



PESTICIDE POISONINGS. ARE YOU PREPARED?

James Roberts, MD, MPH, Medical University of South Carolina

Amy K. Liebman, MPA, MA, Migrant Clinicians Network



April 20, 2017, 1 PM ET

Disclosure and Disclaimer

Faculty:

James Roberts, MD, MPH and Amy K. Liebman, MPA, MA

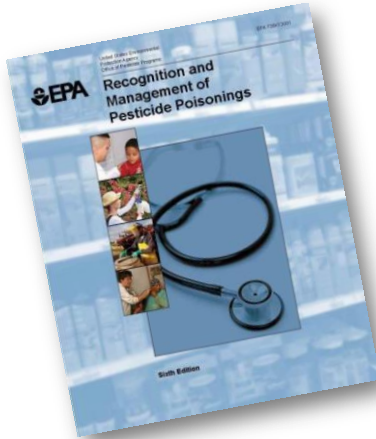
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Learning Objectives



- Describe acute and chronic health effects of pesticide exposure
- Recognize multiple types and categories of pesticides
- Identify the clinical effects of active ingredients in commonly used pesticides
- Understand how to use the *Recognition and Management of Pesticide Poisonings, 6th ed*

Recognition and Management of Pesticide Poisonings, 6th Edition

<http://www2.epa.gov/pesticide-worker-safety/recognition-and-management-pesticide-poisonings>





Pesticide Exposure

- 10,000-20,000 occupational exposures per year in US (EPA 1996)
- Inconsistent and incomplete surveillance system
- Latinos farmworkers most exposed
- Over 1 billion pounds of pesticides used each year, mostly in agriculture

World-wide

3 million poisonings

200,000 deaths

Significant underreporting

~ 25 million poisonings (if all cases counted)

98% under-reporting to surveillance systems (Studies from Central America)

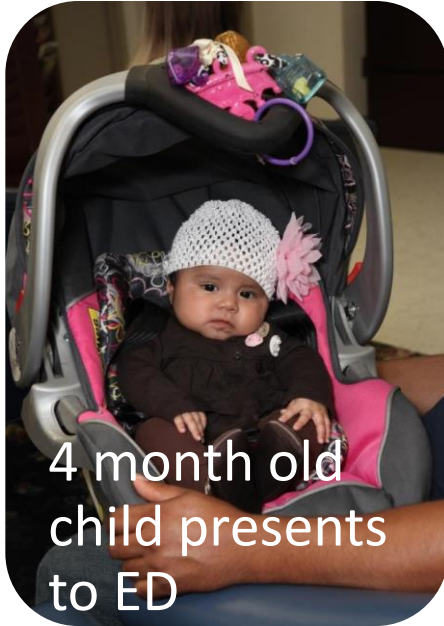


If You Remember One Thing...



- **80%** of children with organophosphate poisoning were transferred with the wrong diagnosis
- Acute pesticide poisoning may not be relatively common...
- But you need a high index of suspicion so that it is not missed

Zweiner RJ, et al. Organophosphate and carbamate poisoning in infants and children
Pediatrics 1988;81:121-6.



4 month old
child presents
to ED

Photo © earldotter.com

- ✓ Fussy, decreased appetite, vomiting, diarrhea, lethargic, limp
- ✓ Apnea reported, and en route “eyes roll back in head”
- ✓ ED exam: Limp, miosis, poor respiratory effort, increased amount of secretions
- ✓ HR 178, RR 34, T 98.6

What is your diagnosis?



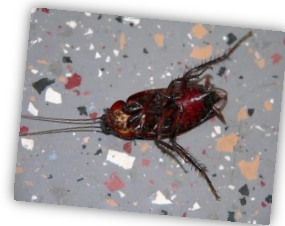
4 Month old now in ICU



- Further hx: 5 previous hospital admissions, 1 of which OP suspected
- Sepsis workup negative
- Received fentanyl, pralidoxime, atropine
- RBC and plasma cholinesterase levels decreased
- Initial urine, blood, and breast milk samples negative for pesticides/metabolites
- Baby stabilized, remained in hospital during investigation of home, dad's work

