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Toxicity of Pesticides

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- All pesticides must be toxic, or poisonous, to kill the pests they are intended to control; but because pesticides are toxic, they are potentially hazardous to humans and animals as well as to pests. Since pesticide toxicity varies widely, it is very important for people who use pesticides to have at least a general knowledge of the relative toxicity of the products they are using

Dose-Time Relationship

- The effect of a pesticide, or any substance for that matter, is dependent on a number of factors. The most important factor is the dose-time relationship. Dose is the quantity of a substance that a surface, plant, or animal is exposed to. Time means how often the exposure occurs. Thus, the dose- time relationship is how much of the substance is involved and how often the exposure to the substance occurs. This relationship gives rise to two different types of toxicity that pesticide applicators must know and understand. They are acute and chronic toxicity.

Kinds of Toxicity

- ***Acute* toxicity** refers to how poisonous a pesticide is to a human, animal, or plant after a single short-term exposure. Acute toxicity is used to describe effects which appear promptly, or within 24 hours of exposure. A pesticide with a high acute toxicity is deadly even when a very small amount is absorbed. Acute toxicity levels are used as a way to assess and compare how poisonous pesticides are. The acute toxicity of a pesticide is used as the basis for the warning statements on the label. Acute toxicity may be measured as acute oral toxicity, acute dermal toxicity, and acute inhalation toxicity.

***Chronic* toxicity**

- is the delayed poisonous effect from exposure to a substance. Chronic toxicity of pesticides concerns the general public, as well as those working directly with pesticides because of potential exposure to pesticides on/in food products, water, and the air. It is measured in experimental conditions after three months of either continuous or occasional exposure.

- A material that has high acute toxicity does not necessarily have high chronic toxicity. Nor does a chemical with low acute toxicity necessarily have low chronic toxicity. For many pesticides, the toxic effects following single acute exposures are quite different from those produced by chronic exposure.

- While you cannot change the inherent toxicity of pesticides, you can limit the possibility of poisoning by preventing and/or limiting exposure. In other words, the risk of harm from pesticide exposure is equal to how poisonous the pesticide is, multiplied by the amount and route of exposure to the pesticide, or:

TOXICITY X EXPOSURE =RISK

Routes of Entry

- There are three specific ways in which pesticides may enter your body. You may be poisoned no matter how they enter. Sometimes you can even be poisoned without knowing it, especially if the pesticide enters through the skin or lungs.

Inhalation (breathing in)
Absorption (through the skin or eyes)
Ingestion (eating, swallowing)
Transfer across the placenta of a pregnant woman to the unborn baby

Dermal Route

- Wet, dry, or gaseous forms of pesticides can be absorbed through the skin. This may occur if pesticides are allowed to get on the skin while mixing or applying, or if pesticide-contaminated clothing is not removed promptly and properly cleaned before being worn again. Oil or paste forms allow greater absorption through the skin than water-based pesticides. Some pesticides do not pass through the skin very readily. Others are quickly absorbed through the skin and can be as dangerous as if they were swallowed.

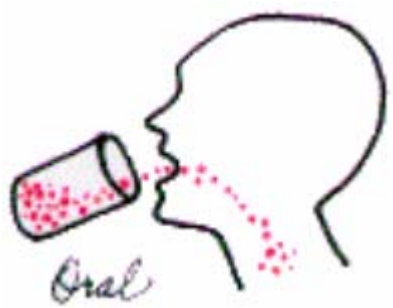
- Skin varies in its capacity to act as a barrier to pesticide absorption. The eyes, ear drums, scalp and groin area absorb pesticides more quickly than other areas on the body. Damaged or open skin can be penetrated by a pesticide much more readily than healthy, intact skin. Once they are absorbed through skin, pesticides enter the blood stream and are carried throughout the body.

