Stock-poisoning Plants of Western Canada

by

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INTRODUCTION

The 2008 revision updates the 1983 version (38) in a series of such manuals produced by Agriculture and Agri-Food Canada. It describes the plants that most frequently cause poisoning and injury to livestock in Western Canada, as well as a number of plants that are potentially dangerous. Species found in both native rangelands and cultivated croplands are included. The use of grain concentrates for livestock feed is not addressed. The scientific name, botanical description, geographic distribution, toxic principle and conditions of poisoning are described for each major poisonous plant. Some additional species, with which certain toxic plants can be confused, are also described. Line drawings accompany some of the entries. Biochemical information on the toxic principle and its mode of action has been derived from current scientific literature. Original references are provided to document controversial topics. The reader is referred to recent reviews of the literature that give more detailed coverage of clinical topics by Radostits et al.(69), Burrows and Tyrl (17), Cheeke (19), Mayland et al.(51) and chemical structures by Majak and Benn (42). Recommendations aimed at prevention of livestock poisoning are given and some possible treatments are suggested.

Poisonous plants contain toxic substances that harm livestock. Some plants produce the toxin themselves while in others the toxin is produced by micro-organisms growing on or inside the plant. Some have spines that cause physical injury. Some plants cause sickness or death immediately after ingestion while with other plants clinical symptoms may not become apparent for several days or weeks. Chronic poisoning occurs when the damage is permanent. Early signs of poisoning can be detected in many cases and steps can be taken to prevent fatalities or further losses. Some plants cause skin blisters, abortions, birth defects or loss of weight. Incorrect mineral content of plants may lead to malnutrition or neurological disease. Another possible source of poisoning is drinking water. High levels of contaminants may make the water unpalatable, reducing consumption and animal performance. Water may contain toxic levels of organic contaminants, minerals, or toxic microorganisms.

Poisoned animals are usually hard to treat. There is often no antidote available or none that can be given under range conditions. Fewer losses will result if poisonous plants are recognized and considered in management plans. It is our hope that this manual will reduce the number of incidents that are misdiagnosed as disease or genetic flaws. The latter has caused breeding-stock producers not to report symptoms, and therefore has allowed feed-induced damage to continue unchecked. When losses occur it is important to assemble a detailed case history, including age and reproductive status of the affected animals, how long they have been in the herd, plus current and previous feeding regimes. Consult a veterinarian when stock dies to determine the cause of death and prevent further losses. After infectious disease has been ruled out, and it is suspected that poisoning by plants has occurred, take or send a specimen (include leaves, stems and roots) to the nearest applicable government agency or university. Positive identification can be made and control measures suggested. In cases of fatal poisoning, it may be possible to determine the cause from toxic plant fragments in stomach contents.

On native pastures control of native poisonous plants by methods other than livestock management is not widely practiced and is becoming less acceptable with the increased demand to maintain natural biodiversity on public land. The use of herbicides for the control of native poisonous plants may be effective in special cases where there are a few dense patches of the target species. Native plant communities are complex mixtures of species viewed either as edible or inedible and many herbs and shrubs are valuable as forage, both for livestock and wildlife. Loss of economically and ecologically valuable plants from herbicide usage is often greater than the gain. Also, herbicide use is severely restricted near sources of water. Digging up the entire plant is a practical way to eradicate small patches of a few species such as water-hemlock, but great care must be taken because of its high toxicity to humans.

Heavy losses caused by poisonous plants are often associated with one of the following conditions:

(1) The amount of toxin in a plant can fluctuate and it can vary with the **growth stage** of the plant. Livestock losses can be reduced if grazing programs are altered during hazardous stages of growth. Some poisonous plants have a short period of high toxicity. Plant habitat can also affect plant toxicity.

(2) **Overgrazing** causes good forage species to diminish in population density and vigor, allowing less palatable plants to increase. Many toxic plants are not palatable to livestock. Overgrazing not only occurs from having too many animals on a pasture but can also be the result of untimely grazing. Native pastures may be grazed too early in the spring, before the forage species, particularly grasses, have grown sufficiently. In Western Canada, death camas, low larkspur, saskatoon, chokecherry, arrowgrass and greasewood are some of the earliest range plants to show new growth.

(3) **Moving livestock** at any season of the year, and particularly during the spring and early summer, gives the animals an opportunity to graze poisonous plants. When animals are moved, their grazing habits are disturbed. If they are hungry, they are apt to quickly eat large amounts of easily available forage and to graze plants they would normally avoid. This can happen when cattle are held without feed overnight in a corral then trucked to a spring range site. Toxic introduced weeds often grow along roads. Any road improvement operation, such as that precipitated by a new logging sale, may cause the introduction of a new and toxic weed from seed carried on road building equipment. Therefore, areas that are known to have significant populations of poisonous plants should be avoided during trailing.

(4) Losses from poisonous plants may increase during **drought** because lack of feed forces stock to eat any plant that is available. Drought-reduced hay crops may necessitate the harvesting and storage of unfamiliar crops or weeds that require gradual introduction into the diet or dilution with other feed to minimize toxicity. There is also evidence that, under moderate environmental stress, some poisonous plants increase the production of toxic compounds.

(5) Ruminants (cattle, sheep, goats, deer, and llamas) and monogastric animals (horses, pigs, and rabbits) may differ in their **susceptibility** to poisonous plants. Plants that contain glycosides, where toxins are bound to sugars, can be much more hazardous to ruminants because microflora of the rumen can rapidly release the toxic agent from the intact glycoside. Susceptibility can also vary among ruminants.

(6) Livestock can reach through a fence to eat plants in a **garden** where aerial portions of domestic plants may be toxic. As well gardeners may discard toxic tree trimmings and other garden waste in adjoining pastures.

(7) Drinking **water** may be contaminated with toxic blue-green algae.

A comprehensive list of the poisonous plants of the Yukon has been published (48b).
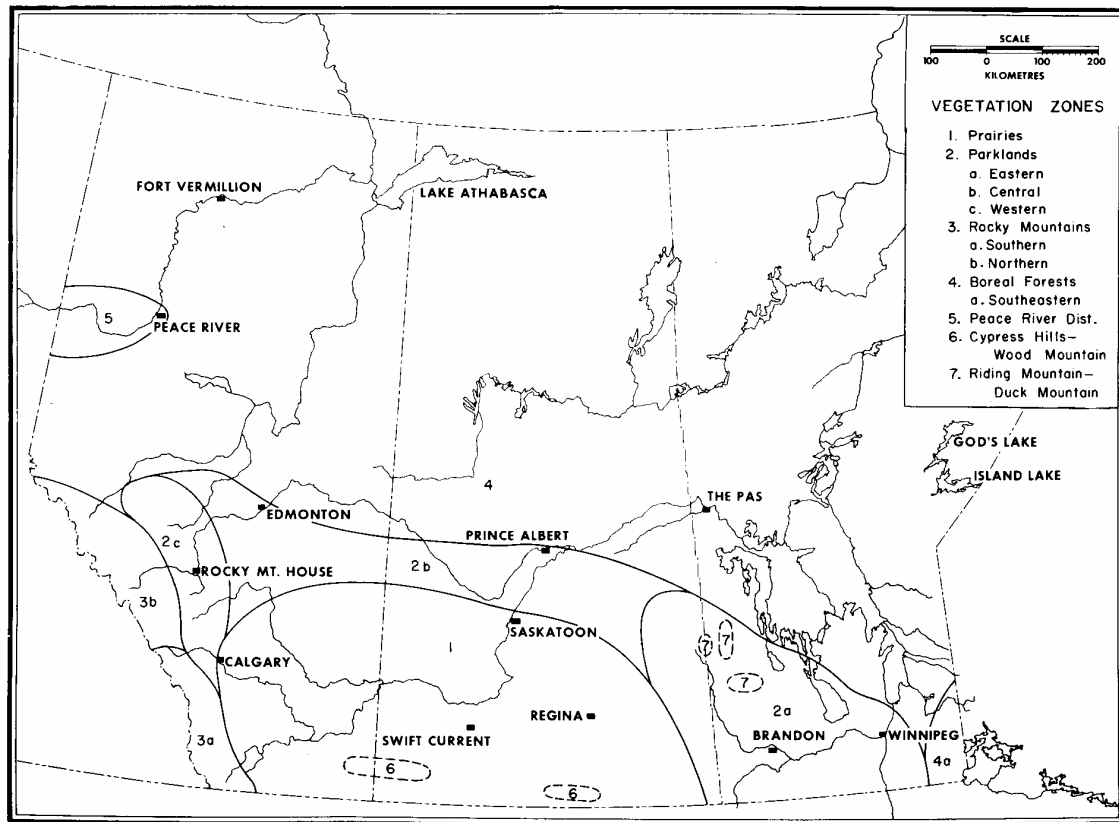
Map of the Vegetation Zones in the Prairie Provinces (38).

MAJOR NATIVE SPECIES

Saskatoon (*Amelanchier alnifolia* var. *alnifolia* (Nutt.) and var. *cusickii* (Fern.) C.L. Hitchc.), Rose Family, Rosaceae

DESCRIPTION AND HABITAT – Saskatoon is a shrub or small tree growing 1-10 m high, sometimes spreading by rhizomes to form dense patches. The leaves are simple, toothed only at the tip to about halfway along the margin. They are alternate, a characteristic that helps distinguish non-flowering stems from those of the non-toxic snowberry shrubs (*Symphorycarpos albus* (L.) Blake and *S. occidentalis* Hook.) which both have opposite leaves. Saskatoon flowers are white and grow in rather open, few-flowered racemes. The fruit is a sweet, purplish berry with many seeds. Saskatoon occurs throughout Western Canada in moist coulees, on rock-covered slopes, along creeks, and in open woodlands. It often occurs together with chokecherry.
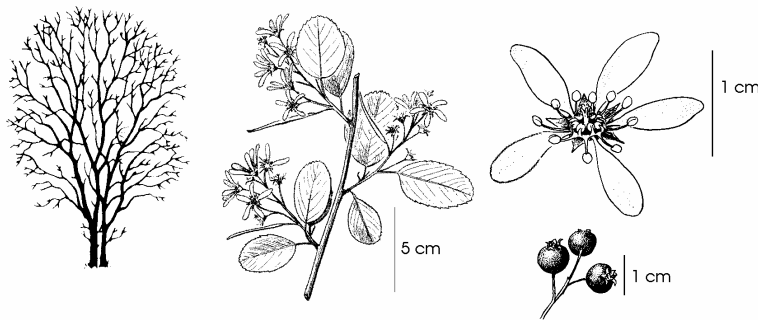
Saskatoon
(Amelanchier alnifolia)

TOXIC PRINCIPLE – Saskatoon is one of a group of cyanogenic (cyanide-generating) plants. These plants contain unique glycosides, compounds containing a sugar attached to a toxic component. Specific plant enzymes can trigger the release of the toxin from the glycoside. In the intact plant, membranes within the cell separate the enzymes from the glycoside. However, when plant cells are broken down, as when chewed or when disrupted by digestive microbes, the enzyme comes in contact with the glycoside breaks it down, and releases the toxin. In cyanogenic plants the toxin released is hydrogen cyanide. Because of the enzymatic activity of rumen bacteria, ruminants are more susceptible to cyanogenic plants than are monogastric mammals such as horses.

