



webinar:

## PESTICIDE POISONINGS. ARE YOU PREPARED?

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Wednesday, February 19, 2014

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# *Disclosure Statement*

➤ *Faculty: Matthew Keifer, MD, MPH*

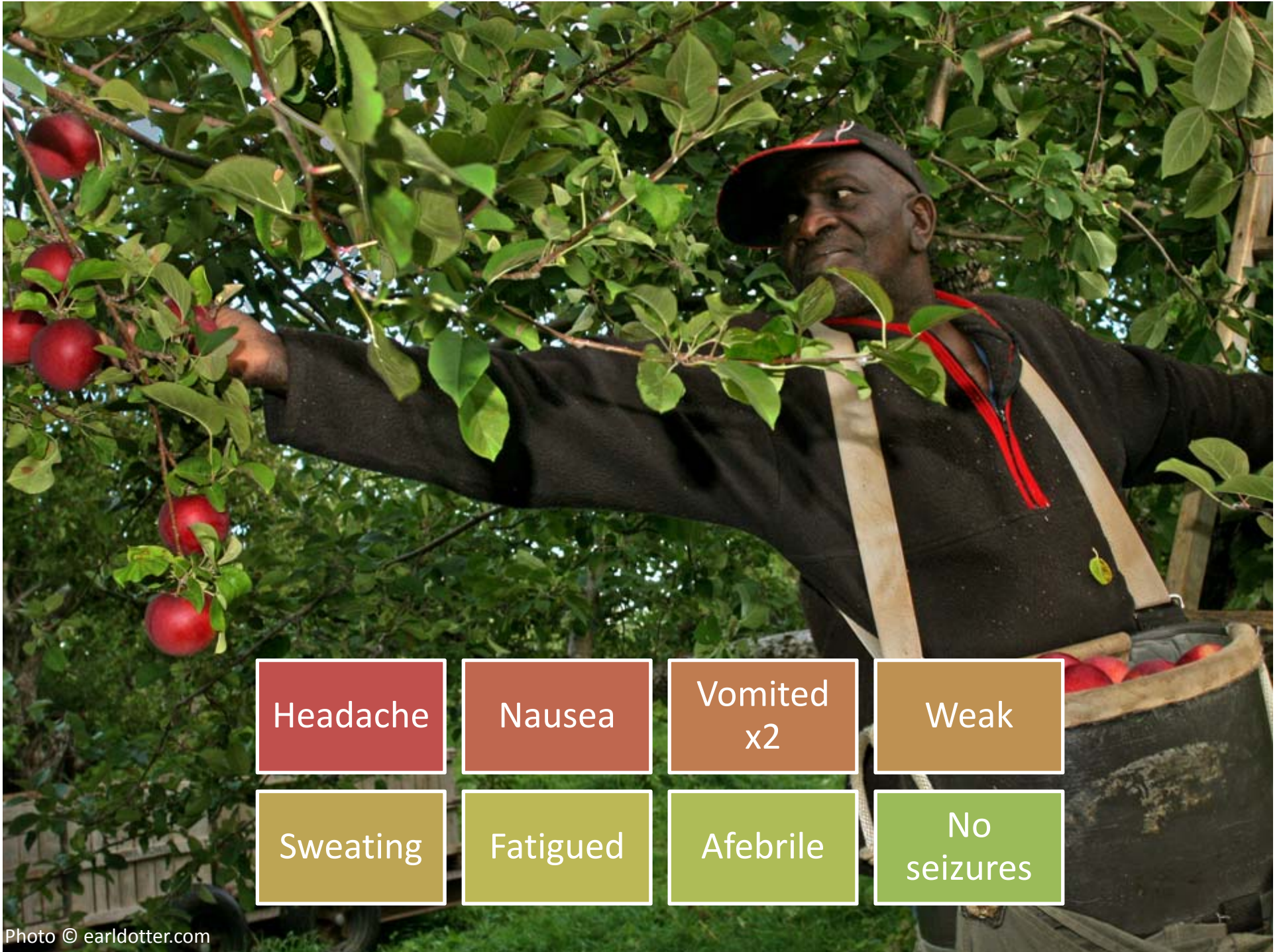
➤ *Amy K. Liebman, MPA, MA*

➤ *Disclosure: We have no real or perceived vested interests that neither relate to this presentation nor do we have any relationships with pharmaceutical companies, biomedical device manufacturers, and/or other corporations whose products or services are related to pertinent therapeutic areas.*

# Learning Objectives



1. Better recognize signs and symptoms of pesticide overexposure
2. Identify key decision points in diagnosing pesticide exposures
3. Demonstrate an understanding of how to use *Recognition and Management of Pesticide Poisonings, 6<sup>th</sup> ed*



Headache

Nausea

Vomited  
x2

Weak

Sweating

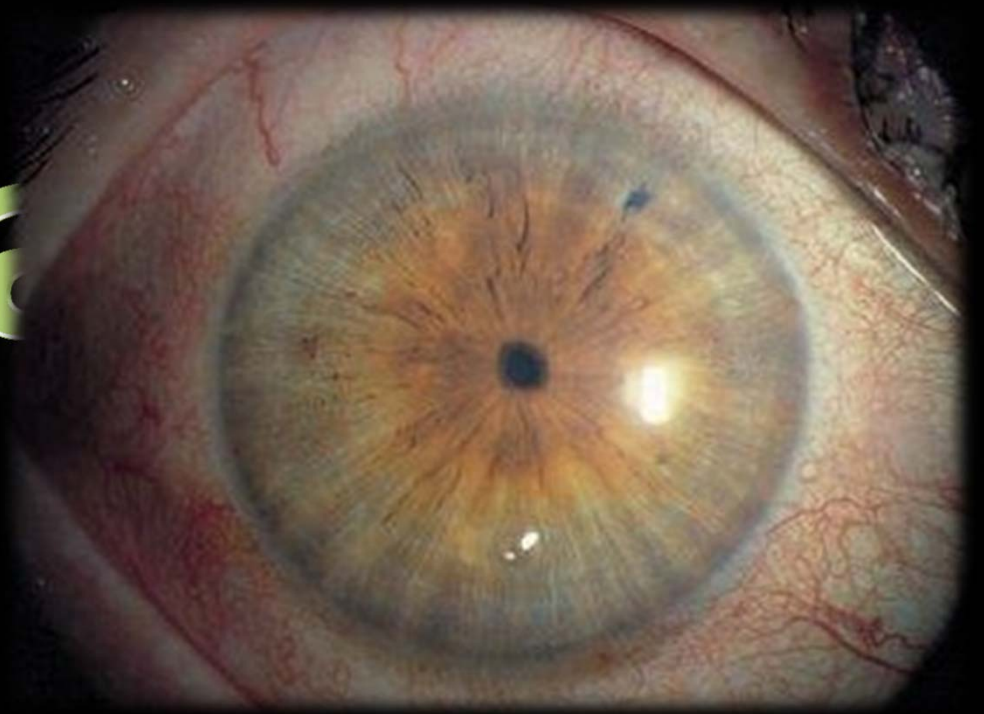
Fatigued

Afebrile

No  
seizures

- ✓ Profusely sweating
- ✓ Thin
- ✓ Muscle fasciculations
- ✓ Miosis

Exa



What do you think  
this looks like?



