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Major Toxic Plants and Their Effect on Livestock: A Review

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Abstract
Poisonous plants are major causes of economic loss to the livestock industry. Each year these plants adversely affect 3 to 5 percent of the cattle, sheep, goat, and horses that graze ranges. These losses result from death of livestock, abortions, photosensitization, decreased production, emaciation, and birth defects. In addition to these losses are those of increased management costs associated with such things as fencing, altered grazing programs, and loss of forage. Suggestions are included for the prevention of livestock poisoning by plants. Various species and classes of livestock are reported to be affected by toxic plants particularly in the dry and early rainy seasons when feed is in short supply. A more extensive survey is required to document all poisonous plants in the rangelands and to identify the major toxic principles in the different species possibly pharmacological activity. This review presents the current knowledge on the identity of plants known to have poisoned livestock and research conducted into these toxic plants. Many of the plants identified are considered toxic on the basis of world literature. Research is needed to determine not only which plants represent a potential risk for animal health and production but also their photochemistry and toxicology. It is strongly recommended that veterinarians document plant poisoning cases through government reporting services and that university and government veterinarians, scientists, and extension agents investigate episodes of plant toxicosis and publish their findings. This would help identify toxic species for further photochemical and toxicological studies and possibly pharmacological activity.
Keywords: Bladder tumors, Bracken fern, Castor oil**,** Kochia, Photosensitization and toxicity.

INTRODUCTION

Toxic plants affecting both large and small animals are a major concern for the practicing veterinarian and livestock producer in every country. In countries with higher plant biodiversity, the number of problematic toxic plants may be greater. Ethiopia also possesses three traditionally recognized agro climatic zones. These are Kola (a hot zone of 500-1500 m above sea level), Weina Dega (intermediate zone of 1500 m and 2500 m), and Dega (a cold zone usually cited as being 250 m above sea level). These ecological zones has favorable to various species of plants and animals. Apart from the globally listed ones, therefore, some factors are inherent in Ethiopia to contribute to phytotoxicity (Mekonnen, 1994).

A variety of poisonous plants have caused extensive losses to the livestock industry in many parts of the world mainly East Africa including Ethiopia. They are still significant problems in numerous areas. Poisonous plants produce their toxic effects after being ingested and/or absorbed by animals which include physical upset, loss of productivity and death. Therefore, even though plants have vital nutritious role and providing the normal atmospheric oxygen, they will cause life threatening effect if they are toxic. In addition to this, some chemicals which are in natural environment i.e. soil, water or industrial origin may also be toxic to livestock (Bah, 2013). Ingestion or contact with some industrial products or by-products disposed on grazing area cause physical upset or death of an individual (Peattie *et al*., 1983) . Plant poisoning is due to either accidental ingestion of material eaten along with grass or willful consumption of poisonous plants when pasture is dry while most poisonous plants remain green all the year round. It is also more likely to occur in animals which have been moved from one part of the country to another. New importations are unfamiliar with the strange ingestion of their fresh surrounding. The livestock industry worldwide is encountering large economic losses due to poisoning by poisonous plants and their secondary compounds. Livestock poisoning occurs when animals consume these plants in large amount in a short period or graze over a prolonged period of time (Panter *et al*., 2007). Most of the poisonous plants remain green and attractive for hungry and thirsty animals. The poisonous plants reported in this review include both herbaceous and woody species. Animals under nutritional stress may be less able to detoxify plant toxins and may suffer relatively greater harm from the metabolic effects of the toxins. Thus, proper observation of grazing animals, good grazing management with prior knowledge of poisonous plants in the rangelands, and strategic supplemental feeding may help to mitigate the problem. Quantifying the magnitude of economic loss from poisonous plants to livestock production is not an easy task. This is because separation between losses due to diseases, accidents, and predators from losses caused by ingestion of poisonous plants can be difficult. Low reproductive performance and weight loss can be caused by disease and inadequate nutrition as well as by consumption of poisonous plants. It is known that some adverse effects of poisonous plants such as birth defects occur long after ingestion of poisonous plants (Holechek, 2002). Therefore the objectives of over viewing major toxic plants are:

- To assess major toxic plants of livestock and their seasonal variation.

- To evaluate the impact they pose on livestock industry.
- To explore possible recommendations in order to reduce losses on live stock for proper prevention and control of side effects.

MAJOR POISONOUS PLANTS THAT AFFECT LIVESTOCKS

Poisonous plants are a major cause of economic loss to the livestock industry. Many poisonous plants can function as useful forage. Poisoning occurs when conditions develop that allow or cause the animal to eat too much too fast. Most of these plants are safe for the average adult to eat in modest quantities. Notable major toxic plants of livestock importance are discussed as follows (Cook *et al*., 2009).