Cyanide is extremely toxic because it blocks aerobic cellular respiration, preventing oxygen utilization and decreasing energy production. Symptoms of cyanide poisoning include rapid or deep breathing, muscular spasms, staggering, convulsions, and bright red mucous membranes. Coma and death follow from lack of oxygen. For mature cattle, the lethal dose of hydrogen cyanide is 2-4 mg/kg body weight. The cyanogenic glycoside in Saskatoon is prunasin. Saskatoon leaves or twigs can contain as much as 3% prunasin which is equivalent to a content of 0.27% hydrogen cyanide on a dry matter basis. Therefore 3 kg of fresh leaves or 1.2 kg of fresh twigs can be enough to kill a 500-kg

(1100-lb) animal. Rate of consumption and digestion will moderate the action of the toxin. Poisoning usually occurs too rapidly for treatment under pasture or range conditions. Intraperitoneal injection of sodium nitrite and sodium thiosulfate is the classical treatment for cyanide poisoning.

CONDITIONS OF POISONING – Saskatoon begins growth early in the spring, commonly before much grass is available for grazing, and somewhat earlier than chokecherry. The dormant twigs can be toxic in winter. The buds and young foliage are highly toxic, especially during the early flowering stage. There are several varieties of this species, and intensive cyanide surveys have only been done on two. In the interior of BC, var. *cusickii* can be six times more toxic than var. *alnifolia*, especially in spring (16). The two varieties are readily distinguishable only when flowering. The more toxic variety has longer petals (more than 12 mm) and smooth leaves. Pubescence on *alnifolia* leaves disappears as leaves mature. At Kamloops, var. *cusickii* plants were limited to the river bottoms and on fairly open hillsides to less than 1000 m elevation, while var. *alnifolia* plants predominated at higher elevations, with a few specimens at low elevations. Ripe saskatoon berries contain very low levels of prunasin and they are not toxic. A survey of saskatoon shrubs in a nursery in Beaverlodge, Alberta showed that the foliage in all selections was cyanogenic.

Because the window for positive identification of the variety is short, a field "scratch and sniff" method can be developed by range managers during the bloom season when prunasin levels are high, for use throughout the year. Vigorously scratching the bark on a young twig with a thumbnail can damage enough cells to allow the release of hydrogen cyanide. Benzaldehyde, which is released at the same time, has the odour of almonds. Therefore, if a scratched stem gives off a strong almond smell, the shrub is probably toxic. Managers should ensure that stock is not forced by insufficient forage supplies or other stress to browse toxic saskatoon bushes. The foliage is quite palatable, to wild animals like deer and moose, as well as to cattle. Browsing one year can alter the shrub's growth pattern (which is largely the extension of the existing stems) causing growth of many shorter and easily-accessible shoots the following spring. In high-traffic areas, range managers should check cyanide content in saskatoon plants that have produced significant new growth in response to previous browsing.

Chokecherry *(Prunus virginiana* L.), Rose Family, Rosaceae

DESCRIPTION AND HABITAT – Chokecherry is a tall deciduous shrub that suckers readily and can produce a dense clump. The bark is marked with small lighter coloured bumps, which can be used to distinguish it from saskatoon during the winter. The small white saucer-shaped flowers have 5 rounded petals. They are borne in long clusters at branch tips, in May and June. The small cherry-like fruits are red or purplish to black, with a bitter, astringent taste. The hanging clusters of fruit remain on the shrub until late fall. The leaves are smooth on the upper surface, an oval with a toothed edge and sharp point.
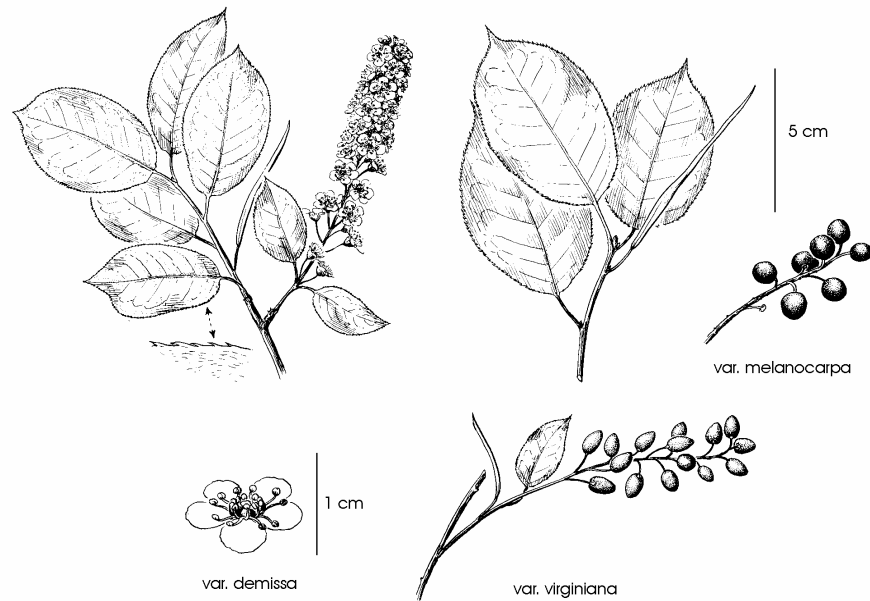
Chokecherry
(Prunus virginiana)

Chokecherry occurs throughout Western Canada, and is often found along fence lines. It is common in moist coulees and on creek banks in arid areas, and occurs abundantly on light soils. There are three varieties. The largest variety, *virginiana,* is in Saskatchewan and Manitoba. It is a large shrub or tree, up to 6 m tall, with thin leaves with a hairless lower surface, and crimson to deep red fruit. In Alberta and east of the Cascade Mountains of BC, the variety is *melanocarpa* (Nels.) Barnaby. It is rarely over 4 m tall and has rather thick leaves without hair on the lower surface, and deep purple to black fruit. The smallest variety is *demissa* (Nutt.) Torr. Usually 2-4 m tall, with a hairy lower surface on the leaves, it is only found west of the Cascades in BC. Lytton Mountain, at the confluence of the Thompson and Fraser Rivers, is the northern tip of the Cascade Mountain Range which intersects the national boundary at Manning Park.

TOXIC PRINCIPLE – Chokecherry contains the cyanogenic glycoside prunasin, as in saskatoon. The fruit is safe in small amounts, and all cyanide is released in cooking. Chokecherry is somewhat unpalatable to livestock, but it is browsed when other forage is scarce or when the new foliage emerges in the spring. The toxicity of the plant varies considerably, but the foliage is toxic at all stages of growth, and the buds, flowers, and twigs are potentially dangerous, the latter even in winter. When compared to saskatoon, the cyanide potential of chokecherry foliage is twice as great at the bloom stage, three times as great at the green fruit stage and ten times higher at the red berry stage. Accordingly, as little as 1.5 kg of fresh chokecherry leaves could be lethal when consumed at bloom stage, and 3 kg at subsequent stages of growth. Unlike saskatoon, the toxicity of chokecherry foliage does not diminish during the growing season. The cyanide

potential in twigs is similar to that in saskatoon, namely that 1.2 kg of fresh twigs can be lethal for a 500-kg cow.

Seaside arrowgrass (*Triglochin maritima* L.), Arrowgrass Family, Juncaginaceae

Seaside arrowgrass
(Triglochin maritimum)
toxic

Marsh arrowgrass
(Triglochin palustre)
toxic
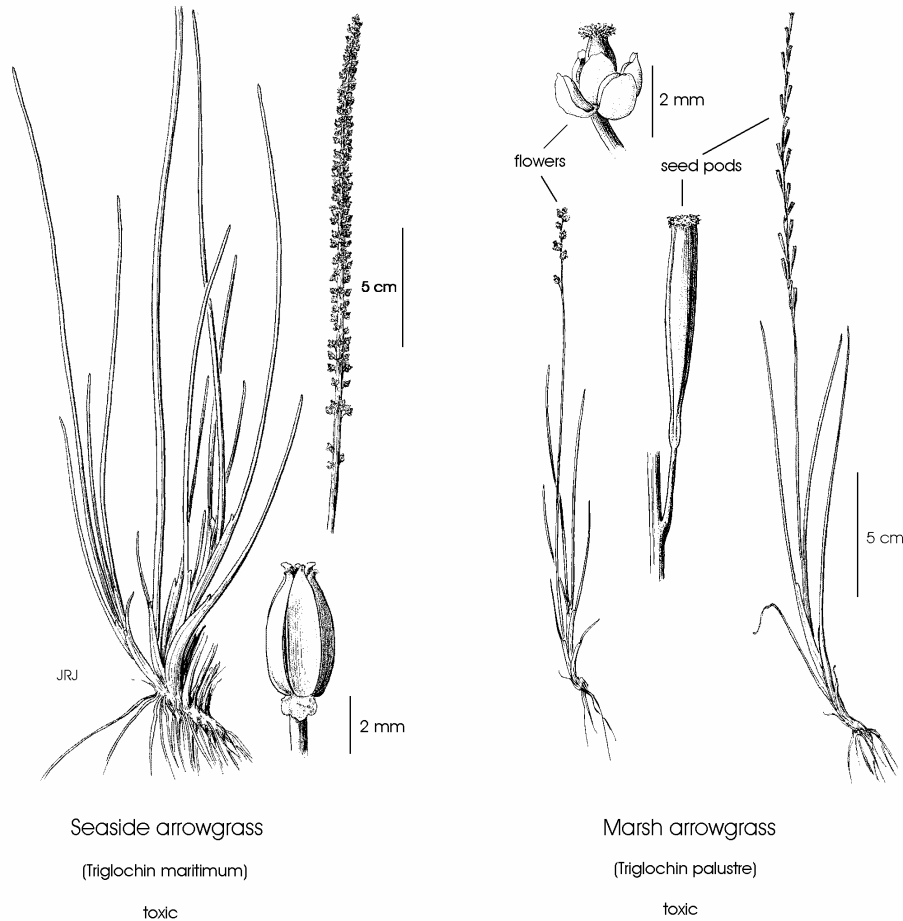
DESCRIPTION AND HABITAT- Seaside arrowgrass is an erect, rush-like or grass-like perennial herb 30-120 cm tall. It is clumped and not branched. Leaves are erect, basal, and much shorter than the flower stalks rising from a stout, often woody rhizome covered with papery remains of old leaves. New leaves are cylindrical, but flattened or dished on one side, smooth, shiny, and bright green with a spongy interior consisting of many narrow vessels. When crushed, they have a distinct "marshy" odour. The wind-pollinated flowers are 2 mm long and greenish, clustered along a spike-like flower stalk. Seedpods are three sided, about 6 mm long, lobed, and oblong. Seaside arrowgrass is widely distributed throughout Western Canada, up to 1,600 m elevation. Habitat type, rather than elevation, appears to limit its occurrence. It is common in salt marshes and on saline soils. In marshes, water is at or near the surface during most or all of the growing season.