**INDEX**  
**Signs and Symptoms**

SYMPTOMS/ SIGNS/DISEASE CATEGORIES	CHARACTERISTIC OF THESE POISONINGS	MAY OCCUR IN THESE POISONINGS
Conjunctivitis (irritation of mucous membranes, tearing)	Chloropicrin Acrolein Copper compounds Organotin compounds Cadmium compounds Metam sodium Paraquat Diquat Acrolein Chloropicrin Sulfur dioxide	Thiophthalimides Thiram Thiocarbamates Pentachlorophenol Chlorophenoxy compounds Chlorothalonil Picloram Creosote Aliphatic acids Strobilurin fungicides

Diplopia	Organophosphates N-methyl carbamates Nicotine	
Photophobia		Organotin compounds
Constricted visual fields	Organic mercury	
Optic atrophy		Thallium
Miosis	Organophosphates N-methyl carbamates	Nicotine (early)
Dilated pupils	Cyanide Fluoride	Nicotine (late)
Non-reactive pupils	Cyanide	

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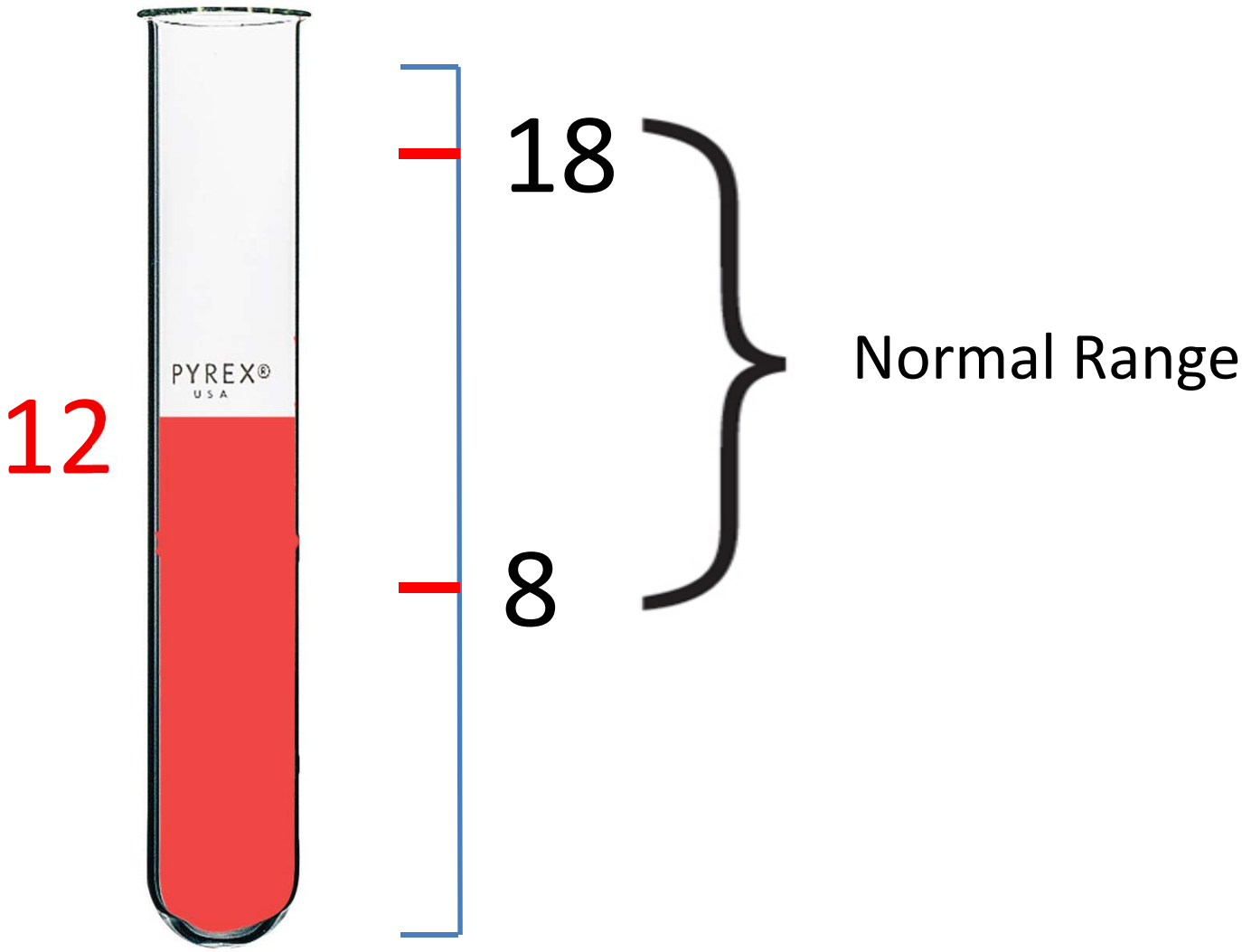
Organophosphate (pg 43)