Bracken fern (western bracken)

Bracken fern *(Pteridium aquiline var. pubescens*) is poisonous to cattle, sheep and horses; sheep, however, are more resistant. Bracken contains thiaminase inhibitors that lead to the development of thiamine deficiency in horses that can be remedied by giving thiamine. Literature has indicated that bracken fern is also carcinogenic. Milk from cows that graze bracken fern may be hazardous to humans. All portions of the fern both green and in harvested hay are poisonous to livestock (Panter *et al*., 2011). .

Signs and lesions of bracken in cattle and sheep include: high fever, loss of appétit, depression, difficulty in breathing, excessive salivation, nasal and rectal bleeding, bloody urine, anemia, leucopenia, thrombocytopenia, hemorrhagic syndrome, hemorrhages on mucous membranes, plastic bone marrow and bladder tumors in cattle; in horses the sign include: loss of weight and condition, emaciation, progressive in coordination, marked depression, arched back with legs apart, twitching muscles, general body weakness, fast pulse, inability to stand, convulsions or spasms, pericardial and epicedial hemorrhage (Davis *et al*., 2011).

Bracken fern is widely distributed in many places around the world. Bracken fern grows on burned over areas, in woodlands and other shaded places , on hillsides, open pastures and ranges in sandy or gravelly soils. The plant starts growth in the early spring and usually remains green until the leaves are killed by frost (Stegelmeier *et al*.,1999). Poisoning often occurs during late summer when other feed is scarce, or when animals are fed hay containing bracken fern. The disease occurs after cows have consumed large amounts of the plant and is manifested in an acute usually fatal form. The disease is more chronic in horses. Cattle are affected by a nor-sesquiterpene glycoside called ptaquiloside, which causes bleeding and damage to the bone marrow. The disease has a delayed onset: Cattle may graze the plant for several weeks and then get sick and die. Poisoned animals seldom recover. If consumed over time, ptaquiloside can also cause cancer in the urinary bladder and gastro intestinal tract. These tumors often bleed, causing red urine (enzootic hematuria or redwater disease). Bracken fern poisoning in horses can occur when they are fed hay containing about twenty percent bracken fern over a period of 30 days. Signs of poisoning include weight loss, incoordination and lethargy. Horses may stand with their legs apart as so bracing themselves and may assume a crouching position with an arched back. Muscle tremors develop and the animal is unable to stand despite violent attempts to do so. Death will occur in several days to a week (Gardner *et al*., 2011).

Fig 1: Bracken fern has broad triangular leaves or fronds **(Panter *et al*., 2011).**

Animals seldom eat bracken fern if sufficient forage is available; so grazing should be delayed until

adequate forage is available. Young shoots are the most toxic and are relatively palatable in early growth stages. To eliminate livestock losses do not overgraze pastures and ranges. Make sure sufficient forage is available at all times to animals in infested areas. If necessary, supplement forage near the end of the grazing period. Do not feed hay contaminated with bracken fern. Poisoning can be treated with thiamine hydrochloride, saline cathartics, and possibly activated charcoal. Few cattle have recovered after signs of acute poisoning appear; however, horses in early stages of poisoning may be saved by intravenous injections of thiamine hydrochloride. The thiamine hydrochloride treatment should be given under the direction of your local veterinarian. In areas where cultivation is practical, the plants can be destroyed by cultivating the soil for two to three years (Ralphs *et al*., 2011).

Poison hemlock (*Conium maculatum*)

The plant species *Conium maculatum* (poisonous hemlock) is one of the most toxic members of the plant kingdom. There are numerous reports of deaths for a wide range of animal species including humans. *Conium maculatum* has a worldwide distribution; it is a very common plant species of a member of the family *Apiaceae* (formerly *Umbelliferae*) and the carrot family. The majority of the *Apiaceae* (*Umbelliferae*) plants produce different volatile oils in clearly delimited tissues of the fruits. It is known that *Conium maculatum* produces and contains piperidine alkaloids but the synthesis and accumulation sites have not yet been unequivocally identified. The location of secretory structures and the presence of essential oils and alkaloids were investigated (Corsi and Biasci, 1998). The concentrations and the relative proportions of the different Conium alkaloids appear to depend on different factors like temperature, moisture, time and age of the plant. The consumption of varying parts of the plants (leaves, fruits) can cause different degrees of clinical effects and there appears to be different susceptibility to toxicity between species. The primary time of year for poison hemlock is spring; often when there is insufficient forage available. At this time the plant may also be more palatable. The toxicity increases throughout the growing season and the roots become toxic only later in the year. Once dried, the toxicity is considered to be reduced but not eliminated. The plant causes different signs and lesions in different species of animals (Dougall and Maureen, 1996).