Inhalation Route

- Whether as dusts, spray mist, or fumes, pesticides can be drawn into your lungs as you breathe. Inhalation of pesticides can occur during the mixing of wettable powders, dusts, or granules. Poisoning can also occur while fumigating or spraying without a self contained breathing apparatus or a proper respirator in enclosed or poorly ventilated areas such as greenhouses or apartments. The largest particles that are inhaled tend to stay on the surface of the throat and nasal passages, and do not enter the lungs. Smaller particles can be inhaled directly into the lungs. The number of particles needed to poison by inhalation depends upon the concentration of the chemical in the particles.

- Even inhalation of dilute pesticides can result in poisoning. Once they are absorbed through the surfaces of the lungs, chemicals enter the blood stream and are distributed to the rest of the body.

Oral Route



- Pesticides can enter the body through the mouth (also called ingestion). This can occur when hands are not properly washed before eating or smoking. They may be swallowed by mistake, if they are improperly stored in food containers. Ingested materials can be absorbed anywhere along the gastrointestinal tract; the major absorption site is the small intestine. Once absorbed, they eventually enter the blood stream, and circulate throughout the body.

Which Route Is More Important?

- You can be poisoned no matter which way pesticides enter your body. While there are few chemicals that are equally poisonous by all routes of entry, some pesticides can enter all three ways and poison you. (For example, parathion is toxic regardless of how it is absorbed).

- The dermal and inhalation routes of pesticide entry are likely to be the most important routes of pesticide applicator exposure. It is unlikely that you would purposely eat or drink the chemicals you are using, but you may breathe them in, splash them on your skin, or expose yourself to pesticide "fallout."

- Healthy skin can slow the absorption of a pesticide when dermal contact occurs. Liquid pesticides containing solvents and oil based pesticides are absorbed quickly compared to dry pesticides. The applicator must know that damaged skin (chapped, cut, or abraded) has lost its ability to slow the entry of a pesticide into the body.

Effects of Toxicity

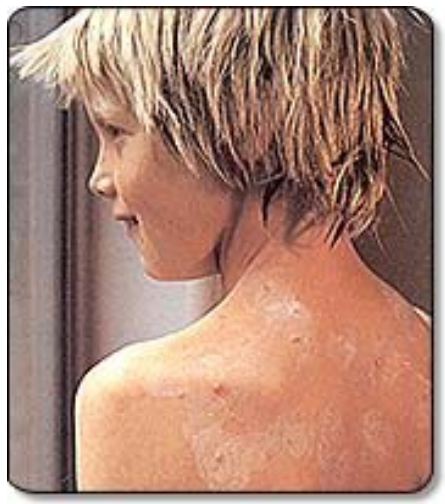
- **In addition to being acute or chronic,** toxic effects can be any of the following:
- **Local or systemic**
- **Immediate or delayed**
- **Reversible or irreversible**
- **Additive, antagonistic, or synergistic**

- **Exposure to pesticides may** also result in the following:
- **Reproductive effects**
- **Teratogenic effects**
- **Carcinogenic effects**
- **Oncogenic effects**
- **Mutagenic effects**
- **Neurotoxicity**
- **Immunosuppression**

PLAN OF ACTION FOR ACUTE PESTICIDE POISONINGS

- **Contact Medical Personnel**
- **Maintain Vital Signs**
- **Eliminate Further Contamination**

1. **Ingested Pesticides**
2. **Pesticides on the Skin**
3. **Pesticides in the Eye.**
4. **Inhaled Pesticides**



Emergency Action in Case of a Major Crash or Spill

If intoxication occurs due to direct contact with the pesticide:

Step 1: Move the patient well away from any contaminated area and from the vicinity of pesticides. Quickly remove any contaminated clothing. Wash exposed area with soap and water.
Step 2: Start the first aid treatment immediately (see page 2).
Step 3: Call a physician as quickly as possible (but do not abandon the first aid treatment)
Step 4: If medical help cannot be obtained or is delayed, transport the patient to the nearest poison control centre, hospital or physician's office. Take the pesticide label or the container or any available records of pesticides used, and any other information such as the notes in this handbook. See back cover for a list of poison control centres in Saskatchewan

Organophosphorous Compounds:

Common names: Azinphos-methyl (Guthion), methamidophos (Monitor), chloropyriphos (Lorsban Dursban), diemthoate (Cygon), diazinon, Malathion, etc.

