) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.21 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

In a 5-generation chronic study, groups of 14 female and 10 male CD-1 mice were maintained on 60 ppm dimethoate in the drinking water. The actual dosage of dimethoate was reported to be 9.5-10.5 mg/kg/day. This treatment had deletrious effects on reproductive performances (vide supra) and resulted in increased mortality of the pups during the first week after birth; however, no histopathlogical changes were observed in the ovaries, testes, liver or kidneys of treated rats. [Budreau CH, Singh RP; Toxicol Appl Pharmacol 26: 29-38 (1973) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.21 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

Dimethoate was tested at five doses ranging from 1-6 mM in an Escherichia coli system using induction of 5-methyltryptophan resistance as the measured endpoint. Under these conditions, dimethoate was mutagenic with a potency similar to that of bidrin, but less than dichlorvos and much less than N-Methyl-N'-nitro-N-nitrosoguanidine, a known powerful mutagen. [Mohn G; Mutat Res 20: 7-15 (1973) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.15 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

Dimethoate tested at doses ranging from 0-5000 ug/plate, for mutagenicity in Escherichia coli strain WP2 hor and Salmonella typhimurium strains TA1535, TA100, TA1538 and TA98. Positive results were obtained in strains WP2 hor and TA100. [Moriya M et al; Mutat Res 116 (3-4): 185-216 (1983) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.18 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

Cultures of the holotrichous ciliate, Tetrahymena pyriformis, were treated with 0, 1, 10, 50, and 100 ug/ml dimethoate. Cell number was estimated each day for 5 days, and organisms were also examined microscopically. Dimethoate produced approx 84% maximum growth inhibition, within 2 days. At 100 ug/ml, dimethoate triggered a general mucocyst discharge in Tetrahymena. Cells became round and swollen, and finally burst. [Kumar S, Lal R; Environ Pollut 57 (4): 275-80 (1989)]**PEER REVIEWED**

Acute toxicity tests of dimethoate were performed using less than 24 hour old Daphnia magna according to Commission of the European Communities guidelines. Static tests were conducted either in 50 ml beakers filled with 50 ml test solution (open system) or in 100 ml stoppered glass bottles containing 100 ml of test solution (closed system). All acute and chronic test results were reported in mg/l. The 48 hr LC50 for > 99% dimethoate were 1.7 (open) and 2.0 (closed); the 48 hr EC50, 1.5 (open) and 1.8 (closed); the 48 hr no observed lethality concn, 0.6 (open) and

0.9 (closed); the 48 hr no observed effect concentration, 0.4 (open) and 0.9 (closed). Chronic toxicity was determined from a 23 day renewal static test with duplicated runs. The daphnids were housed individually in 50 ml beakers filled with 50 ml test solution. The test solution was changed on days 2, 5, 7, 9, 12, 14, 16, 19, and 21. The 23 day EC50 for > 99% dimethoate were 0.23 and 0.11; the 23 day EC50, 0.19 and 0.11; the 23 day no observed lethality concn 0.17 and 0.08; and the 23 day no observed effect concn, 0.10 and 0.08. [Beusen JM, Neven B; Bull Environ Contam Toxicol 42 (1): 126-33 (1989)]**PEER REVIEWED**

The comparative ability of dimethoate to inhibit the growth of repair proficient (rec+ hrc+) and repair deficient (rec- and rec- her-) strains of Proteus mirabilis was investigated. Dimethoate (10 mg in 0.1 ml ethanol/plate) resulted in a 41% increase in the size of the zone of inhibition when the rec+ hrc+ strain was compared with the rec- her- strain. [Adler BR et al; Biol Zentralbl 95: 463-69 (1976) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final) p.18 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

The addition of dimethoate to soil at 10 or 100 mg/kg did not result in significant differences in the number of bacteria solubilizing tricalcium phosphate or in the number of bacteria mineralizing calcium glycerophosphate, but an increase in the population of phospholipase-producing organisms solubilizing lecithin occurred ... At 10 mg/kg, an increase in carbon dioxide production occurred for 2 wk after treatment, followed by a decrease to control levels. At 100 mg/kg, the increase in carbon dioxide output was slower and longer. [WHO; Environ Health Criteria Number 90: Dimethoate p.27 (1989)]**PEER REVIEWED**

Exposure /of Channa punctatus/ for 24 hr, 96 hr, or 14 days to dimethoate concn of 10.8, 8.0, or 5.0 mg/l, respectively, produced moderate vacuolation of the liver and a high degree of cytoplasmic granulation, which developed for up to 96 hr of exposure. The 14-day exposure added little in the way of vacuolation or granulation ... The hematological response to dimethoate included reduced erythrocyte counts and hemoglobin concn, an elevated mean corpuscular hemoglobin and color index indicating that the insecticide exerted an effect similar to the production of anemia ... The signs of the toxicity of dimethoate in fish (Channa punctatus) included jumping, erratic movement, imbalance, and death ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.27 (1989)]**PEER REVIEWED**

Dimethoate inhibited AChE activity in the brain, liver, and muscle of some

fresh-water teleosts (Channa gachua and Cirrhina mrigala), exposed to sublethal concn of 35% EC formulation (0.9-2.4 and 0.6-1.6 mg/l, respectively) ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.27 (1989)]**PEER REVIEWED**

... reported that acute (5-hr) and short-term (up to 32 days) exposure of the fish, Channa gachua, to dimethoate @ 6.2 mg/l and 1.5 mg/l, respectively, produced histological changes in the gills. On acute exposure, there was erosion at the distal end of the gill filaments and loss of cell membrane. With exposure to a concn of 1.5 mg/l, the basement membrane started separating, and the damage to the gill was found to be more significant with increasing exposure time, with vacuolization occurring after 32 days. [WHO; Environ Health Criteria Number 90: Dimethoate p.27 (1989)]**PEER REVIEWED**

The exposure of the fish Heteropneustes fossilis to a dimethoate concn of 10 mg/l led to an increased level of glycogen by the end of the second week in both the liver and the kidney, and to a slight decrease in the protein contents at the end of the eighth day ... A sharp rise in the activity of succinate dehydrogenase in both organs was noted during the first two wk of this study. [WHO; Environ Health Criteria Number 90: Dimethoate p.29 (1989)]**PEER REVIEWED**

Dimethoate at a concn of 0.05 mg/l produced morphological changes in the melanophores of Bufo melanostictus tadpoles and an increase in pigmented areas of the skin ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.29 (1989)]**PEER REVIEWED**

Dimethoate had a very low toxicity for some aquatic organisms in Sudan, such as Oreochromis niloticus, Gambusia affinis, Pseudagrion spp., Crocothemis erythraea, and Lanistes carinatus. Under laboratory conditions, it did not kill any animal at concn lower than 80 mg/l ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.29 (1989)]**PEER REVIEWED**

The toxicity of dimethoate for 11 freshwater species was studied ... The relative susceptibility tests indicated that Daphnia magna was the organism most sensitive to dimethoate, while the microorganisms P.fluorescens, M.aeruginoso, and S.pannonicus were generally less sensitive indicators of toxicity. The susceptibility of aquatic species to a chemical may vary by more than two-three orders of magnitude. The data demonstrate that the sublethal criteria studied were not necessarily the most sensitive toxicological criteria. [WHO; Environ Health Criteria

Number 90: Dimethoate p.29 (1989)]**PEER REVIEWED**

Dimethoate was only slightly repellent to foraging honey-bees. The self-limiting dose for foraging was 20-25 times the lethal oral dose (2.9-3.9 ug/bee versus 150 ng/bee) ... Residual toxicity has been supported by several observations. Nectar from plants sprayed with 0.1% dimethoate was lethal for honey-bees for at least 2-3 days ... or 10 days ... also showed the possible toxic levels of residues in the nectar for up to 10 days after treatment of lemon trees with dimethoate at a rate of 1.12 kg ai/ha. The high bee mortality observed, immediately after treatment, was attributed to dimethoate residues on the plant surface. [WHO; Environ Health Criteria Number 90: Dimethoate p.32 (1989)]**PEER REVIEWED**

Hens did not show any evidence of delayed neurotoxicity ... The effect of dimethoate on esterase levels following the oral dosing of pheasants and following long-term feeding to pheasants and pigeons was investigated ... Dimethoate inhibited brain-alpha-naphthyl acetate esterase more than brain-cholinesterase and triacetin esterase in acute studies ... A characteristic of dimethoate was the elevation of phenyl benzoate esterase levels, showing that after initial liver damage, dimethoate is able to induce certain enzymes. [WHO; Environ Health Criteria Number 90: Dimethoate p.32 (1989)]**PEER REVIEWED**

No visible signs of intoxication were seen in horse receiving dimethoate orally at doses of 25 or 50 mg/kg. Single doses of dimethoate at 40 mg/kg were effective in removing Gasterophilus spp. from infected horses, but toxic signs appeared in animals treated with higher levels of 60-80 mg/kg ... Mild signs of intoxication occurred in sheep at 75 mg/kg, including slight salivation, lachrymation, transitory diarrhea, rhinitis, and anorexia. Doses lower than 15 mg/kg were essentially asymptomatic in calves. The data with dimethoate indicate an appreciable margin of safety between the lowest dose that kills first instar Hypoderma lineatum (5 mg/kg), and the doses that produce mild toxicity (15-20 mg/kg), or severe, reversible toxicity (40 mg/kg) ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.33 (1989)]**PEER REVIEWED**

... described cases of suspected dimethoate intoxication in cattle grazing on pasture that had been sprayed and a poor response to atropine treatment. Chemical analysis of liver, kidney, and brain tissue did not reveal any organophosphorus cmpd or metabolites. Whole blood-ChE was depressed in 3/14 animals. [WHO; Environ Health Criteria Number 90: Dimethoate p.33 (1989)]**PEER REVIEWED**

After spraying barns (for calves) and pigsties with dimethoate, only 16-29% of the initial concn still persisted after 8 wk. Nevertheless, the animals showed a decrease in ChE ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.33 (1989)]**PEER REVIEWED**

A single dose of 300 mg technical dimethoate/kg bw did not cause skin irritation in male and female rabbits ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.36 (1989)]**PEER REVIEWED**

... Dimethoate did not have any irritant effect on the rabbit eye after introduction of 10 mg of dry material into the conjunctival sac. [WHO; Environ Health Criteria Number 90: Dimethoate p.36 (1989)]**PEER REVIEWED**

The effects on experimental animals of repeated oral or inhalation exposure to dimethoate ... In the various studies, which ranged from 5 1/2-12 mo in duration, inhibition of cholinesterase (ChE) in the erythrocytes was a more sensitive indicator of exposure to dimethoate than ChE inhibition in plasma. ChE activity in the brain was measured in one study only. [WHO; Environ Health Criteria Number 90: Dimethoate p.37 (1989)]**PEER REVIEWED**

In a study ... no effects were observed on ChE inhibition in rats administered dimethoate in the diet @ 32 mg/kg. In their first study (12 mo) on rats, ... observed inhibition of ChE in erythrocytes @ 50 mg/kg diet, but not @ 10 mg/kg. In the second study (5 1/2 mo), inhibition of ChE in erythrocytes was found at both 20 and 10 mg/kg, but not @ 5 mg/kg ... /different/ studies ... did not show any inhibition of blood-ChE in rats administered a 40% formulation of dimethoate @ 0.5-1 mg/kg bw, corresponding to 0.2-0.4 mg dimethoate/kg bw. From all available data on the rat, a dietary level of dimethoate of 5 mg/kg, corresponding to 0.25 mg/kg bw, can be considered as the no-observed-adverse-effect level. [WHO; Environ Health Criteria Number 90: Dimethoate p.37 (1989)]**PEER REVIEWED**

From limited studies on the dog ... it can be concluded that a level of 10 mg dimethoate/kg diet, corresponding to 0.25 mg/kg bw, does not result in ChE depression in erythrocytes. [WHO; Environ Health Criteria Number 90: Dimethoate p.37 (1989)]**PEER REVIEWED**

ChE inhibition was not observed in an inhalation study in which rats were exposed for 14 hr/day, over 3 mo, to 0.01 mg dimethoate/cu m (measured concn) ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.42

(1989)]**PEER REVIEWED**

Ip administration of 40 mg dimethoate/kg bw, given as a single dose on the day of mating or on the 9th day of gestation, or given for the first 14 days of gestation in mice, caused a high incidence of embryonal loss ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.42 (1989)]**PEER REVIEWED**

Cygon 4E (containing 47.3% dimethoate) was given to female rats by intubation from the 6th to the 15th day of gestation @ dose levels of 3, 6, 12, or 24 mg/kg bw. The 24 mg/kg dose was toxic for the dams (8/20 manifested clonic spasms and muscular tremors during the treatment period, 7 recovered, and 1 died on the 16th day of pregnancy). Doses of 12 and 24 mg/kg were associated with an increase ($P < 0.05$) in the numbers of anomalous litters (each having at least one anomalous fetus) and wavy-ribbed fetuses. The 3 and 6 mg/kg doses (equal to 1.42-2.84 mg dimethoate/kg) did not produce any evidence of teratogenicity or embryotoxicity in the rats ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.43 (1989)]**PEER REVIEWED**

Cygon 4E (47.3% dimethoate) was given to cats in gelatin capsules @ doses of 3, 6, or 12 mg/kg on the 14th-22nd days of pregnancy. At the levels tested, the cmpd did not produce any effects on the incidence of pregnancy. In the 12 mg/kg group, forepaw polydactily was observed in 8 out of 39 fetuses. Cygon 4E @ 3 or 6 mg/kg (1.42-2/84 mg dimethoate/kg) did not produce any effects ... (the effects observed in ... investigations may be due to the other components in the formulation). [WHO; Environ Health Criteria Number 90: Dimethoate p.43 (1989)]**PEER REVIEWED**

... Reported that dimethoate administered orally was not teratogenic in CD-1 mice @ dose levels of 10 or 20 mg/kg bw, and that these levels were not lethal to the dams. The two highest dose levels of 40 and 80 mg/kg produced maternal toxicity. [WHO; Environ Health Criteria Number 90: Dimethoate p.43 (1989)]**PEER REVIEWED**

Results of various mutagenicity tests using various concn of dimethoate: positive with Escherichia coli (5-methyltryptophane resistance mutation); negative and positive with Salmonella typhimurium (four different tests); induction of mitotic gene conversions with Saccharomyces cerevisiae; negative with Schizosaccharomyces pombe; negative with Chinese hamster ovary cells (sister chromatid exchange increase); negative unscheduled /DNA synthesis/ with rat hepatocytes culture; negative with Drosophila

melanogaster, and increase in sex-linked recessive lethals at 10 mg/kg not at 20 mg/kg with Drosphila melanogaster; positive with host-mediated assay mouse (Salmonella typhimurium); negative with dominant lethal mutation assay mice strain Q; negative with dominant lethal test in mice; a significant increase in frequency of polychromatic erythrocytes with micronuclei - 0.85% with micronucleus test- mice- bone marrow; positive at increased concn (50 and 100 mg/kg bw) with chromosome abnormalities - mice - bone marrow and increase in number of mitosis, chromatid and chromosome type aberrations; and increase in number of chromatid and chromosomal breaks in Syrian golden hamsters /from table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.44 (1989)]**PEER REVIEWED**

Negative mutagenicity results were reported ... for commercial mixtures of insecticides containing dimethoate. Two formulations were tested: dimethoate + fenitrothion (dose 60 mg/kg bw, corresponding to 9 mg/kg of each) and dimethoate + malathion + methoxychlor (dose 100 mg/kg bw corresponding to 9.5 mg dimethoate/kg). a single ip injection did not induce chromosome aberrations in bone marrow cells, spermatogonia, or primary spermatocytes of the mouse. No significant increases in pre- or post-implantation fetal lethality were observed in a dominant lethal mutation assay. [WHO; Environ Health Criteria Number 90: Dimethoate p.43 (1989)]**PEER REVIEWED**

Male mice (strain AB Jena/Halle, random bred) were injected ip with 14C-methyl-labelled dimethoate @ 0.35 mmol/kg. The extent of methylation was in the range of 1-10 umol N-7 methylguanine/mol guanine; the values in the kidneys were higher than those in the liver. The excretion half-life of N-7 methylguanine was 23-160 hr. [WHO; Environ Health Criteria Number 90: Dimethoate p.43 (1989)]**PEER REVIEWED**

Groups of 40, 10-wk old Wistar rats were injected im with 15 mg dimethoate/kg bw (twice weekly) or with isotonic saline until spontaneous death occurred. The mean life spans were 711 and 570 days in the saline and dimethoate-treated animals, respectively. No malignant tumors occurred in the saline-treated group, but 4/35 animals developed benign tumors. In the dimethoate group, 6/30 rats had malignant tumors (a spleen reticulosarcoma, a spleen fibrosarcoma, an ovarian alveolar sarcoma, a liver hepatocellular carcinoma, a malignant reticuloma, and a soft tissue spindle-cell sarcoma) and 5/30 had benign tumors. The first malignant tumor (a splenic fibrosarcoma) was noted after 410 days ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.47 (1989)]**PEER REVIEWED**

Dimethoate (tech grade, 90-100% pure) was given in the feed to Osborne Mendel rats and to B6C3F1 mice. Groups of 50 male and 50 female rats (10 animals in each control group) received "time-weighted average doses" of 155 or 310 mg/kg for male rats and 192 or 384 mg/kg for female rats. After 80 wk, all groups received control diets, and the studies were concluded at wk 114. Groups of 50 male and 50 female B6C3F1 mice were given dimethoate in the diet at concn of 0 (10 "matched" controls), 250, or 500 mg/kg for 60-69 wk (males) or for 80 wk (females). The studies were ended after 94 wk. Tremors and hyperexcitability were observed in exposed animals; rats and mice that survived to termination were generally in poor condition. Survival was reduced in the high-dose groups of rats. Several non-neoplastic lesions occurred more frequently in dimethoate-exposed animals. No increases in neoplasia were reported to be associated with dimethoate administration, for any of the organs or tissues examined histologically ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.48 (1989)]**PEER REVIEWED**

... New well-performed long-term/carcinogenicity studies in rats and mice were submitted. ... No indication for carcinogenicity was found. In the mouse study, the lowest dose of 25 mg/kg produced decreased body weight, decreased cholinesterase activity in erythrocytes, and also slight extramedullary hematopoiesis in the spleen. ... Administration of dimethoate to rats for 2 yr ... also resulted in a decrease in body weight, decreased cholinesterase activity in erythrocytes and the brain, and slight anemia. No effects were observed at 1 mg/kg in diet (0.05 mg/kg bw) ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.48 (1989)]**PEER REVIEWED**

When administered to rats @ 5-30 mg/kg bw orally or 15 mg/kg im, twice a wk, until death, dimethoate caused hyperplasia in the bone marrow, mainly in granulocytopoiesis ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.49 (1989)]**PEER REVIEWED**