Signs and lesions in cattle:

Hemlock is more poisonous to cows than to other animals. The general symptoms of poisoning of cattle are: arthrogryposis, depression, diarrhea, gait incoordination, lateral rotation of limbs, muscle spasms, salivation, teeth grinding, torticollis, trembling, coffee-coloured urine and vomiting.Elimination of plant toxicants via milk by lactating animals is considered a minor route of excretion; however, it may be important when the health of the neonate or food safety in humans is considered. The alkaloids of *Conium maculatum* can be excreted via milk in cattle (Panter and James, 1992).

Sheep and goat:

The general symptoms of poisoning for sheep are ataxia, frequent urination and defecation, salivation, kinked tail, trembling, weakness and death (Lopez *et al*., 1999). Conium seed induced cleft palate and multiple congenital contractures in all kids born to pregnant, with hemlock treated goats. Multiple congenital contractures included torticollis, scoliosis, lordosis, arthrogryposis, rib cage anomalies, over extension and flexure and rigidity of the joints. (Panter and Jemes, 1992).

The hemlock belonging to the invasive weed species has wide adaptation ability. The hemlock is infected often by one or more virus strains such as ring spot virus, carrot thin leaf virus (CTLV), alfalfa mosaic virus (AMV) or celery mosaic virus (CeMV) (Howell and Mink, 1981). The mechanical control of the plant means hand pulling or grubbing. Hand pulling works are easier with wet soils and with small infestations. It is best to pull or grub out the plant prior to flowering. If extensive areas are covered by hemlock, chemical control is simpler. In this case one can use a synthetic auxin-like compound, such as 2, 4-D (2, 4-dichloro-phenoxy-acetic acid), which practically does not kill grasses (few exceptions are only). However, hemlock is a prolific seed producer, and repeated in next year may be needed to achieve complete control. In alfalfa the poison hemlock can be controlled with other selective herbicide such as hexazone (Panter and Keeler, 1989).

Nitrate-accumulating Plants

Many plants, both crops (oat hay, sorghum, corn, sudangrass, Johnson grass and beets) and weeds (careless weed, kochia, pigweed, Russian thistle and nightshade) can accumulate nitrate. Plants containing more than 1.5 percent nitrates (as KNO3) dry weight may be lethal to livestock. Nitrate poisoning can also occur in animals that consume nitrate fertilizers, machine oil and some natural well and pond waters. Plants differ in their ability to accumulate nitrate. Often the type of soil present and the form of nitrogen there influences the amount of nitrate that accumulator plants may contain. Drought conditions or cloudy weather may enhance nitrate accumulation. Treatment of nitrate-accumulating plants with 2, 4-D may also cause plants to accumulate excessive amounts of

nitrate. Nitrate accumulates primarily in the vegetative tissue of plants while the seed remains safe (Panter *et al.*, 2011).

Livestock may be poisoned after eating either harvested or non harvested plants, feed materials high in nitrate, nitrogen fertilizer, etc. Nitrates are converted to nitrite in ruminants. Nitrite causes the production of met hemoglobin, a form of hemoglobin that cannot carry oxygen.Thus, the effects of nitrate poisoning result largely to oxygen starvation or suffocation.The amount of plant material required to poison an animal depends on the amount of nitrate in the plant and on the rate at which the plant is eaten.

Animal consuming nitrate containing plants shows an acute symptoms like blue coloration of membranes of mouth, eyes and other mucous membranes (cyanosis), shortness of breath, staggering gait, death, chocolate-brown blood ,muddy cyanotic mucus membranes, congestion of rumen and abomasums (Ralphs *et al*.,2011)**.**

Nightshades (*Solanum spps*)

There are several species of nightshades that are toxic to horses, cattle, swine, sheep and poultry. The principal species that serve as examples of the genus are black nightshade *(Solanum nigrum),* silver leaf nightshade *(Solanum eleagnifolium),* and buffalo burr (*Solanum rostratum).* The toxins include a combination of a number of sugars and at least six different steroidal amines combined to form a variety of glycoalkaloids. One example is the toxin solanine. Drying does not destroy the toxin. Nightshade species are not very palatable to livestock. However, these plants often grow as weeds in hay and silage crops and small grains where they can be harvested with the crop and then fed to livestock (Panter *et al*., 2011).