Toxicology: Irreversible inhibitors for cholinesterase. Loss of enzyme function allows accumulation of acetycholine at cholinergic neuroeffector junctions (muscarinic effects) and at skeletal myoneural junctions and in automatic ganglia (nicotinic effects). They also impair nerve impulse transmission in the brain causing disturbances in sensorium, motor function and respiratory drive.

Routes of absorption: Ingestion, dermal, inhalation

Toxicity: Highly toxic: parathion, azinophos methyl, methamidophos

Moderately toxic: chloropyriphos, dimethoate, and diazinon

Slightly toxic: Malathion

Summary of treatment:

- Ensure good oxygen supply

- Give atropine sulfate until atropinization is achieved.

Adults: 0.4-2 mg. I.V. every 15-30 minutes for 2-12 hours

Children (< 12 yr): 0.05 mg/kg body weight I.V. Every 15-30 minutes.

- Draw blood for cholinesterase

-Give 2-PAM

Adults: 1.0 g I.V. at 0.5 g per minute, repeat in 1 hour if needed.

Carbamate Insecticides:

Common names: Aldicarb (Temik), carbofuran (Furadan), carbaryl (Sevin), methomyl (Lannate), etc.

Toxicology: Reversible inhibitors for cholinesterase. Effects similar to organophosphorous exposure. The reversibility of the inhibition tends to mitigate the toxicity.

Routes of absorption: Ingestion, inhalation, dermal

Toxicity: Highly toxic: aldicarb, carbofuran, methomyl

Moderately toxic: propoxur, carbaryl

Summary of treatment: Similar to organophosphorous compounds except that

1)Pralidoxime (2-PAM) is of no value in poisonings by cholinesterase-inhibiting carbamate compounds.

2)Enzyme activities commonly revert to normal within a few minutes or hours and therefore, cholinesterase activity measurement is not a reliable indicator of poisoning by carbamate compounds.

FUMIGANTS

Halogen Fumigants:

Common names Methyl bromide, ethylene dibromide, ethylene dichloride, chloropicrin, acrolein, methyl bromide.

Toxicology: All depress the central nervous system causing respiratory arrest and occasionally seizures. They increase cardiac irritability. Chloropicrin is intensely irritating to mucous membranes and can cause pulmonary edema.

Routes of absorbtion: Highly volatile and rapidly penetrate the lining of the respiratory tract.

Toxicity: Highly toxic: chloropicrin, acrolein, methyl bromide
Moderately toxic: ethylene dibromide, ethylene dichloride.

Summary of Treatment:

- Symptomatic treatment, treat acute pulmonary edema, shock and seizures.
- Dimercaprol (BAL) is a specific antidote for methyl bromide intoxication.

RODENTICIDES

Coumarins and Indandiones:

Common names: Warfarin, brodifacoum, comafuryl (Fumarin), pindone (Pival), and diphacinone (Diphacin).

Toxicology: They depress the hepatic synthesis of prothrombin and factors VII, IX, and X essential to blood clotting. Direct damage to capillary permeability occurs concurrently.

Routes of absorption: Ingestion

Toxicity: Low in single doses and high in multiple doses.

Most frequent symptoms: After repeated ingestion for several days: hematuria, nosebleed, hematomata, bleeding gums and melena. Abdominal pain and back pain probably reflect internal hemorrhage. Possible palor, petechial rash and widespread bruising.

Summary of Treatment:

- Vitamin K orally (specifically phytonadione, vitamin KI).
- If large amount is ingested induce vomiting.
- Determine prothrambin time.
- If severe bleeding administer Aquamephtyon I.M.
- Blood transfusion may be needed

Chlorophenoxy Compounds:

Common names: 2,4-dichlorophenoxyacetic acid (2,4-D), dichlorprop, MCPA, MCPB, mecoprop, etc.

