Intoxication by agricultural chemical poisonings

KVASNITSKA O.S.

Definition

- **Pesticides** (Latin pestis is a plague, contagion, caedere – to kill) are chemical matters which are used in agriculture for a fight against diseases and pests of cultural plants and destroying weeds. Application of such matters is basis for the increase of the productivity, at the same time the wide use of pesticides, in agriculture, constantly multiplies the contingent of persons which contact with them

Where Are Pesticides Used?

- Forests to control insects and under-story vegetation;
- Landscapes, parks, and recreational areas to control weeds, insects, and disease pests;
- Rights-of-way along railroads and under electric wires to control vegetation;
- Houses, schools, and commercial and office buildings to control insects, rodents, and fungi;
- Boat hulls to control fouling organisms;

Where Are Pesticides Used?

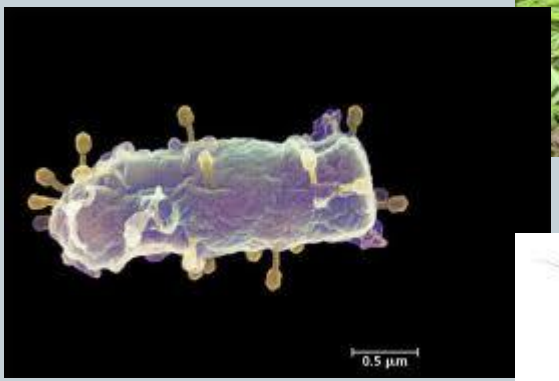
- Aquatic sites to control mosquitoes and weeds
- Wood products to control wood-destroying organisms
- Food preparation areas to control insects and rodents
- Human skin to kill or repel insects
- Household pets to control fleas and ticks
- Livestock to control insects and other pests.

Main groups of pesticides

- 1. **Insecticides** – substances which are used for a fight against insects
- 2. **Fungicides** – for treating of plants from mycotic diseases
- 3. **Defoliants** – preparations which are used for the delete of leaves of plants

Main groups of pesticides

4. **Herbicides** – for destroying weeds
5. **Bactericides** – against bacteria
6. **Acaracides** – for destroying of the mites
7. **Rodenticides** – against rodents
8. **Ovicides** – against larvae and caterpillar



Classification of pesticides according the chemical structure:

- 1. **Chlorine organic connections** (chloridan, heptachlor, chlorten, polychlorpinen).
- 2. **Phosphorus organic connections** (karbofos, chlorofos, metaphos, thiophos).
- 3. **Mercury organic connections** (granosan, mercuran, mercur- gexan).
- 4. **Connections of arsenic** (arsenat sodium, arsenat calcium, parisian greenery).
- 5. **Derivates of carbamic acid** (bethanol, carbin, sevin and other).

Classification of pesticides according the chemical structure:

- 6. **Cyanides** (cyanic acid, cyanamid of calcium).
- 7. **Preparations of copper** (burgundy liquid, blue vitriol).
- 8. **Sulphur and its connections** (colloid sulphur, sulphuric anhydride, ground sulphur).
- 9. **Preparations of vegetable origin** (anabasine, nicotine, piretrum).

Agriculture Pesticide Applications

Aerial

Boom sprayer

Enclosed cab

Air blast sprayer

Backpack wand

Agriculture Jobs

Orchard thinner

Mixer loader

Flagger

Picker

90% of pesticides used today are synthetic

ROUTES OF EXPOSURE

OP's are readily absorbed:

Across the SKIN with skin contact

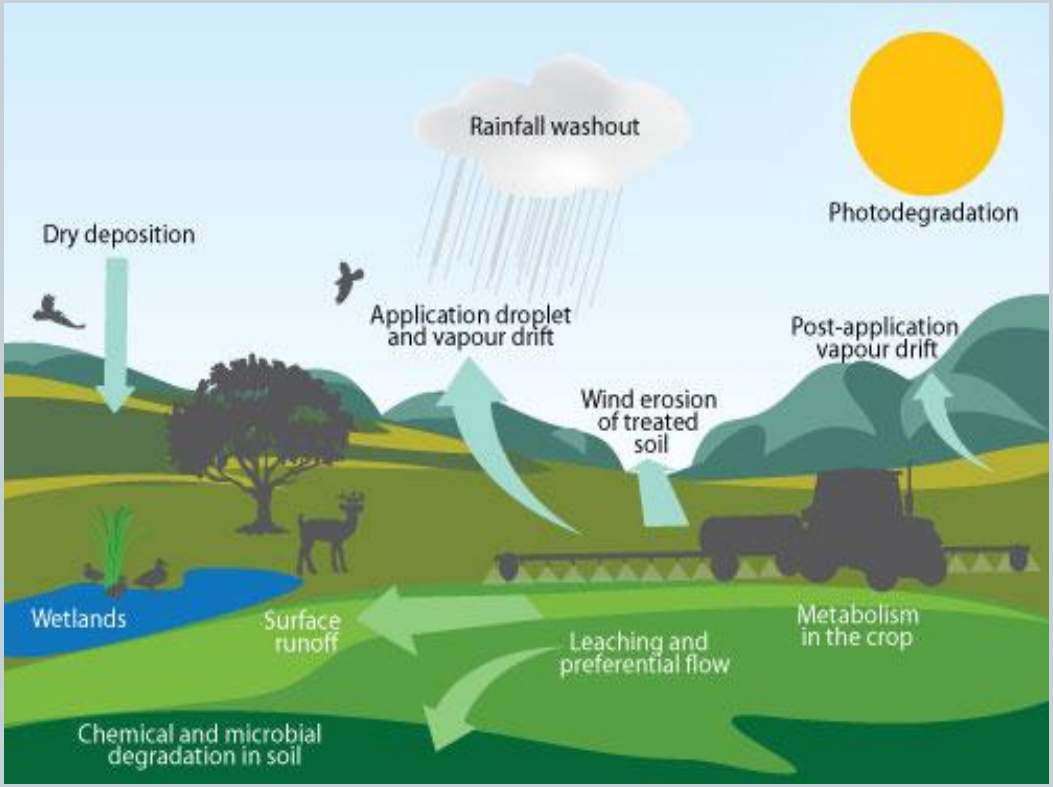
In the lungs with INHALATION of pesticide contaminated air/dust

In the gut by INGESTION of pesticide residue on food/dirt/dust

Source: EPA Protect Yourself from Pesticides-Guide of Agricultural Workers

The pesticide cycle

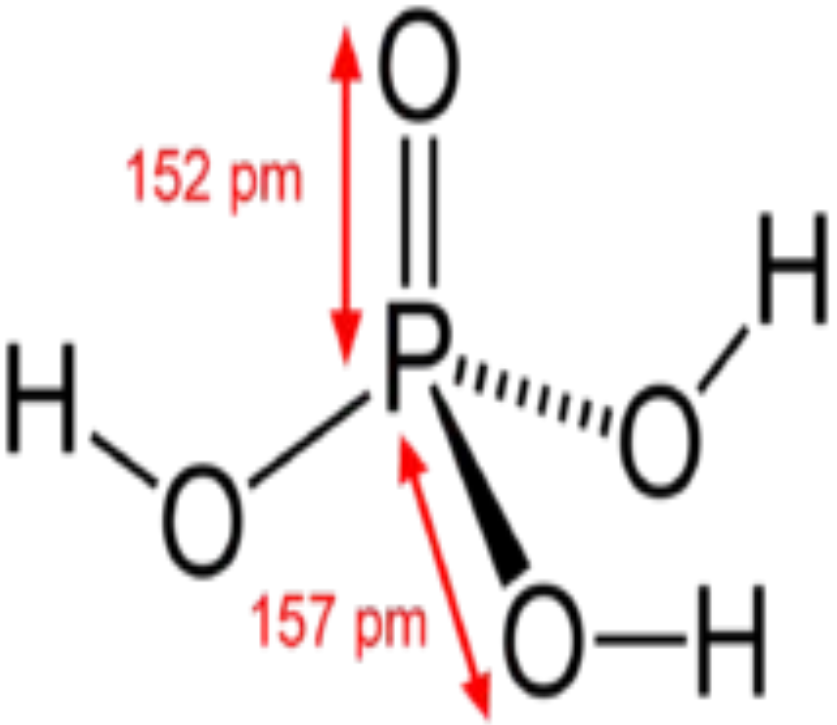
Pesticide use has helped increase agricultural productivity, pesticides may move from agricultural land into the broader environment, thus contributing to environmental contamination of surface and ground waters



Processes involved in the movement of pesticides from the site of application (Cessna et al. 2005)

Intoxication by phosphorus organic connections.