or

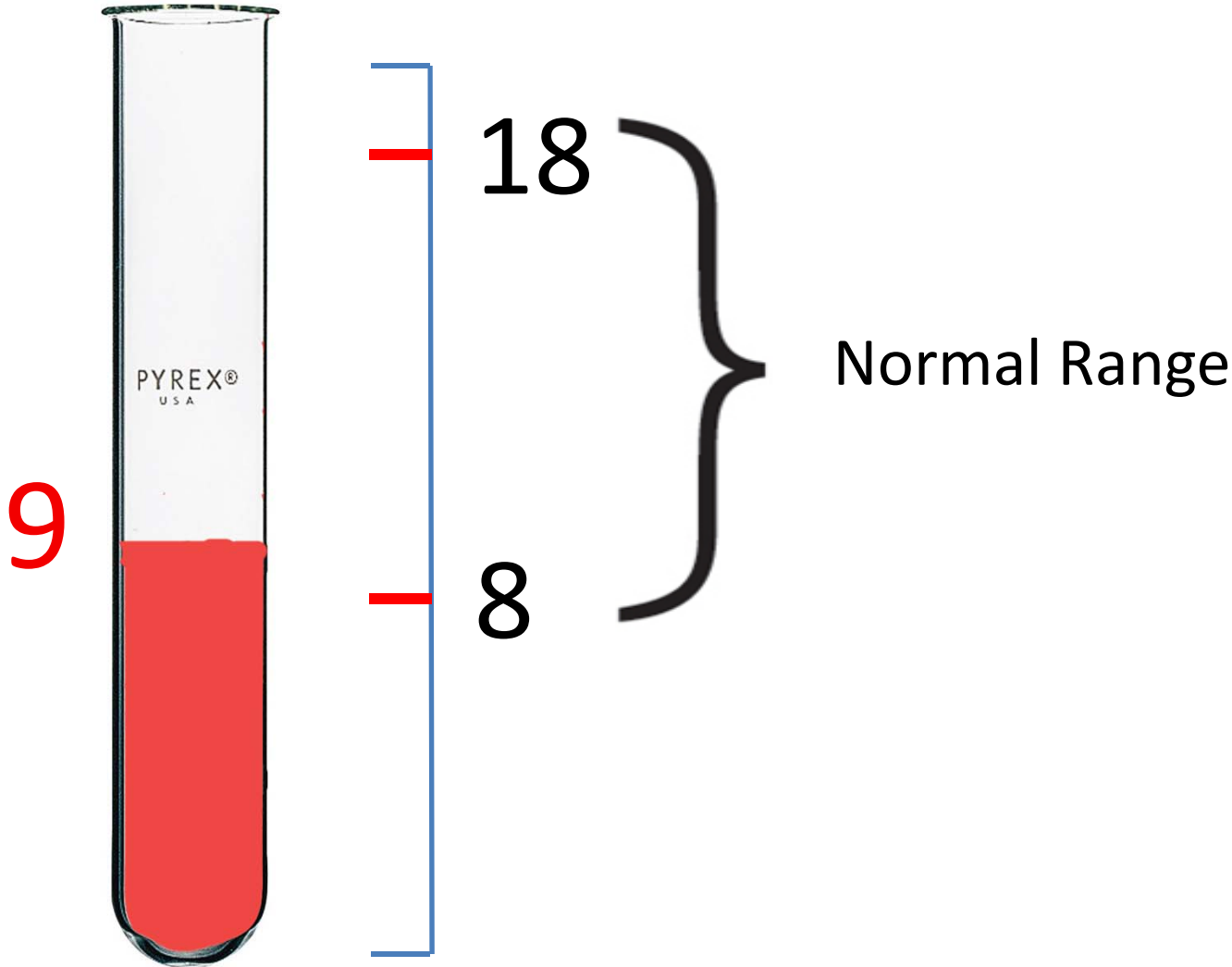
N-Methyl Carbamate

( pg 56)?

# Cholinesterase Test



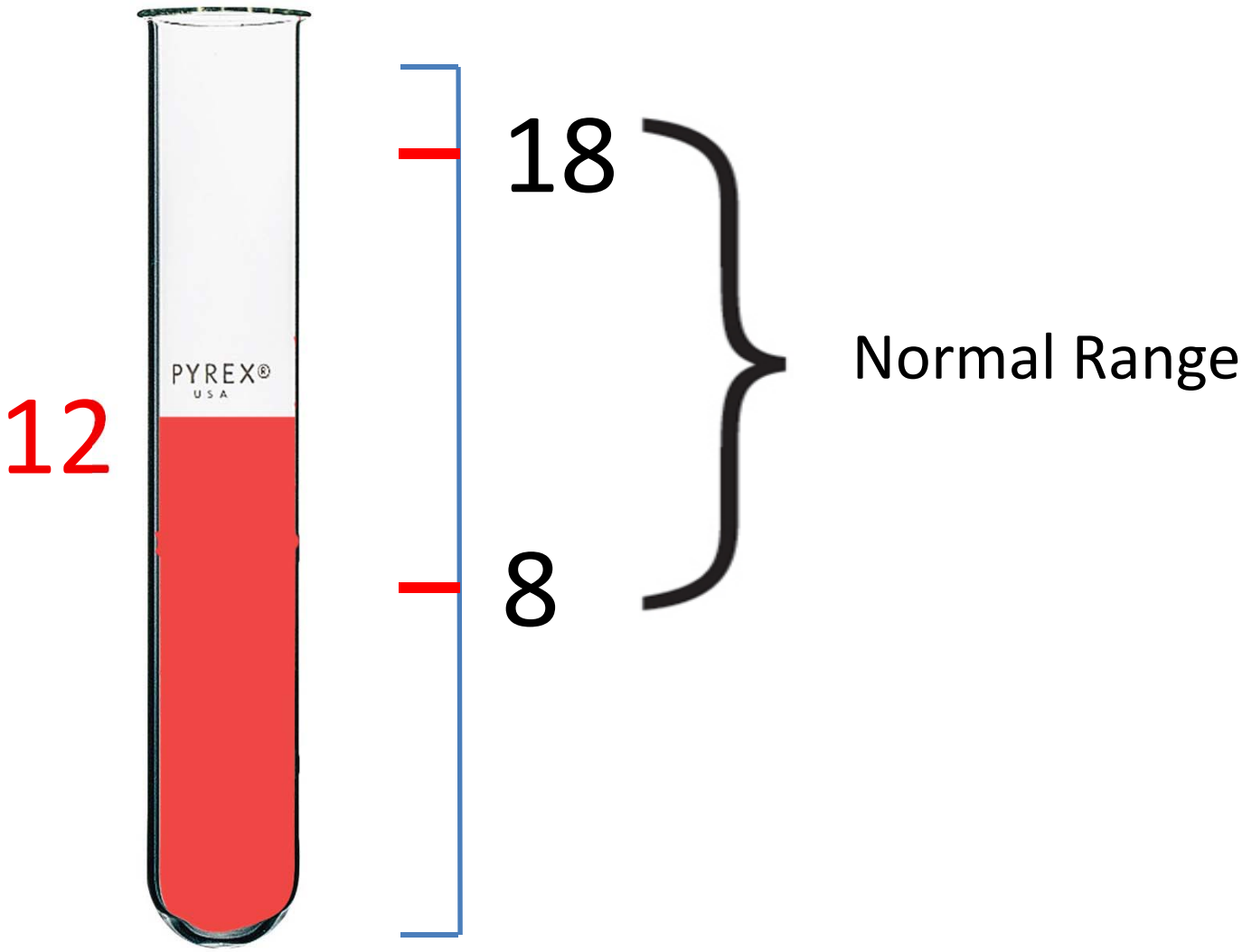
# Cholinesterase Test



Does this rule out OP  
(organophosphate)  
poisoning?



# Cholinesterase Test



# ChE Normal - Cholinergic Syndrome?

What are two explanations for a normal ChE in the presence of a clear cholinergic syndrome?

1. A true depression of a normally high ChE level
2. It is not a OP poisoning

**HIGHLIGHTS**

Muscarinic, nicotinic, CNS effects

Absorbed by inhalation, ingestion, skin

Lipophilic

Poisonings tend to be of shorter duration than OPs

**SIGNS & SYMPTOMS**

Ma  
diz  
Ma  
inn

## N-Methyl Carbamate Insecticides

### Signs and Symptoms of Poisoning

As with organophosphate poisoning, the signs and symptoms are based on excessive cholinergic stimulation. Carbamate poisonings tend to be of shorter duration than organophosphate poisonings because of the reversibility of the AChE binding and the more rapid metabolism of carbamates.<sup>7</sup> However, as mentioned in the next section of this chapter, blood cholinesterase levels may be misleading because of *in vitro* reactivation of a carbamylated enzyme.<sup>8,9</sup> This falsely normal or near-normal level can make the diagnosis more difficult in the acute presentation in the absence of an exposure history.