The effects of dimethoate on the heart were investigated in rabbits ... and guinea-pigs and rats ... After oral administration of 150 mg dimethoate/kg to rabbits, the effects observed included bradycardia and increased atrio-ventricular and intraventricular conductance, with complete recovery after 4-7 days. In rats and guinea-pigs, a dose-effect relationship was established for heart rate disturbances, and atrio-ventricular block. An electron-microscopic study of the myocard did not reveal any changes. The ip doses that were tested ranged from 500 to 1500 mg/kg bw. [WHO; Environ Health Criteria Number 90: Dimethoate p.49 (1989)]**PEER REVIEWED**

In anaesthetized guinea-pigs treated with lethal doses of dimethoate, cardiac failure and serious ECG disturbances developed in the early phase of intoxication. The toxic cardiac phenomena appeared to be unrelated to the degree of cholinesterase inhibition, but were correlated with the myocardial dimethoate concn. Cardiac failure and mortality were first observed at a level of about 110 ug/g, while a level of 221 ug/g resulted in death in all cases. The present investigation refers to the direct effect of dimethoate on the myocardium, independent of its anticholinesterase action ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.49 (1989)]**PEER REVIEWED**

A marked increase in the toxicity of dimethoate was noted in male and female mice after pre-treatment with phenobarbital and with chlorinated hydrocarbons (DDT and dieldrin) ... The toxicity of dimethoate was increased from an ip LD50 of 198 mg/kg bw to 58.5 mg/kg by pre-treatment of mice for 3 days with sodium phenobarbital. [WHO; Environ Health Criteria Number 90: Dimethoate p.49 (1989)]**PEER REVIEWED**

Liquid formulations of technical dimethoate in the solvents, 2-methoxy- and 2-ethoxyethanol, showed increased toxicity after storage. After storage for 7 mo in England and 9 mo under tropical conditions, the oral LD50 for the rat decreased to 30-40 mg/kg and 15 mg/kg, respectively (from an initial 150-250 mg/kg). The most toxic conversion product was 0,0-dialkyl S-(N-methylcarbamoylmethyl) phosphorothioate with an oral LD50 for the rat of 1 mg/kg ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.50 (1989)]**PEER REVIEWED**

Dimethoate is sufficiently nontoxic to mammals that it has been administered to livestock with considerable success for the control of bots. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 362]**PEER REVIEWED**

In one test, involving over 800 sheep, there was no intoxication; the dosage ranged from 15 to 21 mg/kg. However, in some other tests the rate of intoxication was as high as 50 or even 66%, and there were a few deaths, even though the dosage was no higher than 25 mg/kg and in most instances was less. It was eventually realized that the formulation manufactured for injections was safe when it was not over 2 months old but toxic when it had been stored for 2 years. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

The compound is not irritating to the skin or eyes. Erythrocyte

cholinesterase is more susceptible than plasma cholinesterase to inhibition by dimethoate. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

A dietary level of 800 ppm (about 20 mg/kg/day) leads to toxic effects in rats in a few days. Minor toxic effects are produced by a dietary level of 125 ppm. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

Reports of the no-effect dietary level in rats have varied from 32 ppm to 5 to 10 ppm to 1 ppm (about 0.05 mg/kg/day). [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

In dogs, the no-effect dietary level for erythrocyte and plasma cholinesterase is 10 and 50 ppm (0.2 and 1.0 mg/kg/day), respectively. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

When rats were given 24 intraperitonel injections in 34 days, the highest rate showing no toxic effects was 3 mg/kg/day, and the rate producing no significant inhibition of plasma, erthyrocyte, or brain cholinesterase was 0.7 mg/kg/day. In similar but subcutaneous testing, guinea pigs showed no toxic effects at 16 mg/kg/day and no cholinesterase inhibition at 4 mg/kg/day. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

No morphological change or cholinesterase inhibition was found in calves fed dietary levels of 6.3 and 3.5 ppm, respectively. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

When weaning rats were maintained on diets containing 3.5 and 26% protein as casein and were later poisoned with dimethoate, there was no difference in their susceptibility (LD50, 147 and 152 mg/kg, respectively). However, other groups maintained on a normal proportion (24%) of protein derived from various plant and animal sources were distinctly less susceptible to the compound (LD 50, 358 mg/kg). Dimethoate was the only insecticide studied by Boyd that was not at least somewhat more toxic when administered in association with a diet severely deficient in protein. It was Boyd's view that, for a range of compounds, the difference between casein and mixed protein changed the toxicity by a factor of no more than 0.5 to 2.0. [Hayes, Wayland J., Jr. Pesticides Studied in Man.

Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

... An increase in malignant tumors but no increase in benign tumors in rats receiving dimethoate at rates of 5, 15, and 30 mg/kg orally and 15 mg/kg intramuscularly for 511 to 627 days. The increase did not correspond to dosage. Changes in the liver and blood were reported also. In what seems to have been the same experiment, ... severe hyperplasia of the hematopoietic parenchyma of the bone marrow and myeloid metaplasia, especially in the liver and spleen. All types of blood cells were involved, but the greatest effect in most animals was on granulocytopoiesis. The material studied was a commercial product, presumably of eastern European origin. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

Dominant lethal tests were more positive in male mice that received a single sublethal dose of dimethoate than in those that received one-twelfth of that dose daily for 30 days. It was concluded that the compound is a potential mutagen. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

The addition of dimethoate to the drinking water of mice at a concentration of 60 ppm (9.5 to 10.5 mg/kg/day) interfered with reproduction, survival of pups, and growth rate of those that did survive. It had no teratogenic effect. This dosage caused a 66% reduction of plasma cholinesterase activity of the adults and a reduction in their rate of weight gain during the first 2 weeks of exposure, but it did not increase mortality among them. In a three-generation study, the slightly lower concentration of 50 ppm did not decrease reproductive performance or increase mortality of the pups. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

A dosage of 12 mg/kg/day during the critical period of pregnancy increased the incidence of polydactyly in cats and wavy ribs, extra ribs, fused sternebrae, hydroureter, dilated bladder, and runted fetuses in rats; dosages of 3 and 6 mg/kg/day were without teratogenic effect in either species. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

This study reports the absence of increase cell proliferation in the liver or kidney after exposure in the diet to the mutagenic organophosphate

insecticides dimethoate, dioxathion, and dichlorvos following dietary exposure for 2 weeks at the same dose levels and routes of exposure that did not increase the tumor incidence in either organ in 2-year carcinogenesis assays. The present studies support the tenet that chemically induced cell proliferation may be a necessary prerequisite for chemically carcinogenesis, since in rat liver and kidney there was neither cell proliferation after 2 weeks nor tumor development after 2 years dietary exposure to the mutagenic organophosphate insecticides dimethoate dioxathion and dichlorvos. [Cunningham ML et al; Fundam and Appl Toxicol 23 (3): 363-9 (1994)]**PEER REVIEWED**

Wistar rats were continuously treated with 7.0, 10.5, 14.0, 28.0 mg/kg (1/1OO, l/75, 1/50, and l/25 LD50) of dimethoate per os including the pregnancy of dams and the lactation period of pups as well as in adulthood for three subsequent generations. The electroencepharogram of male rats of each generation was recorded at the age of 12-13 weeks. The mean frequency, mean amplitude, electroencephalogram index and the power spectrum were analysed. The data showed that the overall electroencephalogram activity of the treated rats was greater than of the controls. The mean frequency was higher, the mean amplitude and the electroencephalogram index were lower, the activity of the lower frequency wave bands decreased, that of the higher frequency wave bands increased. The changes of the mentioned electroencephalogram parameters were most pronounced in the third generation. [D'esi I et al; Neurotoxicology 15 (3): 731-4 (1994)]**PEER REVIEWED**

The neurotoxic effect of different chronic doses of dimethoate were examined in a three generational study of Wistar-rats. Treatment and investigation began with the F1 generation. The rats were 30 days old at the start of treatment, prior to mating. Rats were administered 7.0 to 28.0 mg/kg of dimethoate daily by gavage. None of the treated rats gave any indication of intoxication throughout the study. Brain tissue cholinesterase activity was significantly diminished in the rats receiving the 28 mg/kg dosing. Electroencephalogram (EEG) mean amplitudes were decreased in a dose dependent manner, most dramatically in the first generation. At the highest dose group of all generations significant changes were observed in the mean electroencephalogram frequency bands. This was also true in the 14 and 28 mg/kg dose groups of the third generation. The first generation revealed significant decreases in the low frequency wave bands at all dose levels. Similar findings occurred in the third generation at three of the doses tested. The fast frequency wave bands showed significant increases in the first generation at the 14 and 28 mg/kg dose level and at all four doses in the third generation. [Desi I

et al; Neurotoxicol 15 (3): 731-4 (1994)]**PEER REVIEWED**

Wistar rats were continuously treated with 7.0, 10.5, 14.0, 28.0 mg/kg (1/100, 1/75, 1/50 and 1/25 LD50) of dimethoate per os including the preqnancy of dams and the lactation period of pups as well as in adulthood for three subsequent generations. The electroencephalogram of male rats of each generation was recorded at the age of 12-13 weeks. The mean frequency, mean amplitude, electroencephalogram index and the power spectrum were analysed. The data showed that the overall electroencephalogram activity of the treated rat 5 was greater than of the controls. The mean frequency was higher, the mean amplitude and the electroencephalogram index were lower, the activity of the lower frequency wave bands decreased, that of the higher frequency wave bands increased. The changes of the mentioned electroencephalogram parameters were most pronounced in the third generation. [Desi I et al; Neurotoxicol (Little Rock) 15 (3): 731-4 (1994)]**PEER REVIEWED**

The effects of dimethoate, carbaryl, and permethrin on hepatic glutathione metabolism regulating enzymes were studied in mice. Male Swiss-albino-mice were gavaged once with 35 mg/kg dimethoate, 166.7 mg/kg carbaryl, or 200 mg/kg permethrin or once daily for 5 days with 17.5 mg/kg dimethoate, 83.3 mg/kg carbaryl, or 100 mg/kg permethrin. Mice were killed after the last dose and the livers were removed and assayed for protein glutathione-S-transferase (GST), glutathione-reductase (GSHRe), and gamma-glutamyl-transferase (GGT). Hepatic protein content was not altered by any of the pesticides when they were administered once. All compounds significantly increased the protein concentration when given daily for 5 days. When given once dimethoate significantly increased glutathion-reductase activity. Neither carbaryl nor permethrin significantly affected glutathione-reductase activity. None of the compounds significantly affected glutathione-S-transferase or gamma-glutamyl-transferase activity. Repeat dosing with dimethoate carbaryl and permethrin significantly increased glutathione-S-transferase activity. Repeat dosing with dimethoate significantly increased glutathione-reductase activity and decreased gamma-glutamyl-transferase activity. glutathione-reductase and gamma-glutamyl-transferase activity were not significantly affected iy carbaryl or permethrin. /It was/ concluded that the glutathione detoxification system appears to have the ability to manage single exposures to pesticides with only minor changes in one of the enzymes regulating its metabolism. Multiple exposures can induce significant disturbances in these enzymes. Dimethoate has the greatest effect on glutathione detoxification enzymes of the pesticides tested. [El-Sharkawy AM et al; Bulletin of Environ Contam and Toxicol 52

(4): 505-10 (1994)]**PEER REVIEWED**

The changes of evoked potentials were studied in a three generation experiment following the chronic treatment of Wistar-rats with dimethoate at 7.0, 10.5, 14.0, or 28.0 mg/kg orally. A dose dependent increase was noted in all three measured evoked potentials with the main effects being observed at the highest dose levels. The changes observed in the third generation tended to be greater than the changes in the first two, but were not significantly different. The latencies of the visual evoked potentials were largest in the third generation. The latencies of auditory evoked potentials were different in all three generations and there was no dose dependent relation in the second. The changes of the latency of somatosensory evoked potentials showed a dose dependent relationship in all three generations, and were milder in the third. /It was/ concluded that the nervous system effects of dimethoate as determined by the three measured parameters increased only slightly across generational lines. [Nagymajtenyl L et al; Neurotoxicology 15 (3): 741-4 (1994)]**PEER REVIEWED**

The three organophosphorous insecticides dimethoate dichlorvos and parathion-methyl were investigated in subchronic experiments on bone marrow cell chromosomes. In the literature these compounds were reported to exhibit both positive and negative results in mutagenicity tests demanding further investigations in subchronic tests. The treatment of different groups of male Wistar rats lasted for 6 weeks with 5 treatment days per week at doses of 1/100, 1/75, and 1/50 of the LD50. Following the last treatment bone marrow cell chromosomes were prepared. The frequency of cells revealing any aberrations as well as numeric and structural aberrations were evaluated. In this test both dimethoate and dichlorvos demonstrated mutagenic effects following subchronic treatment of Wistar rats while parathion-methyl at doses of 1/100, 1/75, and 1/50 of LD50 displayed no significant mutagenicity. [Neh'ez M et al; Ecotoxicol Environ Safety 29 (3): 365-71 (1994)]**PEER REVIEWED**

Administration of organophosphorous pesticide Malathion and Rogor (both a 0.2 ug/kg body wt/day) upto ten days was found to decrease the division rate in the primary spermatocytes of mice. The concurrent administration of vitamin B-comp1ex (0.3 ml of 1% polybion) or ascorbic acid 10.25 ml of 1% Redoxon) with the pesticide could nullify the meiotic inhibition caused by the pesticides. The vitamins were not found to produce any significant effect on the division rate. Possible mechanism(s) behind this vitamin mediated nullification of meiotic inhibition are discussed. [Hoda Q et al; Int J Vitam Nutr Res 63 (1): 48-51 (1993)]**PEER REVIEWED**

The effect of dimethoate at two dosage levels (6.25 and 12.50 mg/kg bw) on male reproduction tissues and their tissue residues in rats were studied. The tested doses were given orall to male rats for 65 consecutive days. Sex organs weight analysis semen picture, testosterone levels and histopathology of the male genital organs were the criteria used to evaluate the reproductive efficiency of the treated rats. There was a dose-related decrease in the weights of most genital organs and sperm motility associated with an increase in the percentages of dead and morphologically abnormal spermatozoa of treated rats. A decrease in plasma testosterone levels was observed in the treated groups. Histological examination revealed that dimethoate caused testicular lesions characterized by moderate to severe degenerative changes of spermatogonial cells and by partial arrest of spermatogenesis. Sections from liver revealed that the central veins and hepatic sinusoids appeared dilated, with some areas of haemorrhage. The highest concentrations from dimethoate were found in liver and tests and the lowest in skeletal muscle. Dimethoate and its metabolite analog were still present in a detectable concentration 21 days after stopping its oral administration. [Afifi NA et al; Dtsch Tierarzti Wochenschr 98 (11): 419-23 (1991)]**PEER REVIEWED**

The effect of an organophosphorus pesticide (dimethoate) on the urinary excretion of hydroxyproline (total nondialysable dialysable and free fractions) and hydroxylysylglycosides glucosylgalactosyl hydroxylysine and galactosehydroxylysine was investigated in two groups of female albino rats fed with normal and high protein diets. In comparison to controls dimethoate treated animals were found to excrete significantly decreased amounts of urinary hydroxyproline fractions from 7th day onwards. The excretion of total hydroxylysylglycoside in urine parallels the excretion of hydroxyproline. The urinary output of both glu-gal-hyl and gal-hyl was also appreciably lower from dimethoate treated animals. The normal ratio of glu-gal-hyl and gal-hyl found in the urine of dimethoate treated animals was discussed in light of decreased turn over of collagen in both bone and skin. The effect of dimethoate in rats fed with high protein diet was comparatively less than those fed with normal diet. [Reddy PN et al; Life Sci 49 (8): 1309-18 (1991)]**PEER REVIEWED**

NATIONAL TOXICOLOGY PROGRAM STUDIES:

A bioassay of the carcinogenicity of technical-grade dimethoate was conducted using Osborne-Mendel rats and B6C3F1 mice. The test material was administered in feed to groups of 50 rats of each sex at either of two concentrations for 80 weeks, followed by 35 weeks of obervation. The time weighted average doses for rats were 155 and 310 ppm for males and 192 and 384 ppm for females. All surviving rats were killed between 113 and 115

weeks. Dimethoate was administered in feed to groups of 50 male and 50 female mice at two concentrations. Female mice received diets containing 250 and 500 ppm of dimethoate for 80 weeks; male mice received the same dosage. However, high-dose males were returned to the control diet at 60 weeks, and low dose males at 69 weeks. All surviving mice were killed between 93 and 94 weeks. Tremors and hyperexcitability, both indications of dimethoate toxicity, were observed in the treated animals. Pathologic evaluation revealed no significant increase in tumors associated with dimethoate treatment in either species of animal, and it is concluded that there was no carcinogenic effect under the conditions of the experiment. [NCI; Toxicology and Carcinogenesis Studies of Dimethoate p.V Report No. 4 (1977) NIH Pub No. 77-804]**QC REVIEWED**

NON-HUMAN TOXICITY VALUES:

LD50 Rat female oral 240-336 mg/kg technical material /From table/ [Sanderson DM, Edson EP; Br J Ind Med 21: 52-64 (1964) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.25 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LD50 Mouse subcutaneous 60 mg/kg technical material /From table/ [Sanderson DM, Edson EP; Br J Ind Med 21: 52-64 (1964) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.25 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LD50 Hamster male sc 60 mg/kg technical material /From table/ [Sanderson DM, Edson EP; Br J Ind Med 21: 52-64 (1964) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.25 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LD50 Rabbit oral 300 mg/kg technical material /From table/ [Sanderson DM, Edson EP; Br J Ind Med 21: 52-64 (1964) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.25 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LD50 Guinea pig oral 350-400 mg/kg technical material /From table/ [Sanderson DM, Edson EP; Br J Ind Med 21: 52-64 (1964) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.25 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LD50 Mice female oral 60 mg/kg technical material /From table/ [Sanderson DM, Edson EP; Br J Ind Med 21: 52-64 (1964) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.25 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LD50 Rat oral 500-680 mg/kg [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

LD50 Guinea pig oral 600 mg/kg [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

LD50 Rabbit oral 400-500 mg/kg [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

LD50 Rat percutaneous > 800 mg/kg [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

LD50 Guinea pig percutaneous > 1000 mg/kg [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

ECOTOXICITY VALUES:

LC50 Gammarus lacustris (scuds) 0.20 mg/l/96 hr (95% confidence limit 0.15-0.27 mg/l), mature, temp 21 deg C. Static bioassay without aeration, pH 7.2-7.5, water hardness 40-50 mg/l as calcium carbonate and alkalinity of 30-35 mg/l. /Technical, 97.4%/ [U.S. Department of Interior, Fish and Wildlife Service. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Resource Publication No. 137. Washington, DC: U.S. Government Printing Office, 1980.31]**PEER REVIEWED**

LC50 Pteronarcys californica (stoneflies) 0.043 mg/l/96 hr (95% confidence limit 0.036-0.051 mg/l), second year class, temp 21 deg C. Static bioassay without aeration, pH 7.2-7.5, water hardness 40-50 mg/l as calcium carbonate and alkalinity of 30-35 mg/l. /Technical, 97.4%/ [U.S. Department of Interior, Fish and Wildlife Service. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Resource Publication No. 137. Washington, DC: U.S. Government Printing Office, 1980.31]**PEER REVIEWED**