Organophosphate poisoning



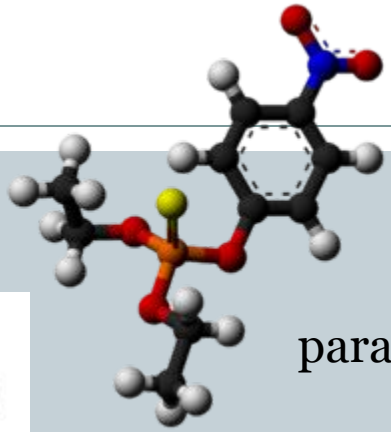
http://en.wikipedia.org/wiki/Organophosphate_poisoning

Organophosphates are used in:

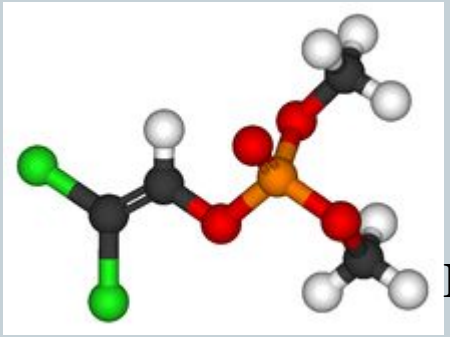
- Pesticides sprayed and dusted onto cereals, fruit and vegetables
- De-wormers and systemic ‘pour-ons’ applied to farm animals
- Fly sprays and vaporizing strips used in industrial, commercial and domestic premises
- Flea collars and treatment for pests
- Anti-lice shampoo

Chemical names for organophosphates active ingridients

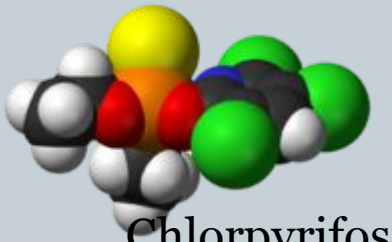
- Methyl parathion
- Ethyl parathion
- Malathion
- Diazinon
- Fenthion
- Dichlorvos
- Chlorpyrifos
- Trichlorfon



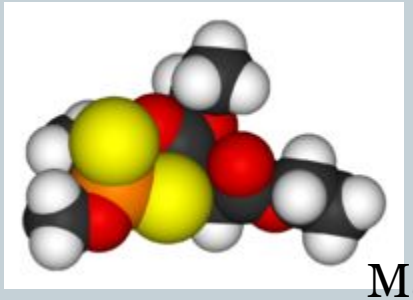
parathion



Dichlorvos



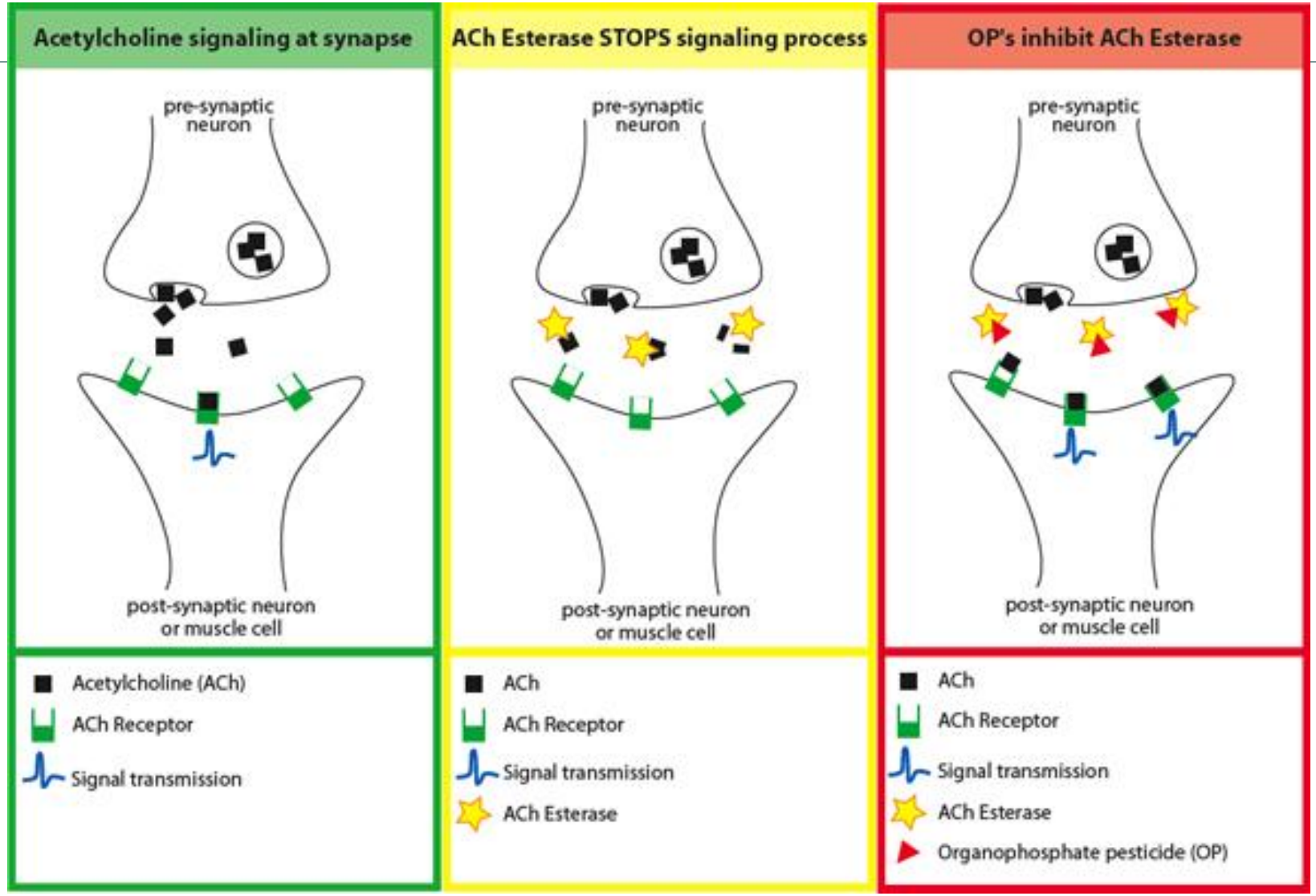
Chlorpyrifos



Malathion

http://en.wikipedia.org/wiki/Organophosphate_poisoning

Pathophysiology

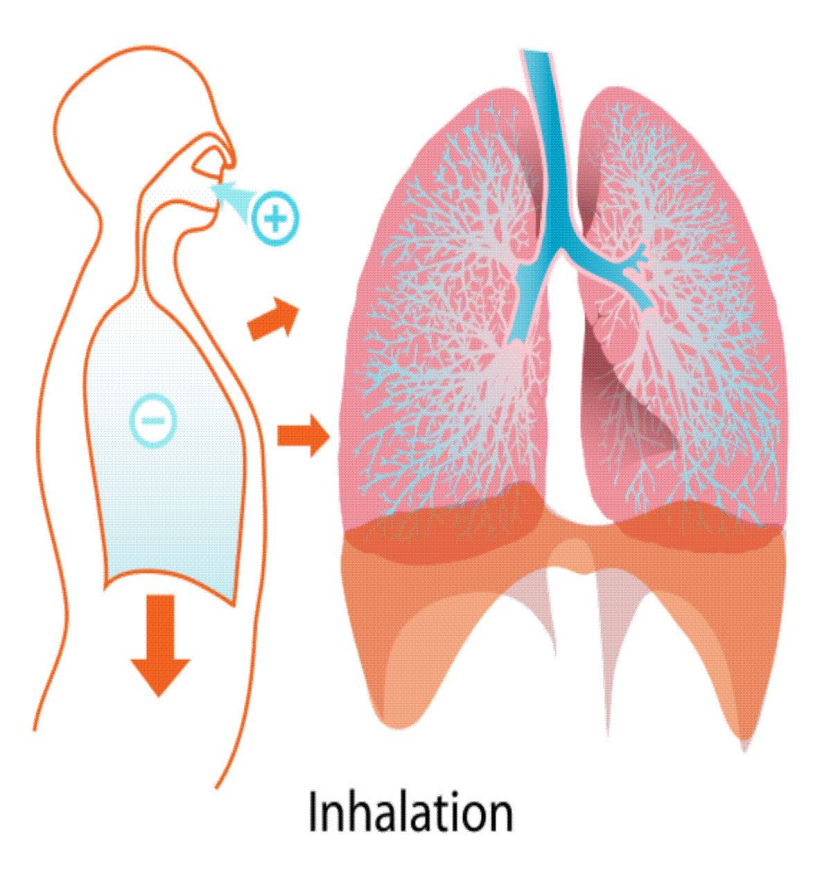


2007 Pediatric Environmental Health Specialty Unit (PEHSU), Department of Environmental & Occupational Health Sciences.University of Washington opchild@u.washington.edu

Common causes of OP poisoning

- **Inhalation**

The agricultural use without adequate protection. Airborne inhalation during application of pesticides to pets or household surfaces and carpets in unventilated areas. Even handling of flea collars for pets may adversely affect a person (sprays or flea collars)



http://trialx.com/curebyte/2012/10/23/inhalation-photos-and-related-clinical-trials/

Common causes of OP poisoning

- **Ingestion**

Consumption of domestic drinking water stored in contaminated, discarded poison containers

Consumption of fruit and vegetables that have been treated with pesticides, and not washed properly

http://toolboxes.flexiblelearning.net.au/demosites/series3/315/resources/ohs/hazards/08hazardoussubstances.htm

