Propoxur Poisoning Statistics Summary
Prepared By Jerome Blondell

FEB 12 1987

Acute hazards to propoxur (Baygon) can be measured by counting or estimating the number of deaths, hospitalizations, and visits to physicians outside of the hospitals.

Mortality

During four years (1961, 1969, 1973, 1974), when all accidental deaths due to pesticides in the U.S. were counted, there were no deaths due to propoxur (1). No accidental propoxur deaths were reported in California from 1965 to 1977 or from 1981 to 1985 when all accidental deaths from pesticides were reported (2).

Hospitalized Poisonings

Based on a 12% sample of the nation's hospitals, propoxur was estimated to have caused an average of 32 hospitalizations each year during the time period 1971 through 1976, accounting for 1 percent of the total pesticide poisonings (3). This estimate is based on 24 observed nonoccupationally-related cases.

During the time period 1974 to 1976, propoxur was associated with 13 nonoccupational hospitalizations per 1 million containers of propoxur estimated in U.S. homes by an EPA survey of household pesticide usage (1976-1977) (4). The average ratio of poisonings for all pesticides was 14 cases per million containers.

Physician-treated Poisoning

Based on data obtained from California (the only state which enforces mandatory reporting of occupational pesticide incidents), physicians treated an average of 8.6 propoxur poisonings each year from 1981 through 1985 (2). An additional 4.6 cases per year were reported as either due to skin or eye injury. Of the total 66 illnesses reported in this period 20 were hand applicators, 42 were due to exposure to residues or drift, and 4 were due to other causes. Between 1981 and 1985, two people were reported hospitalized for occupational propoxur poisoning for a total of 26 days and 12 workers were off work for a total of 66 days.

(1). Hayes, W.J. and Vaughn W.K. Mortality from pesticides in the United States in 1973 and 1974. Toxicology and Applied Pharmacology 42:235-252, 1977.

(2). From Various Reports issued by the California Department of Food and Agriculture, Sacramento, California. Included are reports number HS-322, HS-544, HS-545, HS-985, HS-1098, HS-1186, HS-1188, HS-1304, HS-1305,HS-1370 and HS-1371.

(3). Keefe T.J., Savage E.P., Munn S., Wheeler H.W. Evaluation of Epidemiologic Factors From Two National Studies of Hospitalized Pesticide Poisonings, U.S.A. EPA Report.

(4). Based on calculations from (3) and the EPA Report 540/9-80-002 National Household Pesticide Usage Study, 1976-1977. by E.P. Savage, T.J. Keefe, and H.W. Wheeler.

4.30
→ Lynn - for the files

National Research Council Canada | Conseil national de recherches Canada

Associate Committee on Scientific Criteria for Environmental Quality | Comité associé sur les critères scientifiques concernant l'état de l'environnement

Environmental Secretariat | Secrétariat de l'environnement

ANNOUNCEMENT OF PUBLICATION

EFFECTS OF PROPOXUR ON ENVIRONMENTAL QUALITY WITH PARTICULAR REFERENCE TO ITS USE FOR CONTROL OF BITING FLIES

With the phasing out of DDT for biting fly control in 1968, the need for a replacement became urgent. Several compounds were recognized as effective against mosquitos but were relatively ineffective against adult blackflies. Because mosquitos and blackflies create coexisting problems in many parts of Canada, there was a need for a single insecticide that could be used to control both.

Propoxur, one of the principal biting fly adulticides used in Canada today, is effective against both insects. Particular public attention has recently been given to its use in emergency aerial spray operations to control mosquitos that are vectors of encephalitis in municipalities in Ontario and Manitoba.

The scientific information developed in support of the registration of this insecticide has been exhaustively reviewed and assessed by a panel of experts, including some from outside Canada. The assessment covered application technology, environmental persistence, metabolic processes, as well as the mammalian and environmental toxicology of propoxur within the context of its use for the control of biting flies. Of particular concern to the panel were the subtle effects on rats and monkeys at dosages lower than those usually associated with cholinesterase depression or signs of carbamate toxicity. The question was raised of the possible association of such observations with carbamate toxicity in general, and the panel recommended that studies should be commissioned to determine the toxicological significance of these observations.

In other recommendations, the expert panel stressed the need for standardized experimental procedures and for improved field data that could be directly related to assessing the ecological implications of the use of propoxur and other candidate pesticides for biting fly control in Canada.