LC50 Salmo gairdneri (rainbow trout) 6.2 mg/l/96 hr (95% confidence limit 4.1-9.3 mg/l), wt 1.5 g, temp 21 deg C. Static bioassay without aeration, pH 7.2-7.5, water hardness 40-50 mg/l as calcium carbonate and alkalinity of 30-35 mg/l. /Technical, 97.4%/ [U.S. Department of Interior, Fish and Wildlife Service. Handbook of Acute Toxicity of Chemicals to Fish and

Aquatic Invertebrates. Resource Publication No. 137. Washington, DC: U.S. Government Printing Office, 1980.31]**PEER REVIEWED**

LC50 Salmo gairdneri (rainbow trout) 20.0 ppm/24 hr /Conditions of bioassay not given/ [Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 541]**PEER REVIEWED**

LC50 Chingatta 4.48 mg/l/96 hr /30% Emulifiable concentrate; conditions of bioassay not given/ [Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 541]**PEER REVIEWED**

LC50 Lapomis macrochirus (bluegill) 6.0 mg/l/96 hr, wt 0.3 g, temp 24 deg C. Static bioassay without aeration, pH 7.2-7.5, water hardness 40-50 mg/l as calcium carbonate and alkalinity of 30-35 mg/l. /Technical, 97.4%/ [U.S. Department of Interior, Fish and Wildlife Service. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Resource Publication No. 137. Washington, DC: U.S. Government Printing Office, 1980.31]**PEER REVIEWED**

LC50 Lapomis macrochirus (bluegill) 28.0 ppm/24 hr /Conditions of bioassay not given/ [Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 541]**PEER REVIEWED**

LC50 Coturnix japonica (japanese quail) 346 mg/l in 5-day diet (95% confidence limit 303-394 mg/l) age 14 days /Technical, 99%/ [U.S. Department of the Interior, Fish and Wildlife Service, Bureau of Sports Fisheries and Wildlife. Lethal Dietary Toxicities of Environmental Pollutants to Birds. Special Scientific Report - Wildlife No. 191. Washington, DC: U.S. Government Printing Office, 1975.19]**PEER REVIEWED**

LC50 Ring-necked pheasants 332 mg/l in 5-day diet (95% confidence limit 293-376 mg/l), age 10 days /Technical, 99%/ [U.S. Department of the Interior, Fish and Wildlife Service, Bureau of Sports Fisheries and Wildlife. Lethal Dietary Toxicities of Environmental Pollutants to Birds. Special Scientific Report - Wildlife No. 191. Washington, DC: U.S. Government Printing Office, 1975.19]**PEER REVIEWED**

LC50 Mallards 1011 mg/l in 5-day diet (95% confidence limit 707-1372 mg/l), age 10 days /Technical, 99%/ [U.S. Department of the Interior, Fish and Wildlife Service, Bureau of Sports Fisheries and Wildlife. Lethal

Dietary Toxicities of Environmental Pollutants to Birds. Special Scientific Report - Wildlife No. 191. Washington, DC: U.S. Government Printing Office, 1975.19]**PEER REVIEWED**

LD50 Redwinged blackbird oral 6.60-17.8 mg/kg [Schafer EW Jr et al; Arch Environ Contam Toxicol 12 (3): 355-82 (1983)]**PEER REVIEWED**

LD50 Starlings oral 31.6 mg/kg [Schafer EW Jr et al; Arch Environ Contam Toxicol 12 (3): 355-82 (1983)]**PEER REVIEWED**

LC50 Mosquito fish 40-60 mg/l/96 hr /Conditions of bioassay not specified/ [Hussar, D.A. (ed.). Modell's Drugs in Current Use and New Drugs. 34th ed. New York, NY: Springer Verlag Publishing Co., 1988. 298]**PEER REVIEWED**

LD50 Honey bees 0.9 mg/bee [Hussar, D.A. (ed.). Modell's Drugs in Current Use and New Drugs. 34th ed. New York, NY: Springer Verlag Publishing Co., 1988. 298]**PEER REVIEWED**

LC50 Salmo gairdneri (rainbow trout) 58.0 mg/l/24 hr; 27.0 mg/l/48 hr /Rogor 40, 32% dimethoate, from table; Conditions of bioassay not specified/ [Alabaster JS; Int Pest Control 11 (2): 29-35 (1969) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.28 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LC50 Cyprinus carpio (carp) 22.39 mg/l/168 hr /From table; Conditions of bioassay not specified/ [Basak PK, Konar Sk; Ind J Fish 25 (1-2): 141-55 (1978) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.28 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LD50 Anas platyrhynchos (mallards) male, oral 41.7 mg/kg (95% confidence limit 30.1-57.8 mg/kg), age 3-4 mo /Sample purity 97%/ [U.S. Department of the Interior, Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

LD50 Anas platyrhynchos (mallards) female, oral 63.5 mg/kg (95% confidence limit 45.8-88.1 mg/kg), age 3-4 mo /Sample purity 99.8%/ [U.S. Department of the Interior, Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

LD50 Phasianus colchicus (pheasant) male, oral 20.0 mg/kg (95% confidence limit 15.9-25.2 mg/kg), age 3-4 mo /Sample purity 97%/ [U.S. Department of

the Interior, Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

LC50 Coturnix japonica (Japanese quail) oral 496 ppm in 5-day diet (95% confidence limit 373-659 ppm), age 14-day /Dimethoate (Cygon 2E)/ [Hill, E.F. and Camardese, M.B. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to Coturnix. Fish and Wildlife Technical Report 2. Washington, DC: United States Department of Interior Fish and Wildlife Service, 1986.61]**PEER REVIEWED**

LC50 Daphnia magna 2.50 mg/l/48 hr./From table; Conditions of bioassay not specified/ [USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.29 (1984) ECAO-CIN- B PO81]**PEER REVIEWED**

EC50 Skeletonema costatum (marine algae), effect: decreased dry weight is 9.5 mg/l/96 hr. /Conditions of bioassay not specified/ [Ibramhim EA; Aquat Toxicol 3 (1): 1-14 (1983) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.30 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

EC50 Skeltonema costatum (marine algae), effect: protein content 10.65 mg/l/96 hr. /Conditions of bioassay not specified/ [Ibrahim EA; Aquat Toxicol 3 (1): 1-14 (1983) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.30 (1984) EACO-CIN-PO81]**PEER REVIEWED**

EC50 Skeletonema costatum (marine algae) cell count 11.2 mg/l/96 hr. /Conditions of bioassay not specified/ [Ibrahim EA; Aquat Toxicol 3 (1): 1-14 (83) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.30 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

EC50 Skeletonema costatum (marine algae) carbohydrate and chlorophyll a content 10.8 mg/l/96 hr. /Conditions of bioassay not specified/ [Ibrahim EA; Aquat Toxicol 3 (1): 1-14 (1983) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.30 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LC50 Stonefly 0.14 mg/l (48-hr) and 0.043 mg/l (96-hr) /From table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.28 (1989)]**PEER REVIEWED**

LC50 (mg/l): Rainbow trout (Salmo gairdnerii) 20 mg/l (24-hr) and 6.2-8.5 mg/l (96-hr) /From table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.28 (1989)]**PEER REVIEWED**

LC50 Saccobranchus fossilis 5.14 mg/l (24-hr), 4.80 mg/l (48-hr), 4.67 mg/l (72-hr), and 4.57 mg/l (96-hr) /From table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.28 (1989)]**PEER REVIEWED**

LC50 Channa punctatus 68 mg/l (24-hr), 54 mg/l (48-hr), and 47 mg/l (96-hr) /From table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.28 (1989)]**PEER REVIEWED**

LC50 Long nosed killfish (Fundulus) 1.0 mg/l (48-hr) /From table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.28 (1989)]**PEER REVIEWED**

LC50 Scud 0.9 mg/l (24-hr), 0.4 mg/l (48-hr), and 0.20 mg/l (96-hr) /From table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.28 (1989)]**PEER REVIEWED**

LC50 Red Crayfish 1.0 mg/l (48-hr) /From table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.28 (1989)]**PEER REVIEWED**

The median tolerance limit of the fresh-water teleost, Channa punctatus for dimethoate is 20.5 mg/l ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.27 (1989)]**PEER REVIEWED**

... Determined the TLm values of dimethoate for Channa gachua for 24, 48, 72, or 96 hr to be 5.2, 5.0, 4.6, or 4.5 mg/l, respectively. The safe concn of dimethoate calculated on the basis of TLm values was approx 1.4 mg/l. [WHO; Environ Health Criteria Number 90: Dimethoate p.27 (1989)]**PEER REVIEWED**

The estimated 48- and 72-hr TLm values for zebrafish Brachydanio rerio embryos, exposed to dimethoate, were 940 mg/l and 259 mg/l, respectively ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.29 (1989)]**PEER REVIEWED**

The oral LD50 for the honey-bee (Apis mellifera L) ranges from 93 to 150 ng/bee ... The contact LD50 is 98-120 ng/bee ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.29 (1989)]**PEER REVIEWED**

Acute oral LD50 of dimethoate for birds (mg/kg): 25-50 for hen; 15-25 for pheasant; > 40 for duck; 22 for sparrow; and 26 for blackbird /from table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.32 (1989)]**PEER REVIEWED**

Acute oral LD50 for farm animals (mg/kg bw): > 50 for horse; 80 for sheep; and 70 for cattle /from table/ [WHO; Environ Health Criteria Number 90: Dimethoate p.33 (1989)]**PEER REVIEWED**

METABOLISM/PHARMACOKINETICS:

METABOLISM/METABOLITES:

... Detoxification pathway ... involves the hydrolysis of carboxyester or carboxyamide linkages in some insecticides by tissue or plasma carboxylesterases (sometimes called aliesterase). Malathion and dimethoate are examples. [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. 534]**PEER REVIEWED**

RABBIT & RAT LIVER MICROSOMES CONVERTED DIMETHOATE TO OXYGEN ANALOG & DES-N-METHYL DERIVATIVES. [Menzie, C. M. Metabolism of Pesticides, An Update. U.S. Department of the Interior, Fish, Wild-life Service, Special Scientific Report - Wildlife No. 184, Washington, DC: U.S. Government Printing Office, l974.166]**PEER REVIEWED**

AFTER DIMETHOATE WAS ADMINISTERED TO RATS, THE FOLLOWING CMPD WERE FOUND IN URINE: 1. DIMETHOATE, 2. DIMETHOXON, 3. DIMETHOATE CARBOXYLIC ACID, 4. DIMETHYLPHOSPHORODITHIOATE, 5. DIMETHYL PHOSPHOROTHIOATE, 6. DIMETHYLPHOSPHATE, 7. MONOMETHYLPHOSPHATE, 8. PHOSPHOROTHIOATE, 9. FORMATE, AND 10. N-METHYL 2-GLUCURONATE ACETAMIDE. [Menzie, C. M. Metabolism of Pesticides, An Update. U.S. Department of the Interior, Fish, Wild-life Service, Special Scientific Report - Wildlife No. 184, Washington, DC: U.S. Government Printing Office, l974.166]**PEER REVIEWED**

OXIDATIVE DESULFURATION OF DIMETHOATE TO GIVE O-ANALOG TOOK PLACE RAPIDLY IN RABBITS & RATS. BOTH DIMETHOATE & O-ANALOG UNDERWENT SUBSEQUENT OXIDATIVE N-DEALKYLATION COUPLED WITH FORMATION OF N-HYDROXYMETHYL INTERMEDIATES. [The Chemical Society. Foreign Compound Metabolism in

Mammals. Volume 2: A Review of the Literature Published Between 1970 and 1971. London: The Chemical Society, 1972. 287]**PEER REVIEWED**

Rats given dimethoate excreted dimethylphosphoric acid, dimethylthiophosphoric acid, & other minor metabolites in the urine. [Matsumura, F. Toxicology of Insecticides. 2nd ed. New York, NY: Plenum Press, 1985. 270]**PEER REVIEWED**

/DIMETHOATE/ ... IS MORE THAN 300 TIMES MORE TOXIC TO HOUSEFLIES THAN TO MICE, DIFFERENCE WHICH HAS BEEN ATTRIBUTED TO ITS MORE RAPID METABOLIC DEACTIVATION IN MAMMALS, TO GIVE DIMETHOATE ACID & O,O-DIMETHYLPHOSPHORODITHIONATE. [Parke, D. V. The Biochemistry of Foreign Compounds. Oxford: Pergamon Press, 1968. 204]**PEER REVIEWED**

OXIDATION OF DIMETHOATE TO DIMETHOXON IS BELIEVED TO TAKE PLACE IN VIVO, & SINCE DIMETHOXON IS 100 TIMES MORE INHIBITING TO BRAIN ACETYLCHOLINESTERASE THAN DIMETHOATE, DIMETHOXON MUST PLAY DOMINANT ROLE IN MAMMALIAN TOXICITY. [The Chemical Society. Foreign Compound Metabolism in Mammals. Volume 1: A Review of the Literature Published Between 1960 and 1969. London: The Chemical Society, 1970. 282]**PEER REVIEWED**

Metabolism of (32)P-dimethoate (10-40 mg/kg bw) following oral and im administration /was studied in sheep/. ... Identified urinary metabolites were dimethyl phosphoric acid, dimethyl phosphorothioic acid, and dimethyl phosphorodithioic acid. ... [Chamberlain WF et al; J Econ Entomol 54 (4): 733-40 (1961) as cited in USEPA/ECAO; Health and Environmental Effects Profile for Dimethoate (Final Draft) p.11 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

10 mg (32)P-dimethoate/kg body weight in young Hereford bulls or steers following oral or im administration. ... Major metabolic products /included/ ... dimethyl phosphate and dimethyl phosphorothioate, along with several unidentified metabolites. [Kaplanis JN et al; J Econ Entomol 52 (60): 1190-4 (1959) as cited in USEPA/ECAO; Health and Environmental Effects Profile for Dimethoate (Final Draft) p.11 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

... Dimethoate acid (ie, carboxyamidase product) /was detected/ in a steer treated with dimethoate, along with other metabolites such as

N-desmethyldimethoate, dimethylphosphoric acid, dimethylphosphorothioic acid & dimethylphosphorodithioic acid. [Matsumura, F. Toxicology of Insecticides. 2nd ed. New York, NY: Plenum Press, 1985. 270]**PEER REVIEWED**

In plants, as well as oxidation to the phosphorothioate, there is also hydrolysis to O,O-dimethylphosphorodithioate, -phosphorothioate, & -phosphate. The ester group is demethylated & the methylamino group is hydrolytically cleaved. In animals, metabolism is the same as for plants. [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

DIMETHOATE WAS APPLIED DERMALLY TO LACTATING COW. THIN-LAYER CHROMATOGRAPHY INDICATED THE PRESENCE, IN ADDITION TO DIMETHOATE, OF DIMETHOXON, DIMETHOATE ACID & DESMETHYL DIMETHOATE IN THE BLOOD; & OF DIMETHOXON, DIMETHYLTHIO (OR DITHIO) PHOSPHATE & POSSIBILY DESMETHYL DIMETHOATE IN THE MILK. [Menzie, C.M. Metabolism of Pesticides. U.S. Department of the Interior, Bureau of Sport Fisheries and Wildlife, Publication 127. Washington, DC: U.S. Government Printing Office, 1969. 160]**PEER REVIEWED**

FROM URINE SAMPLES FROM A STEER TREATED WITH LABELED DIMETHOATE, THE FOLLOWING METABOLITES WERE ISOLATED & IDENTIFIED: O,O-DIMETHYL S-CARBOXYMETHYL PHOSPHORODITHIOATE, O-METHYL S-(METHYLCARBAMOYLMETHYL) PHOSPHOROTHIOIC ACID, O,O-DIMETHYL PHOSPHORIC ACID, O,O-DIMETHYL PHOSPHOROTHIOIC ACID, & O,O-DIMETHYL PHOSPHORODITHIOIC ACID. OF 34 TISSUES TESTED FOR RESIDUE LEVELS, LIVER & KIDNEY CONTAINED GREATEST AMT OF TOTAL DIMETHOATE EQUIV & OF ACTUAL DIMETHOATE. SIMILAR RESULTS WERE OBTAINED WITH SHEEP. [Menzie, C.M. Metabolism of Pesticides. U.S. Department of the Interior, Bureau of Sport Fisheries and Wildlife, Publication 127. Washington, DC: U.S. Government Printing Office, 1969. 160]**PEER REVIEWED**

In pheasant, the toxic thiolate analog tends to accumulate & its conversion to one or the other metabolites is much slower than in mammals.

