Jimsonweed (Datura stramonium):

Jimsonweed is an annual herb which grows up to 5 feet tall and emits a rank odor. It has a pale green stem with spreading branches. Leaves are ovate with green or purplish coloration. Flowers are white or purple with a 5-pointed corolla up to four inches long and set on short stalks on the axils of branches. Seeds are contained in a hard, spine capsule, about 2 inches in diameter, which split lenghtwise into four parts when ripe. It is distributed all over the world especially in the United States.

All parts of Jimsonweed are poisonous. Leaves and seeds are the usual source poisoning, but are rarely eaten due to its strong odor and unpleasant taste. Poisoning can occur when hungry animals are on sparse pasture with Jimsonweed infestation. Most animal poisoning results from feed contamination. Jimsonweed can be harvested with hay or silage subsequently poisoning occurs upon feeding the forage. Seeds can contaminate grains and is the most common poisoning which occurs in chickens. Poisoning is more common in humans than in animals. Children can be attracted by flowers and consume Jimsonweed accidentally. In small quantities, Jimsonweed can have medicinal or hallucinogenic properties, but poisoning readily occurs because of misuse.

Jimsonweed toxicity is caused by tropane alkaloids. The total alkaloids in the plant can be as high as 0.7%. The toxic chemicals are atropine, hyoscine (also called scopolamine which acting as an antagonist at both peripheral and central muscarinic receptors, is thought to be the primary compound responsible for the toxic effects of these plant), and hyoscyamine, which possess strong

anticholinergic proprieties. Such alkaloids are easily absorbed from mucous membrane, skin and intestinal tract. Hyoscyamine and scopolamine are metabolized and in the liver by hydrolysis and excreted unchanged in the kidney.

Clinical signs of Jimsonweed poisoning:

Jimsonweed poisoning occurs in most domestic animals: cattle, goat, horses, sheep, swine and poultry. Human poisoning occurs more frequently than livestock after ingestion of leaves and flowers, tea brewed from plant parts, and smoking of stramoniun cigarette. The actual effects are reported to be: cycloplegia (loss of power in the ciliary muscle of the eye) and mydriasis (extreme dilation of the pupil), flushed, warm and dry skin, dry mouth, urinary retention, rabid heart beat, hypertension or hypotension and blurred vision (which is the most persistent symptoms). In the case of over dose the effects are hyperthermia, coma, respiratory arrest and seizures. The effects of Datura have been described as a living dream: consciousness falls in and out, people who don't exist or are miles away are conversed with, etc. The effects can last for days. In this case, the user would ignore most stimuli and respond to unreal ones.

The doses that cause noticeable effects and the doses that can kill are very close with datura. This makes overdosing on Datura stramonium very easy. This can be fatal; it can cause fevers in the (40-43°C) range which is a range that can kill brain cells, and lead to brain damage. Fatalities usually result from trauma or drowning rather than direct toxic effect of Datura.

Early signs:

Rabid pulse.
Rabid breathing.
Diluted pupils.
Restlessness.
Nervousness.
Muscular twitching.
Depression.
Diarrhea.
Wight loss.
Blurred vision.

Fatal causes:

Weak pulse.
Irregular breathing.
Coma.
Higher body temperature.
Convulsions.
Retained urine.

Clinical presentation:

Datura ingestion produces the classic presentation of anticholinergic poisoning syndrome describe by the phrase "blind as a bat, mad as a hatter, red as a beet, hot as hell, dry as a bone". Symptoms typically begin within 2 to 6 hours after ingestion, but consumption of Datura tea has resulted in symptoms within 5 to 10 minutes. Progression effects from cigarettes prepared from Datura stramonium are dryness of the mouth which is the earliest sign of intoxication followed by papillary dilation. Symptoms may continue for 24 to 48 hours because alkaloids delay gastric emptying and absorption, but papillary dilution may continue up to 1 week.

Treatment:

Most patient respond well to supportive and protective care. The patient should be placed in a calm, reassuring environment with a familiar person. Gastric emptying and activated charcoal may be useful within several hours postingestion, but this not well studied. The antidote for anticholinergic poisons is physostigmine (an alkaloid of physostigma; it is a reversible inhibitor of the cholinesterases, and prevents destruction of acetylcholine; used as a cholinergic agent). The initial adult dose of physostgmine is 1 to 2 mg intramuscularly or intravenously over 2 to 5 minutes. Improvement usually occurs with 15 to 20 minutes. Repeated dose may be repeated in 20 to 30minutes. Repeated doses should be given only if the symptoms reappear. And Diazepam (a skeletal muscle relaxant, sedative, and antianxiety agent; also used as an anticonvulsant) has been used for agitation.