De most organophosphate compounds and (3) it frequently invalidates the measurement  
Co of blood cholinesterase activity as a diagnostic index of poisoning (see below).

mixed poisonings

Consider GI decontamination

**CONTRAINDICATED**

Adrenergic amines without specific indication (e.g., hypotension)

# Page 56

**Toxicology**

The **N-methyl carbamate** esters cause reversible carbamylation of acetylcholinesterase (AChE) enzyme, allowing accumulation of acetylcholine, the neuromediator

on post mortum.<sup>10</sup> Respiratory depression combined with pulmonary edema is the usual cause of death from poisoning by N-methyl carbamate compounds.

**Signs and Symptoms of Poisoning**

As with organophosphate poisoning, the signs and symptoms are based on excessive cholinergic stimulation. Carbamate poisonings tend to be of shorter duration than organophosphate poisonings because of the reversibility of the AChE binding and the more rapid metabolism of carbamates.<sup>7</sup> However, as mentioned in the next section of this chapter, blood cholinesterase levels may be misleading because of *in vitro* reactivation of a carbamylated enzyme.<sup>8,9</sup> This falsely normal or near-normal level can make the diagnosis more difficult in the acute presentation in the absence of an exposure history.

The objective of atropine antidotal therapy is to antagonize the effects of excessive concentrations of acetylcholine at end-organs having muscarinic receptors. Atropine does not reactivate the cholinesterase enzyme or accelerate disposition of organophosphate. Recrudescence of poisoning may occur if tissue concentrations of organophosphate are high when the effect of atropine wears off, and multiple doses may be required. Atropine is effective against muscarinic manifestations, but it is ineffective against nicotinic actions, specifically muscle weakness and twitching, and respiratory depression. Despite these limitations, atropine is often a life-saving agent in organophosphate poisoning. The lack of response to a test dose of atropine can help differentiate poisoning by anticholinesterase agents from other conditions.

#### Test Dosage of Atropine

- **Adults: 1 mg**
- **Children under 12 years: 0.01 mg/kg**

Note, however, that lack of response with no evidence of atropinization (atropine refractoriness), may also indicate a more severe poisoning. The adjunctive use of humidified atropine has been reported to improve respiratory distress, decrease bronchial secretions, and increase oxygenation.<sup>43</sup>

#### Dosage of Atropine

In *moderately severe poisoning* (hypersecretion and other end-organ manifestations without central nervous system depression), the following dosage schedules have been used.

- **Adults and children over 12 years: Initial dose 1-3 mg IV. Repeat in 3-5 minutes if no change in clinical symptoms. Dose may be doubled with each administration until the patient is atropinized. Once adequate atropinization has been achieved, the patient can be maintained on an atropine continuous infusion at about 10%-20% of the loading dose and titrated to effect.**<sup>4,44,45,46</sup>
- **Children under 12 years: There is less agreement regarding pediatric dosing. Recent studies recommend beginning with 0.02 mg/kg body weight, and doubling the dose every 5 minutes until atropinization is achieved.**<sup>4,44</sup> *Patients seen in a pediatric ICU setting were given 0.05 mg/kg every 15 minutes.*<sup>31</sup> *Since children sometimes present differently than adults and have more CNS findings, aggressive atropinization should proceed when there are muscarinic signs such as bradycardia, salivation, diarrhea and miosis that can be observed to change with adequate atropine.*<sup>31</sup>



What tells you have  
achieved  
atropinization?



mates may reverse with smaller dosages of atropine than those required to reverse organophosphates, though the required dosage is still considerably larger than that required to atropinize a non-poisoned patient.<sup>17,18</sup> A common dosing pitfall is giving too little atropine initially to achieve timely atropinization. Severely poisoned individuals may exhibit remarkable tolerance to atropine and require large doses.<sup>14</sup> (See dosage below.)

The objective of atropine antidotal therapy is to antagonize the effects of excessive concentrations of acetylcholine at end-organs having muscarinic receptors. Atropine does not reactivate AChE or accelerate excretion or breakdown of carbamate. Multiple doses of atropine may be necessary, as recrudescence of poisoning can occur if tissue concentrations of toxicant remain high when the antidotal effect wears off. Atropine is effective against muscarinic manifestations, but is ineffective against nicotinic actions, specifically muscle weakness and twitching, and respiratory depression. Despite these limitations, atropine is often a lifesaving agent in N-methyl carbamate poisonings.

Reassess the clinical situation after an adequate loading dose has been given. If symptoms persist, but the history is consistent with carbamate poisoning, then continue atropine therapy. However, if the clinical picture is unclear, clinicians should reassess and consider alternative causes of poisoning, such as pyrethroid insecticide poisoning, which may present a similar clinical picture.

In moderate to severe poisoning (hypersecretion and other end-organ manifestations and/or central nervous system depression) the following dosage schedule has proven effective:

#### Dosage of Atropine

##### Adults and Children Over 12 Years

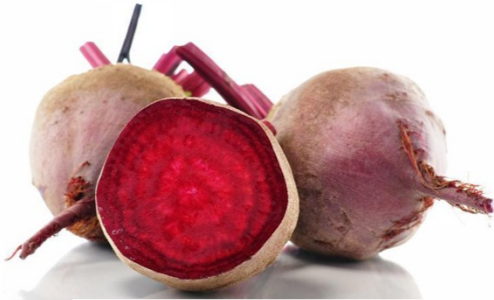
- **Initial Dose: 1-3 mg IV. Repeat in 3-5 minutes if no change in clinical symptoms. Dose may be doubled with each administration until the patient is atropinized. Once adequate atropinization has been achieved, the patient can be maintained on an atropine continuous infusion at about 10%-20% of the loading dose and titrated to effect.<sup>18,19,20,21</sup> Clear breath sounds and absent pulmonary secretions are the primary end point. Other signs of atropinization including flushing, dry mouth and dilated pupils; tachycardia (pulse of 140 per minute) may occur. Early in therapy, monitor for improving blood pressure and heart rate (above 80 beats/ minute), normal pupil size and drying of the skin and axillae.<sup>20,21</sup> Autoinjectors containing 2.0 mg atropine for IM injection are also available.**

**WARNING:** Poisonings in which liquid carbamate pesticide concentrates have been ingested may be complicated by hydrocarbon aspiration. Pulmonary edema and poor oxygenation in these cases will not respond to atropine and should be treated as a case of acute respiratory distress syndrome.