Organophosphate/Carbamate Acute Toxicity

- Phosphorylates acetylcholinesterase (AChE)
 - Excess Ach accumulates in nerve ending
- Classic findings: Hyper-secretion (muscarinic)
 - Salivation, lacrimation, bronchorrhea, perspiration, diarrhea, miosis
 - *Less common in children than adults*
- Skeletal muscle (nicotinic effects)
 - Excitatory (Muscle fasciculations)
 - Inhibitory (Weakness & paralysis) – *this is actually a delayed finding in some cases of OP poisoning*



Organophosphate Toxicity

- **Central effects**
 - Sensory/behavioral disturbance, incoordination
 - Respiratory depression, coma, seizures
- **Autonomic ganglia**
 - Classically see bradycardia--- sinus arrest
 - Tachycardia and hypertension from nicotinic receptors may precede bradycardia
- **Cause of death**
 - respiratory depression (central) exacerbated by excess pulmonary secretions
- **Children v. Adults**
 - Seizures in 8-39% of children v. 2-3% in adults
 - Lethargy and coma 55%-100% of pediatric cases

Roberts JR and Reigart JR. Recognition and Management of Pesticide Poisonings, 6th ed. 2013

Treatment

- Recovery depends on generation of new enzyme
- Airway, oxygenation, and ventilation
- Atropine reverses some cholinergic effects
 - Frequent doses and higher doses are needed
 - Generally less effective against nicotinic or CNS actions
- Pralidoxime (organophosphate only)
 - Reactivates AChE
- Address possible exposures & report incident

Infant's Home Environment

- Dad and uncle both farmworkers, living with their families in one farm trailer
- Both trained as pesticide handlers
 - Helped move pesticide containers but did not open or spray them
 - Wear same clothes home from field, but report washing them separately
- Moved to different trailer at discharge

4 Month at Home

- Tested wipe samples from original trailer
 - 2 different OPs
- Submitted new sample of infant's urine to CDC for further testing
 - Acephate and dimethyl OP metabolites
- Set up decon room away from main living area
 - Still came home and ate lunch at table
 - Changed clothes, but no shower before holding baby



Photo © earldotter.com

Understanding Pesticide Use

Survey of patients in 4 pediatric practices

- Insecticides are applied as a spray or powder in 66% of homes
 - 19% once a month
 - 14% two times a month or more often
- 12% said their doctor discussed pesticides
- Information sources for parents?
 - Pediatricians– 52%

Roberts JR Unpublished data

Medical School and Residency Training

- In medical school, ~ 7 hours on environmental health (EH) related topics (over all 4 years)¹
- US pediatric residency spends an average of two hours on EH related material²
 - ✓ Highly dependent on presence of faculty with expertise
- Sample of clinicians participating in MCN programs-
 - ✓ **78%** of respondents had 2 hours or less of EOH training

¹Schenk M, et al. *Acad Med* 1996; 71:499-501

²Roberts JR, Gitterman B. *Amb Peds* 2003;3:57-59

Physician/Medical Student Knowledge of Pesticides

- Clinician to recognize pesticide poisoning by clues in the history and PE
- My experience with students/ residents
 - They often equate “Pesticide” with “Insecticide”
 - Most can recall generalities of OP poisoning
 - Not differences between kids and adults
 - A differential diagnosis of pesticides?
 - “Rat poison” equates checking for bleeding
 - No institutional memory of convulsants (strychnine)



Which of the following is not an insecticide?



Commonly Presenting Signs and Symptoms

Seizures

Nausea, vomiting, diarrhea

Respiratory distress, pulmonary edema

Headaches and Mental status changes

- drowsiness
- lethargy
- coma

Skin findings

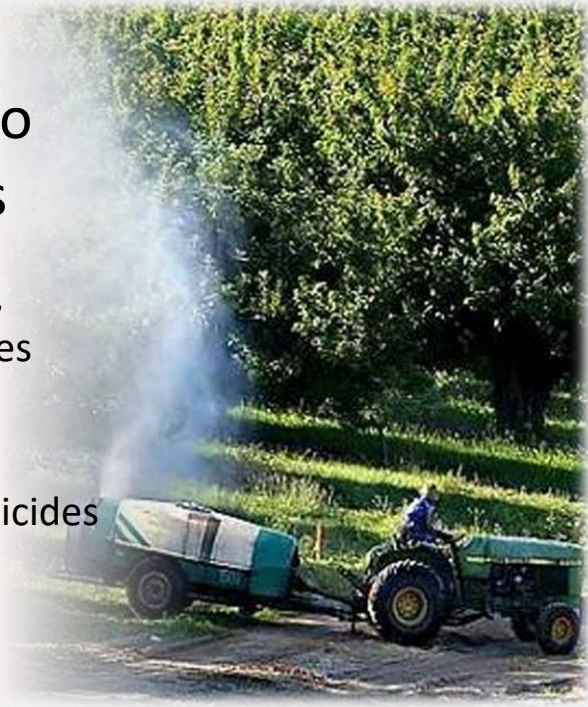
- rash
- blistering
- contact dermatitis