English version (235 pages)
Publication No. NRCC 18572
Available through prepaid purchase orders from

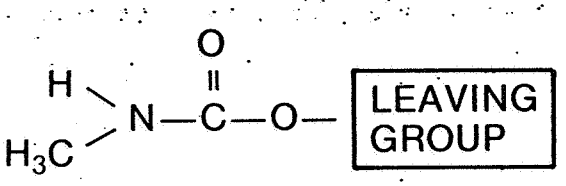
Version française (à paraître)
Publication nº CNRC 18573
En vente, moyennant commandes prépayées, au Service des

Publications, NRCC/CNRC
Ottawa, Ontario, Canada K1A 0R6

Price/prix: $8.00

CARBAMATE CHOLINESTERASE-INHIBITING PESTICIDES

GENERAL CHEMICAL STRUCTURE



COMMON COMMERCIAL PESTICIDE PRODUCTS*

Highly toxic:** aldicarb† (Temik), oxamyl (Vydate), carbofuran (Furadan), methomyl (Lannate, Nudrin), formetanate HCl (Carzol, Dicarzol), aminocarb (Matacil), dimetilan (Snip Fly Bands).

Moderately toxic*:** promecarb (Carbamult), methiocarb (Mesurol, Draza), propoxur (Baygon), pirimicarb (Pirimor, Aphox, Rapid), bufencarb (Bux), carbaryl (Sevin).

TOXICOLOGY

Insecticides of this class cause reversible carbamylation of acetylcholinesterase enzyme, allowing accumulation of acetylcholine at cholinergic neuroeffector junctions (muscarinic effects), and at skeletal muscle myoneural junctions and in autonomic ganglia (nicotinic effects). Poison also impairs CNS function. The carbamyl-enzyme combination dissociates more readily than the phosphorylated enzyme produced by organophosphate insecticides. This lability tends to mitigate the toxicity of carbamates, but also limits the usefulness of blood enzyme measurements in diagnosis of poisoning. Carbamates are absorbed by inhalation, ingestion, and dermal penetration. They are actively metabolized by the liver, and the degradation products are excreted by the liver and kidneys.

---

* Listed approximately in order of decreasing toxicity.

** Acute oral LD_{50} in the rat less than 50 mg/kg.

*** Acute oral LD_{50} in the rat above 50 mg/kg.

† This carbamate is a systemic, i.e., it is taken up by the plant and translocated into foliage and sometimes into the fruit.

A few carbamate insecticides are formulated in methyl (wood) alcohol. In cases of ingestion of these formulations, the toxicology of the methanol must be taken fully into consideration: severe gastroenteric irritation, acidosis, CNS injury, and neuropathy.

FREQUENT SYMPTOMS AND SIGNS OF POISONING

DIARRHEA, NAUSEA, VOMITING, ABDOMINAL PAIN, PROFUSE SWEATING, SALIVATION, and **BLURRED VISION** are frequently reported. Other common symptoms have been dyspnea, tremor, muscle twitching, ataxia, and headache. Temporary paralysis of the extremities has also occurred. Most reported illnesses have not exceeded a few hours, and the prognosis is generally better than in organophosphate intoxications. However, in severe poisonings, one should anticipate the possibility of **RESPIRATORY DEPRESSION,** pulmonary edema, and convulsions. Continuing absorption of intermediate quantities may cause protracted **MALAISE,** weakness, and anorexia, resembling influenza.

CONFIRMATION OF DIAGNOSIS

CAUTION: If there is strong clinical evidence of poisoning, treat patient immediately. **DO NOT WAIT** for laboratory confirmation.

Depressions of plasma and/or RBC cholinesterase activities may be observed following absorption of extraordinary amounts of carbamate insecticide. However, enzyme activities commonly revert to normal within a few minutes or hours. They are not, therefore, reliable detectors of carbamate poisoning; i.e., intoxication may exist when blood cholinesterase activities are normal. The rapid methods for cholinesterase estimation (ACHOLTEST, ChE-tel, MERCKOTEST) are more likely to detect depressions than the longer test methods.

Table 1 in the chapter on **ORGANOPHOSPHATE PESTICIDES** lists the approximate lower limits of normal plasma and red cell cholinesterase-inhibiting carbamates. A normal value does not preclude carbamate insecticide poisoning. Whenever possible, comparison of the test sample with pre-exposure values offers the best confirmation of excessive carbamate absorption: a depression of 25% or more is strong evidence of excessive absorption.

Consult the chapter on **ORGANOPHOSPHATE PESTICIDES** for detailed interpretation of the blood cholinesterase tests.

Measurement of carbamate metabolites in urine within 48 hours of exposure represents a specific and sensitive method for confirming absorption of several pesticides of this class. A number of government and university laboratories in contact with poison control centers can perform these tests. Specimens for such analysis should be collected as promptly as possible after exposure.