This probably explains the greater susceptibility of the pheasant. [Menzie, C.M. Metabolism of Pesticides. U.S. Department of the Interior, Bureau of Sport Fisheries and Wildlife, Publication 127. Washington, DC: U.S. Government Printing Office, 1969. 160]**PEER REVIEWED**

In the locust, the main product was the ... thiolate analog (dimethoxon). Using the green rice leafhopper ... the mono- & di-methylphosphates & orthophosphate were observed. Homogenates of houseflies, 4th & 5th instar larvae of rice stem borer ... adults of apterous form of green peach aphid ... & adult male American coackroaches degraded dimethoate. Demethyl dimethoate, the carboxy acid analog & dimethyl phosphorothioate were identified. [Menzie, C.M. Metabolism of Pesticides. U.S. Department of the Interior, Bureau of Sport Fisheries and Wildlife, Publication 127. Washington, DC: U.S. Government Printing Office, 1969. 161]**PEER REVIEWED**

... (30 mg/kg bw) dimethoate was administered to albino rats (strain unspecified) of both sexes in vivo and in vitro, using either (32)P- or (14)C-dimethoate. Oxidation to the more toxic dimethoxon was assumed to occur in vivo; however, no dimethoxon was detected in the urine or feces of rats treated with dimethoate. The recovered radioactive metabolites following administration of (32)P-dimethoate were identified as monomethylphosphate, dimethylphosphate, thiophosphoric acid, dimethoate carboxylic acid, dimethylphosphorothioic acid, dimethoate carboxylic acid, dimethylphosphorothioic acid, and dimethylphosphorodithioic acid. Following administration of dimethoate labeled with (14)C in the methoxy groups, the observed urinary metabolites were monomethylphosphate, dimethylphosphate, dimethoate carboxylic acid, dimethylphosphorothioic acid, traces of mono- and dimethylphosphates and dimethylphosphorodithioic acid. Methylamine liberated by these pathways is oxidatively demethylated to CO2 and formate. Approximately 80% of the nonphosphorus moiety librated by the action of esterases on the S-C bond are excreted in the urine as glucuronide conjugates. [Hassan A et al; Biochem 18: 2429-38 (1969) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.11 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

Dimethoxon, an active metabolite of dimethoate, is thought to be 75-100 times more potent than dimethoate for the inhibition of rat brain cholinesterase ... . [Sullivan, J.B. Jr., G.R. Krieger (eds.). Hazardous Materials Toxicology-Clinical Principles of Environmental Health. Baltimore, MD: Williams and Wilkins, 1992. 1020]**PEER REVIEWED**

The results of in vitro and in vivo studies showed that the main metabolic

pathways of dimethoate were hydrolysis and oxidation. ... The presence of the oxygen analogue dimethoxon (omethoate) has been demonstrated in insects, plants, and mammals; it appears to be the metabolite responsible for the toxic action of dimethoate ... The highest levels of this metabolite were found in insects, particularly in those highly susceptible to dimethoate. The oxygen analogue was produced in larger quantities in insects than in rats. The enzymes mediating the hydrolysis of the carboxyamide bond are much less effective in insects than in mammals ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.24 (1989)]**PEER REVIEWED**

It has been shown that cleavage of dimethoate by rats and cows occurs initially at the C-N bond to produce the carboxy derivative. ... A second hydrolytic pathway involves an esterase action on the S-C bond ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.25 (1989)]**PEER REVIEWED**

Oxidative metabolism of dimethoate predominated over hydrolytic metabolism in the cell culture system. In the whole rat, the opposite was true. Metabolism of dimethoate in human embryonic lung cells was much the same as metabolism in rats. ... In vitro and in vivo studies showed that dimethoate is biotransformed to the P=O analogue via the liver cytochrome P450 system. ... Concn of 0.1-10 mmol dimethoate/l led to a linear decrease in the rates of N-demethylation and P-hydroxylation. Similarly, in microsomes from rats treated with dimethoate in vivo, increased activity of desulfuration (140%, $P < 0.01$), and decreased activity of hydroxylation and demethylation were seen ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.25 (1989)]**PEER REVIEWED**

In vivo studies on mice showed dimethoate toxicity to be markedly increased by phenobarbital pre-treatment, as a result of induction of hepatic microsomal enzymes including the mixed function oxidases responsible for the conversion of P=S to P=O ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.25 (1989)]**PEER REVIEWED**

It has been found that, while dimethoate undergoes rapid degradation in the rat liver, very little occurs in other tissues. ... The ability of the liver to degrade dimethoate in various species decreased in the order: rabbit > sheep > dog > rat > cattle > hen > guinea-pig > mouse > pig. For the hen, cattle, mouse, sheep, and rat there was a reasonable good straight-line relationship between the LD50 values and the degradation ability of the liver ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.25 (1989)]**PEER REVIEWED**

After administration of 32(P)-dimethoate to rats, dimethoate, dimethoxon, dimethoate carboxylic acid, dimethylphosphorodithioate, dimethylphosphorothioate, dimethylphosphate, monomethylphosphate, phosphorothioate, formate, and N-methyl 2-glucuronate acetamide were found in the urine ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.25 (1989)]**PEER REVIEWED**

Dimethoxon (omethoate), a metabolite of dimethoate, is 75-100 times more potent than dimethoate in inhibiting AChE, suggesting that this metabolite plays a dominant role in mammalian toxicity ... The LD50 of omethoate in rats is 25-28 mg/kg bw ... it is about 10 times more toxic than dimethoate. [WHO; Environ Health Criteria Number 90: Dimethoate p.50 (1989)]**PEER REVIEWED**

Dimethoate is rapidly metabolized. In one very thorough study in rats, about 60% of the administered dose was excreted in 24 hours in the urine and expired air. It was concluded that dimethoxon is 75 to 100 times more potent than dimethoate as an inhibitor of rat brain acetylcholinesterase and that toxicity of the insecticide is due mainly to its conversion to dimethoxon. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

Data to guide an exposure assessment were obtained by giving sugar peas containing overtolerance dimethoate residues (17 ppm; 8% oxon) and a bolus dose of dimethoate to a healthy adult male. The dimethoate tolerance on peas was and remains 2 ppn. Serial total urine samples were collected and analysed for dimethoate and its oxon dimethylphosphate dimethylphosphorothioate (DMTP) and dimethylphosphorodithioate. The dose of dimethoate administered was approx 0.1 mg/kg body weight and produced no symptoms of toxicity. Dimethylphosphates appeared in the urine within 2 hr. The major metabolite (about 60%) was dimethylphosphorothioate. Only traces (< 0.5%) of dimethoate and oxon were recovered from urine. Acetylcholinesterase inhibition was not observed although urinary metabolites were prominent indicating that they are better indicators of acute exposure than cholinesterase inhibition. The results obtained using a bolus dose were virtually identical to those from the trial with overtolerance peas and indicated that dimethoate is readily absorbed and its urinary metabolites are readily eliminated following exposures to low doses (0.1 mg/kg body weight). [Krieger RI and Thongsinthusak T; Food Chem Toxlcol 31 (3): 177-82 (1993)]**PEER REVIEWED**

ABSORPTION, DISTRIBUTION & EXCRETION:

QUALITATIVELY, METB OF DIMETHOATE IN MAMMALS IS ESSENTIALLY

IDENTICAL TO
THAT OCCURRING IN INSECTS. THERE ARE ... QUANTITATIVE DIFFERENCES. IN
GENERAL, DIMETHOATE, IS MUCH MORE RAPIDLY DEGRADED IN MAMMALS
&
ELIMINATED VIA URINE. FOR EXAMPLE, 87-90% OF ORAL DOSE GIVEN CATTLE
WAS
FOUND IN URINE AFTER 24 HR, MAINLY AS HYDROLYSIS PRODUCTS.
[White-Stevens,
R. (ed.). Pesticides in the Environment: Volume 1, Part 1, Part 2. New
York: Marcel Dekker, Inc., 1971. 168]**PEER REVIEWED**

... AFTER INGESTION OF DIMETHOATE, RATS ELIMINATE 60% OF DOSE IN 24 HR
IN
URINE & EXPIRED AIR. [The Chemical Society. Foreign Compound
Metabolism in Mammals. Volume 1: A Review of the Literature Published
Between 1960 and 1969. London: The Chemical Society, 1970. 282]**PEER
REVIEWED**

... Dimethoate rapidly penetrated isolated sections of /mouse/ small
intestine, colon, and rectum, with the highest rates of penetration
occurring in the colon and rectum. The age of the mice had no significant
effects on the extent of penetration. /Initial dose not specified/ [Shah
PV, Guthrie FE; Toxicol Appl Pharmacol 25: 621-4 (1973) as cited in
USEPA/ECAO; Health and Environmental Effects Profile for Dimethoate (Final
Draft) p.9 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

... Following aerial spraying with 38% dimethoate animal tissues contained
higher concentrations of dimethoate than did soil, water or plants. ...
The tissue containing the highest concentrations were brain and intially,
lung. ... [Fedrorenko AP et al; Vestn Zool 4: 89-92 (1981) as cited in
USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.9-10
(1984) ECAO-CIN-PO81]**PEER REVIEWED**

Dimethoate is poorly absorbed through the skin but rapidly absorbed by the
oral route ... . [Sullivan, J.B. Jr., G.R. Krieger (eds.). Hazardous
Materials Toxicology-Clinical Principles of Environmental Health.
Baltimore, MD: Williams and Wilkins, 1992. 1020]**PEER REVIEWED**

When a combination of dimethoate and omethoate (dimethoxon) was given to
cows in dosages of 1 and 0.1 mg/kg body weight, respectively, for 14 days,
only residues of the metabolite omethoate were observed in the milk
(0.004-0.125 mg/kg). Three days after the application, neither compound
could be detected. When dosages of 0.5 mg dimethoate/kg and 0.05 mg

omethoate/kg were given for 14 days or when corn silage containing 1-7 mg dimethoate/kg (resulting in dosages of 0.06-0.36 mg/kg body weight) was given for 28 or 42 days, no residues were detected in the milk ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.21 (1989)]**PEER REVIEWED**

... Checked the blood levels of dimethoate in cats and rats after single oral doses of 50, 75, or 200 mg/kg in the cat and 300 mg/kg in the rat. The determinations were carried out 15, 30, 60, 90, 120, and 180 min after dosing. Dimethoate was detected in the blood of cats and rats after 30 min, and reached a maximum level after 60-90 min. Nearly 80% of the dimethoate in the blood was found in the erythrocytes; only 15-20% was found in the serum. With repeated daily oral intake of dimethoate at doses of 20 mg/kg or 10 mg/kg, the maximum blood level occurred on the 5-10th day of the study. The same pattern in blood levels was observed with repeated inhalation of dimethoate for 4 h/day over 3 months, at a mean concentration of 5 mg/m3 air. Dimethoate was detected in blood from the second day and reached its maximum by the 7-lOth day. [WHO; Environ Health Criteria Number 90: Dimethoate p.24 (1989)]**PEER REVIEWED**

Daily application of 50 mg dimethoate/kg on the skin of rabbits resulted in a maximum concentration in the blood at about the third day ... When dimethoate was applied to the skin of rats for 1, 2, 4, 12, or 24 hr in a single dose of 560 mg/kg, the maximum concentration in the skin was reached after 12 hr of exposure and was correlated with the maximum inhibition of ChE activity in the serum and liver. The concentrations of dimethoate in the blood, liver, and kidney were maximal after 2 hr of exposure ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.24 (1989)]**PEER REVIEWED**

About 45% of the 32P-dimethoate administered orally at 50 mg/kg to rats was excreted in the urine, while only 5.8% was eliminated in the feces, 72 hr after treatment ... The values in rats after dermal application were 30.6% and 6.5%, respectively. More than 95% of the 32P materials in the urine and feces after oral or dermal administration in rats were hydrolytic products ... [WHO; Environ Health Criteria Number 90: Dimethoate p.25 (1989)]**PEER REVIEWED**

Twenty-four hr after ip and oral administration of dimethoate to rats @ doses of 0.25, 2.5, or 25 mg/kg, dimethylphosphorodithioate, dimethylphosphorothioate, and dimethyl phosphate were detected in the urine at concn of 12-14%, 11-15%, and 12-13%, respectively ... Neither the route of exposure nor the dose had any influence on the types of

metabolite formed. [WHO; Environ Health Criteria Number 90: Dimethoate p.26 (1989)]**PEER REVIEWED**

About 89-90% of an oral dose of 10 mg/ dimethoate/kg was eliminated in the urine of cattle at the end of 24 hr. The same percentage of an intramuscular dose of 10 mg/kg was excreted after 9 hr. Only 3.7-5% of the oral dose and about 1.1% of the intramuscular dose were eliminated in the feces after 72 hr and 24 hr, respectively ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.26 (1989)]**PEER REVIEWED**

In human beings, 76-100% of radioactivity was reported to be excreted in the urine, 24 hr after oral dosing with 32P-dimethoate ... . [WHO; Environ Health Criteria Number 90: Dimethoate p.26 (1989)]**PEER REVIEWED**

Following application of (32)P-labeled dimethoate to the backs of cattle at the rate of 30 mg/kg, the concentration of active ingredient reached a maximum level of 0.02 ppm in blood and milk in about 3 hours and then dropped to 0.01 ppm by 9 hours. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

Dimethoate was degraded rapidly by rat liver but very little by other rat tissues. The ability of the livers of different species decreased in the order rabbit > dog = sheep > rat > cow > hen > mouse = guinea pig > pig. At least for the rat, sheep, mouse, cow, and hen, there was a reasonably good straight line relationship between the LD50 values and the products of the rate of metabolism multiplied by the proportion of the body constituted by the liver. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

Cleavage of dimethoate by rats and cows occurs initially at the methyl-phosphate, phosphate-sulfur, sulfur-carbon, and particularly at the carbonyl-nitrogen bonds. In 24 hours, 81% of an oral dose of dimethoate was excreted by male rats, compared with 19% of a dose of the separately administered P==O derivative. Dimethoate is more readily attacked at the C-N bond than is true of the P=O derivative, and this is a major route of degradation. Within 48 hours after dosing, almost 90% of (32)P-dimethoate was recovered in the urine of male rats, and most of the rest was recovered in the feces. Only a little over a total of 50% was excreted by female rats in 48 hours, but later excretion was approximately as slow in females as in males. At 168 hours after treatment, the total (32)P remaining in the tissue was 2 to 5 times greater in female than in male

rats. After 24 hours, the highest concentration of dimethoate equivalent was in the liver, but at 72 and 168 hours the highest concentrations were in the skin and bones, reflecting, the conversion of (32)P to inorganic phosphate. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

Following oral administration of (32)P-dimethoate, rats excreted 50% of the activity in the urine and 25% in the feces in 24 hours. Nine days after dosing, only 0.9 to 1.1% of the (32)P remained in the animals. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 363]**PEER REVIEWED**

In a Russian study, dimethoate residues were detected in fish for 30 day after application at 1.5 kg/hectare. Residue accumulation occurred in the kidneys at levels as high as 159 mg/kg. [Korzhevenko GN et al; J Toxicol Environ Health 8 (1-2): 169-84 (1981) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.30 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

When (32)P-dimethoate was given to volunteers orally, it was absorbed and excreted rapidly, 76 to 100% of the radioactivity appearing in the urine in 24 hours. [Hayes, Wayland J., Jr. Pesticides Studied in Man. Baltimore/London: Williams and Wilkins, 1982. 364]**PEER REVIEWED**

Dimethoate accumulation in liver and muscle tissues of a fresh-water fish, Clarias bactrachus L, was measured by gas liquid chromatography. Fish were exposed to a sub-lethal concentration of the pesticide in the laboratory water tank for a period of eight days. The accumulation increased up to two days, followed by decrease on the 4th and 8th days of exposure in both liver and muscle tissues. Maximumn accumulation was found in liver. After eight days of exposure, fish were released into pesticide-free water to study the rate of loss of the pesticide. It was found that liver and muscle tissues were cleared of dimethoate after four and eight days respectively. [Begum G et al; Pesticide Science 40 (3): 201-5 (1994)]**PEER REVIEWED**

Data to guide an exposure assessment were obtained by giving sugar peas containing overtolerance dimethoate residues (17 ppm; 8% oxon) and a bolus dose of dimethoate to a healthy adult male. The dimethoate tolerance on peas was and remains 2 ppn. Serial total urine samples were collected and analysed for dimethoate and its oxon dimethylphosphate dimethylphosphorothioate (DMTP) and dimethylphosphorodithioate. The dose of dimethoate administered was approx 0.1 mg/kg body weight and produced

no symptoms of toxicity. Dimethylphosphates appeared in the urine within 2 hr. The major metabolite (about 60%) was dimethylphosphorothioate. Only traces (< 0.5%) of dimethoate and oxon were recovered from urine. Acetylcholinesterase inhibition was not observed although urinary metabolites were prominent indicating that they are better indicators of acute exposure than cholinesterase inhibition. The results obtained using a bolus dose were virtually identical to those from the trial with overtolerance peas and indicated that dimethoate is readily absorbed and its urinary metabolites are readily eliminated following exposures to low doses (0.1 mg/kg body weight). [Krieger RI and Thongsinthusak T; Food Chem Toxlcol 31 (3): 177-82 (1993)]**PEER REVIEWED**

MECHANISM OF ACTION:

Organophosphorus derivatives act by combining with and inactivating the enzyme acetylcholinesterase (AChE). ... The inactivation of cholinesterase by cholinesterase inhibitor pesticides allows the accumulation of large amounts of acetylcholine, with resultant widespread effects that may be ... separated into 4 categories: (1) Potentiation of postganglionic parasympathetic activity. ... (2) Persistent depolarization of skeletal muscle ... (3) Initial stimulation following depression of cells of central nervous system ... (4) Variable ganglionic stimulation or blockade ... /Cholinesterase inhibitor pesticides/ [Dreisbach, R.H. Handbook of Poisoning. 12th ed. Norwalk, CT: Appleton and Lange, 1987. 113]**PEER REVIEWED**

Phosphorothioates, like dimethoate, require metabolic oxidation of the phosphorous-sulfur (P=S) bond before producing toxicity. Due to this oxidation step, these substances can exhibit a slower onset of symptoms as compare to cmpd possessing a direct-acting P=O bond ... . [Sullivan, J.B. Jr., G.R. Krieger (eds.). Hazardous Materials Toxicology-Clinical Principles of Environmental Health. Baltimore, MD: Williams and Wilkins, 1992. 1020]**PEER REVIEWED**

The ester and amide groups of dimethoate are cleaved in reactions that vary with the organism and that contribute to the selective toxicity of the compound. [WHO; Environ Health Criteria Number 90: Dimethoate p.24 (1989)]**PEER REVIEWED**

They act principally by inhibition of acetyl cholinesterase (AChE) at the cholinergic synapses. /organophosphorus insecticides/ [WHO; Environ Health Criteria Number 90: Dimethoate p.50 (1989)]**PEER REVIEWED**

INTERACTIONS:

The effects of various classes of insecticides were studied on N-demethylation of dimethylnitrosamine (DMN) by mouse liver enzymes. Organochlorine insecticides increased the activities of dimethylnitrosamine demethylases I and II. Dimethoate, an organophosphorus cmpd, was the only insecticide tested to inhibit the N-demethylation of dimethylnitrosamine, with more pronounced effect on dimethylnitrosamine demethylase I. [Mostafa MH et al; Environ Res 32 (1): 57-61 (1983)]**PEER REVIEWED**

Carbon disulfide pretreatment potentiated the anticholinesterase action of parathion and EPN, but suppressed that of dimethoate and diazinon. Carbon disulfide had no significant effect or a slightly suppressive effect on the other compounds. Some of these effects were contrasted with the repeated alteration of the toxicity following phenobarbital pretreatment. [Yasoshima M, Masuda Y; Toxicol Lett 32 (3): 179-84 (1986)]**PEER REVIEWED**

PHARMACOLOGY:

THERAPEUTIC USES:

MEDICATION (VET): ALSO USED EXPTL IN MANY SPECIES BY MANY ROUTES (SPRAY, ORAL, SC, IM, POUR ON, ETC), & HAS SHOWN PARTICULAR EFFECTIVENESS AGAINST GRUBS IN CATTLE & REINDEER (10 & 30 MG/KG UPPER SAFE LIMITS, RESPECTIVELY) & OESTRUS OVIS IN SHEEP (25 MG/KG SC- AVOID USE IN HOT OR TIRED ANIMALS). OLDER MATERIALS MAY BE MORE TOXIC TO ANIMALS.