Saline soils are free of surface water during most or all of the growing season and often are covered with a "white alkali" crust. Arrowgrass grows with sedges, rushes and slough grasses. After heavy grazing, its stubble can be difficult to distinguish from that of the slender members of the rush genus (***Juncus***) which has cylindrical stems.

TOXIC PRINCIPLE – As with saskatoon and chokecherry, arrowgrass is one of a group of cyanogenic plants. The cyanogenic glycoside in seaside arrowgrass is triglochinin; the highest concentration is found in new growth of leaves and flower spikes in spring (48). Fresh arrowgrass plants can contain as much as 3% triglochinin which is equivalent to 0.22% hydrogen cyanide on a dry matter basis. In short, 3.5 kg fresh arrowgrass can be lethal if consumed by a 500-kg animal. Poisoning usually occurs too rapidly for treatment under pasture or range conditions. Intraperitoneal injection of sodium nitrite and sodium thiosulfate is the classical treatment for cyanide poisoning.

CONDITIONS OF POISONING – Arrowgrass starts to grow earlier in the spring than do the associated grasses, rushes, and sedges. It also has a more rapid regrowth. This plant absorbs large amounts of salts from the soil, which can make it attractive to cattle, particularly when they are lacking salt. Triglochinin levels in arrowgrass are higher on non-saline sites than on saline sites. When cattle are moved to a new pasture they may be hungry, thirsty, and craving salt. This plant may be available at watering sites. Toxin levels in leaves can be substantially elevated during severe moisture stress (48). Furthermore, late in the season drought may reduce supplies of other forage, thus increasing consumption of arrowgrass. When made into hay, the plant gradually loses its toxicity during storage.

RELATED SPECIES – **Marsh arrowgrass** (*Triglochinin palustris* L.) also contains triglochinin. Very similar to seaside arrowgrass, it is a much smaller plant, up to 60 cm tall in contrast to 120 cm for seaside arrowgrass, and often occurs in smaller clumps. Its leaves have sharp-pointed tips (compared to blunt tips), and its seed pods are slender (compared to ovoid pods). Although not as common as seaside arrowgrass, it is as widely distributed in Western Canada. The species is also found elsewhere in North America and in Europe. Reports from these areas consider it as dangerous as seaside arrowgrass.

Meadow death camas (*Zigadenus venenosus* Wats. var. *gramineus* (Rydb.) Walsh.) Lily Family, Liliaceae

DESCRIPTION AND HABITAT – Death camas is a small, slender perennial herb 20-50 cm tall, with smooth, basal, yellowish green, grass-like leaves 3-6 cm wide. Thicker than those of grasses, they are "V" shaped in cross section with a keel on the underside. The many cream coloured lily-like flowers are less than 1.3 cm across and are borne on short stalks in a tightly packed cluster along the main spike-like stem. The plant grows from a dark-coated bulb, 2-4 cm long, which is generally 6-15 cm below ground. This bulb resembles an onion. Leaves appear early in the spring, before most of the grasses, and flowering occurs from April to early June. Seeds are formed in upright capsules about 7-12 mm long during the latter part of June and early July, after which leaves quickly

wither and adhere to the stem, which dies but remains standing. Death camas is common in southern Saskatchewan and Alberta, usually in upland draws and depressions. It is found in south central BC, east of the Cascade Range in mid to upper grasslands, and in openings in montane forests at all elevations.

leaves flat
in cross-section

5 cm

leaves V-shaped
in cross-section

bulb coat

Meadow death-camas
(Zigadenus venenosus)

toxic

Nodding onion
(Allium cernuum)

not toxic but can be confused
with death-camas
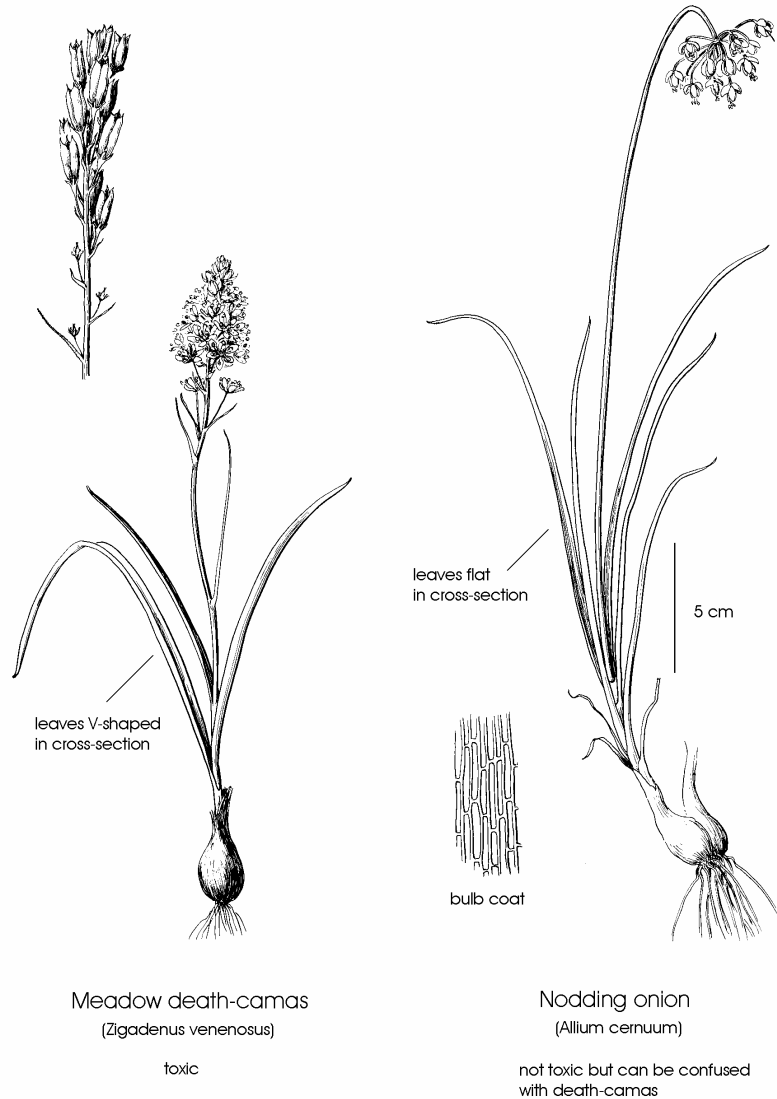
TOXIC PRINCIPLE – Death camas is one of a group of poisonous plants containing steroidal alkaloids. These compounds cause a reduction in blood pressure. Zygacine is the major alkaloid in death camas and it is also the major alkaloid in the very similar coastal variety, **deadly death camas** (Z. *venenosus* var. *venenosus*). Symptoms of poisoning are excessive frothy salivation, often followed by vomiting and then muscular weakness, incoordination, staggering, and finally, collapse and death. Animals may remain in a coma for hours before death. All parts of the plant are poisonous. About 0.2 kg of fresh plants per 50 kg of body weight constitutes a lethal dose for sheep (36). Rate of consumption and digestion will moderate the action of the toxin. Zygacine concentrations are higher from the vegetative to the bud stage of growth when the levels can exceed

0.4% on a dry matter basis (50). The toxin is retained in dried fresh tissue, so death camas is dangerous in hay. There is only a small decline in content as the plant matures and it can still be considered toxic after the leaves have faded and died. Subcutaneous administration of atropine sulphate and picrotoxin has been recommended during the early stages of poisoning.

CONDITIONS OF POISONING – The plant is tall enough to be grazed before most grasses are, so it is very dangerous during early spring. Cattle are occasionally poisoned, but sheep are the most often affected.

RELATED SPECIES – **White camas** (*Zigadenus elegans* Pursh) resembles meadow death camas in appearance but is taller, up to 60 cm, and more robust. Leaves are 6-12 mm broad and bluish green. Flowers are larger than those of death camas, 10-15 mm across, greenish or whitish cream, and are more scattered along the main stem, often occurring on branched stalks. White camas is found throughout Western Canada but it is not common. It prefers moist locations in rich soils. White camas does not contain zygacine (50) but it does contain other steroidal alkaloids, which may explain its apparent lesser toxicity. The resemblance of death camas to wild onion (*Allium* spp. L) has been the cause of human poisoning. The distinctive odour of all wild onion tissue is an easy tool for separating the plants, unless one's hands smell of onion before touching a camas specimen. At Kamloops, nodding onion (*Allium cernuum* L.) is relished by cattle and it often grows with meadow death camas. This onion has a round head of pink flowers in contrast with the spike of cream or white flowers in camas. If flowers are lacking, as in early spring and after grazing, the separation can be made using the leaves: nodding onion leaves are flat, rather than the V shaped cross section of death camas. For identification of this onion in the dormant season, note that the papery outer coat of the onion bulb is marked with long thin cells in regular vertical rows (34).