Common causes of OP poisoning

- **Absorption and ingestion**

Failure to wash hands after handling pesticides or pet flea and tick control products

http://nasdonline.org/document/196/Fact7/d000145/preventing-agricultural-chemical-exposure-a-safety-program-manual.html

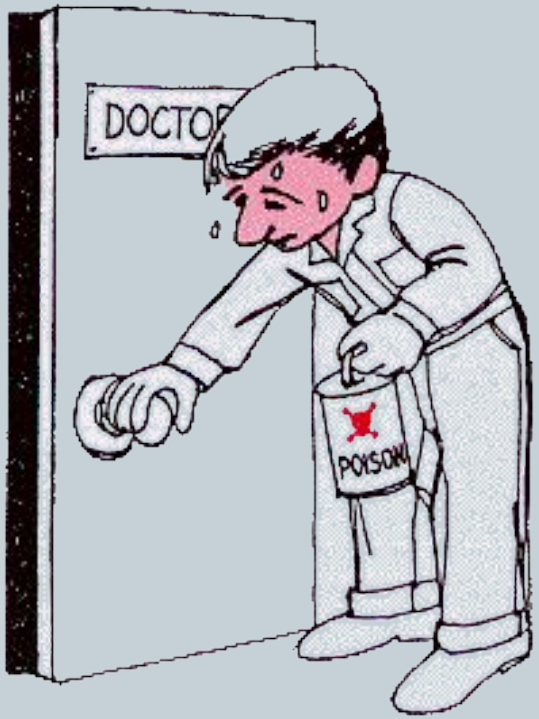
Clinical picture

Symptoms of acute OP poisoning develop during or after exposure, within minutes to hours, depending on the method of contact. Exposure due to inhalation results in the fastest appearance to toxic symptoms, followed by the gastrointestinal route and, finally, the dermal route.

http://blog.ecosmart.com/index.php/2008/09/19/the-history-of-pesticides/

Commonly reported early symptoms

- Headache
- Nausea
- Dizziness
- Hypersecretion (sweating and salivation)
- Muscle twitching
- Weakness
- Tremors
- In coordination
- Vomiting
- Abdominal cramps
- Diarrhea
- Paralysis



http://www.extension.org/pages/17854/symptoms-of-pesticide-poisoning

Clinical picture

- Basic symptoms of the acute poisoning by phosphorus organic pesticides are owing to muscarinic action, nicotinic action and by the central action of acetilcholine.

Muscarinic effects
(result of excitation of M- cholinoreceptions)

- Increased contractions of smooth muscle: GI tract and ureters
- Increased secretions of gland cells: lacrimal, sweet, salivary, gastric, intestinal, pancreatic
- Bradicardia
- Bronchoconstriction
- Miosis: constricted pupils

http://www.extension.org/pages/17854/symptoms-of-pesticide-poisoning

Nicotinic effects (excitation of M- cholinoreceptions and defect of striated muscles)

- Muscle weakness
- Fasciculations: small, local contractions of muscles visible through the skin, representing a spontaneous discharge of a number of fibers innervated by a single motor nerve filament
- Areflexia: absence of reflexes
- Paralysis
- Hypertension
- Tachycardia: rapid heart rate, >100 beats per min

CNS Effects

(toxic influence of acetilcholine on the cortex of cerebrum and medulla)

- Confusion
- Seizures
- Oppression and paralysis of vitally important centers of medulla

The types and severity of cholinesterase inhibition symptoms depend on:

- Toxicity of pesticide
- Amount of pesticide involved in the exposure
- Route of exposure (inhalation is fastest, followed by ingestion, then dermal)
- Duration of exposure

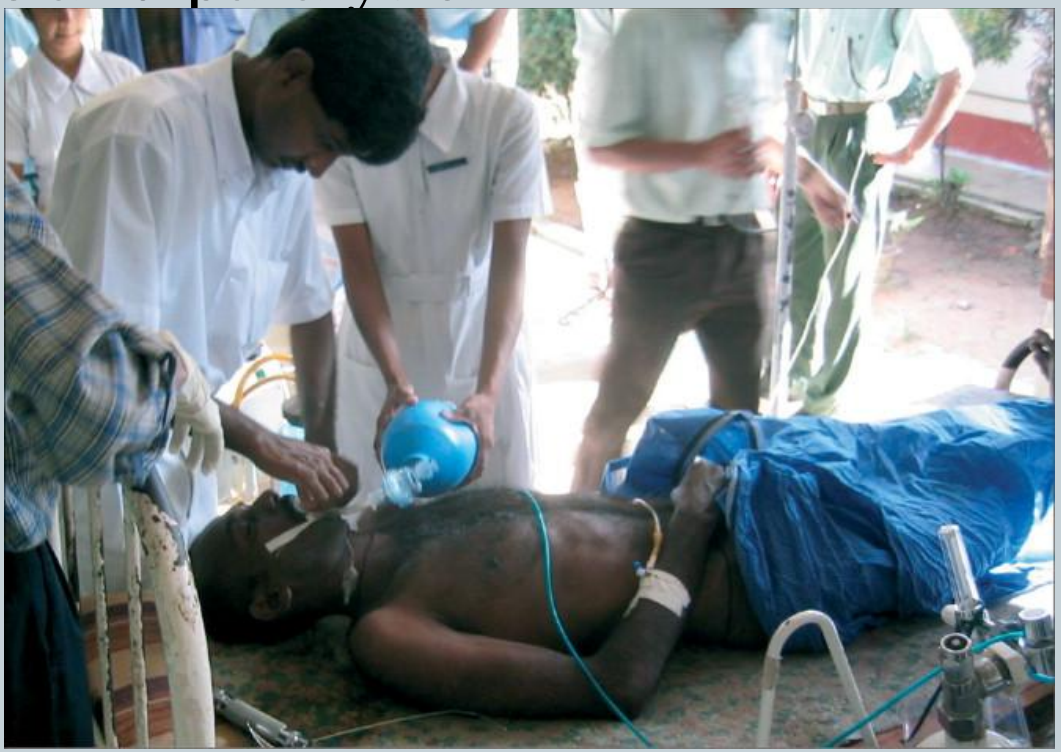
The easy form of acute intoxication

- - tachycardia which later changes on bradycardia, and raises the arterial blood pressure;
- - the decrease of cholinesterase is marked in blood;
- - a disease at the easy form of motion is finished, as a rule, by convalescence.

At middle degree of severity of acute intoxication

- to the symptoms of previous stage addition;
- - a fever with increase of temperature of body to 40 °C, excitation which later changes for depression, feeling of fear, appears inadequate reaction on external irritants;
- - headache increases, appears expressed salivation and tearing, hyperhidrosis, a muscle weakness grows;
- - violation of breathing shows up by hard inhalation and exhalation, with mass of dry whistling and moist large vesicles, little vesicles and vesicular rales;
- - appear the signs of oxygen insufficiency, tachycardia which changes on bradycardia, decrease of arterial blood pressure, a heart is extended, tones are quiet;

- **The heavy (comatose) form of intoxication** meets rarely, sometimes it finished lethally. In the clinic of heavy form distinguish three stages: excitation, convulsive and paralytic.



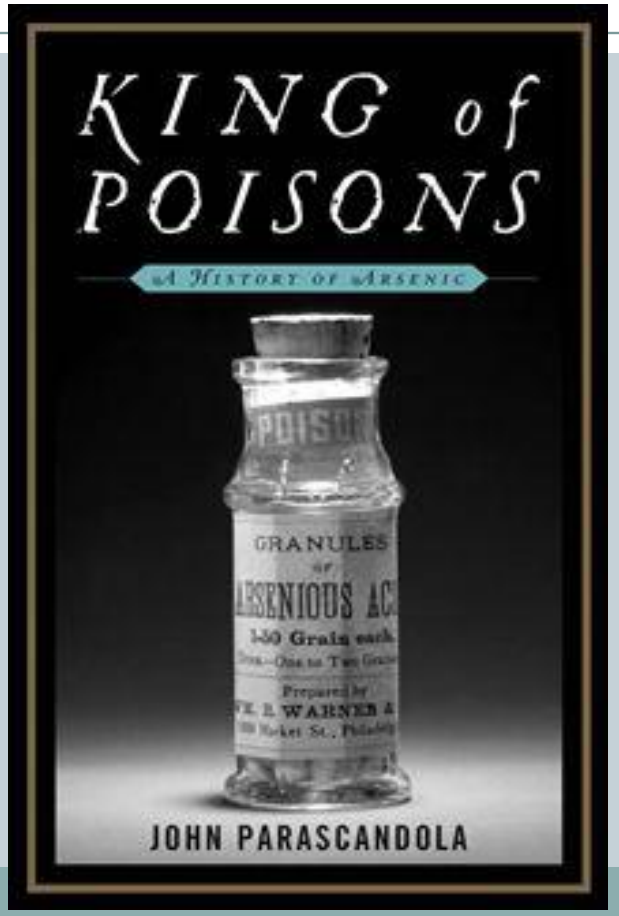
Management of a patient with severe organophosphorus poisoning in a Sri Lankan district hospital.
www.thelancet.com Vol 371 February 16, 2008

- **Chronic poisonings** by phosphorus organic connections it is needed to differentiate with astenovegetative neuroses, myocardial dystrophy. By an important laboratory index which confirms the diagnosis of acute intoxication there is decrease of activity of cholinesterase to 50 % and anymore.