Cardiovascular

- tachycardia
- bradycardia
- hypotension

Worker Exposure to Pesticides

- Mixing, Handling, Applying Pesticides
- Working in Fields/Orchards Treated with Pesticides
- Drift



Exposure to Family Members

- ✓ “Take home” exposure
- ✓ Drift
- ✓ Home application of pesticides
- ✓ Lawn and vegetable and flower gardens

25 year old with mental status changes



- A semi-comatose patient is brought in by EMS
- Patient initially complained of stinging, burning and numbness on hands, arms and face after working in the fields
- Experienced headache, dizziness, nausea, vomiting
- Mental status changes eventually occurred
- Initially not clear what he was exposed to

- Increased oral secretions were noted
- Cyanosis and crackles present
- Muscle fasciculations were noted
- HR variable, tachycardic at times
 - Now bradycardic and occasional PVC's
- Seizure activity within 30 minutes

Is it another case of OP poisoning?

What signs or symptoms pointed you away from Organophosphates?



Patient Management

- Appropriate decontamination takes place
 - Showered, clothes removed and bagged
 - Hospital employee protection
- Airway and breathing support
 - Seizures controlled with lorazepam
- Treatment with atropine and pralidoxime while awaiting cholinesterase levels

- Our patient is a little better, but still sick and appears different than at presentation
 - Now has flushed and hot, dry skin
 - Mydriasis, increasing tachycardia
- Otherwise responded to supportive care
 - Seizures stopped, more alert
 - Cholinesterase levels within normal limits
- Co-worker confirms use of cypermethrin
 - Type II (“Cyano”-pyrethroid)



Pyrethrins/Pyrethroids

- Used worldwide since the 1970s
- Derived from the Chrysanthemum
- Pyrethrins—Short acting, unstable to heat/light, knockdown effect
 - Used for flying indoor pests, (wasp killer)
- Pyrethroids are synthetically modified
 - Outdoor control, agriculture, ectoparasites
 - Very common in consumer products

Chapter 4- Pyrethrins and Pyrethroids

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Pyrethrins HIGHLIGHTS

Strongly lipophilic
Crude pyrethrum is a dermal & respiratory allergen
Easily absorbed by GI tract & pulmonary membranes
Relatively low mammalian toxicity

SIGNS & SYMPTOMS

Contact dermatitis
Rhinitis, asthma

TREATMENT

Antihistamines
Epinephrine for anaphylaxis as required
Topical corticosteroid for contact dermatitis
Flush eyes as necessary
Consider gastric emptying or charcoal adsorption

CHAPTER 4

Pyrethrins and Pyrethroids

PYRETHRINS

Pyrethrum is the oleoresin extract of dried chrysanthemum flowers. The extract contains about 50% active insecticidal ingredients known as pyrethrins. The ketoalcoholic esters of chrysanthemic and pyrethroic acids are known as **pyrethrins**, **cinerins** and **jasmolins**. These strongly lipophilic esters rapidly penetrate many insects and paralyze their nervous systems. Both crude pyrethrum extract and purified pyrethrins are contained in various commercial products, commonly dissolved in petroleum distillates. Some are packaged in pressurized containers ("bug bombs"), usually in combination with the synergists piperonyl butoxide and n-octyl bicycloheptene dicarboximide. The synergists retard enzymatic degradation of pyrethrins. Pyrethrum and pyrethrin products are used mainly for indoor pest control. They are not sufficiently stable in light and heat to remain as active residues on crops. The synthetic insecticides known as pyrethroids (chemically similar to pyrethrins) have the stability needed for agricultural applications. Pyrethroids are discussed separately below.