Ponderosa Pine (*Pinus ponderosa* Dougl.), Pine Family, Pinaceae

DESCRIPTION AND HABITAT – Ponderosa pine is a large coniferous tree typically 20-30 m, but up to 50 m tall. The straight dark green needles are 15-30 cm long and they usually occur in bundles of three. The tree has a characteristic thick and fire-resistant bark, in older trees divided into large reddish-orange plates that freely scale off.
It is a chief component of the vegetation of the drier region of southern interior BC, occurring in pure open stands, especially at lower elevation sites that have been subject to frequent forest fires. It also occurs in mixed stands with Douglas-fir and western larch at elevations up to 1500 m. The problem areas are confined to the valleys of the Fraser, Nicola, Thompson, Similkameen and Okanagan Rivers, eastward along the international boundary to Nelson and the southern portion of the Rocky Mountain Trench.

TOXIC PRINCIPLE – The diterpene acid, isocupressic acid, is the abortive agent (29). It interferes with the blood flow to the uterus and can induce abortion in cattle in the last 3 months of pregnancy. Premature births, stillbirths, weak calves that die shortly after birth and retained placenta can occur. Abortions can occur within 48 hours after fresh or dried

Ponderosa pine
(Pinus ponderosa)
toxic

needles
in bundles
of 3

10 m

5 cm
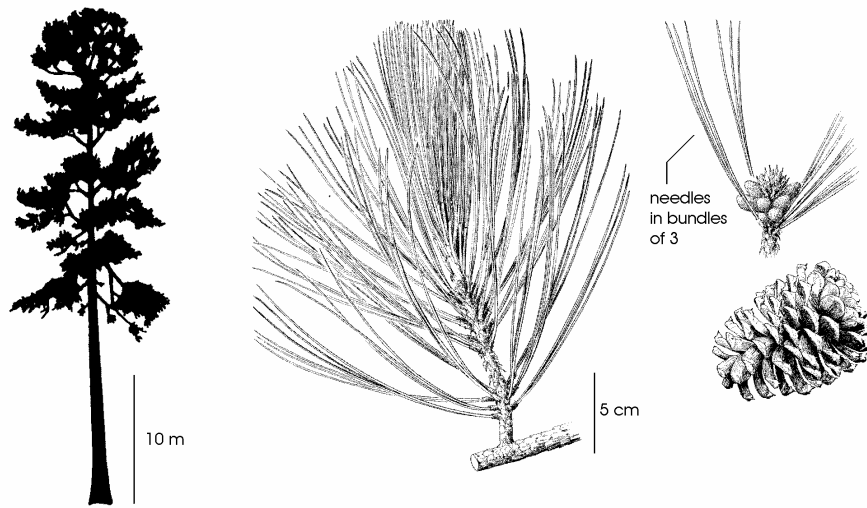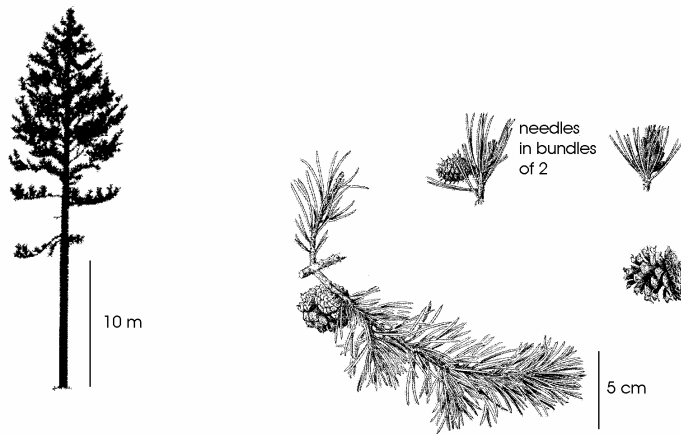
Lodgepole pine
(Pinus contorta)
toxic when a large amount is consumed

needles
in bundles
of 2

10 m

5 cm

pine needles have been consumed. Maternal toxicity and death have also been reported. A high incidence of retained placenta and illness of the cow help differentiate this from other causes of abortion. Other abortive agents, vaso-active lipids, have been isolated that affected reproduction in guinea pigs (26). Tap water extracts of ponderosa needles did not induce abortions   However, abortions have been reported at Kamloops where cows apparently drank snow melt-water occurring on ponderosa needles (J. McGillivray pers commun). Clusters of abortions have coincided with a thaw after a period of cold weather. Cows will periodically choose to drink from puddles, even though water is supplied in a trough. Isocupressic acid is partially water-soluble, with solubility

increasing under alkaline conditions (K.E. Panter pers commun). In ponderosa pine habitat, alkaline water-collection areas are common, so if snow melt-water collects in such an alkaline depression it can theoretically dissolve high levels of this toxin.

CONDITIONS OF POISONING – Sheep and goats are not affected. Cows will consume dead needles, and often there is little snow cover under the trees. It is advisable to winter the bred cows in areas free from ponderosa pine. When this is impossible, it may be prudent to prune the lower branches of the trees and clear the pasture of any fallen trees after storms. It may even be worthwhile to rake up needles. Logging operations in areas where bred cows are pastured should be discontinued because cows nibble on the slash even when they are adequately fed. It is dangerous to leave a discarded ponderosa Christmas tree where cows can reach it.

The calves born alive are premature but are otherwise normal, so it may be possible to save them with extra care and supplemental feeding. The premature delivery means that cows may initially supply insufficient milk, but typically, they are soon able to meet the calf's requirements. It has been observed that, inexplicably, new introductions to a herd and young cattle are more often affected by pine needles (J. McGillivray pers commun).

RELATED SPECIES – **Lodgepole pine** (*Pinus contorta* Dougl. ex Loud. var *latifolia* Engelm.) is common in the southern interior of BC and in western Alberta. Its needles are much shorter than those of ponderosa, 3-7 cm long, twisted, and usually in bundles of two. The bark is thin with small loose scales; the cones are much smaller, 3-6 cm long, slightly lopsided, and egg-shaped when closed. Early studies at Kamloops showed that lodgepole pine did not cause abortion when the needles were given free choice. Subsequently, isocupressic acid was detected in lodgepole and recent claims have been made that it has the potential for causing abortion in cattle. Levels found in bark and needles at Smithers, BC were slightly lower than those previously reported in ponderosa pine. In a study conducted in Utah (30) abortions were induced by force-feeding ground dried lodgepole pine needles but very high dosages were used.

Timber milkvetch (*Astragalus miser* Dougl. ex Hook var. *serotinus* (Gray) Barneby), Pea Family, Fabaceae (Leguminosae)

DESCRIPTION AND HABITAT – Timber milkvetch is a long-lived perennial, with several fine stems 20-50 cm growing from a tap root which is very resistant to forest fire. The pinnate leaves carry 7-13 small (10-18 mm), oblong leaflets. The pinkish mauve flower, like an elongate sweet pea, is generally 6-12 mm long; with few to many occurring along the flower stalk. It flowers from about mid-May, through July, and into August at higher elevations. The domestic-pea-like pod is 20-25 mm long, holding a number of black or greenish seeds. They mature between mid-July and mid-August and are shed soon after, as the pod splits open.

In BC timber milkvetch occurs in the fescue grasslands, in the ponderosa pine, lodgepole pine and Douglas-fir forests of the Cariboo and Chilcotin regions and in the Thompson, Nicola, Okanagan, Similkameen, Kettle, and Columbia-Kootenay valleys. It is sporadic but widespread, with an elevation range from 800 m to 1700 m. Although much less common than in BC, it also occurs in the fescue grasslands of the foothills of the Rocky Mountains in Alberta, from Jasper south to the border.

2 cm

5 mm
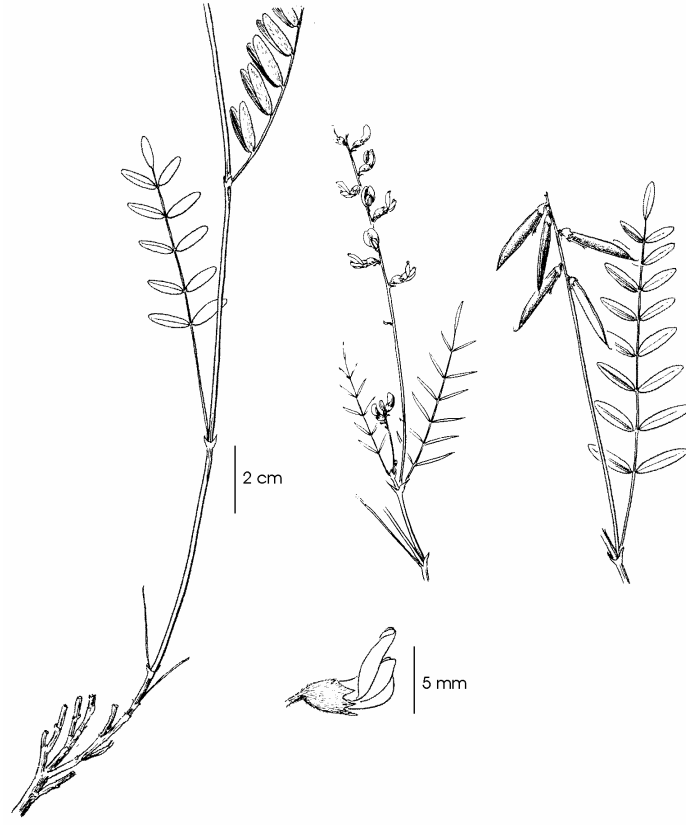
Timber milkvetch

(Astragalus miser var. serotinus)

toxic

TOXIC PRINCIPLE – The toxic principle has been identified as miserotoxin, a glycoside that is rapidly hydrolysed by rumen bacteria, releasing 3-nitropropanol.This nitroalcohol is absorbed and converted to 3-nitropropionic acid, a potent inhibitor of enzymes essential to respiration and energy production. Early signs of poisoning include placidity and stupefaction. Animals tend to close the eyes partially and twitch the ears; excessive frothy salivation is evident. Instability and continual shifting of weight from one leg to the other, a slow and elongated gait, and high-headed carriage may also be observed. Acute poisoning is characterized by staggers, laboured breathing, bluish mucous membranes, and sudden collapse. Chronic poisoning is characterized by general depression, poor body condition, knuckling of the fetlocks, and interference of the hind limbs causing a clicking sound, arching of the back, partial paralysis, diarrhea, difficult respiration and emphysema. Sudden exertion may cause death in both acute and chronic poisoning. In cattle 4 kg fresh timber milkvetch may contain the lethal dose for a mature animal (500 kg). Rate of consumption and digestion will moderate the action of the toxin. Poisoning can be prevented if animals are turned out in good condition and dietary protein levels are adequate. Rumen bacteria have the capacity to detoxify miserotoxin and this capacity is enhanced with protein supplements, which can be provided in a soft molasses block.