Treatment

- Antidote therapy - cholinolitics and reactivates of cholinesterase: at the easy form of intoxication intramuscular enter 1-2 ml of 0,1 % to solution of atropine; at middle and heavy degrees intoxications intensive atropinisation is conducted. Once intramuscular enter 3-5 ml of 0,1 % solution of atropine, and then pass introduction of atropine to supporting. Injections repeat oneself each 5-6 minutes to stopping of muskarinic symptoms and appearance of signs of overdose of atropine (dryness of mycoses, expansion of pupils).
- Respiratory support is given as necessary. Gastric decontamination should be considered only after the patient has been fully resuscitated and stabilised. Patients must be carefully observed after stabilisation for changes in atropine needs, worsening respiratory function because of intermediate syndrome, and recurrent cholinergic features occuring with fat-soluble organophosphorus

Intoxication by arsenic connections



Arsenic (As)

- **Chemistry:**
  - extremely complex because it can exist in metallic form, can be in trivalent and pentavalent state (charge of 3+ or 5+), and can be organic or inorganic
  - widely distributed in nature (variety of forms)

- **Environmental fate**:
  - found in surface and groundwater through runoff
  - accumulates in plants if soil conditions are right
  - bioaccumulates in aquatic ecosystems (so fish consumption is a source)

From: Klaassen et al., Chap. 19, Philp, Chap. 6

Sources of As

- smelting of gold, silver, copper, lead and zinc ores
- combustion of fossil fuels
- agricultural uses as herbicides and fungicides, as insecticides for staining of seed, destroying the pests of garden cultures, rice fields, malarial mosquito maggots and for a fight against rodents
- cigarette smoke
- occupational: largest source is manufacture of pesticides and herbicides

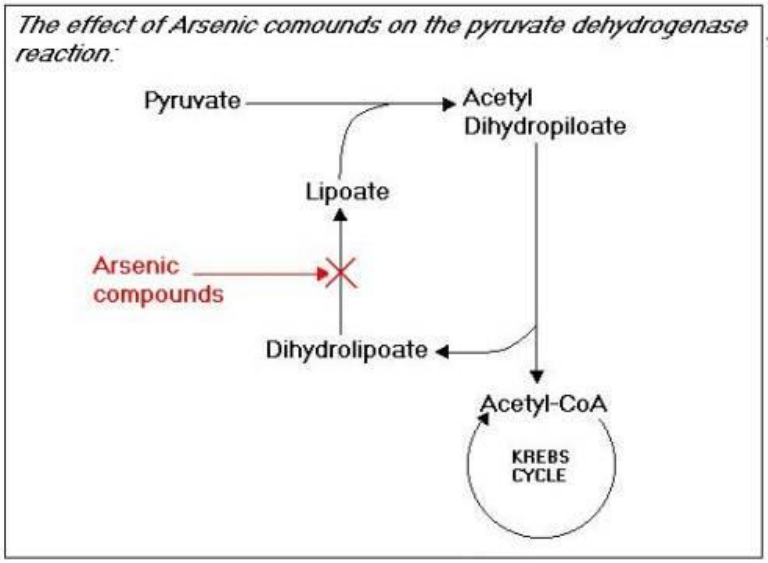
Arsenic (As)

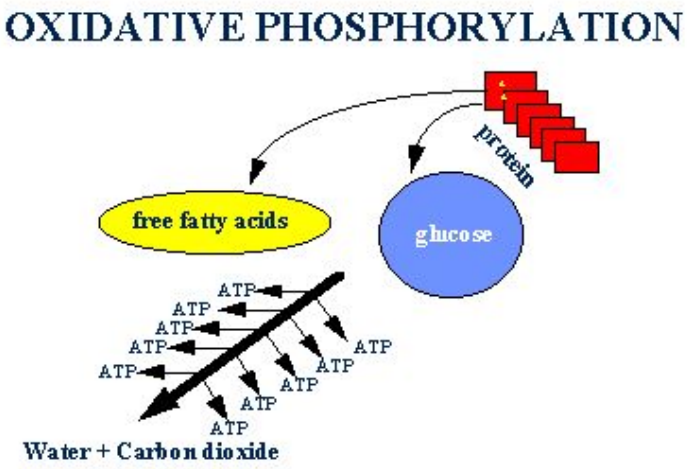
- pharmacokinetics and dynamics:
  - absorbed via inhalation, ingestion and dermal exposure
  - mimics phosphate in terms of uptake by cells
  - Detoxified by methylation: decreased rates lead to increased toxicity (individual susceptibility)
  - Can cross placenta
  - accumulates in liver, kidney, heart and lung - later in bones, teeth, hair, etc.
  - half-life is 10 hr, excretion via kidneys

From: Klaassen et al., Chap. 19, Philp, Chap. 6

Arsenic Toxicity Mechanisms

- **binds to sulfhydryl groups (and disulfide groups), disrupts sulfhydryl-containing enzymes (As (III))**
  - inhibits pyruvate and succinate oxidation pathways and the tricarboxylic acid cycle, causing impaired gluconeogenesis, and redu ce oxidative phosphorylation
    - targets ubiquitous enzyme reactions, so affects nearly all organ systems
- **substitution for phosphorus in biochemical reactions**
  - Replacing the stable phosphorus anion in phosphate with the less stable As(V) anion leads to rapid hydrolysis of high-energy bonds in compounds such as ATP. That leads to loss of high-energy phosphate bonds and effectively "uncouples" oxidative phosphorylation.





The catarrhal form of acute intoxication

- appear from the hit of the aerosol of arsenic on the mycoses of eyes and breathing organs.
- - appearance of weakness, dizziness, nausea, vomit, by sweetish taste in a mouse, feeling of fear, shaking, and painful cramps;
- - there are an irritation and sharp hyperemia of mucosas of overhead respiratory tracts and eyes that shows up burning of eyes, tearing, cold, sneezing, edema of mucus of nose, cough, sometimes with hemoptysis and pain in thorax;
- - the signs of heart insufficiency, astenovegetative syndrome, and also symptoms of defect of gastrointestinal tract, appear later.

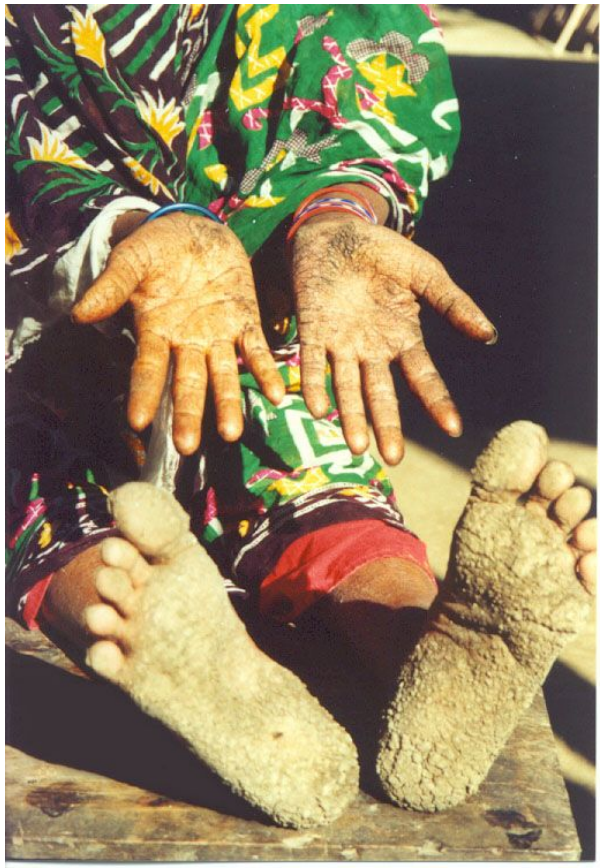
Gastrointestinal form

- at the casual hit of poison in a gastrointestinal tract.
- metallic taste appears in a mouth, dryness, swallowing, incessant vomit (the masses of vomits have a garlic smell), acute abdomen pain, diarrhea.
- the amount of urine diminishes;
- the loss of liquid conduces to acute dehydration of organism;
- an acute weakness, dizziness, develops, sometimes fainting fit, decrease the temperature of body and arterial blood pressure goes down, the collapse state develops;