Oleander (Nerium oleander):(الدّفلة)

Is an evergreen shrub or small tree in the dogbane family *Apocynaceae*. It is the only species currently classified in the genus Nerium. It is native to abroad area all over the world. It typically occurs around dry stream beds. It grows to 2-6 m tall, with spreading to erect branches. The leaves are in pairs or whorls of three, thick and leathery, dark green, narrow lanceolate. The flowers grow in clusters at the end of each branch; they are white, pink or yellow. They are often, but not always, sweetly scented. The fruit is a long narrow capsule 5-23 cm long, which splits open at maturity to release numerous downy seeds.

Cultivation and uses:

Oleander grows well in warm subtropical regions, where it is extensively used as an ornamental plant in landscapes, parks, and along roadsides. It is drought tolerant and will tolerate occasional light frost down to -10°C. It can also be grown in cooler climates in greenhouses, conservatories, or as indoor plants that summer outside. Oleander flowers are showy and fragrant and are grown for these reasons.

Ethenomadical uses:

Records of the medicinal use of oleander date back at least 3500 years. The Mesopotamians in the 15th century BC believed in the healing properties of oleander and the ancient Babylonians used a mixture of oleander and licorice to treat hangovers. Pliny, the Elder of ancient Greece, wrote about the appearance and properties of oleander. Arab physicians first used oleander as a cancer treatment in the 8th century AD. Centuries later; European studies in the 1980s, the oleander extract was found to have six times the immune stimulating activity of the most powerful patented immune stimulators. Then oleander soup was used to treat cancer, hepatitis-C, HIV and other conditions, and is made by

precise directions for boiling, condensing and straining according to the recipe for making the soup. When further condensed and made into a skin creme, the remedy is used to get rid of warts, age spots and pre-cancerous lesions. Oleander leaf extract is also taken to treat congestive heart disease. No one should attempt to make their own oleander remedy without precise directions and oleander should only be taken only after consultation with an experienced herbalist and physician because it is a highly toxic poison in raw form that may be lethal to humans and animals in even doses as small as one leaf.

Oleander toxicity:

Oleander is one of the most poisonous plants and contains numerous toxic compounds, many of which can be deadly to people, especially young children. The toxicity of Oleander is considered extremely high and it has been reported that in some cases only a small amount had lethal or near lethal effects. The most significant of these toxins are oleandrin and neriine, which they are cardiac glycoside; Cardiac glycosides are naturally occurring" plant or animal compounds "whose actions include both beneficial and toxic effects on the heart" Cardiac glycosides that act by inhibiting the cellular membrane sodium-potassium (Na+-K+ ATPase enzyme system) pump with resulting depletion of intracellular potassium and an increase in serum potassium. This result in progressive decrease in electrical conductivity through the heart causing irregular heart activity, and eventual complete block of cardiac activity, and death.They are present in all parts of the plant, but are most concentrated in the sap. Oleander is also known to hold its toxicity even after drying. According to the Toxic Exposure Surveillance System (TESS) in 2002 there were 847 known human poisonings in the United States related to Oleander.

Effect of poisoning:

Reactions to this plant are as follows. Ingestion can cause both gastrointestinal and cardiac effects. The gastrointestinal effects can consist of nausea and vomiting, excess salivation, abdominal pain, diarrhea that may or may not contain blood. Cardiac reactions consist of irregular heart rate, sometimes characterized by a racing heart at first that then slows to below normal further along in the reaction. The heart may also beat erratically with no sign of a specific rhythm. Respiratory reaction consist of difficulty in breathing, Occular reaction consist of dilated pupils, impaired vision. Extremities may become pale and cold due to poor or irregular circulation. Reactions to poisonings from this plant can also affect the central nerves system. These symptoms can include drowsiness, tremors or shaking of the muscles, seizures, collapse, and even coma that can lead to death. Oleander sap can cause skin irritations, severe eye inflammation and irritation, and allergy reactions characterized by dermatitis.

Medical treatment required:

Poisoning and reactions to Oleander plants are evident quickly, requiring immediate medical care in suspected or known poisonings of both humans and animals. Induced vomiting and gastric lavage are protective measures to reduce absorption of the toxic compounds. Charcoal may also be administered to help absorb any remaining toxins. The antidote of cardiac glycoside poisoning digoxin immune fab (should be used in patient who develops hemodynamically significant dysrhythmias or hyperkalemia. Further medical attention may be required and will depend on the severity of the poisoning and symptoms. The cardiac irregularities may be treated using anti-arrhythmic drugs such as potassium chloride, dipotassium EDTA, or atropine sulfate).