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CHAPTERS

The Biological role of toxic secondary metabolites of different origin on insect and humans

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Abstract:-Plant toxins are photochemical or secondary metabolites formed naturally by plants. They protect themselves against various threats like bacteria, Fungi, nematodes, insects, etc. Among secondary plant metabolites, one can find alkaloids, terpenoids, steroids, glycosides, phenolics, tannins, and others. Humans have used these secondary metabolites over thousands of years, both as drugs and agents to kill animals and humans. Some insects are food specialists and can feed on normally toxic plants to other herbivores. Many plant secondary metabolites interfere with the neuro receptors and neurotransmitters in vertebrates. Insects share many neuro receptors with vertebrates. PSM serves as olfactory cues for insects to identify their appropriate food plants. The behavior of insects towards such chemicals cues reminds us of drug addiction in humans and other vertebrates. This Chapter is incorporated the major aspects of plants' secondary metabolites and their impact on insect ecological adaptation.

Keywords:- Phytochemical, secondary metabolites, bacteria, insects, phenolic, glycosides, steroid, terpenoids, alkaloids.

INTRODUCTION

Plant toxins are produced as secondary metabolites that act as a defense mechanism for the plant. As a medicine and toxic use of plants cure disease and pain that are decided by the trial-and-error method. Plants that relieve suffering and cure illnesses along with plant poisons and their antidotes must have been learned through trial and error by early humans. From generation to generation, knowledge was passed by teaching observation, and experience, People of recent days, over 75% of the world's population depends on herbal medicines[1]. In the

USA, only about 10% of the medical pharmacopeia comes directly from plants. However, many North American drugs are chemically synthesized from compounds found originally in plants[1].

In Sanskrit literature is full of records of the use plant In Chinese medicinal records also are available. The second Celestial Emperor (2000 BC), Shen Nung sampled more than 1000 herbs to determine their curative and poisonous properties. Shen Nung died perhaps from sampling one poison too much[2]. Simoons (1998)[3] reviewed how poisoning showed up in various cultures, including the ancient Egyptians, the Greeks, and the Romans, continuing down to modern times.

Around 400 BC, the Hippocratic Oath originated parameter values for many many medical practitioners. Today, it is only administered in approximately 60% of U.S. medical tools at the granting of MD degrees[4]. Older versions of the Oath prohibit the use of poisons by physicians. This caution is not present in today's versions, although it may be covered by the caution to do no harm. Perhaps its omission relates to the treatment with many drugs that in higher concentrations could be deadly. In the mid-nineteenth and early twentieth centuries, the use, of plant-derived poison homicides became more common as the chemical detection of metallic poisons such as arsenic improved[5].

2. Biological role of Plant Toxins

Some plants produce toxins and can be used in treating many diseases. The action of plant toxins is based on their chemical constituent and their action by affecting specific mechanisms, There are a huge variety of plant poisons which are secondary metabolites and they can affect human and animal bodies so plant toxins my medicinal and poisonous.

3. Plant poisonings and secondary metabolites

Homicides poisonings with heavy metals (e.g., antimony, arsenic, others) during medieval times, accidental and intentional were commonplace. These poisons were readily available to people as. active ingredients in a curative potion. In the transfer of property and wealth to heirs Arsenic was known as the nickname of "inheritance powder" as it was often used to hasten the postmortem but by the nineteenth century, chemists were developing procedures for detecting these poisons making them less attractive for nefarious purposes. Around

the twentieth century, there was a transition from metallic poisons to alkaloids and other chemicals of plant origins that were more difficult to detect. Many of these plant poisons had been known for centuries and had made their way into many folk medicines All of these toxins were discovered as having some sort of curative property by ancient people, and many of them are still used today. General symptoms of poisoning caused by these compounds are summarized in Levine *et al.* (2011)[6].