Chronic intoxication

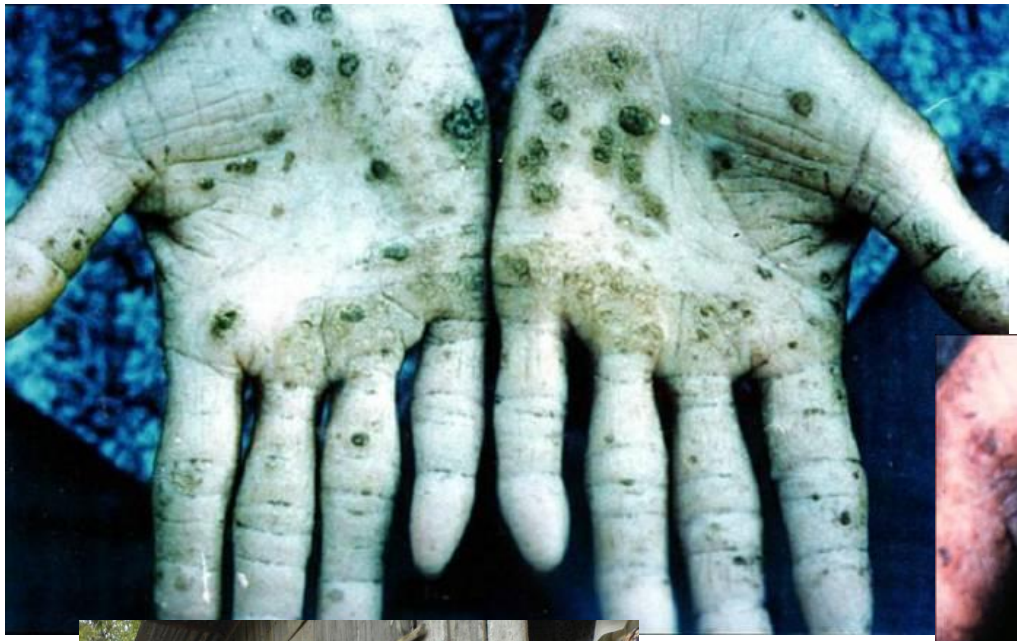
- meets in persons, which long time contact in the terms of productions with pair or dust of connections of arsenic, which get to the organism through respiratory tracts or skin.
- absence of appetite, hypersalivation, periodic nausea and vomit, stomach pain, violation of stool;
- pains in a nose and throat, hoarseness, cough, cold, nose-bleeds, rhinitis, tracheitis, bronchitis;
- rush appears on a skin, ulcers and psilosis;
- heavy violations of metabolism result in considerable weight loss, defect of liver, kidneys, appearance of anemia.

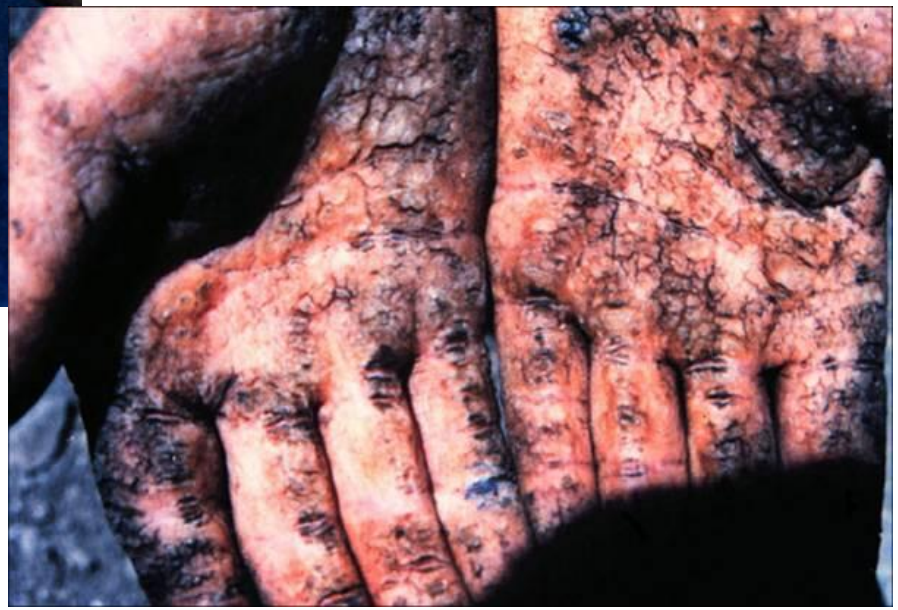
Arsenic poisoning



Typical findings are **skin and nail changes**, such as **hyperkeratosis, hyperpigmentation, exfoliative dermatitis**, and **Mees' lines** (transverse white striae of the fingernails); **sensory and motor polyneuritis** manifesting as numbness and tingling in a "stocking-glove" distribution, distal weakness, and quadriplegia; and inflammation of the respiratory mucosa.Epidemiologic evidence has linked chronic consumption of water containing arsenic at concentrations in the range of 10 to 1820 ppb with vasospasm and peripheral vascular insufficiency culminating in "**blackfoot disease -** a gangrenous condition affecting the extremities.Chronic arsenic exposure has also been associated with a greatly**elevated risk of skin cancer** and possibly of cancers of the lung, liver (angiosarcoma), bladder, kidney, and colon

http://manbir-online.com/diseases/arsenic.htm





http://manbir-online.com/diseases/arsenic.htm

Diagnostic criteria of Chronic arsenicosis.

1. At least 6 months exposure to arsenic levels of greater than 50 mg/L or exposure of high arsenic level from food and air.
2. Dermatological features characteristic of chronic arsenicosis.
3. Non carcinomatous manifestations : Weakness, chronic lung disease, non cirrhotic portal fibrosis of liver with/without portal hypertension, peripheral neuropathy, peripheral vascular disease, non pitting edema of feet/ hand.
4. Cancers : Bowens disease, Squamous cell carcinoma, Basal cell carcinoma at multiple sites, occurring in unexposed parts of the body.
5. Arsenic level in hair and nail above 1 mg/kg and 1.08 mg/kg respectively and/or arsenic level in urine, above 50 mg/L (without any history of taking seafood).

http://www.who.int/water_sanitation_health/dwq/arsenicun4.pdf

Guha Mazumder , (In press)

Dermatological criteria and grading of severity of chronic arsenic toxicity

Grade I	**Mild**	**a) Diffuse melanosis.** **b) Suspicious spotty depigmentation / pigmentation over trunk /limbs.** **c) Mild diffuse thickening of soles and palms**
Grade II	Moderate	a) Definite spotty pigmentation / depigmentation on the trunk and limbs, bilaterally distributed. b) Severe diffuse thickening (with/without wart like nodules of the palms and soles)
Grade III	Severe	a) Definite spotty pigmentation/depigmentation as above with few blotchy pigmented/depigmented macular patches over trunks or limbs. b) Pigmentation involving the undersurface of tongue and/or buccal mucosa. c) Larger nodules over thickened palms and soles occasionally over dorsal aspect of hands and feet. Diffuse verrucous lesions of the soles with cracks and fissures and keratotic horns over palms/soles.

http://www.who.int/water_sanitation_health/dwq/arsenicun4.pdf

Guha Mazumder , (In press)

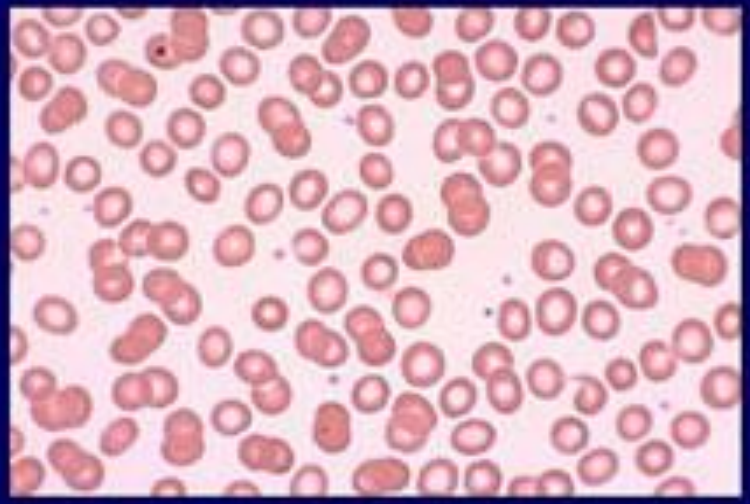
LABORATORY FINDINGS

- When acute arsenic poisoning is suspected, **an x-ray** of the abdomen may reveal ingested arsenic, which is radiopaque. The **serum arsenic level** may exceed 0.9 umol/L (7 ug/dL); however, arsenic is rapidly cleared from the blood. **Electrocardiographic** findings may include QRS complex broadening, QT prolongation, ST-segment depression, T-wave flattening, and multifocal ventricular tachycardia. **Urinary arsenic** should be measured in 24-h specimens collected after 48 h of abstinence from seafood ingestion; normally, levels of total urinary arsenic excretion are less than 0.67 umol/d (50 ug/d).Arsenic may be detected in the hair and nails for months after exposure.Abnormal liver function, anemia, leukocytosis or leukopenia, proteinuria, and hematuria may be detected.Electromyography may reveal features similar to those of Guillain-Barre syndrome.