TREATMENT

CAUTION: Persons attending the victim should avoid direct contact with heavily contaminated clothing and vomitus. Wear rubber gloves while decontaminating skin and hair.

1. Establish **CLEAR AIRWAY** and **TISSUE OXYGENATION** by aspiration of secretions, and if necessary, by assisted pulmonary ventilation with oxygen. Improve tissue oxygenation as much as possible before administering atropine to minimize the risk of ventricular fibrillation.
2. Administer **ATROPINE SULFATE** intravenously, or intramuscularly if IV injection is not possible. Atropine protects the end-organs from excessive concentrations of acetylcholine. It does not reactivate the cholinesterase enzyme. Recrudescence of poisoning may occur if tissue concentrations of carbamate remain high when the effect of atropine wears off. Atropine is the ideal antidote for muscarinic symptoms; it is ineffective against nicotinic actions such as muscle weakness and twitching, and respiratory depression.

   In **MODERATELY SEVERE** poisoning: **Adult dosage,** including children over 12 years: 0.4-2.0 mg repeated every 15 minutes until atropinization is achieved (tachycardia, flushing, dry mouth, mydriasis). Maintain atropinization by repeated doses for 2-12 hours, or longer, depending on severity of poisoning. The appearance of râles in the lung bases, miosis, salivation, nausea, bradycardia, are all indications of inadequate atropinization.

   Dosage for children under 12 years: 0.05 mg/kg body weight repeated every 15 minutes until atropinization is achieved. Maintain atropinization with repeated dosage of 0.02-0.05 mg/kg.

   SEVERELY POISONED individuals may exhibit remarkable tolerance to atropine; twice the doses suggested above may be needed. Persons not poisoned, or only slightly poisoned, may develop signs of atropine toxicity if large doses are given: **FEVER,** muscle fibrillations, and delirium. If these appear while the patient is fully atropinized, atropine should be discontinued, at least temporarily.
3. Pralidoxime (Protopam®-Ayerst, 2-PAM) is of doubtful value in poisonings by carbamate inhibitors of cholinesterase. Atropine alone is almost always an adequate antidote. Pralidoxime is probably contraindicated in poisonings by carbaryl, specifically. If victim of carbamate poisoning exhibits severe muscle weakness and/or respiratory depression, or if poisoning involves a combination of carbamate and organophosphate, a dilute solution of pralidoxime (total dose in 250 ml 5% glucose solution) may be given cautiously IV. The infusion should be terminated if patient's condition worsens. Pralidoxime **dosage:** adults, 1.0 gm; for children under 12 years, 20-50 mg/kg.
4. **OBSERVE** treated patients closely **at least 24 HOURS** to insure that symptoms (possibly pulmonary edema) do not recur as atropinization wears off. In very severe poisonings, metabolic disposition of toxicant

may require several hours or days during which atropinization must be maintained. Markedly lower levels of urinary metabolites indicate that atropine dosage can be tapered off. As dosage is reduced, check the lung bases frequently for râles. If râles are heard, or if nausea, salivation, or bradycardia returns, **RE-ESTABLISH ATROPINIZATION** promptly.

5. **BATHE** and **SHAMPOO** victim with soap and water if there is any chance that **SKIN** and **HAIR** are contaminated.
6. If pesticide has been **INGESTED** in quantity sufficient to cause poisoning, empty the stomach and intestine.

   A. If victim is alert and respiration is not depressed, give **SYRUP OF IPECAC,** followed by 1-2 glasses of water to induce vomiting; adults (including children over 12), 30 ml; children (under 12 years), 15 ml.

      CAUTION: **OBSERVE** victim closely **AFTER** administering **IPECAC.** If consciousness level declines, or if vomiting has not occurred in 15 minutes, proceed immediately to **INTUBATE** the stomach.

      Following emesis, have victim drink a suspension of 30-50 gm **ACTIVATED CHARCOAL** in 3-4 ounces of water to bind toxicant remaining in the gastrointestinal tract.

   B. If victim is **OBTUNDED** or respiration is depressed, empty stomach by **INTUBATION, ASPIRATION,** and **LAVAGE,** using isotonic saline or 5% sodium bicarbonate. Because many pesticides are dissolved in petroleum distillates, emesis and intubation of the stomach involve a serious risk that solvent will be aspirated, leading to chemical pneumonitis. For this reason:

      (a). If victim is unconscious or obtunded and facilities are at hand, insert **ENDOTRACHEAL TUBE** (cuffed, if available) prior to gastric intubation.