Toxicology

Crude pyrethrum is a dermal and respiratory allergen, probably due mainly to non-insecticidal ingredients. Contact dermatitis and allergic respiratory reactions (rhinitis and asthma) have occurred following exposures.^{1,2} Single cases exhibiting anaphylactic³ and pneumonic manifestations⁴ have also been reported. Pulmonary symptoms may be due to inhalation of the hydrocarbon vehicle(s) of the insecticides. The refined pyrethrins are probably less allergenic but appear to retain some irritant and/or sensitizing properties.

Pyrethrins are absorbed across the gastrointestinal tract and pulmonary membranes, but only slightly across intact skin. They are very effectively hydrolyzed to inert products by mammalian liver enzymes. This rapid degradation, combined with relatively poor bioavailability, probably accounts in large part for their relatively low mammalian toxicity. Dogs fed extraordinary doses exhibit tremor, ataxia, labored breathing and salivation. Similar neurotoxicity has been rarely observed in humans, even in individuals who have had extensive contact from using pyrethrins for body lice control or have ingested pyrethrum as an anthelmintic.

In cases of human exposure to commercial products, the possible role of other toxicants in the products should be kept in mind. The synergists piperonyl butoxide and n-octyl bicycloheptene dicarboximide have low toxic potential in humans, which

CHAPTER 4
Pyrethrins & Pyrethroids

Pyrethroids
HIGHLIGHTS

Low systemic toxicity via inhalation and dermal route

Sites of action: sodium & chloride channels; GABA, nicotinic acetylcholine, peripheral benzodiazepine receptors

Type I (e.g., permethrin) usually do not contain a cyano group

Type II (e.g., cypermethrin, fenvalerate) always contain a cyano group

Type II acute poisonings are generally more severe

SIGNS & SYMPTOMS

Type I: fine tremor, reflex hyperexcitability

Type II: severe salivation, hyperexcitability, choreoathetosis

May include dizziness, headache, fatigue, vomiting, diarrhea

Stinging, burning, itching, tingling, numb skin may be reported

Severe cases: pulmonary edema, seizures, coma

TREATMENT

asures, followed by depolarization, conduction block and cell death at very high levels of exposure.⁷ In addition to the calcium and sodium channel sites of action, multiple other sites described (see following), including GABA receptors. They have

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A report of illnesses in 27 farmworkers and 4 emergency responders was related to pesticide drift of the pyrethroid **cyfluthrin**.¹³ In this episode, the most commonly reported symptoms were headache (96%), nausea (89%), eye irritation (70%), muscle weakness (70%), anxiety (67%) and shortness of breath (64%).¹³

Apart from central nervous system toxicity, some pyrethroids do cause distressing paresthesias when liquid or volatilized materials contact human skin. These symptoms

resistance of mammals. However, the possibility of dosing in adults has been shown to metabolize these agents resulting in neurotoxicity. In clinical findings with do not contain a cyano and **fenvalerate**, always amine release from the norepinephrine results in agents. A report of 466 cases notes that eight of these cases, 18% were severe.

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Signs and Symptoms of Poisoning

Type II acute poisonings are generally more severe than Type I.¹⁰ Type I poisoning has been described as characterized by fine tremor and reflex hyperexcitability. Type II poisoning has typically shown severe salivation, hyperexcitability and choreoathetosis. Other signs and symptoms of toxicity include abnormal facial sensation, dizziness, headache, fatigue, vomiting, diarrhea and irritability to sound and touch. In more severe cases, pulmonary edema, muscle fasciculations, seizures and coma can develop.

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water enhance the disagreeable sensations. Sometimes the paresthetic effect is noted within minutes of exposure, but a 1-2 hour delay in appearance of symptoms is more common.^{14,16} Sensations rarely persist more than 24 hours.⁷ Little or no inflammatory reaction is apparent where the paresthesias are reported; the effect is presumed to result from pyrethroid contact with sensory nerve endings in the skin. The paresthetic reaction is not allergic in nature, though sensitization and allergic responses have been

“Other Insecticides” Neonicotinoids (Imidacloprid)

- Introduced in US market in 1990s
- Used in agriculture and for flea control
- Modified from nicotine
- Displaces ACh
 - Selective binding to insect Ach receptors
 - Consequently, less human toxicity



Neonicotinoid Toxicity

- GI effects
 - Vomiting, sore throat, abdominal pain¹
 - Ulceration throughout GI tract (solvent?)
- Excessive nicotinic receptor stimulation
 - Disorientation, agitation, weakness, LOC
- Severe poisoning
 - Rhabdomyolysis²
 - Tachycardia, progressed to v-tach, v-fib³

¹Wu IW, et al. *J Toxicol Clin Toxicol.* 2001;39(6):617-621.