Laboratory Findings

CBC: pancytopenia; basophilic stippling* may be seen on peripheral smear

arsenic poisoning



normal red blood cells

* Also seen in lead poisoning

CDC ATSDR

Treatment

- Vomiting should be induced in the alert patient with acute arsenic ingestion.
- Gastric lavage may be useful; activated charcoal with a cathartic (such as sorbitol) may be tried.
- Aggressive therapy with intravenous fluid and electrolyte replacement in an intensive-care setting may be life-saving.
- **Dimercaprol** is the chelating agent of choice and is administered intramuscularly at an initial dose of 3 to 5 mg/kg on the following schedule: every 4 hr for 2 days, every 6 hr on the third day, and every 12 hr thereafter for 10 days. (An oral chelating agent may be substituted). **Succimer** is sometimes an effective alternative, particularly if adverse reactions to dimercaprol develop (such as nausea, vomiting, headache, increased blood pressure, and convulsions). In cases of renal failure, doses should be adjusted carefully, and **hemodialysis** may be needed to remove the chelating agent-arsenic complex. Arsine gas poisoning should be treated supportively with the goals of maintaining renal function and circulating red-cell mass.

Intoxication by chlorine organic connections.

- Chlorinated hydrocarbon (organochlorine) insecticides, solvents, and fumigants are widely used around the world. This class comprises a variety of compounds containing carbon, hydrogen, and chlorine. These compounds can be highly toxic, and the overwhelming majority have been universally banned because of their unacceptably slow degradation and subsequent bioaccumulation and toxicity.[1]Among the more notable, dichlorodiphenyltrichloroethane (DDT) is an organochlorine pesticide and its invention won Paul Müller the 1948 Nobel Prize in Physiology or Medicine

5 groups of organochlorines insecticides

- **Dichlorodiphenyltrichloroethane** (DDT) and analogues (eg, dicofol, methoxychlor)
- **Hexachlorocyclohexane** (ie, benzene hexachloride) and isomers (eg, lindane, gamma-hexachlorocyclohexane)
- **Cyclodienes** (eg, endosulfan, chlordane, heptachlor, aldrin, dieldrin, endrin, isobenzan)
- **Chlordecone**, kelevan, and mirex
- **Toxaphene**

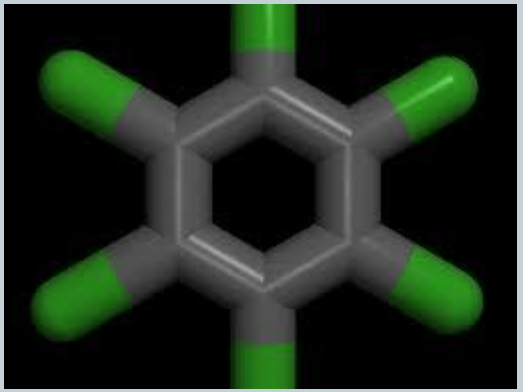
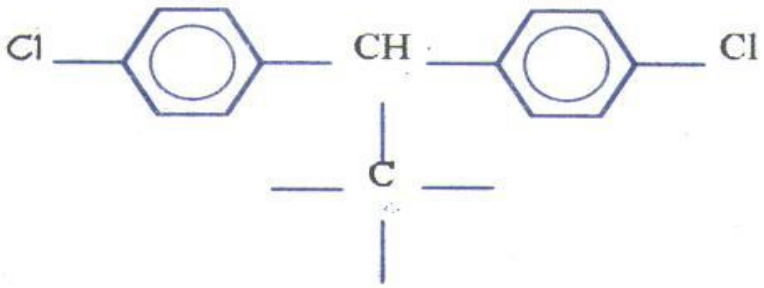


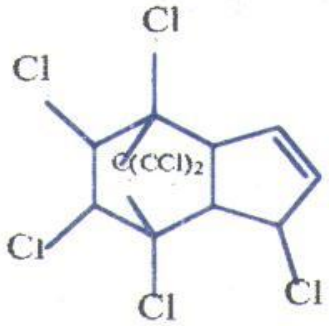
Figure 1: Structural Classification of Organochlorine Insecticides

DICHLORODIPHENYLETHANE



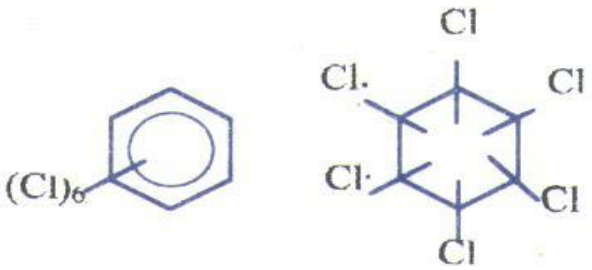
DDT
Dicofol

CYCLODIENES



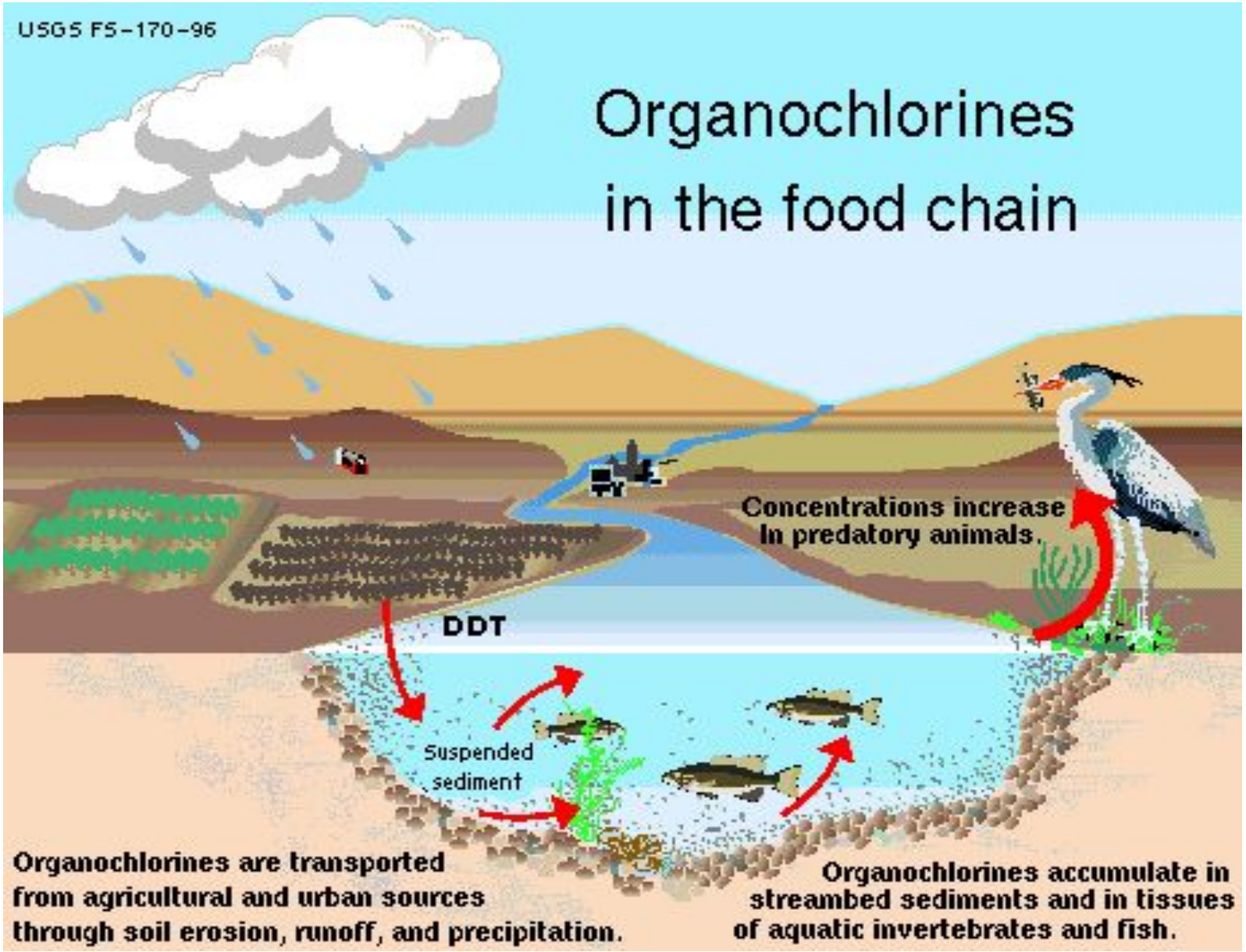
Dieldrin
Chlordane
Endosulfan

CHLORINATED BENZENES
CYCLOHEXANE



Lindane (α–BHC)

http://www.prn.usm.my/old_website/mainsite/bulletin/1996/prn10.html



http://wa.water.usgs.gov/pubs/fs/fs170-96/images/fs-170-96_foodchain.gif

Mechanism of toxicity

- Toxicity in humans is largely due to stimulation of the central nervous system. Cyclodienes (such as endosulfan), hexachlorocyclohexanes (such as lindane), and toxaphene predominately are GABA antagonists and inhibit calcium ion influx, but also may inhibit Ca- and Mg-ATPase, causing calcium ion accumulation at neuronal endplates, thereby causing sustained release of excitatory neurotransmitters. DDT affects potassium and voltage-dependent sodium channels. These changes can result in agitation, confusion, and seizures. Cardiac effects have been attributed to sensitization of the myocardium to circulating catecholamines.
- Some of the more volatile organochlorines can be inhaled while in vapor form or swallowed while in liquid form. Inhalation of toxic vapors or aspiration of liquid after ingestion may lead to atelectasis, bronchospasm, hypoxia, and a chemical pneumonitis. In severe cases, this can lead to acute lung injury (ALI), hemorrhage, and necrosis of lung tissue. In liquid form, they are easily absorbed through the skin and GI tract.

http://emedicine.medscape.com/article/815051-overview#a0104

Clinical presentation

CNS excitation and depression are the primary effects observed from organochlorine toxicity; therefore, the patient may appear agitated, lethargic, intoxicated, or even unconscious. Organochlorines lower the seizure threshold, which may precipitate seizure activity. Initial euphoria with auditory or visual hallucinations and perceptual disturbances are common in the setting of acute toxicity. Patients may have pulmonary complaints or may be in severe respiratory distress. Cardiac dysrhythmias may complicate the initial clinical presentation.