      (b). Keep victim's **HEAD BELOW LEVEL OF STOMACH** during intubation and lavage (Trendelenburg, or left lateral decubitus, with head of table tipped downward). Keep victim's head turned to left.

      (c). **ASPIRATE PHARYNX** as regularly as possible to remove gagged or vomited stomach contents.

      After aspiration of gastric contents and washing of stomach, instill 30-50 gm of **ACTIVATED CHARCOAL** in 3-4 ounces of water through the tube to limit absorption of remaining toxicant.

   C. **SAVE A SAMPLE** of emesis or initial gastric washings for chemical analysis.

   D. If bowel movement has not occurred in 4 hours and patient is fully conscious, give **SODIUM SULFATE,** 0.25 gm/kg in 1-6 ounces of water, as a cathartic. Magnesium sulfate or citrate is equally suitable if renal function is satisfactory.

7. **DO NOT** give morphine, aminophylline, phenothiazines, reserpine, furosemide, or ethacrynic acid.
8. Give adrenergic amines **ONLY** if there is a specific indication, such as severe hypotension.
9. **CONVULSIONS** are **RARE** manifestations of poisoning by carbamates. If they occur, causes other than direct carbamate action should be considered: cerebral anoxia, head trauma, mixed poisoning. Although not tested in these circumstances, **DIAZEPAM** (Valium) is probably the anticonvulsant of choice. **Dosage** for adults and children over 6 years or 23 kg body weight is 5-10 mg given **slowly** intravenously (no more than half total dose per minute), or intramuscularly (deep). Dosage for children under 6 years or 23 kg body weight is 0.1 mg/kg. Repeat this dosage every 2-4 hours if needed to control convulsions. Be prepared to intubate and to assist pulmonary ventilaton mechanically if respiration is depressed. Hypotensive reactions may also occur.
10. Persons who have been clinically poisoned by carbamate pesticides should not be re-exposed to cholinesterase-inhibiting chemicals until symptoms and signs have resolved completely, and normal blood cholinesterase activities have been demonstrated.
11. **DO NOT** administer atropine prophylactically to workers exposed to carbamate insecticides. It is neither practical nor medically sound to do so.

Acute hazards to propoxur (Baygon) can be measured by counting or estimating the number of deaths, hospitalizations, and visits to physicians outside of the hospitals.

Mortality

During four years (1961, 1969, 1973, 1974), when all accidental deaths due to pesticides in the U.S. were counted, there were no deaths due to propoxur (1). No accidental propoxur deaths were reported in California from 1965 to 1977 or from 1981 to 1985 when all accidental deaths from pesticides were reported (2).

Hospitalized Poisonings

Based on a 12% sample of the nation's hospitals, propoxur was estimated to have caused an average of 32 hospitalizations each year during the time period 1971 through 1976, accounting for 1 percent of the total pesticide poisonings (3). This estimate is based on 24 observed nonoccupationally-related cases.

During the time period 1974 to 1976, propoxur was associated with 13 nonoccupational hospitalizations per 1 million containers of propoxur estimated in U.S. homes by an EPA survey of household pesticide usage (1976-1977) (4). The average ratio of poisonings for all pesticides was 14 cases per million containers.

Physician-treated Poisoning

Based on data obtained from California (the only state which enforces mandatory reporting of occupational pesticide incidents), physicians treated an average of 8.6 propoxur poisonings each year from 1981 through 1985 (2). An additional 4.6 cases per year were reported as either due to skin or eye injury. Of the total 66 illnesses reported in this period 20 were hand applicators, 42 were due to exposure to residues or drift, and 4 were due to other causes. Between 1981 and 1985, two people were reported hospitalized for occupational propoxur poisoning for a total of 26 days and 12 workers were off work for a total of 66 days.

(1). Hayes, W.J. and Vaughn W.K. Mortality from pesticides in the United States in 1973 and 1974. Toxicology and Applied Pharmacology 42:235-252, 1977.

(2). From Various Reports issued by the California Department of Food and Agriculture, Sacramento, California. Included are reports number HS-322, HS-544, HS-545, HS-985, HS-1098, HS-1186, HS-1188, HS-1304, HS-1305,HS-1370 and HS-1371.

(3). Keefe T.J., Savage E.P., Munn S., Wheeler H.W. Evaluation of Epidemiologic Factors From Two National Studies of Hospitalized Pesticide Poisonings, U.S.A. EPA Report.

(4). Based on calculations from (3) and the EPA Report 540/9-80-002 National Household Pesticide Usage Study, 1976-1977. by E.P. Savage, T.J. Keefe, and H.W. Wheeler.