*continued next page*



Blind as a bat



Red as a beet



Mad as a hatter






Dry as a bone



Hot as hell

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- 
- 
- 
4. Consider pralidoxime in cases of mixed carbamate/organophosphate poisoning and cases of an unknown pesticide with muscarinic symptoms on presentation (see **Chapter 5, *Organophosphate Insecticides***, subsection *Treatment*, item 5, page 49.<sup>22,23</sup> Pralidoxime has been used in some cases of carbamate poisoning, although other cases have resolved from supportive care alone.<sup>24,25</sup> Pralidoxime is probably of little value in N-methyl carbamate poisonings and is not indicated in isolated carbamate poisonings. **Atropine alone usually is effective.**
  5. Decontaminate concurrently with whatever resuscitative and antidotal measures are needed to preserve life. Contamination of the eyes should be removed by flushing with copious amounts of clean water. For asymptomatic individuals who are alert and physically able, skin decontamination should occur as previously outlined in **Chapter 3, *General Principles***. Specifically, skin and hair should be washed with soap and water. Attending personnel must take precautions including rubber gloves to avoid contamination. Contaminated clothing should be promptly removed, bagged and laundered before returning, and items such as shoes, boots and headgear should be discarded.

# Decontamination



Photo © US Navy

Are we  
done yet?

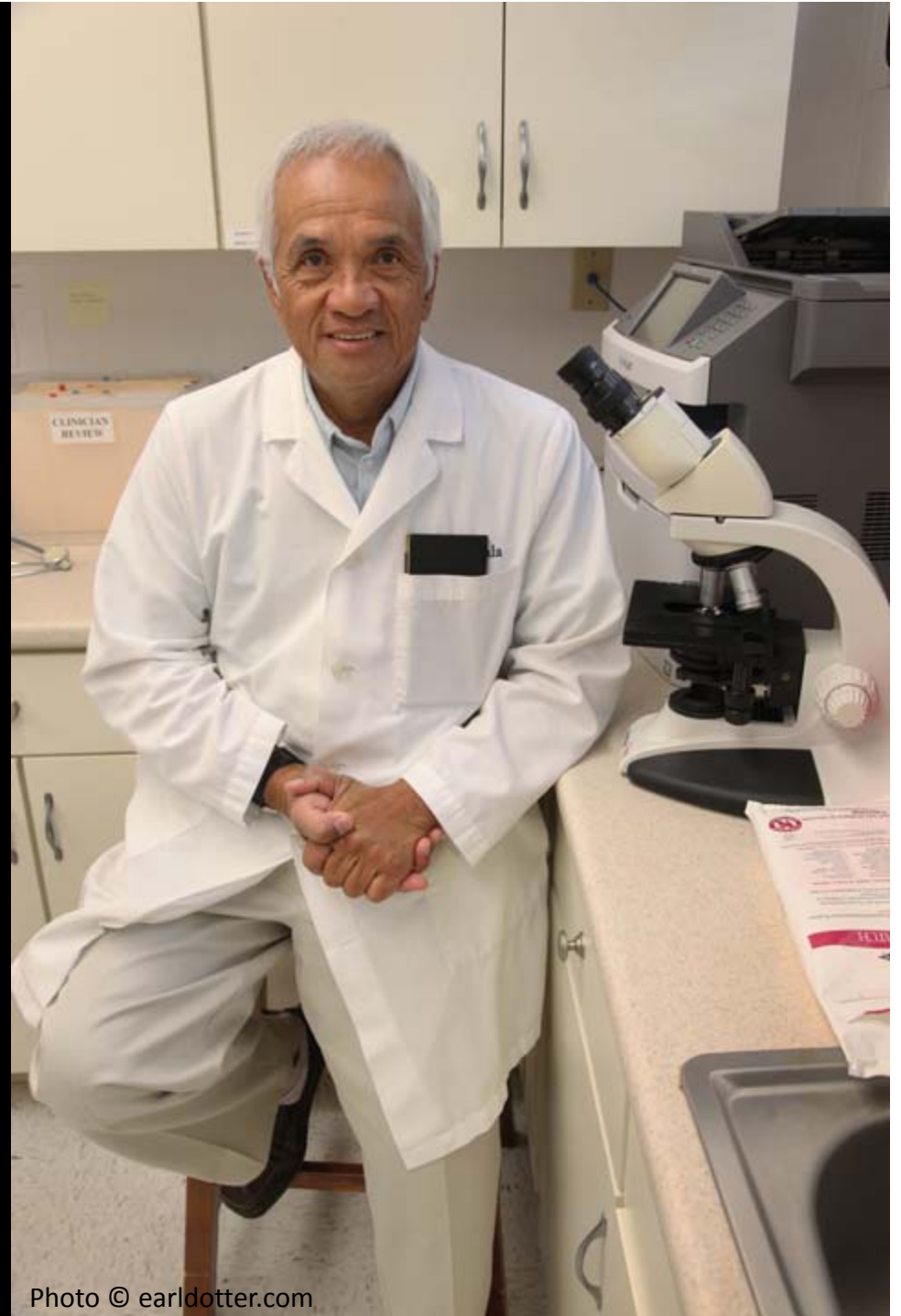


Photo © earldotter.com

### Issues We Face

Migrant Info

Behavioral Health

Cancer

Children's Health

Diabetes

Emergency Preparedness

Environmental and Occupational Health

Pesticides

[Report Pesticide Exposures](#)