²Agarwal R, et al. *Am J Emerg Med.* Sep 2007;25(7):844-845.

³Huang NC, et al. *Am J Emerg Med.* Nov 2006;24(7):883-885.

Fipronil

- Introduced to the US market in the 1990s
- Agriculture, lawn treatments, roach bait stations, household pet application
- Inhibits GABA gated Cl⁻ channels
 - Hyperexcitability of the cell
- High affinity for insects compared to mammals
 - 128x more toxic to insects than mammals



Fipronil

- Majority of cases with mild clinical effects and/or short duration
- Nausea/vomiting, dizziness, headache, abdominal pain
- Altered mental status, agitation, LOC
- Seizures
 - Usually self limiting

Mohamed F et al. *J Toxicol Clin Toxicol.* 2004;42(7):955-963.

Lee SJ et al. *Clin Toxicol* 2010;48:737-744*

*Analysis of surveillance data from 2001-2007, 103 cases

Index of Signs and
Symptoms
Starts on Page 244

Gastrointestinal Tract and Liver

Liver

Kidney

SYSTEM: GI Tract and Liver, cont.	Abdominal pain	Abdominal pain	Organophosphates N-methyl carbamates Paraquat Diquat Nicotine Metaldehyde Fluoride Borate Phosphorous Phosphides Inorganic arsenicals Cadmium compounds Copper compounds Thallium Organotin compounds Neonicotinoids	Chlorophenoxy compounds Aliphatic acids Sodium chlorate Creosote Endothall Aminopyridine Coumarins Indandiones Fumigants (ingested) Cycloheximide
	Ileus	Ileus	Thallium Diquat	Pyriminil
	Constipation	Constipation	Pyriminil	
SYSTEM: Liver	Enlargement	Enlargement	Copper compounds Sodium chlorate Phosphine Carbon tetrachloride Chloroform	Inorganic arsenicals Hexachlorobenzene
	Jaundice (see section on Skin)	Jaundice (see section on Skin)		
M: Kidney	SYMPTOMS/SIGNS/DISEASE CATEGORIES	Proteinuria/hematuria and acute renal failure	Inorganic arsenicals Copper compounds Sodium fluoride Naphthalene Borate Nitrophenols	Cadmium compounds Phosphorus Phosphides Phosphine Chlorophenoxy compounds

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Pesticides Known to Cause Seizures/Tremors

- Insecticides
 - Organochlorines, organophosphates, pyrethroids, nicotine, fipronil
- Rodenticides
 - Strychnine, sodium fluoroacetate, thallium, Al- and Zn phosphide
- Herbicides
 - Diquat, chlorophenoxy compounds (2,4-D)
- Fumigants
 - Cyanide, carbon disulfide, acrylonitrile, methyl bromide

Which pesticide is most commonly reported to the Poison Control Center?



Report of Poison Control Centers' National Poison Data System

Pesticide	Average 2001-2003			2014		
	Total	Mod-Severe Morbidity	Death	Total	Mod-Severe Morbidity	Death
Pyrethroids	17,589	778	1	28,362	878	1
Organophosphates	9,501	567	8	2,921	159	3
Carbamates	3,750	167	1	1,661	73	1
Strychnine	134	17	1	66	9	1
Paraquat	77	12	3	90	8	1
Boric Acid	-	-	-	6,071	12	0

Mowery JR, Spyker DA, et al. *Clinical Toxicology* 2015; 53(10):962-1147

Data Collection of an Acutely Exposed Patient

Acute Pesticide Exposures Clinical Guidelines

III. Data Collection on an Acute Pesticide Exposed Patient

See Evaluation Pesticide Exposure form

1. Patient identification: Name/Age/Sex/Occupation
2. Place of employment
3. Initial and subsequent symptoms and signs*
4. Name of pesticide product including active ingredients, their concentration and EPA registration number
5. Date, time and location when over-exposure occurred
6. How the pesticide was applied, when applied and on what crop or for what use
7. Route(s) of exposure: dermal, ocular, oral, respiratory
8. How much of the product was ingested, if ingested
9. Circumstances of exposure-intentional or accidental, occupational or non-occupational
10. A detailed description of how the exposure happened
11. Others affected or witnessing incident (at work site, home, etc.)
12. If female, assess pregnancy status
13. Treatment already received
 - a. Skin exposure:
 - Was affected area washed? If so, when? If not, proceed with skin decontamination procedures
 - Was any clothing contaminated?
 - If so did they change clothes?
 - b. Ocular exposure:
 - Were the eyes irrigated?
 - If so, with what and for how long?