Black nightshade (both the native and introduced varieties) is an annual six inches to three feet tall. Leaves are simple, ovate to lanceolate, entire to sinuate-dentate. Flowers are white; berries are black when ripe. It grows peripherally in moist areas of fields and pastures of disturbed loamy or gravelly soils. Silver leaf nightshade is a perennial that grows one to three feet tall with white, hairy leaves and stems. Leaves are simple, thick, lanceolate to linear, entire to sinuate. Stems and ribs usually have short stiff spines. Flowers are violet or blue; berries are yellow or orange (Stegelmeier *et al*., 1999). Nightshades are generally unpalatable and are not grazed by livestock except under the stress of overgrazing or in contaminated hay and grain. Poisoning by this group of plants does not always end in death. In acute poisoning the nervous symptoms develop rapidly. Death or recovery occurs within a few hours to one or two days. Death apparently is related to the paralysis. Chronic poisoning is accompanied by emaciation, rough hair coat, anorexia, constipation and ascitis. Signs and lesions of nightshade poisoning includes: labored breathing and expiratory grunt, salivation and nasal discharge, body temperature may be slightly elevated, yellow discoloration of the skin may occur in chronic poisoning, weakness, paralysis, and trembling, increased heart rate, fat may be yellowed and gelatinous, gall bladder may be distended, gastrointestinal irritation including inflammation, hemorrhage, and ulceration (Panter *et al*., 2011).

Losses can be kept at a minimum by good pasture management and weed control. Harvested forage such as hay, grain, or silage can be contaminated with nightshades. Contaminated forage can be fed if it is diluted (mixed) with nightshade-free forage: an on/off feeding strategy should be used. Animals being fed this diluted forage should be kept under close surveillance and immediately removed from the contaminated feed if signs of poisoning appear (Panter *et al*., 2011).

Cocklebur (*Xanthium strumarium*)

Cocklebur *(Xanthium strumarium)* is an annual plant that grows in dry lakebeds, river bottoms, disturbed and flooded areas of fields, pastures and roadways after water disappears. Poisoning occurs primarily in cattle, sheep, horses and swine when the plant is eaten in the cotyledon stage (seedlings). Signs of poisoning include labored breathing, nausea, vomiting and spasmodic contractions of the legs and neck muscles. Cockleburs are widely native annual plants that grow about three feet tall. Leaves of common cocklebur are roughly triangular or heart shaped and is covered with minute hairs on both sides. The brown or greenish brown fruit or burs are covered with stiff, hooked spines and contain two seeds. Cockleburs are found in moist waste place, flood plain, old fields, overgrazed pasture and stock watering place are common habitats (Burrows and Tyrl, 2001).

The toxic compound in cocklebur is concentrated in germinating plants. The seed and the two leaf-cotyledon stage of plants is the most poisonous. Although the toxin remain potent when these portion of the plant are preserved in hay. The toxin is not present in the four stages or the mature plant. Seed also contaminates grain used in concentrate ration feed to the livestock. Sign of cocklebur poisoning in cattle and pig includes depression, weakness, loss of appetite, reluctance to move, incoordination, muscle spasm, lying dawn, paddling of limb, and convulsion followed by coma and death within hours to days of consumption. Cocklebur should be eliminated with mowing, cultivation or herbicide use.While cocklebur is highly susceptible to herbicide, more than one treatment is required for complete elimination, because only one of the two seeds in a bur germinates in the 1st year (Phillips *et al*., 1997).

Fig 2: **Cocklebur** (Burrows and Tyrl, 2001)

2.6. Milkweed (Asclepias syriaca)

Several species of milkweed are poisonous to livestock. Labriform milkweed *(Asclepias labriformis)* is the most toxic milkweed poisoning occurring in chicken, turkeys, rabbits, horses, cattle, sheep, and goats with avian species being less susceptible than mammals. Most livestock losses are a result of hungry animals being concentrated around heavily infested milkweed areas such as corrals, roadways and bedding grounds. However, poisoning may also occur when animals are fed hay contaminated with milk weed. The narrow-leaf whorled milkweed species appear to be more palatable than the broadleaf species. Milkweed may cause losses at any time, but it is more dangerous during the active growing season. Leaves and other above ground parts of the plant are the typical cause of poisonings but roots may also contain toxic constituents. The whorled milkweeds contain a variety of glycosidic substances called cardenolides that are highly toxic. All animals may be affected. Sheep are most at risk but cattle, goats, horses, poultry and pets are also at risk. The primary toxicants are cardiac glycosides that cause gastrointestinal, cardiac and respiratory problems and can cause death if enough is consumed. Resins especially galitoxin in the milky sap may also contribute to the toxicity of milkweed (**Dougall and Maureen, 1996).**