Lead

Heat Stress

Water & Sanitation

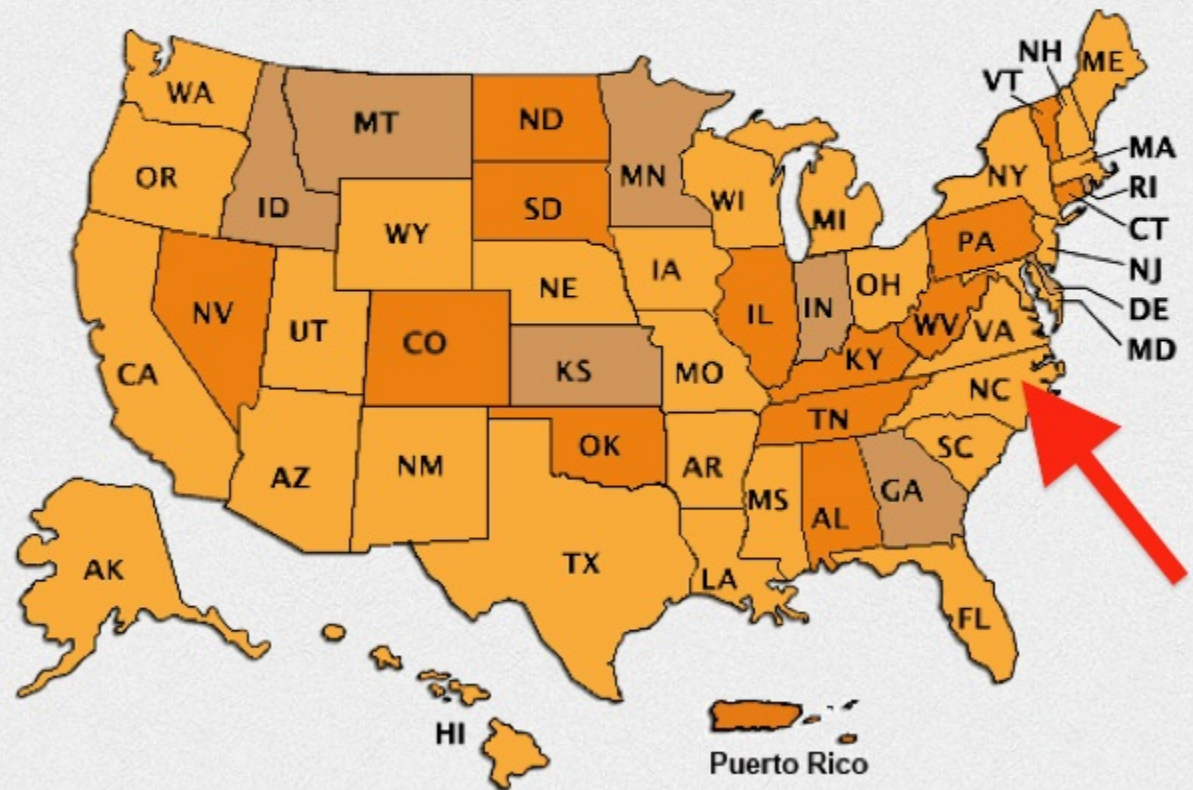
Eye Care

Family Violence

H1N1 Flu

Home » Issues We Face » Environmental and Occupational Health » Pesticides

## Pesticide Exposure Reporting Map



PESTICIDE REPORTING REQUIREMENT:

Required

Optional

None



# Workers' Compensation

Photo © earldotter.com



Which of the following options confirm carbamate overexposure?



# Data Collection of an Acutely Exposed Patient

CHAPTER 2  
Making the Diagnosis

Data Collection on an Acute Pesticide Exposed Patient, continued

Materials to be Gathered:

1. A copy of the pesticide label and/or a copy of the Material Safety Data Sheet (MSDS).
2. A copy of the pesticide application record (tank mix, concentration, etc.) if applicable. This should be available from the pesticide applicator or the grower.
3. 10 cc whole blood, anticoagulated with sodium heparin (refrigerate).
4. 5 cc plasma anticoagulated with sodium heparin (refrigerate).
5. A fresh urine sample (label and freeze).
6. Any contaminated clothing, hats, foliage from the site. Place in clean sealable plastic bag, label, seal and freeze.
7. Other options:
  - a. Fingernail residue. If the worker handled the pesticide or materials with pesticide residue, some pesticide may be lodged under the fingernails. Clean under the nails. Place in clean sealable plastic bag, label, seal and freeze.
  - b. Saliva sample. Some pesticides can be detected in saliva. Have the patient spit repeatedly into a clean glass or plastic container. Seal the container, label and freeze.
  - c. Hair sample. If the head was exposed. Place in clean sealable plastic bag, label, seal and freeze.
  - d. A skin wipe with ethanol-impregnated swab
    - i. Wipe skin that was contaminated if possible. Use a newly opened alcohol wipe. Wipe an area of skin and if possible estimate the size of the area wiped and record this on the sample label. Try to focus on an area that is likely to have been contaminated in the exposure.
    - ii. Place wipe in clean sealable plastic bag, label, seal and freeze.

\*For the pediatric patient, note parents' occupations and child's appearance compared to his/her usual baseline. It is important to ask if the child is acting normally, if there is an abnormal gait, stumbling or ataxia, and if the child has experienced excessive sleepiness, irritability or other personality changes.

Developed by Matthew C. Koeller MD, MPH  
National Farm Medicine Center

# PESTICIDE EXPOSURE ASSESSMENT

To be filled out during clinical assessment. Health provider—ask these questions verbally



Full Name: \_\_\_\_\_ Patient ID \_\_\_\_\_  
Last First

DOB: \_\_\_\_\_  
Street Address Apartment/Unit #

Address: \_\_\_\_\_  
City State ZIP Code

Occupation: \_\_\_\_\_  
 Employer:  Male  Female

Name of pesticide (active ingredients, concentration & EPA registration number): \_\_\_\_\_

**Exposure Information**

**Circumstances:**  
 Intentional  
 Accidental  
 Occupational  
 Non-occupational

**Exposure route:**  
 Dermal  
 Ocular  
 Oral  
 Respiratory

**Method of pesticide application:**  
 Aerial  
 Backpack sprayer  
 Hand sprayer  
 Boom sprayer  
 Air blast  
 Other: \_\_\_\_\_

Amount, if ingested: \_\_\_\_\_

Concentrate or dilutions: \_\_\_\_\_

Crop (if applicable): \_\_\_\_\_

Other exposure details (eg. spills, drift) early twenty? \_\_\_\_\_

Other individuals involved (also exposed, witnessed, assisted)?  
 Who? \_\_\_\_\_

If worker, had patient received Worker Protection Standard training?  
 Yes  No

**Symptoms**

<input type="checkbox"/> Weakness	<input type="checkbox"/> Drooling	<input type="checkbox"/> Blurred vision	<input type="checkbox"/> Chest pain
<input type="checkbox"/> Skin rash	<input type="checkbox"/> Tiredness	<input type="checkbox"/> Excessive sweating	<input type="checkbox"/> Red eyes
<input type="checkbox"/> Headaches	<input type="checkbox"/> Nausea	<input type="checkbox"/> Loss of consciousness	<input type="checkbox"/> Convulsions
<input type="checkbox"/> Shortness of breath	<input type="checkbox"/> Dizziness	<input type="checkbox"/> Vomiting	<input type="checkbox"/> Abdominal pain
<input type="checkbox"/> Muscle twitches	<input type="checkbox"/> Productive cough	<input type="checkbox"/> Confusion	Others: _____

How long after over-exposure did symptoms begin? \_\_\_\_\_ hrs. \_\_\_\_\_ min.

Length of clinical observations: \_\_\_\_\_ hrs. \_\_\_\_\_ min.