THE PESTICIDE EXPOSURES CLINIC

HOSPITAL TOXICOLOGY NETWORK
MEN

Effective Date: _____
 Revision Date: _____
 Approved By: _____

Acute Pesticide Exposures Clinical Guidelines

INTRODUCTION

PURPOSE

DEFINITIONS

PROCEDURE

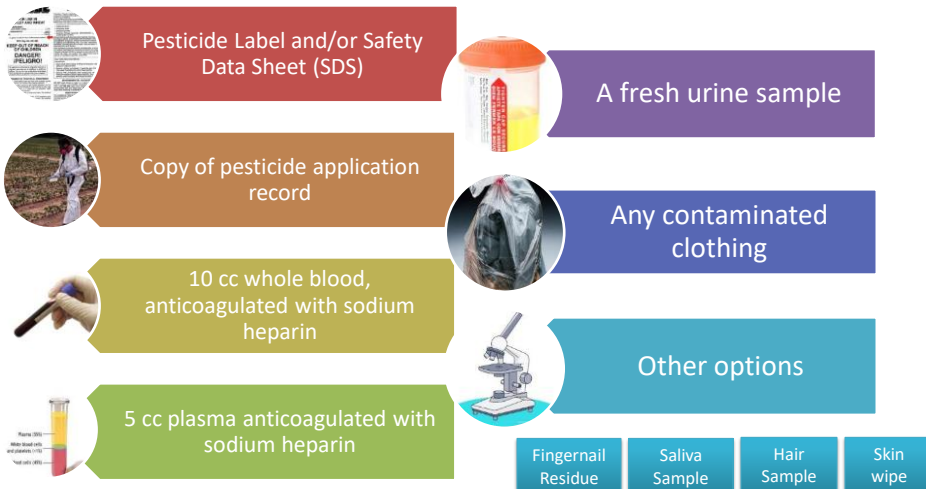
I. Crisis Response

1. Patient response and provider with gloves, ambu bag and respiration if needed.
2. Provide immediate first aid measure: decontaminate, breathing, and circulation.
3. Decontaminate and decontaminate if needed.
4. Identify patient and take patient's vitals.
5. Take records of actions and patient care. See Pesticide Exposure Assessment form (See Resources, esp. 5).
6. Identify chemical, location and exposure.
7. Establish chain of command.
8. Set up triage area: stabilize, monitor and evacuate.
9. Abuse and transport.
10. Consult with specialists: Toxicologist and/or Occupational and Environmental Medicine Specialists.
11. Coordinate transportation of suspected patient and bring it back to the nearest of relevant patient after discharge from hospital.

MEN
Hospital Toxiconet Network

ACUTE PESTICIDE EXPOSURE CLINICAL GUIDELINES

Data Collection on an Acute Pesticide Exposed Patient



Are we
done yet?



Photo © earldotter.com

I am required to report the following:



About **Explore** Connect

Home » Environmental and Occupational Health » Pesticide Reporting and Workers' Compensation

CONTINUING EDUCATION
 Upcoming Webinars
 Archived Webinars
 Online Courses

UNDERSTANDING MIGRATION
 Migrant Health Overview
 Migrant/Seasonal Farmworkers Profile

ISSUES IN MIGRANT HEALTH
 Behavioral Health
 Cancer
 Children's Health
 Diabetes
 Eye Care
 Family Violence

Report Pesticide Exposures

PESTICIDE REPORTING REQUIREMENT:
 Required Optional None

Pesticide Reporting and Workers' Compensation Information for Hawaii Close or Esc Ke

Report Exposure Workers' Compensation

[edit] Report Exposure: Department of Health, Office of Hazard Evaluation and Emergency Response
 Phone: 1-800-222-1222 (Hawaii Poison Control Center)
 Report Online: <http://eha-web.doh.hawaii.gov/eha-cma/Leaders/HEER/Pesticide-Illness-Reporting>