Milkweed is often found in sandy soils of plains and foothills. It grows on ranges and abandoned farms, along roadsides, in pastures, in ditches, and in waste places or overgrazed areas. This plant gets its name from the milky, sticky juice that oozes out quickly when any plant part is broken. The toxicity of milkweed species is quite variable ranging from relatively nontoxic to highly toxic. Animals usually do not eat milkweed unless good forage is scarce or when plants have been frosted. Livestock owners can reduce losses by keeping animals out of milkweed-infested areas. Supplemental feeding usually is beneficial during trailing. Hay contaminated with milkweed can be poisonous to sheep and cattle and should not be fed to them. When possible, rid the areas of milkweed and then manage to prevent reinvasion. Milkweeds can be controlled with 2, 4-D plus picloram or glyphosate as a spot spray (**Dougall and Maureen, 1996).**

Signs and Lesions of milkweed poisoning depression, weakness, and staggered gait**,** difficulty in breathing with expiratory “grunting” sounds, dilation of pupils, weak pulse, loss of muscular control, elevated temperature, respiratory paralysis, degradation and necrosis and gastroenteritis (Cook *et al*., 2009).

Fig 3: Milkweed Greenish-white flowers are borne in umbrella-like cluster (Davis ***et al.*, 2011).**

White Snakeroot (Eupatorium rugosum)

White snakeroot *(Eupatorium rugosum)* contains the same toxin as rayless golden rod (tremetol compounds), and it is toxic to cattle, horses, sheep and goats. It affects all animals, including humans who consume milk from affected animals ("milk sickness"). It damages nearly all muscle types including the heart muscle, disrupting cardiac function. Death may result especially when an animal is stressed. White snake root is most commonly found in moist, shady, wooded areas. The primary danger occurs in late summer throughout the fall especially in overgrazed pastures or where there is insufficient food. All grazing animals can be affected by white snakeroot and the toxin passes in the milk, so nursing animals and humans are also at risk. Clinical signs include: depression, stiff gait, periods of sweating, normal or subnormal body temperature, labored or shallow respiration, muscle tremors, trembling, partial throat paralysis, jaundice, passage of hard feces, prostration, death (death may be sudden with no prior signs) (Hamilton and Mitchell, 1994).

Fig 4: White snakeroot. (Cook, 2009)

Castor oil (Ricinus communis)

Ricinus communis is commonly known as castor oil plant or Palma Christi or "Gulo" in Amharic. The seed contain ricin, an extremely toxic and water-soluble ribosome-inactivating protein; it is also present in lower concentrations in other parts of the plant. According to the 2007 edition of the Guinness Book of World Records, the castor oil plant is the most poisonous in the world though its cousin abrin found in the seeds of the jequirity plant is arguably more lethal. Castor oil long used as a laxative, muscle rub and in cosmetics, is made from the seeds, but the ricin protein is denatured during processing. Because ricin can quickly and repeatedly inactivate hundreds of ribosomes in multiple cells, the LD50 in adults is only about 22 μm/kg when injected or inhaled; ingested ricin is much less toxic due to the digestive activity of peptidases, although a dose of 20 to 30 mg/kg or about 4 to 8 seeds can still cause death via this route (Wedin *et al*., 1986). If ingested symptoms may be delayed by up to 36 hours but commonly begin within two to four hours. These include a burning sensation in the mouth and throat, abdominal pain, purging and bloody diarrhea. Within several days there is severe dehydration, a drop in blood pressure and a decrease in urine. Unless treated death is expected to occur within three to five days. If victims have not succumbed after this time, they often recover. Toxicity varies among animal species: four seeds will kill a rabbit, five a sheep and six an ox or horse, seven a pig and eleven a dog. Poisoning occurs when animals ingest broken seeds or break the seed by chewing. Intact seeds may pass through the digestive tract without releasing the toxin. Ducks have shown substantial resistance to the seeds. It takes an average of 80 to kill them (Union **County College**, 2013).

Fig 5: *The seeds of the castor oil plant contain ricin, one of the world's most lethal toxins* **(Cornell University, 2008)**

Kochia

Kochia (*Kochia scoparia*) or summer cypress is an annual weed. During drought years it has been used both as forage and often as pasture. However, kochia can cause a variety of problems in cattle and sheep. Although not common, it has been associated with oxalate and nitrate poisoning. Diets having much kochia will result in decreased growth rates and death can occur. The principal manifestation of kochia poisoning is photosensitization. Poisoning due to kochia apparently depends on the environmental conditions under which the plant is growing **(Davis** *et al*., 2011).