3.1. Alkaloids

Alkaloids a naturally occurring organic chemicals that contain one nitrogen atom. This compound consists mostly of carbon, hydrogen, and nitrogen but may also contain sulfur and/or oxygen. Rarely, they will include elements such as chlorine, bromine, or phosphorus

3.1.1 Colchicine

One plant poison first approved by FDA in 1961, it is an alkaloid drug favored by the Greeks and Romans and came from a species of crocus (*Colchicum* spp., family: Iridaceae). These plants are the source of the alkaloid drug colchicine (Figure 1) that sometimes is prescribed today for the treatment of gout, arthritis, and constipation-predominant irritable bowel syndrome. Colchicine is known to most biologists as an inhibitor of cell division. The drug comes from crocus corms and seeds. Colchicine can be deadly if misused as there is no known antidote for colchicine poisoning. Multiple system failures occur in 24-72h after consumption of a lethal dose. An unfortunate case of colchicine poisoning occurred in Colorado when a thief misread the label on a bottle he had stolen from a doctor's safe, and he died after ingesting the pills.

3.1.2. POISON HEMLOCK

Poison hemlock, is a well-known toxin plant of the carrot family, known, since ancient Greece, is poison hemlock (*Conium maculatum* L), a member of the carrot family (Apiaceae). In ancient Greece, Important people who received death sentences were allowed to choose their method of death. Socrates selected poisoning with a tea made from poison hemlock. Plato witnessed his death and described in detail the stages of the poison's action[7]. Plato's description fits the symptoms of contemporary poisoning by poison hemlock[8]. The active toxic ingredient is the alkaloid coniine (Figure 1), which causes paralysis of the

respiratory muscles leading to death. As little as 100 mg (1.6mg/kg body weight for a 60kg adult) is a lethal dose (e.g., six to eight leaves of *C. maculatum*). Poison hemlock is widespread in the northern hemisphere and a few cases of poisoning occur each year.

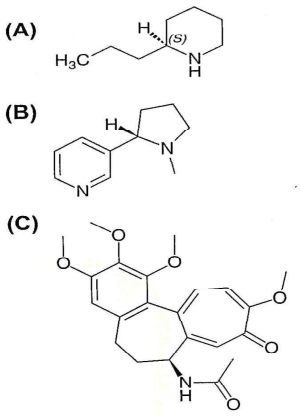


FIGURE 1. Alakaloid toxins.
(A) Coniine;
(B) Nicotine;
(C) Colchicine.

3.1.3 The Tropane alkaloids

Tropane alkaloid is a valuable secondary metabolite found in high concentrations in the family Solanaceae that include potato, and tomato. Deadly nightshade, *Atropa belladonna*; jimson weed or "loco weed" (*Datura* spp.); angel's trumpet (*Brugmansia spp.*); and henbane (*Hyoscyanus niger*) are the sources of several hallucinogenic and potentially lethal tropane alkaloids scopolamine, atropine, and hyoscyamine. (Figure 2)

3.1.4 Scopolamine

Scopolamine is used as an antidepressant and antinausea drug. It is anticholinergic and antimuscarinic. Paradoxically, overdoses can produce depression. It is hallucinogenic but the experiences are generally extremely unpleasant. Scopolamine at one time was administered to pregnant women in labor as "twilight sleep."

3.1.5 Atropine

Atropine is also an anticholinergic, antimuscarinic drug that causes pupil dilation, increases heart rate, and increases secretion of saliva. A fatal dose of atropine is greater than 10mg, whereas scopolamine is toxic at 2-4mg. The name "belladonna" comes from Italy where it was once used to dilate the eyes of women to make them more attractive ("bella") to men.

3.1.6 Hyoscyamine is the levorotatory 1somer of atropine and is also the precursor for the synthesis of scopolamine. Its actions are similar to scopolamine and atropine. Hyoscyamine is named for the genus of henbane which concentrates tropane alkaloids in the leaves and seeds (Figure 3). Perhaps a leader in poisonings among these plants is jimson weed. This plant is featured in the many controversial books by Carlos Castenada (http://en.wikipedia.org/wiki/Carlos_Castaneda) that first came to prominence in the 1960s. Jimson weeds are very hallucinogenic and can be fatal.