Pesticide Reporting Requirements
 Required to Report: Yes
 What to Report?: Any Pesticide-Related Exposure
 Timeframe to Report Injury or Exposure: 24 Hours
 Who is Required to Report?: Physicians, Laboratories.
 Additional Info:

For reporting purposes, pesticide and heavy metal poisoning include acute poisoning or any subacute illness caused by, or believed to be caused by, these toxic agents. Alternative Method: 1. Report by phone to the Hawaii Department of Health 1 (808) 586-4249 on O'ahu, or 1-800-468-4644 ext 64249 # from the Neighbor Islands. 2. Fill out from from web site and mail to the Hawaii Department of Health.

WPS Enforcement
 Worker Protection Standard Enforcement Agency:
 Department of Agriculture: <http://hawaii.gov/hdoa/pi/pest>

required Optional None

Workers Compensation

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- Medical treatment for injured patient
 - Immediate
 - Long term
 - Access to Specialty Care
- Wages
- Return to Work
- Prevention - Hazard Control
- Public Health
 - Surveillance

When is illness or injury work related?

Any injury or illness resulting from or sustained in the course of any occupation or employment.

More than 50% likely due to work

USE THESE WORDS:

- “More likely than not” due to work
- Work “most likely” cause of the condition
- “But for the work” the condition would not exist

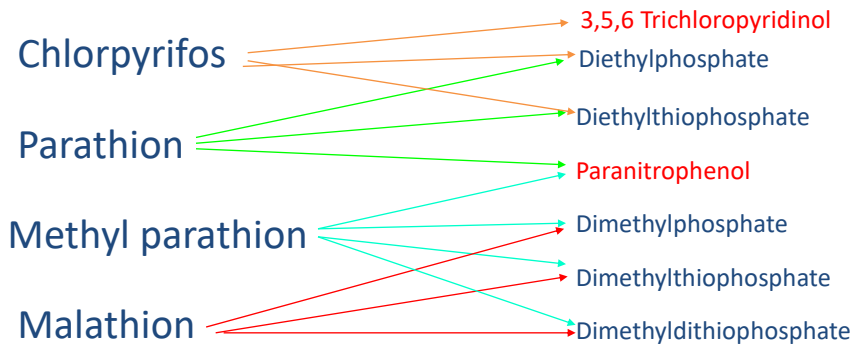
Biomonitoring Data
from CDC

Urinary Pesticide Metabolites in Children

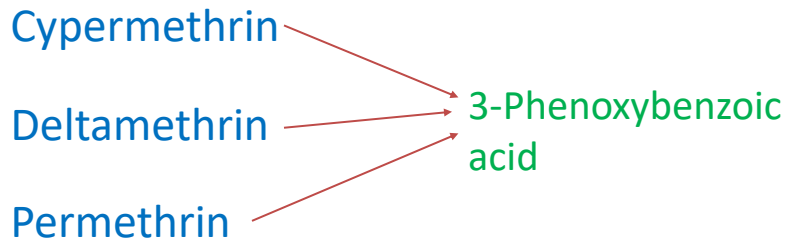
2001-2002 National Health and Nutrition Examination Survey (NHANES)

- Pesticide usage questions
 - Use of pesticide in the last month
 - Performed by professional or non-professional
- Dietary questions
 - Intake of types of foods (greens, dried beans)
- Lab data of urinary metabolites of OP and pyrethroid insecticides
 - Age for survey variables limited to 6 -18 years to maintain consistency with lab data

Organophosphate Metabolites (Found in children's urine)



Pyrethroid Metabolites (Found in children's urine)



Conclusions

- Urinary metabolites of pesticides are higher when:
 - Pesticides have been applied in the past month
 - When applied by a non professional
 - Are applied more often
- Poverty and black race associated with higher organophosphate levels



Chapter 21- Chronic Effects



- Increasing information about Chronic effects
- Neurodevelopmental
 - Growing body of solid longitudinal studies
 - Insecticides (OP) affect memory, cognitive development, reasoning, and IQ
- Birth Defects
 - Some evidence to suggest association
- Cancer
 - Childhood ALL
 - Prostate cancer and NHL



Pesticides and Childhood Cancer

- Leukemia and brain tumors have been noted in many epidemiological studies to be associated with pesticides
- Risk factors
 - parental occupational exposure
 - family use-- pest strips, termite treatment, flea collars for pets
 - Parental exposure to pesticides BEFORE and DURING pregnancy
- Multiple studies
 - Two important review articles

Zahm S and Ward M. *Env Health Persp* 1998;106:893-908.
Infante-Rivard C & Weichenthal S. *J Tox Environ Health* 2007;10:81-99.