CONDITIONS OF POISONING – Miserotoxin levels are highest at early stages of growth but as the plant matures and the biomass increases, the amount of toxin per plant

also increases, allowing more toxin to become available to grazing livestock. There is a potential for using early grazing as a tool to manage rangelands infested with timber milk-vetch since the plant has poor regrowth vigor. Heavy rain after a dry period can promote regrowth, increasing miserotoxin levels in timber milkvetch by as much as 50%. Timber milkvetch is avoided and other forages are preferred when cattle graze fescue grassland ranges in good condition. In contrast, the plant is a preferred species in lodgepole pine forest range where it grows with pinegrass, which is less palatable. First and second-calf heifers are much more susceptible to poisoning than are older cows or yearlings. As well, "naïve" animals, those that have never been exposed to the plant, are also very susceptible. Both of these groups of animals can be protected if they are turned out in good condition and with adequate dietary protein supplementation. Miserotoxin can occur in the nectar and it is poisonous to the domestic honeybee.

RELATED SPECIES – A second variety of this species (*A. miser* var. *miser*) occurs in south eastern BC and southwestern Alberta, where ranges of the two varieties overlap. The variety *miser* typically is equally pubescent on both sides of the leaflet, while the variety *serotinus* is much less pubescent on the upper surface of the leaflet. The variety *miser* has not been tested for toxicity in Canada.
Other species of *Astragalus*, **Canada milk-vetch** (*A. canadensis* L.), **hillside milk-vetch** (*A. collinus* Dougl ex Hook.) and **Robbins' milk-vetch** (*A. robbinsii* (Oakes) Gray) also contain nitro toxins but these are less toxic to ruminants and more toxic to monogastric animals such as horses.

Tall larkspur (*Delphinium glaucum* S. Wats.), Buttercup Family, Ranunculaceae

DESCRIPTION AND HABITAT – Tall larkspur is a perennial with an elongated many-flowered inflorescence reaching up to 2 m. The 4 small petals are light blue, surrounded by 5 deep purple sepals 6-12 mm long. The upper sepal is elongated backwards into the spur for which the genus is named. The leaves are toothed, deeply cleft into three to five main lobes and are alternate on the stem, with short hairs on one or both sides. Lower leaves are 8-20 cm in diameter and round in outline. In the chemical literature, tall larkspur is also known as *D. brownii* Rydb. (37).

Tall larkspur has not been reported in Manitoba or southern Saskatchewan but it is common in the western boreal forest, foothills of the Rocky Mountains, and in the central interior of BC, ranging farther north and at higher elevations than low larkspurs. It is less common in the boreal forest of eastern Alberta and western Saskatchewan. It occurs in the margins of trembling aspen groves, in open forests of balsam poplar, spruce, or lodgepole pine and in willow thickets, as well as in grassy openings in forested areas. It prefers some shade, fairly rich soil, and a good moisture supply.

TOXIC PRINCIPLE – Tall larkspur contains a number of diterpenoid alkaloids, of which methyllcaconitine (MLA), a neurotoxin, is the most poisonous. It occurs in all native species of *Delphinium* in Western Canada. The MLA content of tall larkspur can exceed 0.8% on a dry matter basis. The primary and life threatening result of MLA toxicity is
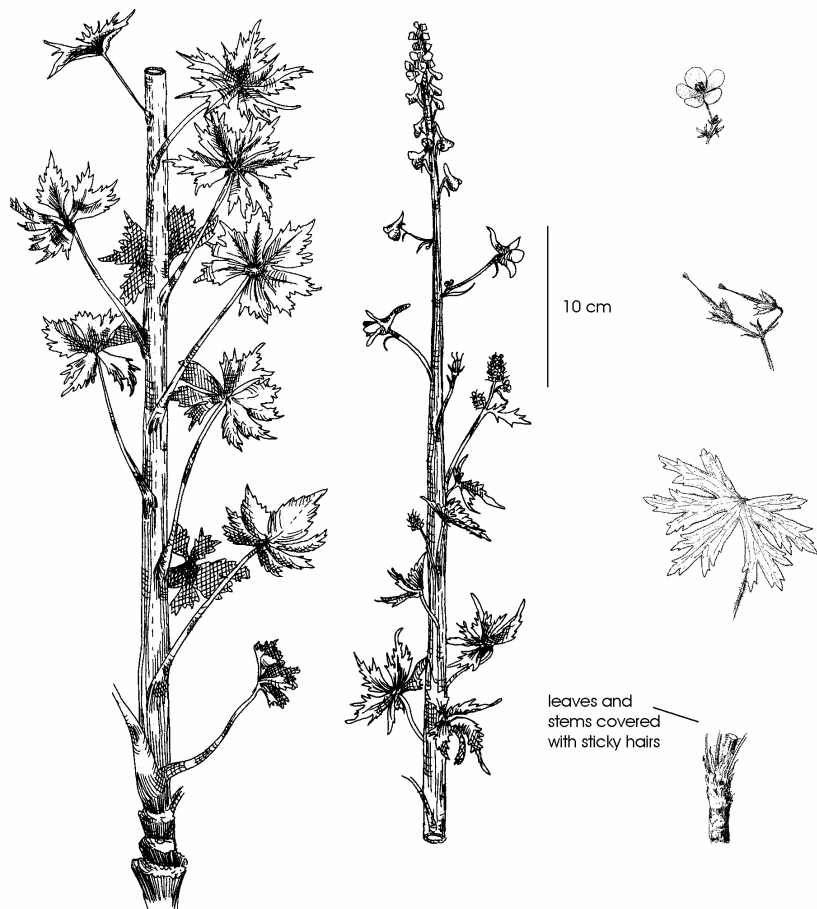
Tall larkspur

(Delphinium glaucum)

toxic

Sticky geranium

(Geranium viscosissimum)

not toxic but may be
confused with tall larkspur

10 cm

leaves and
stems covered
with sticky hairs

neuromuscular paralysis. Symptoms of poisoning include incoordination, muscular tremors and periodic collapse. Poisoned animals, when down, make desperate attempts to regain their feet. The lethal oral dose of MLA is 25 to 40 mg/kg body weight. Less than 10 kg fresh tall larkspur may contain the lethal dose for a mature animal (500 kg). Rate of consumption and digestion will moderate the action of the toxin. Intravenous (into the jugular vein) or intraperitoneal (into the flank) injection of physostigmine is an effective treatment when the animal is down and the treatment can be administered without undue stress to the animal. Treatment has to be repeated at short intervals. Stressful handling may precipitate death and therefore treatment of range cattle is difficult and impractical. Furthermore, physostigmine may not be readily available. Neostigmine, a similar but somewhat less effective drug, may be more available, because it is stocked at hospitals for diagnostic procedures (J. McGillivray pers commun).

CONDITIONS OF POISONING – Tall larkspur is among the first plants to emerge in spring, and its foliage is highly palatable to livestock at early stages of growth. Hence, cattle losses are heaviest early in the spring when tall larkspur can form a large part of the available forage. In the foothills of Alberta, where poisoning has frequently occurred, the plant becomes less palatable to cattle as it progresses to the bloom and pod stages of growth. Sheep can graze tall larkspur without evident damage and, because of this apparent immunity, sheep have been widely used in the control of larkspur. It has been confused with **sticky geranium** (*Geranium viscosissimum* F. & M. var. *viscosissimum*) early in the season because the leaves are very similar. These two plants can be distinguished before the tall flower stalks appear on larkspur: the geranium has fine, sticky hairs on its leaf stems, while leaf stems of tall larkspur are almost hairless and glossy. In the forest the distinction can be very difficult because hairs are barely perceptible on shaded geranium stems, even though they are obvious on plants growing in the sun. When the plants mature the much taller larkspur is easily distinguished from the geranium, which has shorter stems bearing symmetrical pink 5-petalled flowers. Early in the season it may be possible to use the previous year's delphinium flower stems to help in separating these plants from the geranium plants.

RELATED SPECIES – **Low larkspurs** are native perennials, similar in appearance to tall larkspur, but only reaching 50 cm in height, and occurring on drier sites. Leaves are usually hairy, are mostly basal, alternate, and deeply divided into a number of linear segments; they grow up to 5 cm in diameter and are round in outline. Flowers are up to 3 cm across, light blue to purple, with the uppermost sepal forming the characteristic spur up to twice the length of its blade. There are few to 15 flowers on short stalks along the main flowering stem. Roots range from fibrous to tuberous, even within some species. There are three species of low larkspur in Western Canada but none have been reported from Manitoba or eastern Saskatchewan. **Montana larkspur** (*D. bicolor* Nutt.) is common in the Wood Mountain area, west to the Cypress Hills and foothills of the Rocky Mountains in southern Alberta. In BC, it is widespread in the fescue grasslands of the east Kootenay and is replaced by **upland larkspur** (*D. nuttalianum* Pritz. ex. Walpers) farther west. Both species generally occur at elevations of about 700-1500 m, on well-drained sites in grassland and ponderosa pine forest, and often between shrubs. **Menzies' larkspur** (*D. menziesii*. DC. var. *menziesii*) occurs west of the Cascade Mountains.