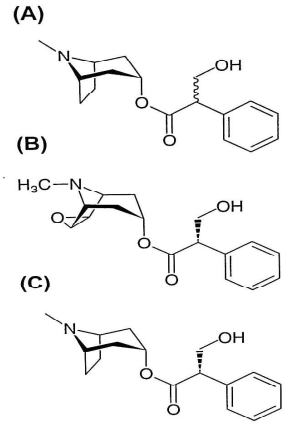
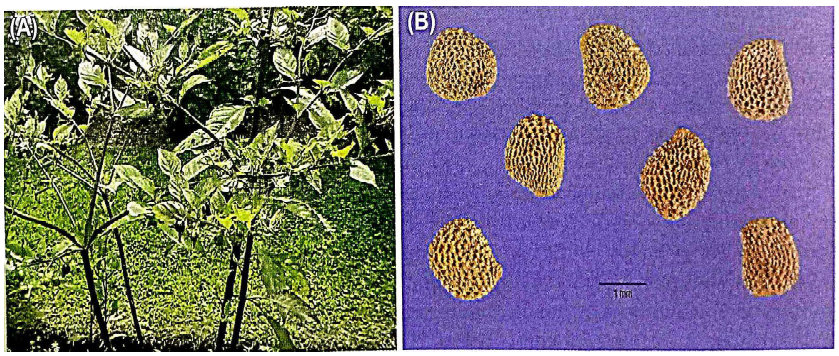


FIGURE 2. Structures of tropane alkaloids.
(A) Atropine;
(B) Scopolamine;
(C) Hyoscyamine.



3.2 Other Alkaloids

3.2.1 STRYCHNINE

Strychnine (Figure 4) is a plant product taken by mouth, inhaled or mixed in a solution, or injected in the vein is a potent alkaloid neurotoxin that blocks cholinergic receptors in skeletal muscles. If taken in excess doses can lead to paralysis of respiratory muscles causing

asphyxia and death. The lethal dose for humans is 32 mg/kg body weight. Strychnine is most commonly derived from the seeds of Strychnic nux-vomica, a native tree of South India, and a related climbing shrub, *Strychnos ignatii*, native to the Philippines called St. Ignatius'bean. It was used as a rodent poison in Europe beginning in the seventeenth century until the present time. Accidental poisonings were not uncommon. It is suggested that the death of Jane Stanford in 1905, a cofounder of Leland Stanford University was a result of strychnine poisoning although it is not clear how this came about[9]. Once strychnine became readily available, it made its way into use for homicides. It has been suggested that it was the poison given to Alexander the Great in 323 BC[10]. However, because of the overt symptoms of strychnine poisoning and its easy chemical detection, it is not the poison of choice today. Nevertheless, it did appear in the San Diego death of Sue Morency who died under unusual circumstances in 1990. She had a body concentration of strychnine that as 4 times the lethal level. Her husband was arrested and charged with homicide[11].

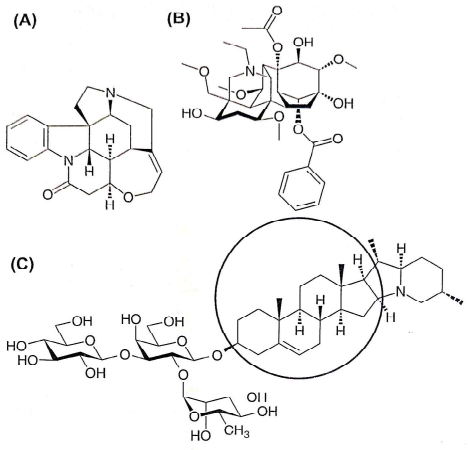


FIGURE 4. Some other alkaloid toxins.
(A) Strychnine;
(B) Aconitine;
(C) Solanine.
Note the inclusion of a steroidnucleus (circle).

3.2.2 ACONITINE

Aconitine is also an alkaloid toxin, (Figure 5) and is produced by the 250 species of Aconitum 90 family *Ranunculaceae* commonly called Wolf's bane and monkshood (Figure 5). All parts of these plants are extremely toxic, especially the roots. In addition to its use in ancient medicines, it was used to make poisoned arrows for hunting (Chinese, Japanese Ainu, Aleuts) and warfare (Chinese).