Kochia weeds can be found growing on abandoned farm land, cultivated land, ditch banks, road sides, waste lands, and other disturbed areas. It thrives in a great variety of environments and soil types. Kochia is very drought tolerant. Kochia weed should not be confused with prostrate kochia (*Kochia prostrata*), which is considered excellent winter forage. Cattle and sheep may become intoxicated after grazing kochia. Cattle are more susceptible to intoxication from kochia than are sheep. Cattle grazing pastures with a high percentage of kochia lose weight or gain it slowly. Cattle grazing near pure stands of kochia may show depression, dehydration, weight loss, photosensitization and discharge from the eyes and nose. They may show signs of polioencephalomalacia and death occurs frequently. The toxin may be any or all of the following compounds: oxalate, nitrate, sulfate, saponin and alkaloid. During periods of drought, livestock producers frequently ask if it is safe to feed kochia to their cattle. In general the advice given is to dilute the kochia with other hay if at all possible and watch the cattle closely for signs of poisoning (Cook et al., 2009)

Lupines

Sheep are frequently poisoned by feeding on lupine. Poisoning usually occurs when hungry animals are allowed to graze lupine. Losses may be especially heavy when hungry sheep are trailed through lupine ranges in late summer. Sheep and cattle have been poisoned by eating lupine plants that have been cut and dried for hay. Cows may give birth to calves with cleft palate and skeletal defects if the cows ingest certain lupines during early gestation (crooked calf syndrome). Five poisonous species are silky lupine (*Lupinus sericeus*), tailcup lupine (*Lupinus caudatus*), velvet lupine (*Lupinus leucophyllus*), silvery lupine (*Lupinus argenteus*), and lunara lupine (*Lupinus formosus*). Some lupine species are not poisonous to livestock and not all species cause birth defects. Piperidine and quinolizidine alkaloids (ammodendrine and anagyrine) are the compounds causing toxicosis and cleft palate as well as skeletal defects. Poisonous species of lupine are toxic from the time they start growth in spring until they dry up in fall. Younger plants are more toxic than older plants; however, plants in the seed stage in late summer are especially toxic because of the high alkaloid content of the seeds. Under proper conditions, some lupines make good forage. The amount of lupine that will kill an animal varies with species and stage of plant growth. It is not safe to let sheep graze species such as *L. argenteus* under any condition. Cattle may be poisoned by eating 1 to 12 lb of lupine without other forage. Smaller amounts are poisonous if cattle eat lupine daily for three to seven days. Hay containing lupines has caused poisoning and death of cattle (**Panter *et al*., 2011).**

To reduce losses poisoning can be reduced by keeping hungry animals away from lupines in the early growth stage, in late summer when the plant is in the highly toxic seed stage, and from dense plant stands at all times. Supplemental feeding is beneficial, especially when animals are trailed through lupine ranges. If animals

are poisoned on lupines, do not try to move them until they show signs of recovery (**Davis *et al*., 2011).**

MAJOR EFFECTS ON ANIMAL HEALTH AND PRODUCTION

Plants were grouped based on the major organ system affected by consumption of the plant. The times of the year with higher precipitation rates are April through May and October through November. The start of the rainy period is accompanied by intense growth of some plant species used for animal feed (especially grasses), a situation that is usually associated with increased accumulation of potentially toxic compounds such as nitrates. However, some plants accumulate more toxins during the dry periods, for example, the native plant *Mascagnia concinna*, which accumulates more cyanogenic glycosides during these periods **(Tokarnia *et al*., 2002).**

Gastrointestinal System

Ricinus communis (castor, higuerilla, palmacristi, ricino) is a naturalized plant one of the most potent lectins known. In general lectins cause necrosis of the cells lining the gastrointestinal tract. All animal species are sensitive to the effects of ricin. The toxicosis however is uncommon and it is usually associated with feeding garden clippings or with contamination of forage grasses with *R. communis* trimmings. Clinical signs include weakness, salivation, profuse aqueous diarrhea, dehydration, teeth grinding, and recumbence; the major postmortem finding is severe gastroenteritis (Aslani *et al*. 2007). Another plant compound highly irritating to the gastrointestinal mucosa is ricinoleic acid, a fatty acid present in *Ricinus communis* seeds, considered to be responsible for the cathartic properties of ricin oil. Ricinoleic acid is an irritant that alters the intestinal epithelium causing loss of water and electrolytes, increased loss of luminal DNA, and decreased enzymatic activity of enterocytes (Bretagne *et al*., 1981).