[Rossoff, I.S. Handbook of Veterinary Drugs. New York: Springer Publishing Company, 1974. 179]**QC REVIEWED**

INTERACTIONS:

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ENVIRONMENTAL FATE & EXPOSURE:

ENVIRONMENTAL FATE/EXPOSURE SUMMARY:

Dimethoate will be released to the environment from its production and use as a contact and systemic insecticide. If released to soil, dimethoate will not adsorb to the soil and will be subject to leaching. Evaporation from dry soil surfaces, and biodegradation in soil may be important removal mechanisms of dimethoate from soil. Soil half-lives of approximately 4 and 2.5 days were reported during drought and moderate rainfall conditions. However, a half-life of 122 days has also been measured in soil. Based on these half-lives and the fact that hydrolysis may be important in water under basic conditions, dimethoate may be susceptible to hydrolysis in moist, basic soils. In water, adsorption to sediment and bioconcentration in fish are not expected to be important transport processes. Photolysis and evaporation are not expected to be important fate processes of dimethoate in water. Dimethoate may be subject to biodegradation in natural waters based on a half-life of 8 weeks for degradation in raw river water. Hydrolysis may be important based on estimated aqueous hydrolysis half-lives of 118 and 3.7 day at pH 7 and pH 9, respectively. In the atmosphere, dimethoate may exist in the vapor- and particulate-phases. Degradation of vapor-phase dimethoate by reaction with photochemically produced hydroxyl radicals (estimated half-life of 5 days) will be important. Particulate-phase dimethoate may be removed from air via dry and wet deposition. Exposure to dimethoate will result mainly from occupational contact during its production and use as a contact and systemic insecticide. General population exposure may also occur through the ingestion of contaminated foods. (SRC) **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE:

Hand sprayers involved in spraying dimethoate using two different spraying techniques on crops inside a greenhouse were exposed to dimethoate through respiratory exposure at concns of 0.059 and 0.001 mg/h, and through dermal exposure at concns of 346 and 10.5 mg/h; machine operators were exposed to

dimethoate via respiratory and dermal exposure at concns of 0.034 and 0.0007 mg/h, and 29.6 and 1.5 mg/h, respectively(1). In a similar study(3), spraymen were dermally exposed to dimethoate at a concn range of 175.8-8322.1 ug/sq cm/day while respiratory exposure ranged 5.1-19.9 ug/day(3). Tractor operators involved in air blast spraying of citrus trees were dermally exposed to 0.05-23 ug/cm2/hr dimethoate while sitting in either an open tractor, in a cab with open windows, or a tractor with an open-cage canopy(2). Tractor operators were dermally exposed to dimethoate at concns of 0.01-0.81 ug/cm2/hr while sitting in a cab with closed windows(2). Workers are potentially exposed to dimethoate via dermal contact with readily dislodged foliar residues(3). After dimethoate was applied to orange trees at 1.4 kg/ha, potential worker exposure was measured to be 0.65, 0.2, 0.18, 0.029, 0.013 ug/sq cm after 3, 10, 17, 30, and 60 days, respectively(3). After dimethoate was applied to lemon trees at 1.4 kg/ha, potential worker exposure was measured to be 0.16, 0.034, 0.025, 0.008 ug/sq cm after 3, 10, 18, and 45 days, respectively(3). Based on several monitoring studies, the general population may be exposed to dimethoate via consumption of contaminated foods(SRC). [(1) Adamis Z et al; Int Arch Occup Environ Health 56: 299-305 (1985) (2) Carman GE et al; Arch Environ Contam Toxicol 11: 651-9 (1982) (3) Copplestone JF et al; Bull World Health Organ 54: 217-23 (1976) (3) Iwata Y et al; J Agric Food Chem 27: 1141-5 (1979)]**PEER REVIEWED**

Dimethoate can be absorbed by man through the unprotected skin, when inhaled as vapors or airborne droplets, or by ingestion. [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 1641]**PEER REVIEWED**

AVERAGE DAILY INTAKE:

The Average daily intake (AVDI) of dimethoate in 8 population groups in 1982-1984 was determined according to the FDA's monitoring program for chemical contaminants in the U.S. food supply (Total Diet Study or Market Basket Study)(1). In 6-11 month old infants, the AVDI was 7 ng/kg-body weight-per day. In 2 yr old toddlers, the AVDI was 6.4 ng/kg-body weight-per day(1). In 14-16 year old females, the AVDI was 2.9 ng/kg-body weight-per day(1). In 14-16 year old males, the AVDI was 2.5 ng/kg-body weight-per day(1). In 25-30 year old females, the AVDI was 10.1 ng/kg-body weight-per day(1). In 25-30 year old males, the AVDI was 7.7 ng/kg-body weight-per day(1). In 60-65 year old females, the AVDI was 7.9 ng/kg-body weight-per day(1). In 60-65 year old males, the AVDI was 7.8 ng/kg-body weight-per day(1). The AVDI of dimethoate in adult males age 16 -19 as determined by the Total Diet Study was 0.001 and 0.003 ng/kg-body

weight-per day for FY80 and FY81/82, respectively(2). The AVDI of dimethoate in toddlers as determined by the Total Diet Study was 0.001 ng/kg-body weight-per day for FY81/82(3). The AVDI estimated from FDA's 1990 Total Diet Study was 0.0081, 0.001, and 0.0023 ug/kg/day in 6-11 month old infants, 14-16 yr old males, and 60-65 yr old females, respectively(4). [(1) Gunderson EL; J Assoc Off Anal Chem 71: 1200-9 (1988) (2) Gartrell MJ et al; J Assoc Anal Chem 69:146-61 (1986) (3) Gartrell MJ et al; J Assoc Anal Chem 69: 123-45 (1986) (4) Winter CK; Rev Environ Contam Toxicol 127: 23-67 (1992)]**PEER REVIEWED**

ARTIFICIAL POLLUTION SOURCES:

Release of dimethoate to the environment will result from its production and use as a contact and systemic insecticide. The USEPA has cancelled the registration of dust formulations of dimethoate and prohibits its application without the use of personal protective equipment(1). [(1) USEPA; Health and Environ Effects Profile for Dimethoate (1985) EPA ECAO-CIN-PO81]**PEER REVIEWED**

ENVIRONMENTAL FATE:

TERRESTRIAL FATE: Soil half-lives of approximately 4 and 2.5 days were reported during drought and moderate rainfall conditions(7). According to all available data in the Pesticide Properties Database, the half-life of dimethoate in soil is 7 days(1). However, a half-life as long as 122 days has been determined for dimethoate in soil(3). Dimethoate will be susceptible to hydrolysis in moist alkaline soils based on a reported base catalyzed rate constant of 756 1/m-hr at pH 9 which corresponds to a half-life of 3.7 days in water(6). Based on experimental Koc values of 18, 36(4), 5.2(5), and 20(1), dimethoate is not expected to adsorb to soil(SRC). After 6 days, average dimethoate losses due to evaporation from uncovered soil columns were 40.4, 32.3, 23.0, and 24.9% in sand, sandy clay loam, loam, and clay loam, respectively; dimethoate losses were directly proportional to water losses(7); this study indicates that dimethoate may be removed from terrestrial surfaces via evaporation(SRC). Biodegradation may be an important fate process in soil(SRC) with 77% degradation reported in clay loam soil in 2 wks compared to 18 and 20% degradation in the same soil that had been autoclaved or irradiated(2). [(1) Wauchope RD et al; Rev Environ Contam Toxicol 123: 1-36 (1991) (2) Getzin LW, Rosefield I; J Agric Food Chem 16: 598-601 (1968) (3) Menzie CM; Ann Rev Entomol 17: 199-222 (1971) (4) Kanazawa J; Environ Toxicol Chem 8: 477-84 (1989) (5) Sabljic A; Environ Sci Technol 21: 358-66 (1987) (6) Ellington JJ et al; Measurement of hydrolysis rate constants for evaluation of hazardous waste Vol2 USEPA-600/S3-87/019 p.6 (1987) (7) USEPA; Health and Environ Effects Profile for Dimethoate EPA ECAO-CIN-PO81

(1985)]**PEER REVIEWED**

AQUATIC FATE: Based on experimental Koc values of 18, 36(1), 5.2(2), and 20(3), dimethoate is not expected to adsorb to sediments or suspended solids(SRC). Based on experimental BCF ranges of 1.1-2.4 and 2.7-6(4), and an estimated BCF of 2(5), dimethoate is not expected to bioconcentrate in fish(SRC). Based on respective neutral (pH 7) and base catalyzed (pH 9) hydrolysis rate constants at 25 deg C of 1.7X10-4/hr and 756 1/m-hr, respective hydrolysis half-lives of 118 and 3.7 days can be estimated for dimethoate in water(6). Photolysis and volatilization from water do not appear to be important removal process of dimethoate from water. Based on a half-life of 8 weeks in river water(7), dimethoate may be removed from water via biodegradation; however, the loss of dimethoate in this study may have been from hydrolysis and oxidation. [(1) Kanazawa J; Environ Toxicol Chem 8: 477-84 (1989) (2) Sabljic A; Environ Sci Technol 21: 358-66 (1987) (3) Wauchope RD et al; Rev Environ Contam Toxicol 123: 1-36 (1991) (4) Chemicals Inspection and Testing Institute; Japan Chemical Industry Ecology - Toxicology and Information Center ISBN 4-89074-101-1 (1992) (5) Kenaga EE; Ecotox Environ Safety 4: 26-38 (1980) (6) Ellington JJ et al; Measurement of hydrolysis rate constants for evaluation of hazardous waste Vol2 USEPA-600/S3-87/019 p.6 (1987) (7) Eichelberger JW, Litchtenberg JJ; Environ Sci Technol 5: 541-4 (1971)]**PEER REVIEWED**

ATMOSPHERIC FATE: According to a suggested classification scheme(2), the vapor pressure of 8.25X10-6 mm Hg at 25 deg C(1) indicates that dimethoate will exist in both the vapor- and particulate-phases in the ambient atmosphere. Particulate-phase dimethoate may be removed from air via dry deposition(SRC), and one monitoring study(4) indicates that dimethoate is removed from the atmosphere via wet deposition. Vapor-phase dimethoate is degraded in the ambient atmosphere by reaction with photochemically formed hydroxyl radicals(SRC); the half-life for this reaction in air can be estimated to be about 5 hrs(SRC) from a structure activity relationship(3). Products of oxidation and hydrolysis accounted for 1.5 and 11%, respectively, of the dimethoate deposited on an untreated glass surface left in a greenhouse for 7 days(5). Therefore, some oxidation and hydrolysis may account for the removal of dimethoate from air. Photolysis will not be an important removal mechanism from air(SRC). [(1) Tomlin C; The Pesticide Manual 10th ed. Bath, England: British Crop Council, Royal Society of Chemistry. p 349 (1994) (2) Eisenreich SJ et al; Environ Sci Technol 15: 30-8 (1981) (3) Meylan WM, Howard PH; Chemosphere 26: 2293-9 (1993) (4) Nations BK and Hallberg GR; J Environ Qual 21: 486-92 (5) USEPA; Health and Environ Effects Profile for Dimethoate EPA ECAO-CIN-PO81 249 pp (1985)]**PEER REVIEWED**

When applied to plants, dimethoate was ... decomposed ... on surface ... by hydrolysis & oxidation. On the plant surface, dimethoate underwent non-enzymatic oxidation to the oxygen analog & hydrolysis to water sol deriv identified as dimethyl phosphoric & O,O-dimethyl phosphorothioic acids, desmethyl dimethoate ... [Menzie, C.M. Metabolism of Pesticides. U.S. Department of the Interior, Bureau of Sport Fisheries and Wildlife, Publication 127. Washington, DC: U.S. Government Printing Office, 1969. 161]**PEER REVIEWED**

ENVIRONMENTAL BIODEGRADATION:

The concn of dimethoate left (initial concn 10 ppb) after various times in raw water from Little Miami river at pH 7.3-8.0 was 10 ppb after 1 hr, 10 ppb after 1 wk, 8.5 ppb after 2 wks, 7.5 ppb after 4wks, and 5.0 ppb after 8 wks(1). Biodegradation may play a minor role in the disappearance of dimethoate in the river water; no experiments were conducted with sterilized river water(1). Percent degradation in chehalis clay loam soil in 2 wk, non-sterile, 77%, autoclaved, 18%, irradiated, 20%(2). Half-lives in soil in June-July averaged 11 days, and less than 2% of applied dimethoate residue detected after 10 months(2). In laboratory experiments at 20-30 deg C half-lives for degradation were 28.9 and 36.7 days(2).However, dimethoate degraded faster when incubated for 30 days in samples of autoclaved sand, sandy clay loam, loam, and clay soils than in similarly treated nonsterile soils(5). Biodegradation appears to depend on the soil type and the microorganisms present in the soil(5). A half-life of 122 days has been observed in soil(7) which also suggests that biodegradation of dimethoate can be slow. In moist soils, dimethoate is readily oxidized to dimethoxon(6), but the role of microbial degradation on the removal of dimethoate from the environment is uncertain(3). Recovery of dimethoate incubated with enrichment cultures using raw sewage: 0 days, 54 ppm; 0.5 days, 54 ppm; 1 day, 52.5 ppm; 6 days, 22.4 ppm; 9 days, 13.5 ppm; 12 days, not detected(4). Using an initial concn of 100 mg/L dimethoate, 0-17 %Theoretical BOD was observed after a 4 week period in a biodegradation screening test using 30 mg/L sludge(8). [(1) Eichelberger JW, Litchtenberg JJ; Environ Sci Technol 5: 541-4 (1971) (2) Getzin LW, Rosefield I; J Agric Food Chem 16: 598-601 (1968) (3) USEPA; Health and Environ Effects Profile for Dimethoate EPA ECAO-CIN-PO81 246 pp. (1985) (4) Barik S et al; Agric Wastes 10: 81-94 (1984) (5) El Boit IOD et al; Int J Environ Studies 11: 113-24 (1977) (6) Duff WG, Menzer RE; Environ Entomol 2: 309-18 (1973) (7) Menzie CM; Ann Rev Entomol 17: 199-222 (1971) (8) Chemicals Inspection and Testing Institute; Japan Chemical Industry Ecology - Toxicology and Information Center ISBN 4-89074-101-1 (1992)]**PEER REVIEWED**

ENVIRONMENTAL ABIOTIC DEGRADATION:

Hydrolysis half-lives at 70 deg F were measured to be 22 hr in River Thames water at pH 8.0, 18 hr in River Irving water at pH 7.5, and 12 hr in ethanol/pH 6.0 buffer solution; it was roughly estimated that hydrolysis rates would be several hundred times slower at environmentally important temperatures(1). Reported hydrolysis half-lives at 70 deg F were 0.8 hr at pH 9 and 21 hr at pH 2(2). Based on respective neutral (pH 7) and base catalyzed (pH 9) hydrolysis rate constants at 25 deg C of 1.7X10-4/hr and 756 1/m-hr, respective hydrolysis half-lives of 118 and 3.7 days can be estimated for dimethoate in water(6). Based on a lack of absorbance of wavelengths of light above 290 nm(3), dimethoate will not be expected to undergo direct photolysis under sunlit conditions. Products of oxidation and hydrolysis accounted for 1.5 and 11%, respectively, of the dimethoate deposited on an untreated glass surface left in a greenhouse for 7 days(5). The rate constant for the vapor- phase reaction of dimethoate with photochemically produced hydroxyl radicals can be estimated from a structure activity relationship(4) to be 7.9X10-11 cu cm/molecule-sec at 25 deg C which corresponds to an atmospheric half-life of about 5 hrs at an atmospheric concn of 5X10+5 hydroxyl radicals per cu cm(SRC). [(1) Ruzicka JH et al; J Chromatog 31: 37-47 (1967) (2) Freed VH et al; Environ Health Perspect 20: 55-70 (1977) (3) Gore RC et al; J Assoc Off Anal Chem 54: 1040-82 (1971) (4) Meylan WM and Howard PH; Chemosphere 26: 2293-99 (1993) (5) USEPA; Health and Environ Effects Profile for Dimethoate EPA ECAO-CIN-PO81 249 pp (1985) (6) Ellington JJ et al; Measurement of hydrolysis rate constants for evaluation of hazardous waste Vol2 USEPA-600/S3-87/019 p.6 (1987)]**PEER REVIEWED**

ENVIRONMENTAL BIOCONCENTRATION:

After a 6 week period in a flow through system at 25 deg C, BCF ranges of 1.1-2.4 and 2.7-6 were determined in carp (cyprinus carpio) using initial concns of 1 and 0.1 mg/l dimethoate, respectively(1). An estimated BCF value of 2 has been reported for dimethoate(2). Based on these BCF values, dimethoate is not expected to bioconcentrate in fish(SRC). [(1) Chemicals Inspection and Testing Institute; Japan Chemical Industry Ecology - Toxicology and Information Center ISBN 4-89074-101-1 (1992) (2) Kenaga EE; Ecotox Environ Safety 4: 26-38 (1980)]**PEER REVIEWED**

SOIL ADSORPTION/MOBILITY:

The Koc values for dimethoate were measured to be 18 and 36 in a clay loam soil and clay soil, respectively(1). In another study, the Koc was measured to be 5.2 in an unspecified soil(2). According to the Pesticide Properties Database, the experimental Koc value for dimethoate is 20(3). According to a suggested classification scheme(4), these Koc values

suggest that dimethoate will have very high mobility in soil. Average dimethoate losses due to leaching of various soil columns with the equivalent of 2.5 cm of rain ranged from 39.6% (clay) to 78.6% (sand)(5). In four soils containing less than 1% organic content, the soil TLC Rf values ranged from 0.89 to 0.97(6). The soil TLC Rf values in two other soils (0.35-1.05% OC) was 0.40-0.50 and was not affected by pH or salt concentration changes(7). A 14-day perfusion flask experiment in soil treated with water, lime, or urea resulted in dimethoate losses from leaching of 79.2%, 83%, or 72.3%, respectively(8). [(1) Kanazawa J; Environ Toxicol Chem 8: 477-84 (1989) (2) Sabljic A; Environ Sci Technol 21: 358-66 (1987) (3) Wauchope RD et al; Rev Environ Contam Toxicol 123: 1-36 (1991) (4) Swann RL et al; Res Rev 85:17-28 (1983) (5) USEPA; Health and Environ Effects Profile for Dimethoate EPA ECAO-CIN-PO81 (1985) (6) Khan S, Khan NN; Soil Sci 142: 214-222 (1986) (7) Sharma SR et al; Ecotox Environ Safety 11: 229-240 (1986) (8) El Beit IOD et al; Int J Environ Studies 12: 235-60 (1978)]**PEER REVIEWED**

No dimethoate migration was observed below 3 inches in field sandy loam soil treated at a rate of 1 lb/acre dimethoate up to three times and analyzed for dimethoate up to 14 days after application(1). It has been reported that the downward movement of dimethoate in the field depends somewhat on the soil type; it increases with increasing soil moisture content(2). It was estimated that dimethoate could move approximately 2.5 cm in 1 month in soil containing 42.9% moisture; movement thereafter will probably decrease over time(3). [(1) Bohn WR; J Econ Entomol 57: 798-9 (1964) (2) Duff WG, Menzer RE; Environ Entomol 2: 309-18 (1973) (3) Graham-Bryce IJ; J Sci Food Agric 29: 489-94 (1969)]**PEER REVIEWED**

Soil sorption constants based on the organic carbon content of 15 pesticides were measured using 2 soils (clay loam and high clay) at 0.01, 0.1 and 1.0 ppm pesticide. The soil sorption coefficients ((ug pesticide/g soil)/(ug pesticide/g water)) for dimethoate were 0.8 + or - 0.3 in clay loam and 0.5 + or - 0.1 in high clay soil. The soil sorption constants were 18 and 36 respectively, with a mean of 27. Significant correlations were found between organic carbon content and water solubility, octanol/water partition coefficient, retention time in reversed phase high pressure liquid chromatography and molecular wt. [Kanazawa J; Environ Toxicol Chem 8 (6): 477-84 (1989)]**PEER REVIEWED**

