EPIDEMIOLOGY OF PLANT POISONING:

- Approx. 5-10% of calls to poison centres (US) involve plants
- Plants are in the top 10 substances that children <6 yr are exposed to
- > 80% plant exposures benign, 7% in a health care visit, death in < 0.001%

IDENTIFICATION AND CLASSIFICATION:

- Physical identification of wild plants can often be difficult
  - Lack of defining structures depending on the specimen condition, time of year and growth stage
  - Lack of familiarity with all the flora (UBC e-flora, guide books)
- Plant poisoning can be classified in several ways:
  - Chemical compounds
  - Pharmacological. mechanistic action
  - **Toxidromes**
- Diagnosis is based on history (including what patient *thought* they were ingesting) and toxidrome

PLANT TOXIDROMES OVERVIEW:

CARDIAC	• Cardiac glycosides • Na channel activator	• Na/Ca channel blocker • K channel blocker
NEUROTOXIC	• Opiate/sedative • Anticholinergic	• Hallucinogenic • Convulsive
CYTOTOXIC	• Cyanogenic comp. • Mitosis inhibitors	• Protein synthesis inhibitors
GI	• Numerous (many plants with more severe systemic toxicity can cause GI symptoms)	
DERMATITIS	• Oxalate plants • Phototoxic	• Phorbol esters

→ clinical effects can very depending on "potency" of plant (growing conditions, maturity, etc) and how much ingested

CARDIAC:

CARDIAC GLYCOSIDES:

INFO	• *Digitalis lanata* (foxglove), oleander, lily of the valley	
MOA	Inhibit Na^+/K^+ATPase	• Increased intracellular Na and Ca • Increased serum potassium
	CV and ECG toxicity	• Sinus/junctional bradycardia • Ventricular tachydysrhythmias
	Increased vagal tone	• Abdominal pain/vomiting • Bradycardia, AV block
LABS	• Cardiac glycosides = digoxin, digitoxin, oleanderin, nerine ○ Potency and elimination half-lives vary • Serum digoxin assay: qualitative only as cross-reactivity with glycosides *other than* digoxin is unknown/variable	
TXT	• GI decontamination • Atropine • DigiFab: empiric dose 10 vials, depends on clinical response	

SODIUM CHANNEL ACTIVATORS:

INFO	• *Aconitum* (monkshood), *Veratum* (false hellebore), *Zygadenus* (death camas), *Rhododendron* (rhodo, azalea)
MOA	• Bind to sodium channels in excitable membranes (nerves, cardiac, skeletal muscle) and prevent closing → persistent depolarized state
S/S	• N/V, oral irritation, burning, sneezing • Bradycardia, hypotension, heart block, QT widening (prolonged sodium influx), ventricular dysrhythmias • Drowsiness, confusion, dizziness, paresthesias, convulsions • Muscle weakness, fasciculations • Fast onset (20 mins – 2 hours)
TXT	• GI decontamination? • Supportive care (fluids, pressors, maintain ventilation [weakness/paralysis]) • Atropine, pacing • Lidocaine not effective but flecainide, amiodaron helpful • Bypass for refractory VT/cardiogenic shock • Poisonings with rhodo generally resolve in hrs, others days

CARDIAC CONTINUED:

SODIUM CHANNEL/CALCIUM CHANNEL BLOCKER:

INFO	• *Taxus* yews – all parts except red fleshy aril are poisonous ○ Fruit: hard seed coat prevents release of taxines unless chewed
S/S	• Onset may be fast (minutes) with teas, or slower (hours) with ingestion of plant material • GI upset, dizziness, tachycardia → bradycardia, convulsions, loss of consciousness • Shock, acidosis • Widened QRS complex, ventricular dysrhythmias, asystole
TXT	• Mainly supportive • Sodium bicarbonate – varying results, maintain pH • Lidocaine, amiodarone – varying results • DigiFab – not indicated • Lipid emulsion therapy • Bypass/extracorporeal membrane oxygenation

TABERNANTHE IBOGA

- Source of ibogaine used in detox programs (opiates, cocaine and stimulants, alcohol, nicotine)
- Can cause potassium channel blockade, QTc prolongation, dysrythmias
  - Mostly in pts with underlying CVD, but also healthy individuals

NEUROTOXIC:

ANTICHOLINERGIC:

INFO	• *Atropa belladonna* (deadly nightshade) • *Datura stramonium* (jimson weed) • *Brugmansia (Datura) aureua* (angel's trumpet)

MOA	• Contain alkaloids atropine, scopolamine
	• Causes typical anticholinergic effects
	• Datura is abused
	  ○ Onset & severity depends on method of ingestion
	  ○ Effects can be prolonged