Circulatory System

Allium cape which includes all types of onions is capable of causing toxicosis in both large and small animals due to its content of organic sulfoxide especially alkyl or alkenyl cysteinyl sulfoxide. After ingestion the organosul-foxides are transformed into a complex mixture of organic sulfur compounds some of which are capable of causing intravascular hemolysis in cattle, sheep and horses. The excessive intake of onions leads to hemolytic anemia and methemoglobinemia which develops within a week of onion ingestion. Clinical signs in cattle include diarrhea, hemoglobinuria, ataxia and coma. Cattle are more sensitive than horses and goats are the most resistant. The hemolytic anemia caused by onion ingestion can also occur in dogs and cats. Another plant that causes intravascular hemolysis is *Brassica oleracea* (col silvestre), several varieties of which are used as forage for ruminants. *B. oleracea* contains the non-protein amino acid S-methyl cysteinyl sulfoxide (SMCO), which is reduced in the rumen to dimethyl disulfide hemolysin. The anemia induced by the intravascular hemolysis may be lethal in cattle which are very sensitive to the hemolytic effects of SMCO (Parton, 2000).

Cardiac glycosides are a specific type of toxic glycosides that affect the cardiac muscle sometimes causing fatal toxicosis. Cardiac glycosides increase the contraction force of the heart by inhibiting the myocardial Na^+-K^+ ATP-ase which can lead to cardiac arrest. Two types of cardiac glycosides are recognized depending on their chemical characteristics; namely cardenolide and bufadienolide glycosides. At least four plants containing cardenolide cardiac glycosides are present: *Digitalis purpurea*, *Nerium oleander* (oleander, delfa, adelfa, azuceno de La Habana), *Thevetia peruviana* (catapis, oleander amarillo), and *Asclepias curassavica* (bencenuco, mataganado). All have sporadically caused toxicosis in herbivores (Poindexter *et al*., 2007).

Urinary System

Urinary bladder tumors in cattle have been associated with the intake of *Pteridium aquilinum*. This weedy plant found worldwide grows in well-drained, acid soils and open lands. Cattle readily eat the plant when it is still young; old plants are normally not eaten unless there are no other plants in the pasture. This plant contains at least two important toxic components: a thiaminase capable of destroying Vitamin B1 and a mutagenic carcinogenic glycoside known as ptaquiloside. The toxicosis results from the chronic intake of ptaquiloside and its major sign is hematuria caused by the development of multiple bleeding tumors in the bladder mucosa (Pedraza *et al*., 1983 and Smith, 1997).

High levels of soluble oxalates that chemically correspond to sodium or potassium salts of oxalic acid are a common cause of plant induced nephrotoxicity. Soluble oxalates are readily absorbed in the systemic circulation where they can react with blood calcium, causing hypocalcaemia. Oxalates eventually form insoluble calcium oxalate crystals that block the renal tubules. Precipitation of calcium oxalate crystals in the kidney leads to anuria, uremia, and acute renal failure. Soluble oxalate toxicosis is more common in ruminants because the plants that contain them are usually more palatable and readily eaten compared with plants containing insoluble oxalates (C**heeke, 1995).** Native or naturalized grasses known to accumulate potentially toxic levels of soluble oxalates include *Brachiaria humidicola* (braquiaria alambre), *Cenchrus ciliaris* (Pasto buffel), *Digitaria*

decumbens (Pasto pangola), *Panicum maximum* (Pasto guinea, India, siempreverde),*Pennisetum clandestinum* (kikuyo), *Pennisetum purpureum* (Pasto elefante) and *Setaria sphacelata* (setaria, Pasto miel). Acute renal failure and perirenal edema have been reported worldwide in cattle, sheep, pigs, and horses that ate these plants. Signs and lesions in cattle include weakness, ataxia, high blood urea levels, proteinuria, perirenal edema, and nephrosis **(**Last *et al*., 2007).

Nervous System

Conium maculatum is commonly found along roadsides and close to irrigation waters. *Conium maculatum* contains at least five main piperidine alkaloids of which of the most important are coniine (mainly in the seeds) and γ-coniceine (in vegetative tissue). The other three alkaloids are N-methylconine, conhydrine and pseudoconhydrine. In world literature the toxicosis has been reported in horses, pigs, sheep and cattle with cattle the most sensitive species. The initial signs of the acute toxicosis include muscle weakness, tremors, incoordination and mydriasis followed by brady-cardia, depression coma and death from respiratory failure. Ipomoea carnea is native to tropical and subtropical country. The toxic compound of this plant was found to be the indolizidine alkaloid swainsonine that inhibits lysosomal hydroxylases, particularly the enzyme α-mannosidase. Livestock exposed to the toxin fail to gain weight and exhibit neurological alterations including failure to apprehend and swallow feed, hypermetria and ataxia (Antoniassi *et al*., 2007).