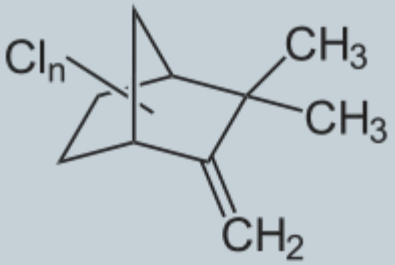
Other symptoms include the following:

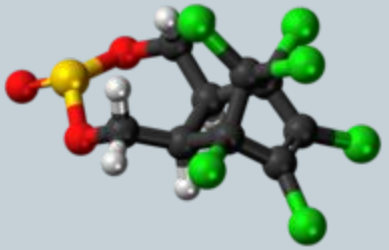
- Pulmonary - Cough, shortness of breath
- Dermatological - Skin rash
- Gastrointestinal - Nausea, vomiting, diarrhea, and abdominal pain
- Nervous system - Headache, dizziness, or paresthesias of the face, tongue, and extremities

http://emedicine.medscape.com/article/815051-overview#a0104

Physical findings

Physical examinations findings depends on type of exposure





Ingestions

- Nausea and vomiting
- Confusion, tremor, myoclonus, coma, and seizures
- Respiratory depression or failure
- Unusual odor - Toxaphene may have a turpentine-like odor. Endosulfan may have a sulfur odor

http://emedicine.medscape.com/article/815051-overview#a0104

Physical findings

Skin absorption or inhalation

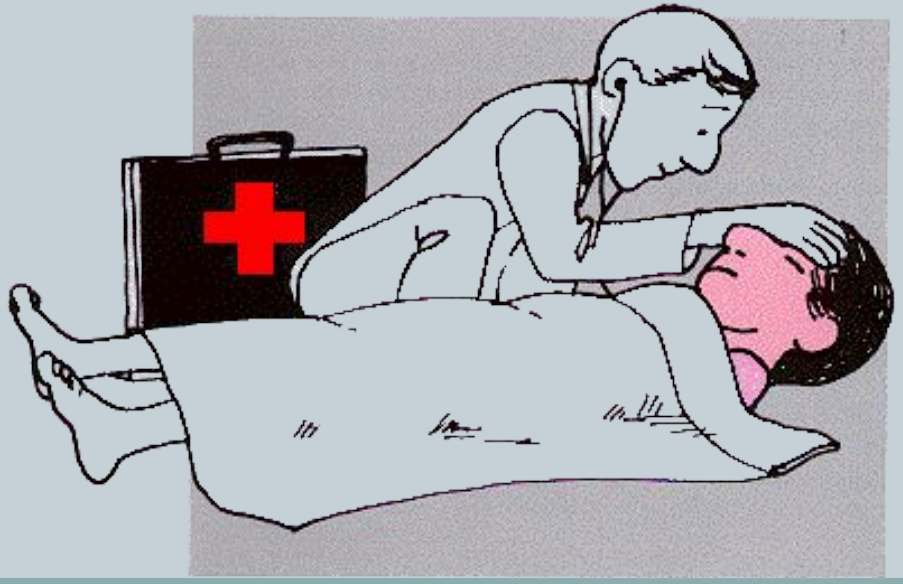
- Ear, nose, and throat irritation
- Blurred vision
- Cough
- Acute lung injury (ALI)
- Dermatitis

http://emedicine.medscape.com/article/815051-overview#a0104

Physical findings

Chronic exposure (meets in persons who constantly contact with chlorine organic connections: workers of compositions and enterprises from the production of chemical poisonings)

- Anorexia
- Hepatotoxicity
- Renal toxicity
- CNS disturbances
- Skin irritation



http://emedicine.medscape.com/article/815051-overview#a0104

Physical findings

- Pulmonary - Increased A-a gradient, hypoxemia
- Cardiovascular - Sinus tachycardia or bradycardia, QT prolongation, nonspecific ST-segment changes
- Gastrointestinal - Transaminitis and hyperbilirubinemia
- Hematological - Leukocytosis and prolonged activated partial thromboplastin time (aPTT)
- Renal - Acidemia, azotemia, creatinine elevation, hyperkalemia

http://emedicine.medscape.com/article/815051-overview#a0104

Prehospital Care

- Dermal decontamination is a priority. Remove clothes.
- Wash skin with soap and water.
- Provide oxygen and supportive care as necessary
- GI decontamination and elimination

Treatment

GI Decontaminant

Activated charcoal is emergency treatment in poisoning caused by drugs and chemicals. The network of pores present in activated charcoal adsorbs 100-1000 mg of drug per gram of charcoal. It does not dissolve in water.

For maximum effect, administer within 30 minutes of ingesting poison.

Multiple dose activated charcoal (MDAC) may be administered at 10-20 g q2-4h without a cathartic

Treatment

- **Bile acid sequestrants**
- These binding agents are used in the treatment of hypercholesterolemia and have been noted to bind certain lipid-soluble drugs and enterohepatically recycled drugs.
- Cholestyramine forms a nonabsorbable complex with bile acids in the intestine, which, in turn, inhibits enterohepatic reuptake of intestinal bile salts.

Treatment

- **Benzodiazepines**
- Mainstay of treatment for hydrocarbon insecticide–induced seizures.
- **Lorazepam (Ativan)**
- Rate of injection should not exceed 2 mg/min. May be administered IM if unable to obtain IV access.
- **Midazolam (Versed)**
- Used as alternative in termination of refractory status epilepticus. Because water soluble, takes approximately 3 times longer than diazepam to peak EEG effects. Thus, clinician must wait 2-3 min to fully evaluate sedative effects before initiating procedure or repeating dose.
- **Diazepam (Valium)**
- Depresses all levels of CNS (eg, limbic and reticular formation), possibly by increasing activity of GABA.

Treatment

- **Anticonvulsants**
- **Class Summary.** Additional options include pentobarbital or propofol for seizure control if status epilepticus does not respond to benzodiazepines or phenytoin or fosphenytoin.

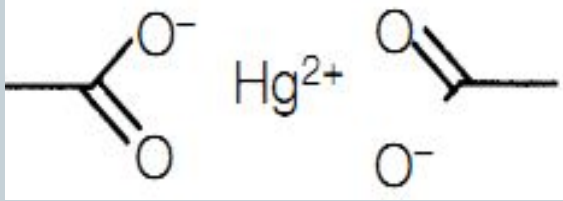
Intoxication by mercury organic connections.

- They are high enough bactericidal and fungicides characteristics and at staining does not have a negative influence on a corn, seed of vegetable and technical crops of bobs. That's why they are basic pesticides that are used for staining of seed.

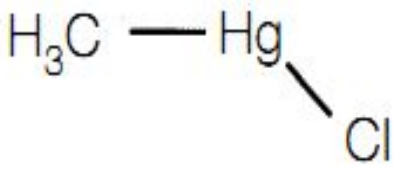
- The organic mercury compounds are of great interest today because they are often found in the food chain and have been used to inhibit bacterial growth in medications. Organic mercury is also found in fungicides and industrial run-off.