Like tall larkspur, the low larkspurs contain MLA as the major neurotoxic alkaloid, but the concentration of MLA in low larkspur is a third of that in tall larkspur. In low larkspur, higher levels of MLA may be promoted under conditions of soil moisture stress, since plants from drier sites showed higher levels of the alkaloid. Early vegetative stages of growth yield the highest levels. Flower buds, flowers and pods of low larkspurs contain much higher levels of MLA than do leaves and stems. Low larkspurs are among the earliest-appearing plants in the spring and are tall enough to be grazed before most of the grasses. In upland larkspur the concentration of MLA is highest during early vegetative stages of growth when the plant is also very palatable to cattle but significant levels of MLA persist during bloom and pod stages. In BC, reports of poisonings have been infrequent, but two incidents in recent memory stand out. In spring of 1974 and 2004 upland larkspur was extremely abundant, tall and with many flowers, so that the

10 cm

Montana larkspur

(Delphinium bicolor)

toxic

Upland larkspur

(Delphinium nuttalianum var. fulvum)

toxic
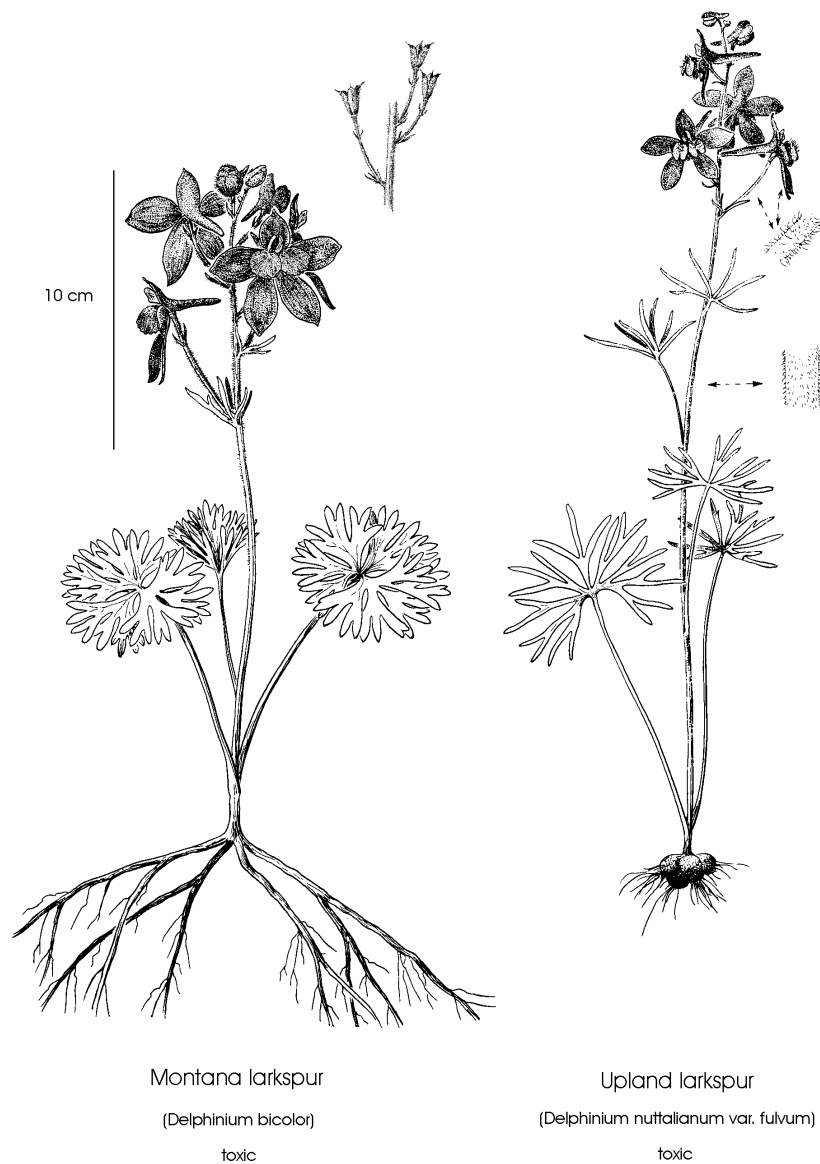
patches of vivid blue stood out on hillsides around Kamloops. In both years significant numbers of cattle died due to larkspur. The first spring was cool and moist with a late snow pack. The spring of 2004 was warm and dry. In 2004 the poisonings occurred over a period of about a week, in locations scattered around Kamloops, and the larkspur was grazed at the late bud to full bloom stage. Field observations indicated that the cattle were not hungry, but were consciously seeking out this plant for unknown reasons. Unlike tall larkspur, the low larkspurs are considered ephemeral: blossoms appear in May and early June, and seeds are formed in the latter part of June or early July, after which the above-ground parts quickly die back and disappear. This characteristic means that these toxic plants can be missed in a late-season evaluation of a range site.

Delphinium-leaved monkshood (*Aconitum delphinifolium* DC.) and **Columbia monkshood** (*A. columbianum* Nutt. var. *columbianum*) are native perennials closely related to tall larkspur. Several publications describe them as very toxic, but laboratory findings in Canada do not support this position. Analyses of all parts of delphinium-leaved monkshood from Jasper, Alberta did not detect MLA (2). Several alkaloids were identified, but the highly toxic diterpenoid aconitine alkaloids that occur in European and Himalayan species of monkshood were not found. The mixture of alkaloids in the local monkshood was similar for all plant parts but the total concentration was significantly highest in the flowers. It was theorized then that a large dose might cause problems to grazing livestock, but cattle graze in monkshood patches in Alberta and no cases of poisoning have been documented. O.T. Edwards analysed the aerial parts of Columbia monkshood from Vernon, BC and found similar results: no MLA or other highly toxic alkaloids (M.H. Benn pers commun).

upper leaves
close to stem

10 cm

pithy stem

tuberous roots
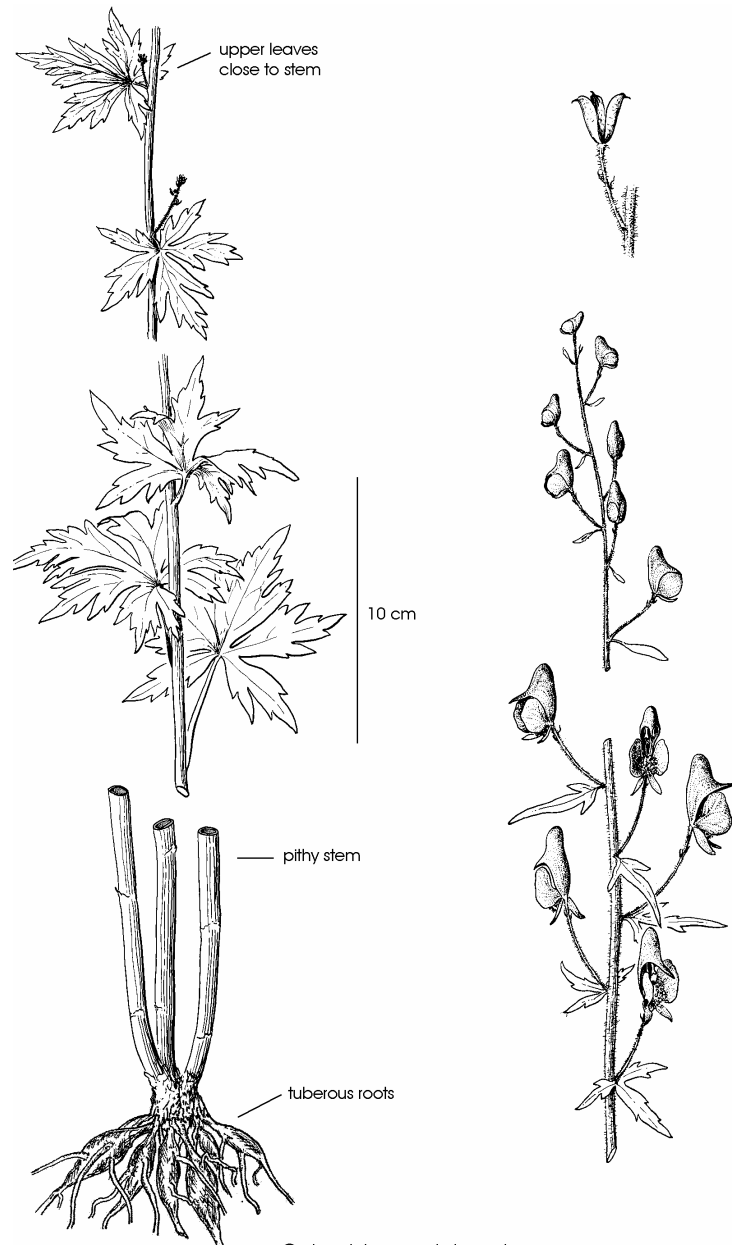
Columbia monkshood
(Aconitum columbianum var. columbianum)

not toxic but may be confused with toxic
introduced monkshood and with tall larkspur

This difference between the native and introduced monkshoods is important because several of the latter are grown in gardens here. In Europe monkshoods have been called "**wolfbane**" because of their historic use to kill wolves. Their reputation evidently led to the belief that our monkshoods are dangerous. We have not attempted to describe here how to separate our native species from introduced monkshoods. We have not found evidence that introduced species are invasive, so garden escapes should be easy to

recognize. The introduced monkshoods are so toxic that care should be taken to ensure that livestock and pets do not eat them; prunings from them should not be left within reach of livestock.

Monkshoods have much the same distribution and habitat as that of tall larkspur and often both are found growing together, though monkshood is much less common. Columbia monkshood grows up to 2 m tall and occurs in moist mountain meadows of the Rocky Mountains in southern BC and Alberta. Delphinium-leaved monkshood is up to 1 m tall and its range extends from the Aleutian Islands south through the mountains into BC and Alberta. It is found in sub alpine forest and alpine tundra, with plants only 10 cm tall in exposed, high sites. They look much like tall larkspur, but can be easily distinguished by flower shape. In contrast with the narrow spur of larkspur, the upper sepal is much inflated and arches forward over 2 large vertical side sepals, forming the "monk's hood" of its common name. Vegetative characteristics can be used to distinguish monkshood from tall larkspur early in the season. Roots of monkshood are short and tuber-like, whereas those of tall larkspur are long and fibrous. Upper leaves of monkshood are close to the stem; those of larkspur have petioles or stalks.

Water-hemlock (*Cicuta* spp.), Carrot Family, Apiaceae (Umbelliferae)

DESCRIPTION AND HABITAT – Water-hemlock is a large native biennial with the umbrella-shaped clusters of small white flowers that are typical of the carrot family. Note that the term "hemlock" is also applied to some completely unrelated plants found in BC, including western hemlock (*Tsuga heterophylla* (Raf.) Sarg.) a coniferous tree up to 60 m in height. Water-hemlock is considered to be the most toxic poisonous plant, so correct identification is important, both to reduce the risk of livestock consuming it, and to minimize tedious removal of patches of similar-looking but innocuous species. There is considerable variation within the genus *Cicuta*, and a large number of common names are in use. Water-hemlock mainly spreads by seed. The base of the stem is somewhat swollen and is characterized by hollow chambers separated by horizontal membranes. Roots, some of them noticeably thickened or "tuberous", are clustered around this stem base. When cut vertically, the chambers in the stem base and in the thicker roots exude a yellowish, aromatic, and extremely poisonous oil. The odor has been described as like raw parsnip (36), or parsley. Four species of water-hemlock, all toxic, are currently recognised in North America (53). Douglas' water-hemlock is described here, while the remaining three (spotted, European, and bulbous) are described in "Related species" below.