SX	• Hyperthermia, coma, seizures, ventilatory failure reported • Deaths uncommon, sometimes related to environmental exposure, drowning, etc, while intoxicated
TXT	• May consider GI decontamination even late if plant materials or seeds ingested (slowed gastric emptying, etc) • Benzodiazepines for agitation, seizures • Phsostigmine (cholinesterase inhibitor) often mentioned, but is Special Access in Canada

CYTOTOXIC:

POISON HEMLOCK	INFO	• *Conium maculatum* • Mistaken for wild carrot and parsley
	MOA	• Contains coniine (similar to nicotine) • Toxic dose: 6g of leaves for adults
	S/S	• Oral & GI irritation, salivation, vomiting • HA, dizziness, confusion → seizures, coma • HTN, tachycardia → bradycardia and shock • Muscle fasciculations, myoclonus, weakness and paralysis
WATER HEMLOCK	INFO	• *Cicuta spp.* – most toxic indigenous N.A. plant • Mistaken for wild parsnip, wild carrot
	MOA	• Contains cicutoxin (roots and stems) • Inhibits GABAergic activity • 1 cm of root = fatal in adults; oral contact with stem caused toxicity in children

	S/S	• GI upset within 15 minutes
		• Sz may occur w/in 30 mins in sever cases
		  ○ Often intractable
		  ○ Can lead to CVS decompensation

	TXT	• Supportive care • Benzos → barbiturates → Propofol • No role for phenytoin

CYTOTOXIC:

CYANOGENIC:

INFO	• *Prunus* (cherries, peaches, plums, apricots, almonds), apples, cassava, hydrangeas, cotoneaster, *Sambucus* spp (elderberry)
	• Poisoning is rare
	  ○ Hard seed coat prevents release of cyanogenic compounds from seeds
	  ○ Cyanogenic compound content varies
	  ○ Onset can be delayed (need hydrolysis to release CN)

S/S	• GI upset (possible from other components of plant) • Lethargy, altered mental status, seizures • Shortness of breath, tachypnea • Initial hypertension → hypotension, CVS collapse • Lactic acidosis (blocks aerobic metabolism)
TXT	• Supportive care • Hydroxocobalamin

MITOSIS INHIBITORS:

INFO	• *Colchicum autumnale* (autumn crocus) ○ Different from spring crocus
MOA	• Autumn crocus contains colchicine → inhibits microtubule formation, arrests mitosis • Rapidly dividing cells (GI, bone marrow) affected worst • No reports of poisoning from plant (DPIC) ○ Several poisonings from pharmaceutical product
S/S	• GI symptoms • Cardiovasacular collapse • Multiorgan system failure • Initial leukocytosis followed by bone marrow failure
TXT	• No antidote (immune Fab experimental, not available) • Supportive care • Colony stimulating factors

PROTEIN SYNTHESIS INHIBITORS:

INFO	• *Ricinus communis* (castor bean), *Abrus precatorius* (rosary pea), *Robinia pseudoacacia* (black locust or false acacia)
MOA	• Contain toxalbumins (ricin, abrin, robin) • Inhibit ribosomes → halts protein synthesis → cell death
S/S	• Minimal toxicity if seeds swallowed intact • GI toxicity if chewed; may limit systemic absorption but severe/prolonged GI sx and hypovolemia are problems • Can cause life-threatening systemic toxicity if injected (mg) or pulmonary toxicity if inhaled
TXT	• No antidotes – treatment is mainly sympatomatic/supportive

LOCAL IRRITANTS:

OXALATE PLANTS:

- Ejection of raphides + other irritant into mucosa causes local irritation
- Mostly home management but
  - Oropharyngeal inflammation may lead to airway obstruction
  - Ocular exposure can lead to severe irritation

PHOTOTOXIC:

INFO	• Giant hogweed, citrus (lime, grapefruit), celery
MOA	• Psoralen, bergapten, etc • Absorb UV wavelength light → DNA damage
S/S	• Initial redness turning to blisters • Scars may take years to heal • Ocular injury may be sight-threatening • Hyperpigmentation fades (weeks → months)
TXT	• No specific treatments • Prevent: decontaminate, avoid sunlight exposure x 48h

PHORBOL ESTERS: *Euphorbia spp*

- Irritant and ribosome inhibitors
- Can cause skin irritation
- Main hazard DPIC has seen = ocular
  - Ocular reactions may progress over 24h, follow-up important
  - Corneal recovery expected in 1-2 weeks
- Treatment: decontaminate, symptomatic care