Structures, physical, and chemical properties of organic mercury compounds

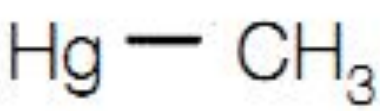
Mercuric (II) Acetate*



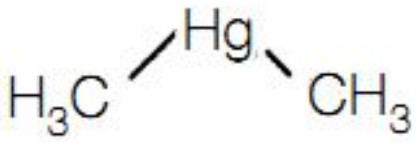
Methylmercuric Chloride



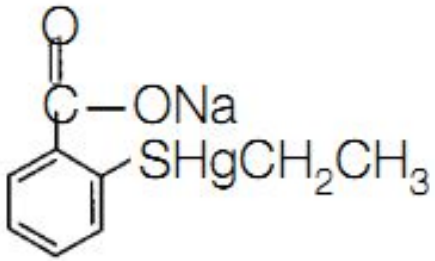
Methyl Mercury



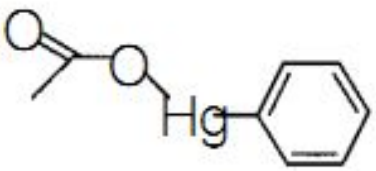
Dimethylmercury



Thimerosal

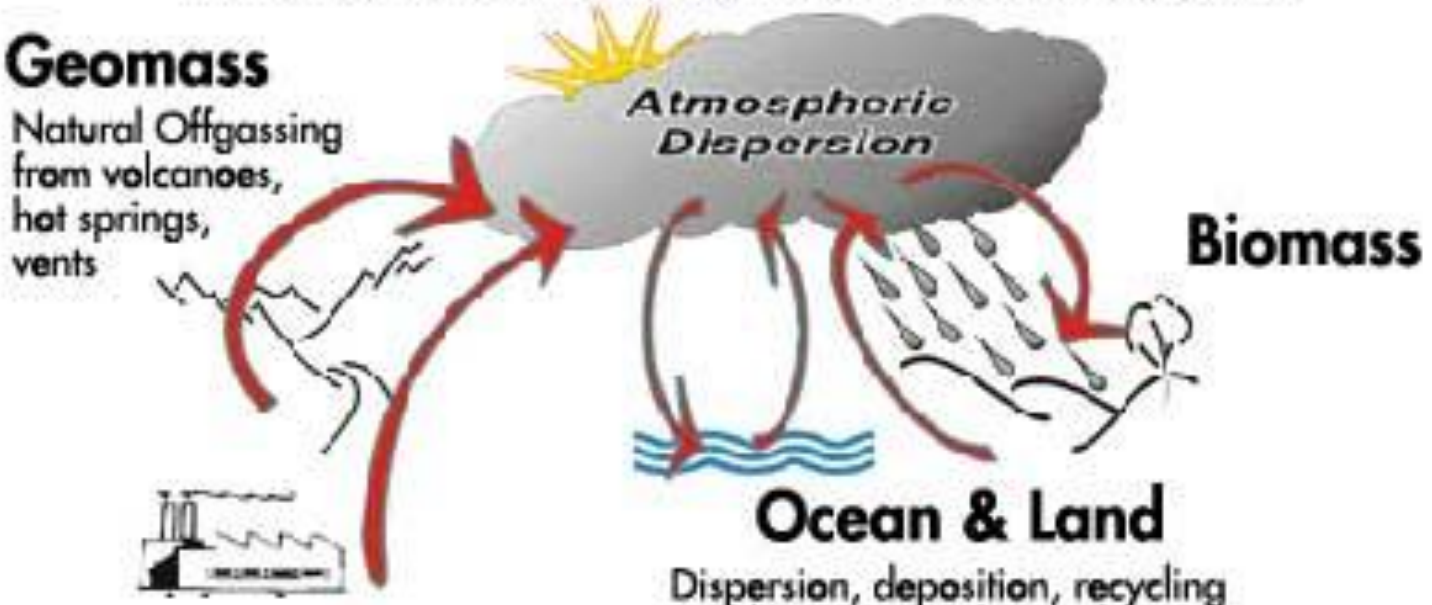


Phenylmercuric acetate



BioGeoChemical Cycling of Mercury

from subsurface to surface distribution and biomass uptake



Geomass

Natural Offgassing from volcanoes, hot springs, vents

Biomass

Ocean & Land

Dispersion, deposition, recycling and biomass uptake involving:

Transformation in steps to methylmerucry (most due to bacterial action)

Bioaccumulation in food chain

Biomagnification in fish and wildlife (Up to 10^7 x increase over background)

Bilogical impact/effects in cell division and reproduction, behavior, and letality

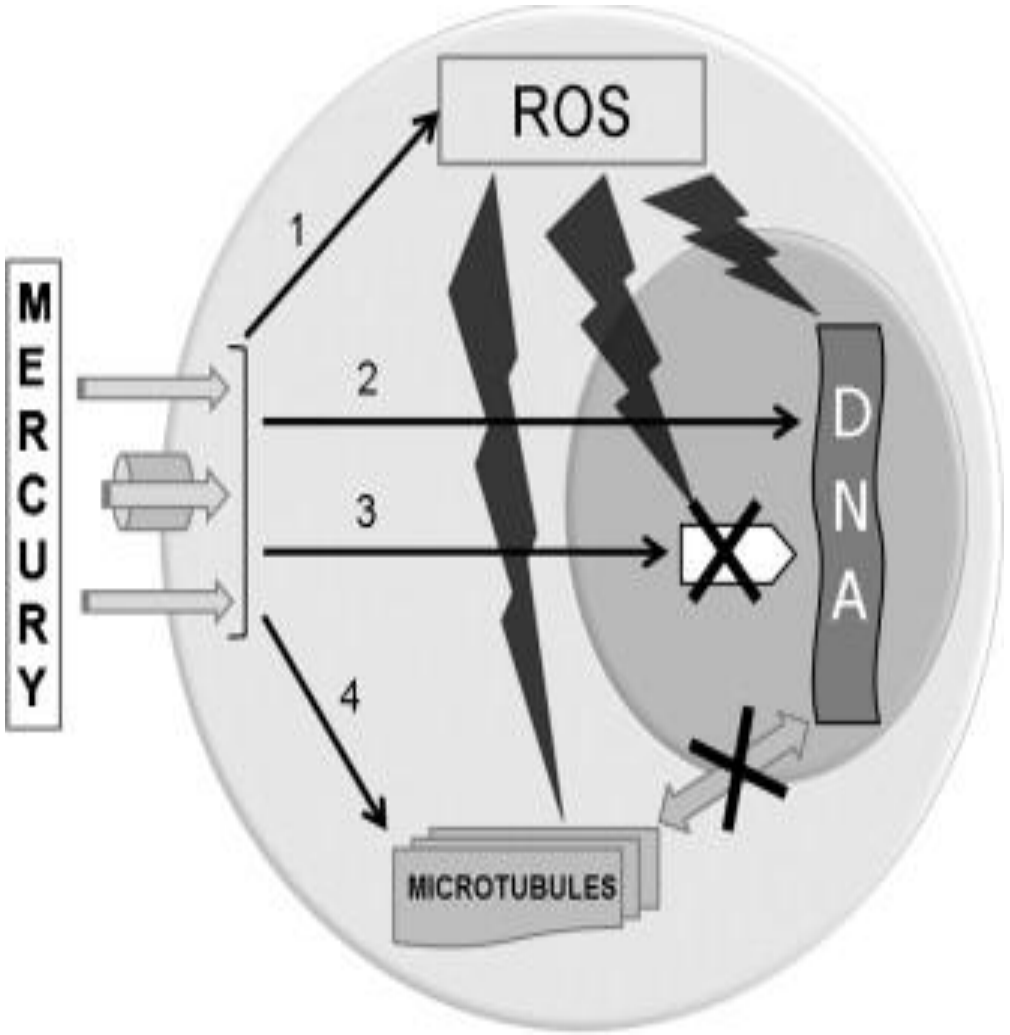
Industrial Releases from mining activities, burning coal and oil, waste disposal of:

- batteries,
- switches,
- thermometers,
- fluorescent lights,

cement factories, crematorums, amalgems

Mechanism of mercury toxicity

- Molecular mechanisms of mercury genotoxicity. Mercury compounds enter the cell through plasmatic membrane or transport proteins (grey cylinder). (1) Inside the cell, they may produce reactive oxygen species (ROS) which react directly with DNA or, indirectly, induce conformational changes in proteins responsible for the formation and maintenance of DNA (DNA repair enzymes, proteins of microtubules). Mercury compounds may be also able to bind directly to: (2) DNA molecules, forming mercury species-DNA adducts, (3) “zinc fingers” core of DNA repair enzymes (white large arrow), affecting their activity and (4) microtubules, avoiding mitotic spindle formation and chromosome segregation.



Minamata disease

Clinical presentation

- Ataxia
- tremors
- unsteady gait
- illegible handwriting, slurred speech
- erythema of the palms and soles
- edema of the hands and feet,
- desquamating rash, hair loss, pruritus
- tachycardia, hypertension, photophobia, irritability, anorexia, insomnia,
- poor muscle tone, and constipation or diarrhea.

- **A diagnosis** we put when we have special clinical picture and information of anamnesis, which specify on a contact with mercury organic connections. The important diagnostic sign of intoxication is a presence of mercury in blood, urine, and at heavy intoxications – in a cerebrospinal liquid.

Treatment

- - To wash a stomach and enterosorbtion;
- - Antidote - Unitiol, intramuscular 5 % solution on a chart: in first days 3-4 times in 6-8 hours, on the second days 2-3 times, on third-seven days 1-2 times per a day;
- - Intravenous enter 10 ml of 30 % solution of thiosulphate of sodium.
- - During acidosis intravenous we give 200 ml of 3-5 % solution of hidrocarbonate of sodium.
- - Symptomatic therapy.
- - Hemotransfusion, hemodialysis.
- - During chronic intoxication - Unitiol, the vitamins of group B, ascorbic acid, and also symptomatic therapy and procedures of physical therapies.

Thanks for attention!