Adult Cancers Hodgkins and NHL

- 23 of 27 studies with positive associations¹
 - Half of studies in farm worker populations
 - Multiple classes of pesticides
- Separate meta-analysis case control studies²
 - NHL OR= 1.35, 95% CI, 1.2-1.5
 - Leukemia OR= 1.35, 95% CI, 0.9-1.2
 - Multiple myeloma OR= 1.16, 95% CI, 0.99-1.36

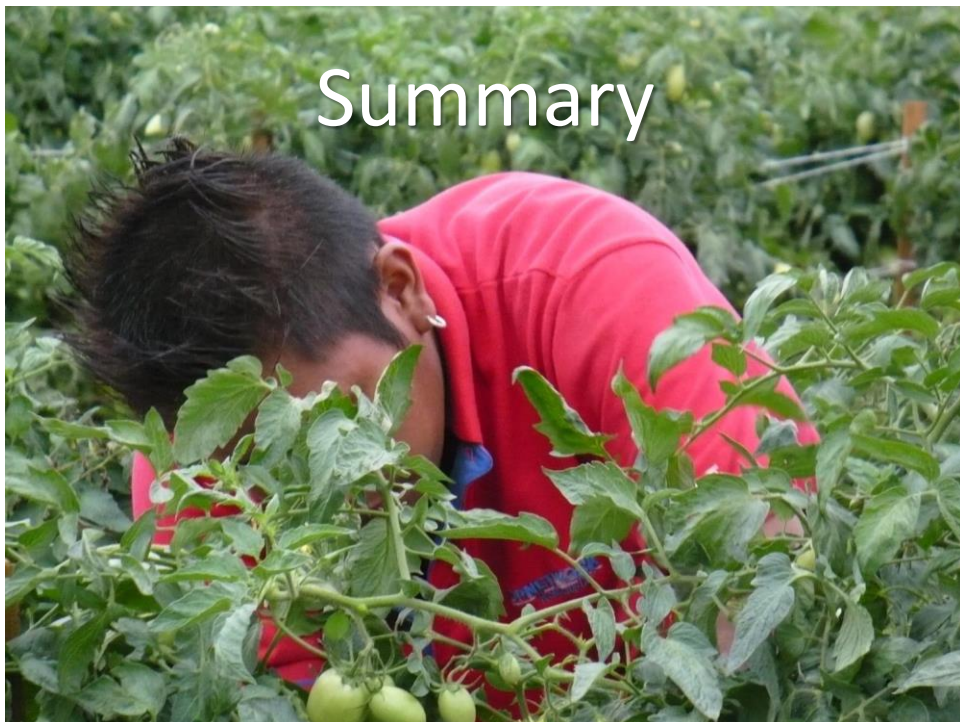


¹Bassil KL, *Can Fam Physician*. Oct 2007;53(10):1704-1711
²Merhi M, *Cancer Causes Control*. Dec 2007;18(10):1209-1226

So what do we do?



- ✓ Recognize your patient's occupation
- ✓ Recognize and treat acute poisoning
- ✓ Report
- ✓ Promote primary prevention
- ✓ Ask about take home exposures
- ✓ Become involved in local/state/federal policy



- Pesticides may have both Acute and/or Chronic effects
 - Higher short term exposure most often associated with Acute effects
 - Chronic effects may occur as late occurrence following a high exposure, or sub-acute exposure
- Acute effects may often be non-specific
 - Helpful patterns or unique symptoms
 - Needs a high index of suspicion



Resources www.migrantclinician.org

Contact



Amy K. Liebman, MPA, MA

Director, Environmental &
Occupational Health

(512) 579-4535

aliebman@migrantclinician.org

James Roberts, MD, MPH

Professor of Pediatrics
Medical University of South
Carolina

(843) 876-8512

robertsj@musc.edu