In **Douglas' water-hemlock,** or western water-hemlock (*Cicuta douglasii* (DC.) Coult. & Rose) thickened roots are largely situated below ground. Stems are stout, growing up to 2 m tall, hollow, and usually smooth. The leaves, 10-80 cm long, are alternate, smooth, with each leaf divided 1-3 times. The leaflets are 3-4 times as long as broad, and have saw-toothed margins. The vein branches in most of the leaflets go to the bottom of the V of the toothed edge: "veins to the valleys", a good characteristic to use in distinguishing three of the water-hemlocks from other members of the carrot family. The root system

Main leaf veins often run to valleys

20 mm

2 mm

Chambers in stem base and roots exude oil

bulbils in axils of basal leaves in fall

thickened tuberous roots
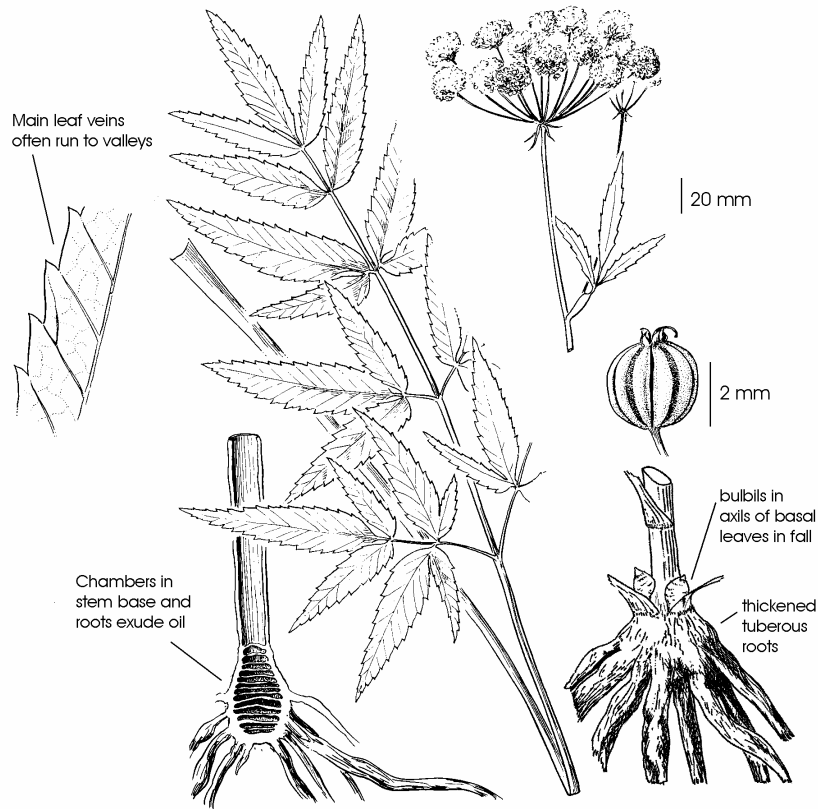
Douglas (Western) water-hemlock

(Cicuta douglasii)

dies after the plant flowers, but late in the season short stem branches, "globose rootstalks" (54) or "bulbils", form at the base of the stem. These winter-hardy stems allow patches of this plant to persist even when surrounding vegetation is too competitive to allow seedlings to survive. Douglas' water-hemlock occurs in western North America, throughout the northern Prairies and BC, except on the Queen Charlotte Islands, in habitats that are normally wet, such as slough margins, wet meadows and along stream banks (54).

TOXIC PRINCIPLE – The toxic principle, the oily substance found in the thickened roots and in lower concentrations in the new growth, is the polyacetylene diol cicutoxin. All species of water-hemlock are poisonous to all livestock, as well as to human beings. Poisonings of humans are on record throughout countries in which water-hemlocks occur. The toxin acts directly on the central nervous system causing extreme and violent convulsions and death from respiratory failure within a few hours of ingestion of the plant. A small dose is lethal and symptoms of poisoning appear rapidly, usually within 30 min after ingestion. The first symptom is excessive salivation and frothing at the mouth. Frothing is followed by tremors, uneasiness, and violent convulsions. Severe pain, especially in the abdomen, is evident. Clamping of the jaws and grinding of the teeth often occur, and the tongue may be lacerated. Relaxed periods with laboured breathing, loss of muscle control and lying down may occur between convulsive seizures (63).

Acute poisoning in sheep has been treated with pentobarbitol and atropine. Usually in a range situation the toxin acts too quickly for treatment to be an option.

CONDITIONS OF POISONING – Its occurrence in wet habitats means that grazing of the foliage in spring may result in roots being pulled out of the soft soil. In wet areas the thick roots may have little or no soil cover, and when a pond dries up, grazing animals may access previously submerged areas and easily uproot the plant. Green seeds are highly toxic (K.E. Panter pers commun). Concentration of the toxin drops during the process of curing but may remain a problem in contaminated hay. The level continues to drop slowly during storage. Therefore the longer contaminated hay is stored before being fed, the lower the risk (K.E. Panter pers commun). Attempts to control the plant by cutting may result in stimulating late-season regrowth that will be attractive to animals in a drought. For small populations in high-use areas, digging out the entire plant may be the best control method. After the top growth dies in the fall the remaining bulblets are easily separated so care should be taken to collect all of them. Wearing gloves is essential.

RELATED SPECIES – A very similar species is **spotted water-hemlock**, or spotted cowbane (*C. maculata* L. var. *angustifolia* Hook.). The most widely distributed *Cicuta* in North America; it is common in BC east of the Coast and Cascade Mountains, and throughout the Prairies. It is as tall and as toxic as Douglas' water-hemlock, but its stems may have purplish spots. As in that species, leaf vein branches generally run to the notches of the leaflet margins, but its slimmer leaflets can distinguish it. They are 5 times as long as broad, in contrast with 3-4 times as broad for Douglas' water-hemlock. The species of water-hemlocks can be distinguished by seed characteristics (53). Spotted water-hemlock is found in the same habitats as Douglas' water-hemlock, but the thick roots tend to be mainly above ground. **European water-hemlock**, (*C. virosa* L. or *C. mackenzieana* Raup), widely distributed in Europe and Asia, occurs infrequently in north-eastern BC and across the northern Prairies. It is smaller than the two more common species, up to 1 m tall, and may be less toxic than the two larger species. The leaflets are narrower than those of Douglas' water-hemlock, and the midvein on the upper leaflet surface is rough-hairy in contrast with a smooth midvein on spotted water-hemlock. Leaflet vein branches run to the leaflet notches. **Bulbous water-hemlock** (*C. bulbifera* L.) also grows up to 1 m tall, and is distinguished by bulblets in the upper leaf axils (flowers are often absent, and if present do not produce seeds). It has very narrow leaflets, and leaf vein branches do not run to the notches of the margins. It is found in northern BC and the Prairie Provinces, and is less toxic than the three other water-hemlocks (53).
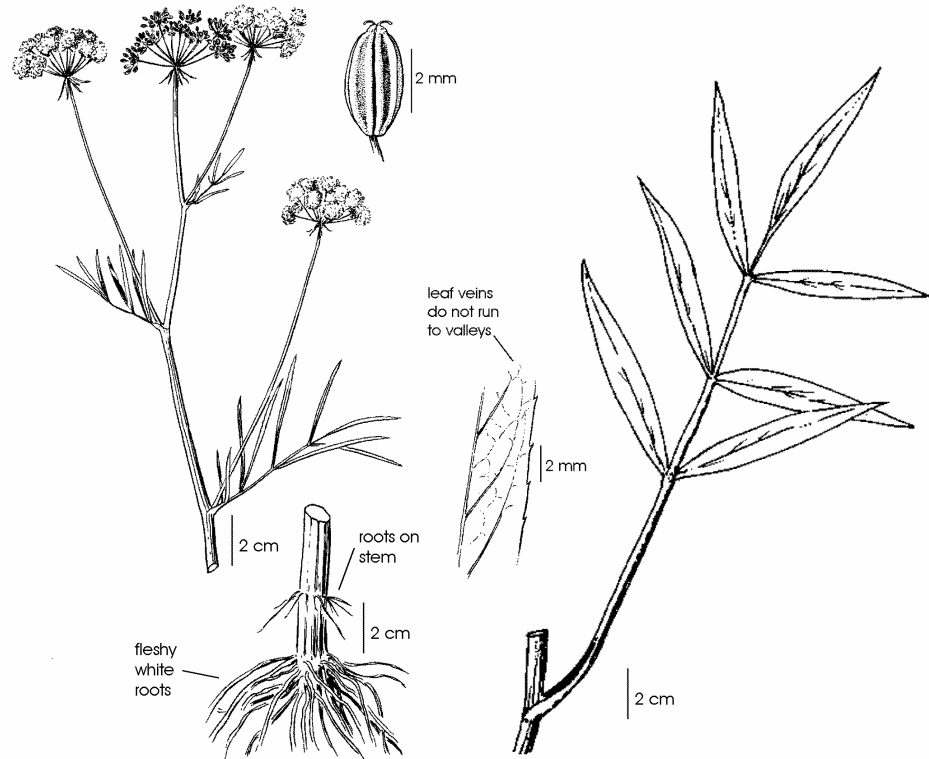
Water - parsnip

(Sium suave)

not toxic but may be confused
with water-hemlock

Water-parsnip, (*Sium suave* Walt.) is a 1.2 m perennial of slough or lake margins, which has often been confused with water-hemlocks. Water-parsnip has once-divided leaves (pinnate) with long, almost linear stemless (sessile) leaflets. They do not have prominent veins that extend to either leaflet tooth tips or bases. The first leaves in the spring are quite different from later leaves; rather fern-like and often submerged. The stem is not enlarged at the base and the root is not conspicuously chambered in cross-section. In fact, the taproot tends to lie horizontally, with the tip dying and becoming dark brown and brittle in older plants. The living root at the stem end bears thick fleshy white roots. This plant tends to grow in areas that are flooded for part of the season. Analysis of roots collected at Kamloops in July 2002 found small amounts of polyacetylenes similar to that observed in Douglas' water-hemlock (M.H. Benn pers commun).