VOLATILIZATION FROM WATER/SOIL:

Based on a vapor pressure of 8.25X10-6 mm Hg at 25 deg C and a water solubility of 23800 at pH 7 and 20 deg C(1), the Henry's Law constant for dimethoate can be estimated to be 1.05X10-10 atm-cu m/mole at 20-25 deg

C(SRC). According to a suggested classification scheme(2), this Henry's Law constant indicates that dimethoate will be essentially nonvolatile from water. After 6 days, average dimethoate losses due to evaporation from uncovered soil columns were 40.4, 32.3, 23.0, and 24.9% in sand, sandy clay loam, loam, and clay loam, respectively; dimethoate losses were directly proportional to water losses(3). The vaporization index for dimethoate was estimated to be 0.2-3 kg/ha/yr, or more(4). [(1) Tomlin C; The Pesticide Manual 10th ed. Bath, England: British Crop Council, Royal Society of Chemistry. p 349 (1994) (2) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Environmental Behavior of Organic Compounds. Washington DC: American Chemical Society pp. 4-9, 5-4, 5-10, 7-4, 7-5, 15-15 to 15-32 (1990) (3) USEPA; Health and Environ Effects Profile for Dimethoate EPA ECAO-CIN-PO81 (1985) (4) Haque R, Freed VH; Res Rev 52: 89-116 (1974)]**PEER REVIEWED**

ENVIRONMENTAL WATER CONCENTRATIONS:

DRINKING WATER: Dimethoate was not detected in water from 54 wells in California, in an area where it was used for at least 15 years(1). Dimethoate was not detected in 82 samples from 80 stations in New York State, 1964-66, including wells, groundwater, and surface water(2). [(1) Maddy KT et al; A Study of Well Water in Selected California Communities for Residues of 1,3-Dichloropropene, Chlorallyl Alcohol, and 49 Organophosphate or Chlorinated Hydrocarbon Pesticides CA Dept Food Agric HS-1002 (1982) (2) Zweig G, Devine JM; Res Rev 26: 17-36 (1969)]**PEER REVIEWED**

GROUNDWATER: According to the U.S. EPA's Pesticides in Groundwater Database, dimethoate was detected at a concn range of 0.38-10 ug/l in 12 of 2,844 wells from 9 different states monitored between 1984-1991(1). Dimethoate was qualitatively identified in 24 groundwater samples from wells in California between May 1779-April 1984(2). [(1) US EPA; Pesticides in Groundwater Database. A Compilation of Monitoring Studies: 1971-1991. National Summary. USEPA Off Pest Programs Prevention Pesticides and Toxic Substances (H7507C) USEPA-734-12-92-001 (2) Cohen DB; ACS Symp Ser 315(Eval Pestic Ground Water): 499-529 (1986)]**PEER REVIEWED**

SURFACE WATER: Dimethoate was detected at a concn range of 9.7-368.4 ng/l in river water from 4 rivers in Southern Italy(1). In 1985, dimethoate was detected in surface waters in the UK at a maximum concn of 0.94 ug/l(2). [(1) Provini A et al; Toxicol Environ Chem 31-2: 157-65 (1991) (2) Croll BT; J Inst Water Environ Manage 5: 389-95 (1991)]**PEER REVIEWED**

RAIN/SNOW: Dimethoate was detected at an average concn of 0.19 ug/l and a

maximum concn of 0.23 ug/l in precipitation samples from Iowa during November 1987- September 1990(1). [(1) Nations BK, Hallberg GR; J Environ Qual 21: 486-92 (1992)]**PEER REVIEWED**

EFFLUENT CONCENTRATIONS:

Dimethoate was qualitatively identified in water that was used to extinguish a burning chemical storage building in Belgium(1). In 1991, dimethoate was detected at an average concn range of 0.4- 1.08 ug/kg in farm ditch water which flows into three rivers in the lower Fraser Valley, British Columbia(2). [(1) Selala MI et al; Bull Environ Contam Toxicol 51: 325-32 (1993) (2) Wan MT; J Environ Sci Health B29: 917-49 (1994)]**PEER REVIEWED**

SEDIMENT/SOIL CONCENTRATIONS:

SOILS: Tadzik SSR, less than or equal to 0.3 ppm(2). Detected, not quantified in 10.6% of cotton field samples(1). [(1) Baratov KB; Izv Akad Nauk Tadzh SSR, Otd Biol Nauk 2: 89-92 (1978) (2) Baratov KB; Khlopkovodstvo 3: 23 (1980)]**PEER REVIEWED**

Dimethoate was detected in sediment samples from 4 of 6 sites in the Sarno and Vulturno rivers in Italy at an average concn range of 12-33 ng/g dry weight(1). [(1) Melluso G et al; Bull Environ Contam Toxicol 52: 13-8 (1994)]**PEER REVIEWED**

ATMOSPHERIC CONCENTRATIONS:

Dimethoate was not detected in U.S. ambient air samples during 1970-71(2). Based on the analysis of 123 ambient air samples collected from 10 U.S. locations during FY 1980, dimethoate was identified in 3.3 percent of the samples with a high concn of 23 ng/cu m(1). [(1) Kutz FW; Res Rev 85: 277-92 (1983) (2) Lewis RG, Lee RE Jr; Air Pollut From Pest and Agric Processes Lee RE Jr ed, CRC Press, Cleveland, OH pp 5-51 (1976)]**PEER REVIEWED**

FOOD SURVEY VALUES:

Dimethoate was qualitatively identified in 1 large fruit sample during Fiscal Year 1976 (FY76), 16 small fruit samples during FY75-76, 33 leaf and stem vegetables, 196 vine and ear vegetable sample, and 46 bean samples during FY74-76, and 1 root vegetable sample during FY75(1). After applying 150 grams dimethoate/ha to lettuce, dimethoate was detected on the outer leaves at concns of 4.61, 1.36, 0.19, and 0.01 ppm, fresh weight, after 2, 4, 7, and 14 days, respectively; it was detected on the inner leaves at a concn 0.01 ppm, fresh weight, after 7 days(2). After applying 300 grams dimethoate/ha to lettuce, dimethoate was detected on

the outer leaves at concns of 8.44, 1.2, 0.32, and 0.06 ppm, fresh weight, after 2, 4, 7, and 14 days, respectively; it was detected on the inner leaves at a concn of 0.04 ppm, fresh weight, after 7 days, respectively(2). Dimethoate was not detected above a detection limit of 0.01 ppm in the edible pulp of lemons and oranges 52-59 days postapplication at 1.4 kg/ha(3). [(1) Duggan RE et al; Pest Res Levels in Foods in the United States From July 1, 1969 to June 30, 1976; Washington DC FDA Div Chem Technol (1983) (2) Szeto SY et al; J Environ Sci Health B19: 225-35 (1984) (3) Iwata Y et al; J Agric Food Chem 27: 1141-5 (1979)]**PEER REVIEWED**

In a US monitoring survey of 6970 produce samples (fruits and vegetables) collected between 1989 and 1991, dimethoate was detected above a detection limit of 0.25 ppm in 3 of 198 grapes, 2 of 153 greens, 1 of 139 lemons, and 2 of 179 peppers(1). Fresh olive samples collected from olive groves in Greece between 1991 and 1992 contained dimethoate residues at a concn range of 0.005-0.07 mg/kg; dimethoate was also detected in one processed (ready to eat) olive sample at a concn of 0.05 mg/kg(2). During October 1, 1981 and September 30, 1986, dimethoate was found in 319 U.S. agricultural commodities at a concn range of > 0-0.05 ppm, 117 samples at 0.05-0.1 ppm, 192 at 0.1-0.5 ppm, 29 at 0.5-1.0 ppm, 20 at 1.0-2.0 ppm, and 8 agricultural commodities at a concn > 2.0 ppm(3). This study does not distinguish between domestic and imported commodities or between surveillance and compliance samples(3). During October 1, 1981 and September 30, 1986, dimethoate was found in 145 U.S. domestic agricultural commodities conducted by surveillance sampling at a concn range of > 0-0.05 ppm, 64 at 0.05-0.1 ppm, 105 at 0.1-0.5 ppm, 22 at 0.5-1.0 ppm, 17 at 1.0-2.0 ppm, and 5 domestic agricultural commodities at a concn > 2.0 ppm(4). During October 1, 1981 and September 30, 1986, dimethoate was found in 170 U.S. imported agricultural commodities conducted by surveillance sampling at a concn range of > 0-0.05 ppm, 50 at 0.05-0.1 ppm, 77 at 0.1-0.5 ppm, 4 at 0.5-1.0 ppm, 3 at 1.0-2.0 ppm, and 3 imported agricultural commodities at a concn > 2.0 ppm(4). During April 1, 1989 - December 3, 1991, dimethoate was detected in 17 of 843 Canadian domestic commodities of fresh apples at a concn range of > 0-0.5 ppm; dimethoate was detected in 6 of 324 fresh bean samples from Canada at a concn range of > 0-0.1 ppm, and it was detected in 21 of 281 fresh lettuce samples at a concn range of > 0-1 ppm(5). In the same Canadian study(5), dimethoate was also detected in imported commodities of fresh beans, apples, broccoli, cucumber, grapes, lettuce, orange, pepper, and pear. [(1) Schattenberg HJ III, Hsu JP; J AOAC Internatl 75: 925-33 (1992) (2) Lentza- Rizos C; J AOAC Internatl 77: 1096-100 (1994) (3) Luke MA et al; J Assoc Off anal Chem 71: 415-20 (1988) (4) Hundley HK et al; J Assoc Off

Anal Chem 71: 875-92 (1988) (5) Neidert E et al; J AOAC Int 77: 18-33 (1994)]**PEER REVIEWED**

Dimethoate has been detected, by FDA's pesticide residue monitoring program, in American foods during fiscal years 1978-1992(1-5); however, the concns and the frequencies of occurrence were not reported(1-5). Dimethoate was qualitatively identified in 167 of 27,065 human food samples during FY88-89(6). Dimethoate was identified between 1983 and 1984 in 1 of 47 composite samples of strawberries from Ontario, Canada at a concn of 0.04 mg/kg(7). [(1) Yess NJ et al; J Assoc Off Anal Chem 74: 273-80 (1991) (2)Yess NJ et al; J Assoc Off Anal Chem 74: 265-72 (1991) (3) FDA; J AOAC Int 76: 127A-48A (1993) (4) FDA; J Assoc Off Anal Chem 73: 127A-46A (1990) (5) FDA; J Assoc Off Anal Chem 74: 121A-41A (1991) (6) Minyard JP, Roberts WE; J Assoc Off anal Chem 74: 438-52 (1991) (7) Frank R et al; Bull Environ Contam Toxicol 39: 272-9 (1987)]**PEER REVIEWED**

FISH/SEAFOOD CONCENTRATIONS:

In a Russian study, dimethoate residues were detected in fish for 30 day after application at 1.5 kg/hectare. Residue accumulation occurred in the kidneys at levels as high as 159 mg/kg. [Korzhevenko GN et al; J Toxicol Environ Health 8 (1-2): 169-84 (1981) as cited in USEPA/ECAO; Health Effects Profile for Dimethoate (Final Draft) p.30 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

ENVIRONMENTAL STANDARDS & REGULATIONS:

FIFRA REQUIREMENTS:

Tolerances are established for total residues of the insecticide dimethoate including its oxygen analog (O,O-dimethyl S-(N-methylcarbamoylmethyl) phosphorothioate) in or on the following raw agricultural commodities: brussels sprouts, alfalfa, apples, beans (dry, lima, snap), broccoli, cabbage, cattle (fat, meat byproducts, meat), cauliflower, celery, collards, corn (fodder, forage, grain), cottonseed, eggs, endive (escarole), goats (fat, meat byproducts, meat), grapefruit, grapes, hogs (fat, meat byproducts, meat), horses (fat, meat byproducts, meat), kale, lemons, lentils, lettuce, melons,milk, mustard ggreens, oranges, pears, peas, pecans, peppers, potatoes, poultry (fat, meat byproducts, meat), safflower seed, sheep (fat, meat byproducts, meat), sorghum (forage, grain), soybeans, soybeans (forage, hay), spinach, Swiss chard, tangerines, tomatoes, turnips (roots, tops), and wheat (grain, green fodder, straw). [40 CFR 180.204(a) (7/1/94)]**PEER REVIEWED**

Tolerances with regional registration, as defined in 180.1(n), are established for total residues of dimethoate including its oxygen analog in or on the following raw agricultural commodities: cherries. [40 CRF 180.204(b) (7/1/94)]**PEER REVIEWED**

A tolerance is established for total residues of the insecticide dimethoate (O,O-dimethyl S-(N-methylcarbamoylmethyl) phosphorodithioate) including its oxygen analog (O,O-dimethyl S-(N-methylcarbamoylmethyl) phosphorothioate) in dried citrus pulp for cattle feed. Such residue may be present therein only as a result of the application of the insecticide to the growing agricultural crop. [40 CFR 186.2100 (4/1/94)]**PEER REVIEWED**

As the federal pesticide law FIFRA directs, EPA is conducting a comprehensive review of older pesticides to consider their health and environmental effects and make decisions about their future use. Under this pesticide reregistration program, EPA examines health and safety data for pesticide active ingredients initially registered before November 1, 1984, and determines whether they are eligible for reregistration. In addition, all pesticides must meet the new safety standard of the Food Quality Protection Act of 1996. Dimethoate is found on List A, which contains most food use pesticides and consists of the 194 chemical cases (or 350 individual active ingredients) for which EPA issued registration standards prior to FIFRA, as amended in 1988. Case No: 0088; Pesticide type: insecticide (acaricide); Registration Standard Date: 04/11/83; Case Status: OPP is reviewing data from the pesticide's producers regarding its human health and/or environmental effects, or OPP is determining the pesticide's eligibility for reregistration and developing the Reregistration Eligibility Decision (RED) document.; Active ingredient (AI): Dimethoate; Data Call-in (DCI) Date(s): 06/28/91, 10/13/95, 02/09/96; AI Status: The producers of the pesticide has made commitments to conduct the studies and pay the fees required for reregistration, and are meeting those commitments in a timely manner. [USEPA/OPP; Status of Pesticides in Registration, Reregistration and Special Review p.113 (Spring, 1998) EPA 738-R-98-002]**QC REVIEWED**

ACCEPTABLE DAILY INTAKES:

FAO/WHO ADI: 0.01 mg/kg [FAO/WHO; Pesticide Residues in Food - 1990. Evaluations Part 1 - Residues p.420 Plant Prod Protect Paper 103/1 (1990)]**PEER REVIEWED**

CERCLA REPORTABLE QUANTITIES:

Persons in charge of vessels or facilities are required to notify the

National Response Center (NRC) immediately, when there is a release of this designated hazardous substance, in an amount equal to or greater than its reportable quantity of 10 lb or 4.54 kg. The toll free number of the NRC is (800) 424-8802; In the Washington D.C. metropolitan area (202) 426-2675. The rule for determining when notification is required is stated in 40 CFR 302.4 (section IV. D.3.b). [40 CFR 302.4 (7/1/94)]**PEER REVIEWED**

Releases of CERCLA hazardous substances are subject to the release reporting requirement of CERCLA section 103, codified at 40 CFR part 302, in addition to the requirements of 40 CFR part 355. Dimethoate is an extremely hazardous substance (EHS) subject to reporting requirements when stored in amounts in excess of its threshold planning quantity (TPQ) of 500/10,000 lbs. [40 CFR 355 (7/1/97)]**QC REVIEWED**

RCRA REQUIREMENTS:

P044; As stipulated in 40 CFR 261.33, when dimethoate, as a commercial chemical product or manufacturing chemical intermediate or an off-specification commercial chemical product or a manufacturing chemical intermediate, becomes a waste, it must be managed according to federal and/or state hazardous waste regulations. Also defined as a hazardous waste is any container or inner liner used to hold this waste or any residue, contaminated soil, water, or other debris resulting from the cleanup of a spill, into water or on dry land, of this waste. Generators of small quantities of this waste may qualify for partial exclusion from hazardous waste regulations (40 CFR 261.5(e)). [40 CFR 261.33 (7/1/94)]**PEER REVIEWED**

STATE DRINKING WATER GUIDELINES:

(AZ) ARIZONA 1.2 ug/l[USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)]**QC REVIEWED**

(CA) CALIFORNIA 140 ug/l[USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)]**QC REVIEWED**

(FL) FLORIDA 5 ug/l[USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)]**QC REVIEWED**

(WI) WISCONSIN 2 ug/l[USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking

Water Standards and Guidelines (11/93)]**QC REVIEWED**

ALLOWABLE TOLERANCES:

Tolerances for total residues of the insecticide dimethoate including its oxygen analog (O,O-dimethyl S-(N-methylcarbamoylmethyl) phosphorothioate) in or on the following raw agricultural commodities, are as follows: 5.0 ppm for brussels sprouts; 2 ppm for alfalfa, apples, beans (dry, lima, snap), broccoli, cabbage, cauliflower, celery, collards, endive (escarole), grapefruit, kale, lemons, lentils, lettuce, mustard greens, oranges, pears, peas, peppers, soybeans (forage, hay), spinach, Swiss chard, tangerines, tomatoes, turnips (roots, tops), and wheat (green fodder, straw); 1 ppm for corn (fodder, forage), grapes, and melons; 0.1 ppm for cottonseed, pecans, safflower seed, and sorghum (grain); 0.1 ppm (negligible residue) for corn (grain); 0.2 ppm for potatoes, and sorghum (forage); 0.05 ppm (negligible residue) for soybeans; 0.04 ppm (negligible residue) for wheat (grain); 0.02 ppm (negligible residue) for cattle (fat, meat byproducts, meat), eggs, goats (fat, meat byproducts, meat), hogs (fat, meat byproducts, meat), horses (fat, meat byproducts, meat), poultry (fat, meat byproducts, meat), and sheep (fat, meat byproducts, meat); 0.002 ppm (negligible residue) for milk. [40 CFR 180.204(a) (7/1/94)]**PEER REVIEWED**

Tolerances with regional registration, as defined in 180.1(n), are established for total residues of dimethoate including its oxygen analog in or on the following raw agricultural commodities: cherries, 2 ppm. [40 CRF 180.204(b) (7/1/94)]**PEER REVIEWED**

A tolerance of 5 ppm is established for total residues of the insecticide dimethoate (O,O-dimethyl S-(N-methylcarbamoylmethyl) phosphorodithioate) including its oxygen analog (O,O-dimethyl S-(N-methylcarbamoylmethyl) phosphorothioate) in dried citrus pulp for cattle feed. Such residue may be present therein only as a result of the application of the insecticide to the growing agricultural crop. [40 CFR 186.2100 (4/1/94)]**PEER REVIEWED**

CHEMICAL/PHYSICAL PROPERTIES:

MOLECULAR FORMULA:

C5-H12-N-O3-P-S2 [Worthing, C.R. and S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 8th ed. Thornton Heath, UK: The British Crop Protection Council, 1987. 298]**PEER REVIEWED**

MOLECULAR WEIGHT:

229.28 [Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 508]**PEER REVIEWED**

COLOR/FORM:

Colorless crystals [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

ODOR:

CAMPHOR-LIKE ODOR [Worthing, C.R., S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 7th ed. Lavenham, Suffolk, Great Britain: The Lavenham Press Limited, 1983. 205]**PEER REVIEWED**

Mercaptan odor [Verschueren, K. Handbook of Environmental Data of Organic Chemicals. 2nd ed. New York, NY: Van Nostrand Reinhold Co., 1983. 541]**PEER REVIEWED**

BOILING POINT:

107 deg C at 0.05 mm Hg /From table/ [USEPA/ECAO; Health and Environmental Effects Profile for Dimethoate (Final Draft) p.2 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

MELTING POINT:

49 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

CORROSIVITY:

Slightly corrosive to iron [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

DENSITY/SPECIFIC GRAVITY:

1.277 @ 65 DEG C [Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 508]**PEER REVIEWED**

OCTANOL/WATER PARTITION COEFFICIENT:

Log Kow = 0.50 & 0.78, 0.78 is the recommended value [Hansch, C. and A. Leo. The Log P Database. Claremont, CA: Pomona College, 1987. 102]**PEER REVIEWED**

SOLUBILITIES:

SOL IN MOST ORG SOLVENTS, EXCEPT SATURATED HYDROCARBONS [Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 508]**PEER REVIEWED**

VERY SOL IN ETHANOL, CHLOROFORM, ACETONE; SLIGHTLY SOL IN DIETHYL ETHER;
INSOL IN PETROLEUM ETHER; SLIGHTLY SOL IN AROMATIC HYDROCARBONS [Sunshine, I. (ed.). CRC Handbook of Analytical Toxicology. Cleveland: The Chemical Rubber Co., 1969. 510]**PEER REVIEWED**

More than 5000 mg/l water @ 20 plus or minus 1.5 deg C [Bowman BT et al; J Environ Sci Health B18 (2): 221-7 (1983)]**PEER REVIEWED**

25 g/l water @ 21 deg C [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

SOL IN CYCLOHEXANONE; LOW SOLUBILITY IN XYLENE, HEXANE [Spencer, E. Y. Guide to the Chemicals Used in Crop Protection. 7th ed. Publication 1093. Research Institute, Agriculture Canada, Ottawa, Canada: Information Canada, 1982. 217]**PEER REVIEWED**

> 300 g/kg alcohol @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 300 g/kg benzene @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 300 g/kg chloroform @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 300 g/kg dichloromethane @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 300 g/kg ketones @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 300 g/kg toluene @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 50 g/kg carbon tetrachloride @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 50 g/kg saturated hydrocarbons @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

> 50 g/kg octan-1-ol @ 20 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

Solubility in water 23.3 (pH 5), 23.8 (pH 7), 25.0 (pH 9)(all in g/l at 20 deg C) [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 1994]**PEER REVIEWED**

SPECTRAL PROPERTIES:

INDEX OF REFRACTION: 1.5334 @ 65 DEG C/D [Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 508]**PEER REVIEWED**

Intense mass spectral peaks: 87 m/z (100%), 93 m/z (82%), 125 m/z (68%), 47 m/z (33%) [Hites, R.A. Handbook of Mass Spectra of Environmental Contaminants. Boca Raton, FL: CRC Press Inc., 1985. 323]**PEER REVIEWED**

Intense mass spectral peaks: 229 m/z [Pfleger, K., H. Maurer and A. Weber. Mass Spectral and GC Data of Drugs, Poisons and their Metabolites. Parts I and II. Mass Spectra Indexes. Weinheim, Federal Republic of Germany. 1985.368]**PEER REVIEWED**

VAPOR PRESSURE:

1.1 mPa @ 25 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

OTHER CHEMICAL/PHYSICAL PROPERTIES:

IT DECOMPOSES ON HEATING, INITIALLY FORMING THE O,S-DIMETHYL ANALOGUE

[Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

The technical grade (96% purity) forms white to greyish crystals; MP 45-47 deg C [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

CHEMICAL SAFETY & HANDLING:

SKIN, EYE AND RESPIRATORY IRRITATIONS:

May cause eye irritation. [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

FLASH POINT:

107 deg C (closed cup) [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

FIRE FIGHTING PROCEDURES:

Dry chemicals, carbon dioxide for small fires. Water spray or foam for larger fires. [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

PRIOR HISTORY OF ACCIDENTS:

The effects of accidental exposure to a vapor created by burning a mixture of 40% dimethoate, 34% xylol, 16% phenol, and 10% of an alkyl aryl sulfate are described in 26 exposed firemen and compared to 20 unexposed subjects. Immediately after the accident, 24% of the exposed individuals experienced respiratory tract and conjunctival irritation, and 24% related that they had suffered diarrhea, involuntary defecation, or abdominal cramps. Other symptoms included headache, anxiety or depression, itching, and decrease in libido in the first few weeks after the accident. One month after the exposure, a nauseating smell was evident in the perspiration of 23% of the exposed individuals, or came out of their hair when it was washed. Breath and/or feces also had this smell in some persons. All individuals were asymptomatic within 8-12 months after the exposure. ... [Matos E; Medicina 42 (4): 381-4 (1982)]**PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING:

/Wear/ impervious gloves, boots, body-covering ... [Farm Chemicals

Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

/Wear/ ... closed collar gowns, rubber gloves and long boots. [ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 187]**PEER REVIEWED**

All applicators, including homeowners & flaggers & personnel involved with mixing, loading, & transferring operations, must wear the protective clothing & equipment enumerated. Pilots are exempt from this equipment. The protective clothing & equipment to be worn is as follows: a. Impermeable gloves (for example, rubber or plastic covered gloves). b. Rubber or synthetic rubber boots or boot covers. c. Long-sleeved shirt & long pants, made of closely woven fabric. d. Wide-brimmed hat. e. Respirators must be worn by flaggers & mixer/loaders. [Environmental Protection Agency/OPTS. Suspended, Cancelled and Restricted Pesticides. 3rd Revision. Washington, D.C.: Environmental Protection Agency, January 1985. 4]**PEER REVIEWED**

PREVENTIVE MEASURES:

Avoid eye, skin, clothing contact. Do not breathe dust. Use with adequate ventilation. ... Wash thoroughly after handling. [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

Maintain good ventilation at the site. No eating and smoking at the site. Wash daily all the garments. [ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 187]**PEER REVIEWED**

/SRP/: Contact lenses should not be worn when working with this chemical. **PEER REVIEWED**

SRP: The scientific literature for the use of contact lenses in industry is conflicting. The benefit or detrimental effects of wearing contact lenses depend not only upon the substance, but also on factors including the form of the substance, characteristics and duration of the exposure, the uses of other eye protection equipment, and the hygiene of the lenses. However, there may be individual substances whose irritating or corrosive properties are such that the wearing of contact lenses would be harmful to the eye. In those specific cases, contact lenses should not be worn. In any event, the usual eye protection equipment should be worn even when contact lenses are in place. **PEER REVIEWED**

SRP: Contaminated protective clothing should be segregated in such a manner so that there is no direct personal contact by personnel who handle, dispose, or clean the clothing. Quality assurance to ascertain the completeness of the cleaning procedures should be implemented before the decontaminated protective clothing is returned for reuse by the workers. All contaminated clothing should not be taken home at end of shift, but should remain at employee's place of work for cleaning. **PEER REVIEWED**

STABILITY/SHELF LIFE:

The biological activity remains practically unvaried for 2 yr under environmental conditions, provided stored in unopened and undamaged original containers, in shaded, cool, well-aired places. ... Crystals may form in formulations stored at < 32 deg F/0 deg C. Stable a minimum of 1 yr at < 25-30 deg C/77-86 deg F. [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

STABLE IN AQ SOLN [Hayes, W.J., Jr., E.R. Laws, Jr., (eds.). Handbook of Pesticide Toxicology. Volume 2. Classes of Pesticides. New York, NY: Academic Press, Inc., 1991. 1016]**PEER REVIEWED**

UNSTABLE IN ALKALINE MEDIA, STABILITY INCR @ PH VALUES BETWEEN 4 TO 7 [Spencer, E. Y. Guide to the Chemicals Used in Crop Protection. 7th ed. Publication 1093. Research Institute, Agriculture Canada, Ottawa, Canada: Information Canada, 1982. 217]**PEER REVIEWED**

... 50% loss occurs in 12 days at pH 9 ... [Worthing, C.R. and S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 8th ed. Thornton Heath, UK: The British Crop Protection Council, 1987. 298]**PEER REVIEWED**

Dimethoate is thermally unstable and decomposes on heating after first being converted to the more toxic dithio-isomer. It is hydrolyzed more rapidly in alkaline medium: 50% hydrolysis @ 70 deg C @ pH 9 in 0.8 hr and 21 hr @ pH 2. [United Nations. Treatment and Disposal Methods for Waste Chemicals (IRPTC File). Data Profile Series No. 5. Geneva, Switzerland: United Nations Environmental Programme, Dec. 1985. 243]**PEER REVIEWED**

STORAGE CONDITIONS:

The biological activity remains practically unvaried for 2 yr under environmental conditions, provided stored in unopened and undamaged original containers, in shaded, cool, well-aired places, inaccessible to animals & unauthorized persons. Recommended temp < 25 deg C/77 deg F. Crystals may form in formulations stored at < 32 deg F/0 deg C. Stable a minimum of 1 yr at < 25-30 deg C/77-86 deg F. Stack containers

to permit air circulation at bottom & inside of piles. Do not contaminate food, feed products. [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

Liquid formulations must be stored above 45 deg F. [Farm Chemicals Handbook 1986. Willoughby, Ohio: Meister Publishing Co., 1986.,p. C-84]**PEER REVIEWED**

CLEANUP METHODS:

Use of granular, activated carbon in the adsorption of pesticides from wastewater is presented. Dimethoate was one of the compounds studied. [Dennis WH Jr et al; J Environ Sci Health B18 (3): 317-31 (1983)]**PEER REVIEWED**

Absorb with paper towels. Place in a plastic bag. Burn in an open pan with help of flammable solvent or in a furnace. [ITII. Toxic and Hazardous Industrial Chemicals Safety Manual. Tokyo, Japan: The International Technical Information Institute, 1988. 187]**PEER REVIEWED**

DISPOSAL METHODS:

Generators of waste (equal to or greater than 100 kg/mo) containing this contaminant, EPA hazardous waste number P044, must conform with USEPA regulations in storage, transportation, treatment and disposal of waste. [40 CFR 240-280, 300-306, 702-799 (7/1/89)]**PEER REVIEWED**

Potential candidate for rotary kiln incineration with a temperature range of 820-1600 deg C with residence times for liquids and gases, seconds; solids, hours. Also, a potential candidate for fluidized bed incineration with a temperature range of 450 to 980 deg C with residence times for liquids and gases, seconds; solids longer. /From table/ [USEPA; Engineering Handbook for Hazardous Waste Incineration p.3-9 (1981) EPA 68-03-3025]**PEER REVIEWED**

This compound should be susceptible to removal from wastewater by air stripping. [USEPA/ORD; Innovative and Alternative Technology Assessment Manual pp. 3-5, 3-11,12 (1980) EPA 430-9-78-009]**PEER REVIEWED**

Group I Containers: Combustible containers from organic or metallo-organic pesticides (except organic mercury, lead, cadmium, or arsenic compounds) should be disposed of in pesticide incinerators or in specified landfill sites. /Organic or metallo-organic pesticides/ [40 CFR 165.9 (a) (7/1/94)]**PEER REVIEWED**

Group II Containers: Non-combustible containers from organic or metallo-organic pesticides (except organic mercury, lead, cadmium, or arsenic compounds) must first be triple-rinsed. Containers that are in good condition may be returned to the manufacturer or formulator of the pesticide product, or to a drum reconditioner for reuse with the same type of pesticide product, if such reuse is legal under Department of Transportation regulations (eg 49 CFR 173.28). Containers that are not to be reused should be punctured ... and transported to a scrap metal facility for recycling, disposal or burial in a designated landfill. /Organic or metallo-organic pesticides/ [40 CFR 165.9 (b) (7/1/94)]**PEER REVIEWED**

Dimethoate is thermally unstable and decomposes on heating after first being converted to the more toxic dithio-isomer. It is hydrolyzed more rapidly in alkaline medium: 50% hydrolysis @ 70 deg C @ pH 9 in 0.8 hr and 21 hr @ pH 2. Mix dimethoate with lime and bury. Recommendable methods: Incineration, hydrolysis, adsorption, & landfill. Peer-review: Large amt - incinerate @ high temp in a unit with effluent gas scrubbing. Adsorb product on vermiculite and landfill. (Peer-review conclusions of an IRPTC expert consultation (May 1985)) [United Nations. Treatment and Disposal Methods for Waste Chemicals (IRPTC File). Data Profile Series No. 5. Geneva, Switzerland: United Nations Environmental Programme, Dec. 1985. 243]**PEER REVIEWED**

Alkaline hydrolysis: Dimethoate is detoxicated with alkaline cmpd. For this purpose, recommended are the preparation DIAS (a mixture of synthetic surfactants and organic solvents and alkali), 3-5% soln of potassium hydroxide, soda ash or lime chloride (1 kg in 4 l of water). The rate of detoxication increases with heating. The overalls polluted with organophosphorus cmpd should be shaken and soaked in soap-and-soda soln for 6-8 hr. Then the overalls should be washed two or three times in a hot soap-and-soda soln and rinsed carefully. Containers should be decontaminated with 5% caustic or washing soda (300-500 g per 10 l of water). The containers should be first filled with this soln, kept for 6-12 hr, then washed with ample water. If soda is not available, wood ash may be used instead. Waste dimethoate and containers should be destroyed in accordance with the sanitary rules. [United Nations. Treatment and Disposal Methods for Waste Chemicals (IRPTC File). Data Profile Series No. 5. Geneva, Switzerland: United Nations Environmental Programme, Dec. 1985. 243]**PEER REVIEWED**

OCCUPATIONAL EXPOSURE STANDARDS:

MANUFACTURING/USE INFORMATION:

MAJOR USES:

Systemic insecticide-acaricide used for a wide range of insects such as aphids, thrips, planthoppers, white flies, mites on ornamental plants, alfalfa, apples, corn, cotton, grapefruit, grapes, lemons, melons, oranges, pears, pecans, safflower, sorghum, soybeans, tangerines, tobacco, tomatoes, watermelons, wheat, other vegetables; residual wall spray in farm buildings for houseflies [Farm Chemicals Handbook 1994. Willoughby, OH: Meister, 1994.,p. C-124]**PEER REVIEWED**

INSECTICIDE FOR DECIDUOUS FRUITS & NUTS & COMMERCIAL/INDUSTRIAL USES [SRI]**PEER REVIEWED**

It is effective against Diptera of medical importance [Worthing, C.R. and S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 8th ed. Thornton Heath, UK: The British Crop Protection Council, 1987. 298]**PEER REVIEWED**

FOR FRUIT FLY LARVAE [White-Stevens, R. (ed.). Pesticides in the Environment: Volume 1, Part 1, Part 2. New York: Marcel Dekker, Inc., 1971. 100]**PEER REVIEWED**

MEDICATION (VET) **QC REVIEWED**

MANUFACTURERS:

Drexel Chemical Company, 1700-1740 Channel Ave, PO Box 9306, Memphis, TN 38113-0306, (901) 774-4370; Production site: Cordele, GA 31051 [SRI; 1995 - Directory of Chemical Producers. p.799 (1995)]**PEER REVIEWED**

METHODS OF MANUFACTURING:

REACTION OF SODIUM DIMETHYLPHOSPHORODITHIOATE WITH PHENYL CHLOROACETATE FOLLOWED BY REACTION WITH METHYLAMINE; REACTION OF METHYL ESTER OF O,O-DIMETHYL DITHIOPHOSPHORYLACETATE WITH METHYLAMINE [SRI]**PEER REVIEWED**

... O,O-dimethyl dithiophosphoric acid ... /was/ added ... to ...

anhydrous sodium carbonate suspended in ... methyl isobutyl ketone. The mixture was then heated to about 65 deg C & ... N-methyl chloroacetamide ... added. The resulting mixture was held at 80 deg C for 1 hr with continued stirring, then cooled to room temp & filtered. The filtrate was washed twice with water, dried over anhydrous sodium sulfate, & filtered. The filtrate was heated under vacuum to remove the methyl isobutyl ketone. The residual product /is/ S-carbamoylmethyl O,O-dimethyl dithiophosphate ... [Sittig, M. (ed.) Pesticide Manufacturing and Toxic Materials Control Encyclopedia. park Ridge, NJ: Noyes Data Corporation. 1980. 314]**PEER REVIEWED**

GENERAL MANUFACTURING INFORMATION:

... LITTLE CROSS-RESISTANCE /IN FLIES/ IS SHOWN TO DIMETHOATE, & THIS ORGANOPHOSPHATE CMPD INDUCES LITTLE OR NO RESISTANCE TO ITSELF. [White-Stevens, R. (ed.). Pesticides in the Environment: Volume 1, Part 1, Part 2. New York: Marcel Dekker, Inc., 1971. 476]**PEER REVIEWED**

A cholinesterase inhibitor, use has been restricted. [Lewis, R.J., Sr (Ed.). Hawley's Condensed Chemical Dictionary. 12th ed. New York, NY: Van Nostrand Rheinhold Co., 1993 410]**PEER REVIEWED**

An insecticidal coating compound containing 3-30% by weight insecticide at 70-97% vinyl dispersion. The vinyl dispersion is a fluid suspension of a vinyl resin with particle size of 0.75-1.6 mu, an inherent viscosity, as determined by ASTM D1234, of 0.80-1.20, and curing temperatures of 20-180 deg in a liquid plasticizer system. Thus, a mixture of vinyl dispersion resin 51.3, butyl benzyl phthalate 28.6, calcium, & zinc stearate 1.7, flucythrinate 7.5, and epoxidized soybean oil 2.86% by weight was coated on cattle ear tags and cured at 148 deg for 5 minutes. [Fishbein R et al; Eur Patent Applic Patent No. 167726 (01/15/86) assigned to American Cynamid Co]**PEER REVIEWED**

PREPN: CASSADAY ET AL, YOUNG, US PATENTS 2,494,283 & 2,996,531 (1950, 1961, BOTH TO AM CYANAMID); BRITISH PATENT 791,824 (1958 TO MONTECATINI),
CA 52, 18222 (1958). [Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989. 508]**PEER REVIEWED**

IT IS INCOMPATIBLE WITH ALKALINE PESTICIDES. ... IT IS A CONTACT & SYSTEMIC INSECTICIDE & ACARICIDE EFFECTIVE AT 300-700 G AI/HA AGAINST
A BROAD RANGE OF INSECTS & MITES ON WIDE RANGE OF CROPS. [Worthing,

C.R. and S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 8th ed. Thornton Heath, UK: The British Crop Protection Council, 1987. 298]**PEER REVIEWED**