Notable changes over observation period (describe): \_\_\_\_\_

Other workers/persons exposed who developed symptoms?  
 Yes  No

# Confirmation of Poisoning

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Eye

SYSTEM: Eye	SYMPTOMS/ SIGNS/DISEASE CATEGORIES	CHARACTERISTIC OF THESE POISONINGS	MAY OCCUR IN THESE POISONINGS
	Conjunctivitis (irritation of mucous membranes, tearing)	Chloropicrin Acrolein Copper compounds Organotin compounds Cadmium compounds Metam sodium Paraquat Diquat Acrolein Chloropicrin Sulfur dioxide Naphthalene Formaldehyde Ethylene oxide Methyl bromide Endothall Toluene Xylene Fipronil	Thiophthalimides Thiram Thiocarbamates Pentachlorophenol Chlorophenoxy compounds Chlorothalonil Picloram Creosote Aliphatic acids Strobilurin fungicides Pyrethrins
	Lacrimation (muscarinic)	N-methyl carbamates	
	Yellow sclerae	Nitrophenols	Agents that cause jaundice (see section on Skin)
	Keratitis	Paraquat	
	Ptosis	Thallium	
	Diplopia	Organophosphates N-methyl carbamates Nicotine	
	Photophobia		Organotin compounds
	Constricted visual fields	Organic mercury	
	Optic atrophy		Thallium
Miosis	Organophosphates N-methyl carbamates	Nicotine (early)	
Dilated pupils	Cyanide Fluoride	Nicotine (late)	
Non-reactive pupils	Cyanide		

**RESTRICTED USE PESTICIDE**

Due to acute inhalation toxicity to humans.

For retail sale to and use by certified applicators or persons under their direct supervision and only for those uses covered by the certified applicator's certification.

# METAM SODIUM

**A SOIL FUMIGANT SOLUTION FOR SPECIFIC CROPS AS LISTED IN THIS LABEL:  
MAY BE APPLIED BY WATER-RUN APPLICATIONS (e.g., CHEMIGATION), SOIL INJECTION OR  
SOIL BEDDING EQUIPMENT TO SUPPRESS AND/OR CONTROL SOIL-BORNE PESTS IN LISTED  
ORNAMENTALS, FOOD AND FIBER CROPS.**

For the control or suppression of Weeds, Diseases and Nematodes. Suppresses and/or Controls Weeds such as Annual Bluegrass, Bermudagrass, Chickweed, Dandelion, Ragweed, Henbit, Lambsquarter, Amaranthus species, Watergrass, Johnsongrass, Nutgrass, Wild Morning Glory and Purslane Nematodes and Symphylids, Soil-borne diseases such as Rhizoctonia, Pythium, Phytophthora, Verticillium, Sclerotinia, Oak Root Fungus and Club Root of Crucifers.

**ACTIVE INGREDIENT:**

Sodium methylthiocarbamate (anhydrous)\*.....32.7%

**OTHER INGREDIENTS:**.....67.3%

**TOTAL:**.....100.0%

\*Contains 3.18 lbs. METAM SODIUM per gallon

**KEEP OUT OF REACH OF CHILDREN**

**ANGER — PELIGRO**

*Si usted no entiende la etiqueta, busque a alguien para que se la explique a usted en detalle.*

*(If you do not understand the label, find someone to explain it to you in detail).*

**FIRST AID**

<b>If on skin or clothing:</b>	<ul style="list-style-type: none"> <li>• Take off contaminated clothing.</li> <li>• Rinse skin immediately with plenty of water for 15-20 minutes.</li> <li>• Call a poison control center or doctor for treatment advice.</li> </ul>
<b>If in eyes:</b>	<ul style="list-style-type: none"> <li>• Hold eye open and rinse slowly and gently with water for 15-20 minutes.</li> <li>• Remove contact lenses, if present, after the first 5 minutes, then continue rinsing eye.</li> <li>• Call a poison control center or doctor for treatment advice.</li> </ul>
<b>If inhaled:</b>	<ul style="list-style-type: none"> <li>• Move person to fresh air.</li> <li>• If person is not breathing, call 911 or an ambulance, then give artificial respiration, preferably mouth-to-mouth if possible.</li> <li>• Call a poison control center or doctor for further treatment advice.</li> </ul>
<b>If swallowed:</b>	<ul style="list-style-type: none"> <li>• Call a poison control center or doctor immediately for treatment advice.</li> <li>• Have person sip a glass of water if able to swallow.</li> <li>• Do not induce vomiting unless told to do so by a poison control center or doctor.</li> <li>• Do not give anything by mouth to an unconscious person.</li> </ul>

**EMERGENCY INFORMATION**

Have the product container or label with you when calling a poison control center or doctor, or going for treatment.  
**FOR THE FOLLOWING EMERGENCIES, PHONE 24 HOURS A DAY:**

For Medical Emergencies, phone:.....1-888-681-4261

For Transportation Emergencies, including spill, leak or fire, phone: CHEMTREC.....1-800-424-9300

For Product Use Information, phone: AMVAC.....1-888-462-6822

SEE SIDE/BACK PANEL FOR ADDITIONAL PRECAUTIONARY STATEMENTS AND DIRECTIONS FOR USE.

EPA Reg. No. 5481-350

Net Weight:

EPA Est. No.  5481-CA-1  1448-MO-1  61842-WA-1

As Marked on Container



**PRECAUTIONARY STATEMENTS  
HAZARDS TO HUMANS AND DOMESTIC ANIMALS**

**DANGER: Corrosive.** Causes skin damage. May be fatal if absorbed through the skin. Do not get on skin or clothing. Prolonged or frequently repeated skin contact may cause allergic reactions in some individuals. Harmful if swallowed. Harmful if inhaled. Irritating to eyes, nose and throat. Avoid breathing vapor or spray mist. Irritating to eyes. Do not get in eyes.

**PERSONAL PROTECTIVE EQUIPMENT (PPE)**

Some materials that are chemical-resistant to this product are barrier laminate or Viton  $\geq 14$  mils. For more options, follow the instructions for category H on the chemical-resistance category selection chart.

Handlers applying while irrigation systems are operating or handlers who may be exposed to liquid spray while repairing a malfunctioning chemigation system or shutting off equipment must wear:

- chemical-resistant coveralls over long-sleeve shirt and long pants,
- chemical-resistant gloves,
- chemical-resistant footwear plus socks,
- chemical-resistant headgear, and
- respirator of the type specified in the respiratory protection section in the PPE requirements on this label.

Handlers wearing chemical-resistant attire are limited to 30 minutes of exposure in any 60 minute period to prevent heat illness, and, as required by the Worker Protection Standard for Agricultural Pesticides, employers of these handlers must take any necessary steps to avoid heat illness.

Except as required above, handlers transferring or loading liquid formulations, handlers operating motorized ground equipment with open cabs, handlers repairing or inactivating irrigation or chemigation equipment during application, and handlers cleaning up spills or equipment, must wear:

- coveralls over long-sleeve shirt and long pants,
- chemical resistant gloves,
- chemical resistant footwear plus socks,
- chemical-resistant apron if transferring or loading the fumigant or cleaning up spills or equipment,
- protective eyewear, and
- respirator of the type specified in the PPE requirements for respiratory protection section in the PPE requirements on this label if triggered.

All other handlers, including handlers operating motorized ground equipment with closed cabs (except for handlers who set up and calibrate chemigation and irrigation equipment and start the application from inside the application block) as stated in this labeling must wear:

- long-sleeve shirt and long pants,
- shoes plus socks, and
- respirator of the type specified in the respiratory protection section in the PPE requirements on this label if triggered.