Toxicology: Moderately irritating to skin, eyes and respiratory and G.I. linings. Large amounts causes severe metabolic acidosis, striated muscle injury (myotonia, muscle weakness) and electrocardiographic changes. Because they are weak uncouplers of oxidative phosphorylation they may produce hyperthermia.

Routes of absorption: Absorbed through skin, gut and lungs.

Toxicity: Low toxicity

Most frequent symptoms: Skin and mucous membrane irritation.

When ingested: vomiting, chest pain, abdominal pain, diarrhea, fibrillary muscle twitching and myotonia. Very large doses: metabolic acidosis, fever, tachycardia, hyperventilation, vasodilation, sweating.

Summary of Treatment:

- Supportive

-Avoid ethanol

-Ascorbic acid, as a hydrogen donor, may have significant antidotal action.

Thiocarbamates:

Common names: Thiram, ziram, nabam, maneb, manocozeb, butylate, EPTC, triallate (Avadex).

Toxicology: None is cholinesterase inhibitor. Some are moderate skin and mucous membrane irritants. A few are sensitizers (e.g. thiram). Thiram and possible EPTC and traillate inhibit aldehyde dehydrogenase giving antabuse-like reaction after alcohol ingestion.

Routes of absorption: Ingestion, inhalation and dermal.

Toxicity: Low toxicity

Most frequent symptoms: Dermatitis, nasal stuffiness, and sneezing cough. Ingestion of large amounts may produce nausea, vomiting, diarrhea, hypothermia and ataxia. Antabuse-like reaction is mainly flushing, headache, sweating, weakness and chest tightness

Summary of Treatment:

- Supportive

-Avoid ethanol

-Ascorbic acid, as a hydrogen donor, may have significant antidotal action.

Nitrophenolic Compounds:

Common names: Dinitrophenol, dinitrocresol, dinoseb, dinocap, etc.
Toxicology: Stimulate oxidate metabolism in cell mitochondria by uncoupling phosphorylation. This leads to pyrexia, tachycardia and dehydration and ultimately depletes carbohydrate and fat stores. Pyrexia and direct action on the brain cause cerebral edema, manifest clinically as a toxic psychosis and sometimes convulsions. Liver parenchyma and renal tubules show degenerative changes.
Routes of absorption: Skin, ingestion and inhalation.
Toxicity: Highly toxic.

Summary of Treatment:

- Supportive

-Do not administer antipyretics or atropine.

- Hemodialysis and hemoperfusion may be considered.

Pyridylium Compounds:
Common names: Diquat (Reglone) and paraquat (Gramoxone)
Toxicology: Injures epithelial tissues by direct irritant effects and by peroxidation of intracellular and extracellular phospholipids and inhibition of surfactant synthesis by lung tissue. The injury is usually reversible however, the pulmonary reaction is sometimes irreversible. Skin contact may cause irritation, fissuring, discoloration and loss of fingernails. Splashes into the eye can cause conjunctivitis and possible protracted opacification of the cornea. Causes damage of the liver parenchymal cells and the kidney tubules. Paraquat is concentrated in lung tissue causing proliferation of connective tissue cells, which fill alveolar spaces leading to asphyxia.
Routes of absorption: Skin and inhalation but more serious after ingestion.
Toxicity**: Paraquat:** high toxicity **Diquat:** moderate toxicity

Most frequent symptoms: Irritation of skin and mucuous membranes. Ingestion causes burning pain from mouth to abdomen, nausea, vomiting, diarrhea and occasionally melena.
After 1-3 days: albuminuria, hematuria, pyuria, elevated BUN and creatinine, possible oliguria, Jaundice may develop. Progressive decline in oxygen tension plus cough, dyspnea, tachypnea, cyanosis and rarely pulmonary edema. These may be delayed as long as two weeks.

Summary of Treatment:

- Supportive

- If by ingestion do not delay treatment even if no symptoms are apparent.

- Evacuate the stomach.

- Bentonite is ideal absorbent, though charcoal can be used.

- Do not administer supplemental oxygen.

- Hemodialysis and/or hemoperfusion.

Thank You