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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 mosquitoes but were relatively ineffective against adult blackflies. Because mosquitoes 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 mosquitoes 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.

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CARBAMATE:
CHOLINESTERASE-INHIBITING
PESTICIDES

GENERAL CHEMICAL STRUCTURE

H₃C \( \text{N} - \text{C} - \text{O} \) \text{LEAVING GROUP} \ O

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, Drazza), 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.

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* Listed approximately in order of decreasing toxicity.
** Acute oral LD₅₀ in the rat less than 50 mg/kg.
*** Acute oral LD₅₀ 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. **BATH**E 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 **OBSTUNED** 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 ventilation 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.
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