Phalaris aquatica (formerly known as Phalaris *tuberosa* and Phalaris *arundinacea*) are two species of phalaris. These grasses are of low palatability and consumption causes diarrhea. In cattle and sheep these grasses may cause acute toxicosis with sudden death of sub chronic toxicosis with transient neurological signs or chronic toxicosis with permanent neurological damage. Clinical signs are mostly neurological and include ataxia, aimless walking, muscular fasciculation, tremors, opisthotonus, excessive salivation, tetanic spasms and limb paddling (Bourke *et al*., 2005).

Musculoskeletal and Connective Tissue

The genus *Senna* (formerly known as *Cassia*) includes several species of plants known to induce myopathy in cattle, horses and pigs that graze on them or that eat feed contaminated with their seeds. Senna toxicosis causes myocardial degeneration, congestive heart failure and generalized degeneration of skeletal muscles. The muscle damage is accompanied by high serum activity of the enzyme aspartate amino transferase (AST) and creatine kinase (CK) and myoglobinuria. At postmortem examination the affected muscles look pale and show whitish striations (Barth *et al*., 1994). *Conium maculatum* may affect the musculo-skeletal system. In addition to being neurotoxic the alkaloids from *Conium maculatum* can also cause congenital malformations in calves particularly contractures of the musculoskeletal system and cleft palate. The sensitive periods for congenital malformations during gestation are days 40 to 100 for contractures and cleft palate and days 40 to 50 for cleft palate only (Panter *et al*., 1988).

Skin

Toxic plant-induced primary and secondary photosensitization is a common cause of skin lesions in livestock throughout the world. Secondary photosensitization results from impaired excretion of phylloerythrin. The flowers and seeds of *Fagopyrum esculentum* contain a conjugated photo-reactive quinone known as fagopyrin (Hagels *et al*., 1995), which has been known to induce primary photosensitization in cattle, sheep, goats and other animal species for many years. Primary photosensitization is caused by the reaction of the photo reactive compound in non-pigmented skin when it is exposed to solar radiation in the ultraviolet range. The photoactive compounds absorb solar energy forming reactive molecules (free radicals) that react with nearby macromolecules causing inflammation, erythema, edema, serous exudation, scar formation and skin necrosis. Several plants of the *Euphorbiaceae* family are potentially toxic due to their Content of phorbol type diterpene esters, which are highly irritating to the skin and mucosa and some are tumor promoters (Goel *et al*., 2007).

ECONOMIC IMPACTS OF POISONOUS PLANTS ON LIVESTOCK

Direct losses of livestock involve the economic impact of poisonous plants on the animal. These losses include such things as: deaths of livestock, abortions, birth defects, weight loss (due to illness or decreased feed intake), lengthened calving interval, decreased fertility, decreased immune response, decreased function due to damage to organs such as the nervous system, lungs, liver, etc., loss of breeding stock due to deaths, functional inefficiency, etc (Nielsen and James, 1991).

Indirect losses include those activities or costs that are incurred by a livestock operation to prevent losses or costs incident to livestock poisonings by plants. Indirect losses (cost) include fences built and maintained to manage livestock at risk due to poisonous plants, herding livestock to prevent poisoning, supplemental feeding to prevent poisoning, altered grazing programs which may result in increased costs or grazing inefficiency, medical costs incident to poisoning and forage lost because it could not harvested at the

proper time or intensity. These costs are difficult to quantify, yet are present (Nielsen and James , 1991).

CONCLUSION AND RECOMMENDATIONS

Toxic plants can adversely affect every organ system and pose a risk to animal health and production. There are potentially dozens of plant species in and along pastures and meadows and sometimes in hay that can cause toxicity problems to livestock. It is important to be aware of these plants and their toxicity symptoms. There are times such as early spring, during summer or droughts when forage supplies are low and this is when one needs to be most aware of what livestock is grazing. There are also situations where regardless of adequate forage, certain animal's just love to browse and end up consuming toxic plants or plant parts. Toxic plants are the major problem of livestock production. For effective prevention and control of plant poisoning it is worthwhile to apply the following fundamental measures as recommendation:

- Identification of plants that are poisons to the livestock.
- Improvement of management and hygienic practice of the pasture land.
- Notifying the problem to the veterinarian personal or toxicologist.
- Creating awareness of the season of most poisonous plants are growing and cause problem on animal health.
- Giving adequate feed during drought season.
- Testing plant toxicity and inspection of pasture.

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