All handlers who set-up and calibrate chemigation and irrigation equipment and start the application from inside the application block must wear:

- long-sleeve shirt and long pants,
- shoes plus socks,
- protective eyewear, and
- respirator of the type specified in the respiratory protection section in the PPE requirements on this label if triggered.

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**Thiocarbamate  
COMMERCIAL  
PRODUCTS**

thiram (Aules, Chipco  
Thiram 75, Fermide 850,  
Femasan, Hexathir,  
Mercuram, Nomersam,  
Polyram-Ultra, Pomarsol  
forte, Spotrete-F, Spotrete  
WP 75, Tetrapom, Thimer,  
Thioknock, Thiotex,  
Thiramad, Thirasan,  
Thiuramin, Tirampa, TMTD,  
Trametan, Tripomol, Tuads)

ziram (Cuman, Hexazir,  
Mezene, Tricarbamix,  
Triscabol, Vancide MZ-96,  
Zincmate, Ziram Technical,  
Ziram F4, Zirberk, Zirex 90,  
Ziride, Zitox)

ferbam (Carbamate WDG,  
Ferbam, Ferberk, Hexaferb,  
Knockmate, Trifungol)

**Confirmation of Poisoning**

Tests to detect these compounds are not readily available.

**Treatment of Strobilurin Toxicosis**

Remove the patient from the source of exposure.

Provide supportive treatment directed to symptoms. Significant acute toxicity is not generally expected; therefore, exposure can be asymptomatic and symptoms usually do not warrant medical attention.

Consider skin decontamination as outlined in **Chapter 3, General Principles**.

Flush eyes with water or normal saline. If eye irritation, redness or swelling persists for more than 15 minutes, recommend consultation with an ophthalmologist.

**THIOCARBAMATES**

Thiocarbamates are commonly formulated as dusts, wettable powders or water suspensions. They are used to protect seeds, seedlings, ornamentals, turf, vegetables and fruit including apples. Unlike the N-methyl carbamates (**Chapter 6**), thiocarbamates have very little insecticidal potency. A few exhibit weak anticholinesterase activity, but most have no significant effect on this enzyme. Overall, they are less of a threat to human health than the insecticidal carbamates. Fungicidal thiocarbamates are discussed in this section, while those used as herbicides are considered in **Chapter 13, Other Herbicides**.

Metam-sodium, thiram and ziram and ferbam are the thiocarbamate pesticides. They are discussed individually.

**Metam-sodium**

Metam-sodium is formulated in aqueous solutions for application as a soil biocide to kill fungi, bacteria, weed seeds, nematodes and insects. All homeowner uses have been canceled in the United States.

**Toxicology**

Although animal feeding studies do not indicate high toxicity of **metam-sodium** by ingestion, its decomposition in water yields methyl isothiocyanate, a gas that is extremely irritating to the eyes and to respiratory mucous membranes including the lower respiratory tract/lungs. Inhalation of methyl isothiocyanate may cause pulmonary edema, manifesting with severe respiratory distress and coughing of bloody, frothy sputum. For this reason, **metam-sodium** must be used outdoors only, and stringent precautions must be taken to avoid inhalation of evolved gas. Metam-sodium can be very irritating to the skin.

Theoretically, exposure to metam-sodium may predispose the individual to "Antabuse" reactions if alcohol is ingested after exposure. Such occurrences have not been reported in the medical literature.

**Confirmation of Poisoning**

There are no tests for metam-sodium or its breakdown products in body fluids.

**Treatment of Metam-sodium Toxicosis**

Decontaminate skin and GI tract, as outlined in **Chapter 3, General Principles**.

If pulmonary irritation or edema occurs as a result of inhaling methyl isothiocyanate, transport the victim promptly to a medical facility. Treatment for pulmonary edema should proceed as outlined in **Chapter 17, Fumigants in the Treatment of Fumigant Toxicosis** subsection beginning on page 166.

Metam-sodium is not a cholinesterase inhibitor. Atropine is not antidotal.

**Thiram**

**Thiram dust** is moderately irritating to human skin, eyes and respiratory mucous membranes. Contact dermatitis has occurred in occupationally exposed workers. A few individuals have experienced sensitization to thiram.<sup>14</sup> Thiram is a common component of latex and possibly responsible for some of the allergies attributed to latex.

**Toxicology**

Systemic human poisonings by **thiram** itself have been very few, probably due to limited absorption in most circumstances involving human exposure. Those that have been reported have been similar clinically to toxic reactions to **disulfiram** (Antabuse), the ethyl analogue of thiram that has been extensively used in alcohol aversion therapy.<sup>16</sup> In laboratory animals, thiram at high dosage has effects similar to those of disulfiram (hyperactivity, ataxia, loss of muscle tone, dyspnea and convulsions), but thiram appears to be about 10 times more toxic than disulfiram.

Neither thiram nor disulfiram is a cholinesterase inhibitor. Both, however, inhibit the enzyme acetaldehyde dehydrogenase, which is critical to the conversion of acetaldehyde to acetic acid. This is the basis for the "Antabuse" reaction that occurs when ethanol is consumed by a person on regular disulfiram dosage. The "reaction" includes symptoms of nausea, vomiting, pounding headache, dizziness, faintness, mental confusion, dyspnea, chest and abdominal pain, profuse sweating and skin rash. In rare instances, Antabuse reactions may have occurred following ingestion of beverages containing alcohol among workers previously exposed to thiram.

**Confirmation of Poisoning**

Urinary xanthurenic acid excretion has been used to monitor workers exposed to thiram, but the test is not generally available.

**Treatment of Thiram Toxicosis**

Decontaminate skin and GI tract as outlined in **Chapter 3, General Principles**.

Infuse appropriate intravenous fluids, especially if vomiting and diarrhea are severe. Monitor serum electrolytes and glucose and replace as needed.

**Treatment of Acetaldehyde Toxicosis (Antabuse reaction)**

Use oxygen inhalation, trendelenburg positioning and intravenous fluids, which are usually effective in relieving manifestations of "Antabuse" reactions.

**Thiocarbamate  
HIGHLIGHTS**

Formulated as dusts,  
wetable powders, water  
suspensions

Less human health threat  
than insecticidal carbamates

**SIGNS & SYMPTOMS**

Skin, eye, respiratory  
irritation

For metam-sodium  
inhalation, respiratory  
distress, bloody sputum

May result in Antabuse-  
like reaction if alcohol is  
consumed after exposure

**TREATMENT**

Decontaminate skin and GI  
tract

For metam-sodium, treat  
pulmonary impacts as for  
fumigant toxicosis

For thiram, ziram and  
ferbam, IV fluids as needed

For Antabuse reaction,  
oxygen, IV fluids and  
trendelenburg positioning



webinar

## **NATIONAL CONVERSATION:** Strengthening the Worker Protection Standard



Wednesday, March 5, 2014 10am PDT

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