FIGURE 5. Monkshood, *Aconitum variegatum.* Härtsfela, Germany, courtesy of Bern Haynold, available http://commons.wikimedia.org/wiki/File:Aconitum_variegatum_110807f.jpg

Aconitine effectively opens sodium channels so that muscles and neurons cannot be repolarized. Thus, aconitine can produce ventricular dysrhythmia of the heart leading to death. It can also cross the blood-brain barrier and produce neural effects. One of the early uses of Aconitum extracts in Europe was to kill wolves, hence another of the plants' many common names is wolf's bane. The lethal dose for humans is 32 mg/kg body weight. Surprisingly, the caterpillars of numerous moth species feed on this plant despite its toxicity to many other animals. The lowest oral dose reported to kill a human is only 29 ug/kg body weight (100x more lethal than strychnine). Reportedly, Cleopatra used aconitine to poison her brother (and husband) Ptolemy XIV so she could replace him with her son (http://en.wikipedia.org/wiki/Aconitum). A promising young Canadian TV and film actor, Andre Noble, died after consuming monks hood while on a hike in Newfoundland[12]. In 2009, the British "Curry Killer," Lakhbir Singh, murdered her lover by feeding him a curry dish laced with aconitine[13].

3.2.3 Solanine, a glycoalkaloid

Genus Potatoes (*Solanum tuberosum*, Solanaceae) that show signs of greening, sprouting, rotting, or physical damage have high concentrations of solanine and should not be eaten (Figure 4C). If one observes green material beneath the skin of a potato, one should not eat the potato because solanine is concentrated in this green layer and there may also be elevated levels in the rest of the potato. Greening in a potato is evidence of excessive exposure to light. Solanine, like other cyanide compounds, is produced as a deterrent to insects and other animals that might feed on the plants. It is found in lower amounts in other food plants such as eggplant and green peppers.

In the USA, each adult human consumes about 65 kg of potatoes/year. Ingestion of potatoes high in solanine and a closely relatedglycoalkaloid, chaconine, has been associated with numerous poisonings and some fatalities[14]. The compounds can cause neurological impairments, vomiting, and diarrhea. Most varieties of potatoes contain less than 5mg/kg. Concentrations of 14 mg/kg potato cause a bitter taste and 20 mg/kg causes a burning sensation in the mouth and throat.

4. Glycosides

Glycosides, in chemistry molecules in which sugar (saccharide) is associated with another functional chemical group via a glycosidic bond. Joining these components is usually formed through an oxygen, sulfur, or nitrogen atom. A glycoside with a sulfur bond would be a thioglycoside, for example, given below:

4.1 Digoxin, a cardiac glycoside

Digoxin is a drug, used in medication to treat cardiac failure and certain arrhythmias and abortion, The family Solanaceae does not have a corner on poisonous/medicinal plants. Although its use is declining its overuse result in death, there are about 20 species of foxglove (Digitalis spp.: figwort family, Scrophulariaceae). Digoxin (Figure 6) is a cardiac glycoside extracted from foxglove. It often goes under the name of digitalis. Some cardiac patients under treatment for congestive heart failure and atrial arrhythmia carry a supply of Digitalis pills for self-medication if they feel symptomatic andare away from professional help, although its use is declining. Overdosing of digitalis can result in death. People have sometimes confused foxglove with comfrey (*Symphytum* spp) and brewed a toxic "comfrey tea" (Figure 6). However, comfrey contains the pyrrolizidine alkaloid retronecine (Figure 7) that is hepatotoxic and linked to liver cancer and probably should not be ingested. Treatments for accidental and purposefully overdoses of these drugs remain a major field of research[10].

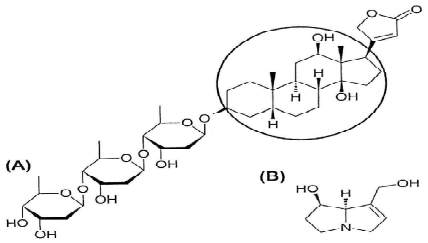


Figure 6: Structure of Comfrey contains the pyrrolizidine alkaloid retronecine