In the manufacture of dimethoate, by-products that cause severe eye irritation have been identified, mainly bis(dimethoxythiophosphoryl) disulfide, which in rabbits produced opacity of the cornea, imflammatory reaction and edema in conjunctiva and lids. [Grant, W.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986. 347]**PEER REVIEWED**

FORMULATIONS/PREPARATIONS:

Emulsifiable concentrate ... dustable powder; aerosol; ULV /ultra low vol/ liquid ... [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

THESE INCLUDE: BIO SYSTEMIC INSECTICIDE (PAN BRITANNICA IND), VITEX (LA LITTORALE) EC (200, 400, OR 600 G TECHNICAL/L); 'ROGOR AS' UL (300 G/L); TURBAIR SYSTEMIC INSECTICIDE (PAN BRITANNICA IND), UL; WP (200 G/KG); GRANULES (50 G/KG). [Worthing, C.R. and S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 8th ed. Thornton Heath, UK: The British Crop Protection Council, 1987. 299]**PEER REVIEWED**

Combinations: Salut and Saluthion (BASF AG) (with chlorpyrifos). [Farm Chemicals Handbook 1994. Willoughby, OH: Meister, 1994.,p. C-124]**PEER REVIEWED**

Mixed formulations: (dimethoate(+) permethrin; dichlorvos; endosulfan; lindane (+) malathion; parathion; chlorpyrifos; fenitrothion; fenvalerate. [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

Technical grade is 98% pure [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

Use of dry formulations is not permitted. [Lewis, R.J., Sr (Ed.). Hawley's Condensed Chemical Dictionary. 12th ed. New York, NY: Van Nostrand Rheinhold Co., 1993 410]**PEER REVIEWED**

IMPURITIES:

(31)P nuclear magnetic resonance was used to analyze technical grade dimethoate. Dimethoate contained O,O,S-trimethyl phosphorodithioate & other unidentified phosphorus contaminants. [Greenhalgh R et al; J Agric Food Chem 31 (4): 710-13 (1983)]**PEER REVIEWED**

CONSUMPTION PATTERNS:
INSECTICIDE FOR VEGETABLES, 31%; CITRUS, 23%; DECIDUOUS FRUITS & NUTS,
15%; ALFALFA, 8%; COTTON, 8%; WHEAT, 8%; OTHER FIELD CROPS-EG, SORGHUM
& SOYBEANS, 8% (1982) [SRI]**PEER REVIEWED**

Insecticide use: 2,959,621 lbs active ingredient per year. [Gianessi LP, Puffer CA; Insecticide use in U.S. crop production. Resources for the Future. November (1992)]**PEER REVIEWED**

U. S. PRODUCTION:
(1978) 1.04X10+9 G (CONSUMPTION-INCL IMPORTS) [SRI]**PEER REVIEWED**

(1982) 5.90X10+8 G (CONSUMPTION-INCL IMPORTS) [SRI]**PEER REVIEWED**

(1976) 2.5x10+6 lb [USEPA; Dimethoate Position Document 2/3 (1979) as cited in USEPA/ECAO; Health and Environmental Effects Profile for Dimethoate (Final Draft) p.2 (1984) ECAO-CIN-PO81]**PEER REVIEWED**

LABORATORY METHODS:

ANALYTIC LABORATORY METHODS:
PRODUCT ANALYSIS IS BY GAS-LIQUID CHROMATOGRAPHY (CIPAC HANDBOOK, 1980,
1A, 1225; CIPA PROC, 191, 3 204, 211) OR BY THIN-LAYER CHROMATOGRAPHY
& ESTIMATION OF PHOSPHORUS IN APPROPRIATE SPOTS ... RESIDUES MAY BE
DETERMINED BY GAS LIQUID CHROMATOGRAPHY ... [TOMLIN C; THE PESTICIDE MANUAL - INCORPORATING THE AGROCHEMICALS HANDBOOK. 10TH ED. CROP PROTECTION PUBLICATIONS, P.350 (1994)]**PEER REVIEWED**

GAS-LIQUID CHROMATOGRAPHY METHOD FOR DIMETHOATE & DIMETHOXON RESIDUES:
WA STELLER & NR PARASELLA, J ASSOC OFF ANAL CHEM, 55, 1280, 1972. [Spencer, E. Y. Guide to the Chemicals Used in Crop Protection. 7th ed.

Publication 1093. Research Institute, Agriculture Canada, Ottawa, Canada: Information Canada, 1982. 217]**PEER REVIEWED**

Relative retention time data for 194 pesticides and metabolites, including dimethoate, are reported for a 15 m SE-30 capillary gas chromatography column under a single temp-programmed regime. The reproducibility of retention time and quantitation is discussed and the performance of electron capture and nickel thermionic detectors is evaluated in relation to pesticide residue analysis. [Ripley BD et al; J Assoc Off Anal Chem 66 (5): 1084-95 (1983)]**PEER REVIEWED**

A thin-layer chromatographic method is reported for the separation of dimethoate, dimethoate (oxygen analog), dioaxthion, disulfoton, fonofos (oxygen analog), and oxydemetonmethyl. The method involves the concurrent use of two Eastman silica gel plates with fluorescent indicator and two solvent systems. One plate is developed in solvent system containing 2,2,4-trimethylpentane:methylcyclohexane:isoamyl alcohol:paraffin oil: toluene:cyclohexane:methylcyclohexane (1:1:1:1:1:1). The pesticides are located by spraying with ammoniacal silver nitrate solution in acetone followed by exposure to long-wave uv light after drying at 100 deg C for 2-3 minutes. A method is also reported for the thin-layer chromatographic separation of diazinon, dimethoate, dimethoate (oxygen analog), dioxathion, fonofos (oxygen analog), oxydemetonmethyl from each other using solvent system containing 2,2,4-trimethylpentane:methylcyclohexane:isoamyl alcohol:parafin oil: acetone (4:2:2:3:1). [Frederici JA, Paul J; Microchem J 34 (2): 211-8 (1986)]**PEER REVIEWED**

EPA Method 8141 is a gas chromatographic method used to determine dimethoate in ground water, soil, and non-water miscible waste. A gas chromatograph with a flame photometric or nitrogen-phosphorus detector is used for this multiresidue procedure. Method detection limits for this compound using a flame photometric detector are 0.26 ug/l for water, and 13.0 ug/kg for soil. M8141 [USEPA/Office of Solid Waste (OSW); Test Methods for Evaluating Solid Waste, Physical/Chemical Methods SW846 Methods (1986)]**PEER REVIEWED**

Method 8270A, Semivolatile Organic compounds by Gas Chromatography/Mass Spectrometry (GC/MS): Capillary Column Technique; Estimated quantitation limit is 20 ug/l for water; quantitation limit not reported for soil [USEPA/Office of Solid Waste (OSW); Test Methods for Evaluating Solid Waste, Physical/Chemical Methods SW846 Methods (1986)]**PEER REVIEWED**

SPECIAL REFERENCES:

SPECIAL REPORTS:

USEPA/ECAO; Health and Environmental Effects Profile for Dimethoate (1985) ECAO-CIN-PO81

Talukdar AR; Nucleus 28 (3): 243-59 (1985). A review with 248 references on the metabolism and toxicologic risks of dimethoate. /Topics also considered include/: mutagenicity, clastogenicity, teratogenicity, factors affecting toxicity, metabolites of dimethoate, their formation and toxicity, degradation pathway, and its use in Indian agriculture.

US Dept of Agriculture; The Biologic and Economic Assessment of Dimethoate. A Report of the Dimethoate Assessment Team to the Rebuttable Presumption Against Registration of Dimethoate. Tech Bull USDA 1663: 322 pp. (1981). A review with 425 references on the uses of dimethoate. Benefit analyses are discussed and other alternatives are /presented/.

Reuber MD; Environ Res 34 (2): 193-211 (1984). The carcinogenicity of dimethoate is considered.

DHEW/NCI; Bioassay of Dimethoate for Possible Carcinogenicity (1977) Technical Rpt Series No. 4 DHEW Pub No. (NIH) 77-804

WHO; Environ Health Criteria Number 90: Dimethoate (1989).

SYNONYMS AND IDENTIFIERS:

RELATED HSDB RECORDS:

1587 [FORMOTHION] (Analog)

SYNONYMS:

L-395 [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

AC-12880 [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational

Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

AC-18682 [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

ACETIC ACID, O,O-DIMETHYLDITHIOPHOSPHORYL-, N-MONOMETHYLAMIDE SALT [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

AMERICAN CYANAMID 12,880 **PEER REVIEWED**

Bi-58 **PEER REVIEWED**

8014 BIS HC **PEER REVIEWED**

Cekuthoate [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

CL 12880 **PEER REVIEWED**

CYGON **PEER REVIEWED**

CYGON 4E **PEER REVIEWED**

CYGON INSECTICIDE **PEER REVIEWED**

DAPHENE [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

De-Fend [U.S. Department of the Interior, Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

DEMOS-L40 **PEER REVIEWED**

Devigon [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

Dimet [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

DIMETATE **PEER REVIEWED**

DIMETHOAAT (DUTCH) **PEER REVIEWED**

DIMETHOAT (GERMAN) **PEER REVIEWED**

Dimethoat Tech 95% [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

DIMETHOGEN [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

O,O-DIMETHYLDITHIOPHOSPHORYLACETIC ACID, N-MONOMETHYLAMIDE SALT **PEER REVIEWED**

O,O-Dimethyl-dithiophosphorylessigsaeure monomethylamid (German) **PEER REVIEWED**

O,O-DIMETHYL-S-(N-METHYL-CARBAMOYL)-METHYL-DITHIOFOSFAAT (DUTCH) **PEER REVIEWED**

O,O-DIMETHYL S-(N-METHYLCARBAMOYLMETHYL) DITHIOPHOSPHATE **PEER REVIEWED**

(O,O-DIMETHYL-S-(N-METHYL-CARBAMOYL-METHYL)-DITHIOPHOSPHAT) (GERMAN)
PEER REVIEWED

O,O-DIMETHYL S-METHYLCARBAMOYLMETHYL PHOSPHORODITHIOATE [The Merck Index.
10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 469]**PEER REVIEWED**

O,O-DIMETHYL S-(N-METHYLCARBAMOYLMETHYL) PHOSPHORODITHIOATE **PEER REVIEWED**

O,O-dimethyl-S-(2-oxo-3-aza-butyl)-dithiophosphat (German) **PEER REVIEWED**

O,O-dimetil-S-(n-metil-carbamoil-metil)-ditiofosfato (Italian) **PEER REVIEWED**

DIMETON **PEER REVIEWED**

DIMEVUR **PEER REVIEWED**

Dithiophosphate de O,O-dimethyle et de s(-n-methylcarbamoyl-methyle) (French) **PEER REVIEWED**

End 24650 [U.S. Department of the Interior, Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153. Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

ENT 24650 **PEER REVIEWED**

EXPERIMENTAL INSECTICIDE 12,880 **PEER REVIEWED**

FIP **PEER REVIEWED**

Fortion NM [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

Fosfamid (USSR) [Farm Chemicals Handbook 1989. Willoughby, OH: Meister Publishing Co., 1989.,p. C-104]**PEER REVIEWED**

Fosfatox R **PEER REVIEWED**

FOSFOTOX **PEER REVIEWED**

FOSFOTOX R **PEER REVIEWED**

FOSFOTOX R 35 **PEER REVIEWED**

Fostion [U.S. Department of the Interior, Fish and Wildlife Service. Handbook of Toxicity of Pesticides to Wildlife. Resource Publication 153.

Washington, DC: U.S. Government Printing Office, 1984. 32]**PEER REVIEWED**

FOSTION MM **PEER REVIEWED**

LURGO **PEER REVIEWED**

S-METHYLCARBAMOYLMETHYL O,O-DIMETHYL PHOSPHORODITHIOATE **PEER REVIEWED**

N-Monomethylamide of O,O-dimethyldithiophosphorylacetic acid [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

NCI-C00135 **PEER REVIEWED**

Caswell number 358 [USEPA/OPP; Catalog of Pesticide Chemical Names and Their Synonyms p.95 (1986)]**PEER REVIEWED**

PEI 75 **PEER REVIEWED**

PERFECTHION **PEER REVIEWED**

PERFEKTHION **PEER REVIEWED**

EPA pesticide code 035001 [USEPA/OPP; Catalog of Pesticide Chemical Names and Their Synonyms p.95 (1986)]**PEER REVIEWED**

PHOSPHAMID **PEER REVIEWED**

PHOSPHAMIDE **PEER REVIEWED**

PHOSPHORODITHIOIC ACID O,O-DIMETHYL ESTER, ESTER WITH 2-MERCAPTO-N-METHYLACETAMIDE [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 469]**PEER REVIEWED**

PHOSPHORODITHIOIC ACID, O,O-DIMETHYL ESTER, S-ESTER WITH 2-MERCAPTO-N-METHYLACETAMIDE **PEER REVIEWED**

PHOSPHORODITHIOIC ACID, O,O-DIMETHYL S-(2-(METHYLAMINO)-2-OXOETHYL) ESTER

PEER REVIEWED

RACUSAN **PEER REVIEWED**

Rebelate [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

ROGOR **PEER REVIEWED**

ROGOR L **PEER REVIEWED**

ROGOR P **PEER REVIEWED**

ROGOR 40 **PEER REVIEWED**

ROGOR 20L **PEER REVIEWED**

ROXION **PEER REVIEWED**

Roxion UA [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

SINORATOX **PEER REVIEWED**

Solut [Farm Chemicals Handbook 1986. Willoughby, Ohio: Meister Publishing Co., 1986.,p. C-85]**PEER REVIEWED**

Systemin **PEER REVIEWED**

SYSTOATE **PEER REVIEWED**

Trimetion [U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety Health. Registry of Toxic Effects of Chemical Substances (RTECS). National Library of Medicine's current MEDLARS file.p. 86/8606]**PEER REVIEWED**

FORMULATIONS/PREPARATIONS:

Emulsifiable concentrate ... dustable powder; aerosol; ULV /ultra low vol/ liquid ... [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

THESE INCLUDE: BIO SYSTEMIC INSECTICIDE (PAN BRITANNICA IND), VITEX (LA LITTORALE) EC (200, 400, OR 600 G TECHNICAL/L); 'ROGOR AS' UL (300 G/L); TURBAIR SYSTEMIC INSECTICIDE (PAN BRITANNICA IND), UL; WP (200 G/KG); GRANULES (50 G/KG). [Worthing, C.R. and S.B. Walker (eds.). The Pesticide Manual - A World Compendium. 8th ed. Thornton Heath, UK: The British Crop Protection Council, 1987. 299]**PEER REVIEWED**

Combinations: Salut and Saluthion (BASF AG) (with chlorpyrifos). [Farm Chemicals Handbook 1994. Willoughby, OH: Meister, 1994.,p. C-124]**PEER REVIEWED**

Mixed formulations: (dimethoate(+) permethrin; dichlorvos; endosulfan; lindane (+) malathion; parathion; chlorpyrifos; fenitrothion; fenvalerate. [Hartley, D. and H. Kidd (eds.). The Agrochemicals Handbook. 2nd ed. Lechworth, Herts, England: The Royal Society of Chemistry, 1987.,p. A153/Aug 87]**PEER REVIEWED**

Technical grade is 98% pure [Tomlin C; The Pesticide Manual - Incorporating The Agrochemicals Handbook. 10th ed. Crop Protection Publications, p.349 (1994)]**PEER REVIEWED**

Use of dry formulations is not permitted. [Lewis, R.J., Sr (Ed.). Hawley's Condensed Chemical Dictionary. 12th ed. New York, NY: Van Nostrand Rheinhold Co., 1993 410]**PEER REVIEWED**

EPA HAZARDOUS WASTE NUMBER:

P044; An acute hazardous waste when a discarded commercial chemical product or manufacturing chemical intermediate or an off-specification commercial chemical product or a manufacturing chemical intermediate.

ADMINISTRATIVE INFORMATION:

HAZARDOUS SUBSTANCES DATABANK NUMBER: 1586

LAST REVISION DATE: 20030214

LAST REVIEW DATE: Reviewed by SRP on 9/14/1995

UPDATE HISTORY:

Complete Update on 02/14/2003, 1 field added/edited/deleted.

Complete Update on 11/08/2002, 1 field added/edited/deleted.

Complete Update on 01/14/2002, 1 field added/edited/deleted.

Complete Update on 08/09/2001, 1 field added/edited/deleted.

Complete Update on 03/09/2000, 1 field added/edited/deleted.

Complete Update on 02/08/2000, 1 field added/edited/deleted.

Complete Update on 02/02/2000, 1 field added/edited/deleted.

Complete Update on 11/18/1999, 1 field added/edited/deleted.

Complete Update on 09/21/1999, 1 field added/edited/deleted.

Complete Update on 08/26/1999, 1 field added/edited/deleted.

Complete Update on 06/01/1999, 1 field added/edited/deleted.

Complete Update on 03/17/1999, 1 field added/edited/deleted.

Complete Update on 02/24/1999, 1 field added/edited/deleted.

Complete Update on 10/20/1998, 1 field added/edited/deleted.

Complete Update on 06/02/1998, 1 field added/edited/deleted.

Complete Update on 02/27/1998, 1 field added/edited/deleted.

Complete Update on 10/23/1997, 1 field added/edited/deleted.

Complete Update on 05/08/1997, 1 field added/edited/deleted.

Complete Update on 03/17/1997, 2 fields added/edited/deleted.

Complete Update on 10/13/1996, 1 field added/edited/deleted.

Complete Update on 09/12/1996, 1 field added/edited/deleted.

Complete Update on 09/11/1996, 1 field added/edited/deleted.

Complete Update on 07/11/1996, 1 field added/edited/deleted.

Complete Update on 01/25/1996, 54 fields added/edited/deleted.

Field Update on 01/21/1996, 1 field added/edited/deleted.

Complete Update on 12/28/1994, 1 field added/edited/deleted.

Complete Update on 11/28/1994, 1 field added/edited/deleted.

Complete Update on 03/25/1994, 1 field added/edited/deleted.

Complete Update on 09/02/1993, 1 field added/edited/deleted.

Complete Update on 04/27/1993, 1 field added/edited/deleted.

Field update on 12/21/1992, 1 field added/edited/deleted.

Complete Update on 09/03/1992, 1 field added/edited/deleted.

Complete Update on 09/26/1991, 1 field added/edited/deleted.

Complete Update on 05/23/1990, 69 fields added/edited/deleted.

Field update on 05/18/1990, 1 field added/edited/deleted.

Field Update on 03/07/1990, 1 field added/edited/deleted.

Field Update on 03/07/1990, 1 field added/edited/deleted.

Field Update on 03/07/1990, 1 field added/edited/deleted.

Field Update on 03/06/1990, 1 field added/edited/deleted.

Express Update on 10/13/1989, 3 fields added/edited/deleted.

Field Update on 05/12/1988, 1 fields added/edited/deleted.

Complete Update on 03/04/1988, 2 fields added/edited/deleted.

Complete Update on 02/24/1988, 75 fields added/edited/deleted.

Complete Update on 03/31/1986

RECORD LENGTH: 224270