Cow parsnip (*Heracleum maximum* Bartr. or *H. lanatum* Michx.) has large conspicuous flat-topped clusters of fragrant white flowers. The entire plant is hairy. Reaching up to 3 m in height, its leaves are usually very large, divided into three large leaflets. It is not semi-aquatic, but grows on stream banks and in moist low ground, in lowlands to moderate elevations in mountains. Due to its content of furanocoumarins it is suspected of causing primary photosensitization (17). Nevertheless, preferential consumption of large amounts of this plant by cattle, without significant symptoms, has been observed.

The concentration of furanocoumarins is much higher in **giant hogweed** (*Heracleum mantegazzianum* Sommier & Lev.) which is very similar in appearance but grows up to 5 m tall. Introduced from the Russian Caucasus as an ornamental, it has now escaped to riparian areas in south coastal BC. The photosensitizing constituents of giant hogweed are so concentrated that skin contact with the sap makes mechanical control risky for workers. However, grazing animals are not generally at risk. In Europe it is controlled by grazing sheep (5).

5 mm

5 cm

Cow parsnip
(Heracleum lanatum)

not toxic but may be confused
with water-hemlock

Negligible amounts of furanocoumarins are contained in **angelicas**, native perennials which can easily be confused with the water-hemlocks. However, in contrast with water-hemlock, angelicas are usually single-stemmed with a thick taproot. Their leaves are twice-divided, with the divisions having three to seven leaflets. Veins usually run to the points of the teeth of the leaflet margins, rather than to the valleys as in water-hemlocks.

Kneeling angelica (*Angelica genuflexa* Nutt.), up to 3 m tall, **Dawson's angelica** or mountain parsnip (*A. dawsonii* S. Wats.), up to 1.2 m tall, and **sharptooth angelica** (*A. arguta* Nutt.) up to 2 m in height, occur in moist meadows and forests in BC and Alberta. In kneeling angelica, which is found throughout BC including the Queen Charlotte Islands, and in the Peace River and boreal forest of Alberta, the leaf axis is sharply bent and the leaflets are reflexed downward, making the leaf look wilted. Dawson's angelica has a whorl of toothed bracts just under the cluster of pale greenish-yellow flowers. Sharptooth angelica has a strong odour, slightly reminiscent of celery, the flowers are sometimes pinkish, and it has a large parsnip-like taproot. Larger specimens of this root

have chambers that exude an oily liquid when cut, and therefore could be confused with the chambered root of water-hemlock. Fresh root samples of sharptooth angelica collected in July 2002 at Merritt BC yielded small amounts of furanocoumarins (M.H. Benn pers commun).

whorl of toothed bracts

leaf veins run mainly to points

2 cm

2 mm

10 cm

petiole sheathing stem more so with upper leaves

2 mm

parsnip-like root

10 cm

Sharptooth angelica

(Angelica arguta)

Dawson's angelica, Mountain parsnip

(Angelica dawsonii)
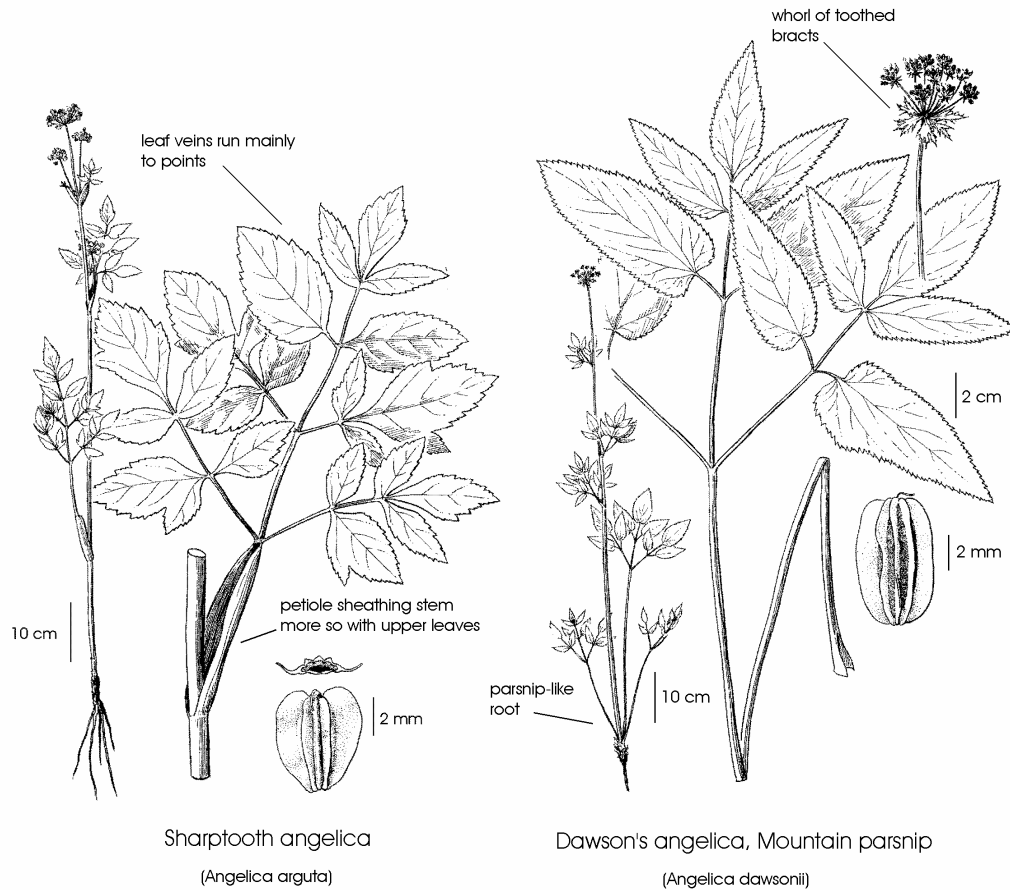
neither is toxic but may be confused with water-hemlock

Leaves of young shoots of an unrelated native species, **red elderberry** (*Sambucus racemosa* L.), can be confused with those of water-hemlock. Although much less toxic than the water-hemlocks, all parts of this shrub contain significant amounts of a cyanogenic glycoside (see Saskatoon). Leaves of water-hemlock are alternate on the stem while those of elderberry are opposite. Another related plant, **poison-hemlock**, is described in the Weeds section.

OTHER NATIVE SPECIES

Bracken (*Pteridium aquilinum* (L.) Kuhn var. *pubescens* Underw), Fern Family,
Dennstaedtiaceae (Polypodiaceae)

This native fern is an herbaceous perennial, growing to 50-210 cm. The aboveground part
of the plant, the frond, is a finely divided leaf with several main branches forming a
broad-based triangle in outline, which is held high on a strong woody petiole. This fern
has a strong, very long-lived hairy rhizome, and the fronds grow singly rather than
grouped in rosettes as in most other ferns. The rhizome enables this plant to survive
attempts at control by burning and cutting. Fronds develop by unrolling. The immature
leaf may be called a "crosier", or a "fiddlehead" because of its similarity to the curled
neck end of a fiddle (violin). Fiddleheads of bracken are consumed by humans in many
countries, despite their toxin content (17). The fiddleheads produced in the Maritime
Provinces of Canada are from the non-toxic Ostrich fern (*Matteuccia struthiopteris* (L.)
Todaro). Bracken is found in eastern Manitoba, the Riding Mountains of Saskatchewan,
southwestern Alberta and BC.

broad-based
triangle outline

2 cm

fronds
grow singly

20 cm
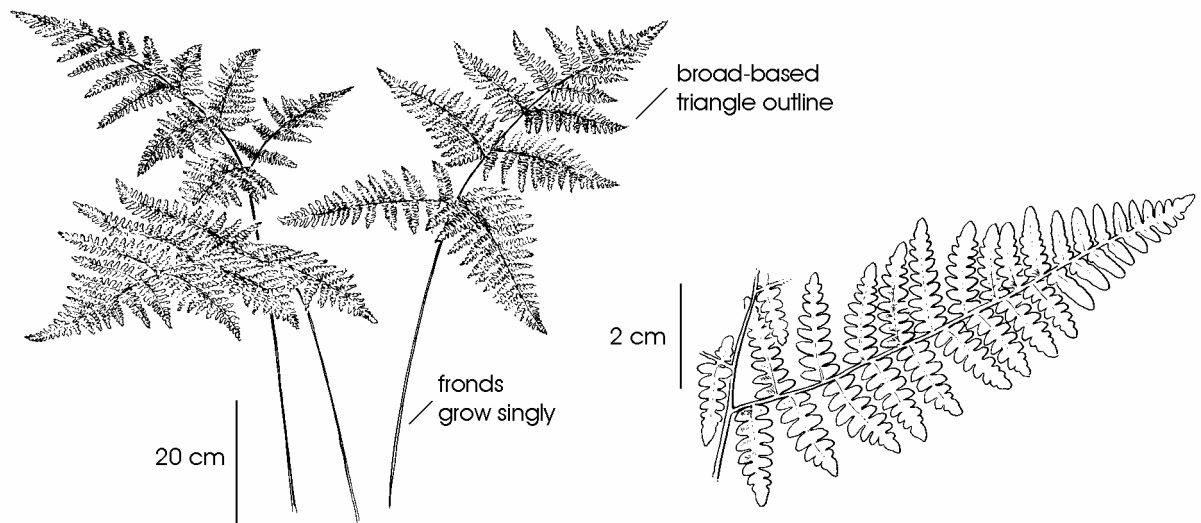
Bracken
(Pteridium aquilinum)

toxic

Bracken contains several toxins, including the enzyme thiaminase and the glycoside
ptaquiloside. Thiaminase splits the B vitamin thiamine, which renders it inactive.
Rhizomes contain more of the enzyme than do fronds. The enzyme content in rhizomes is
highest during summer, while in fronds it is highest during early spring growth and it is
retained in dry bracken. Thiamine deficiency induced by bracken is most common in
horses, but has been observed in pigs consuming the rhizomes. Bracken does not usually

cause thiamine deficiency in ruminants but it was encountered in starving cows which were pulling up and consuming the rhizomes near Chilliwack, BC (R.J. Clegg pers commun). A post mortem may reveal signs of heart failure but low blood levels of thiamine will indicate bracken poisoning. Symptoms in horses include loss of appetite, lethargy, and uncoordinated movement. The horse assumes a crouching stance, with arched neck and feet placed wide apart. In the later stages tremors develop and the animal is unable to remain standing despite frantic efforts to get up. Consumption of hay containing more than 20% bracken produces symptoms in about a month, and removal of the toxic feed will often suffice. Treatment is an intravenous injection of a thiamine solution, which gives quick reversal of symptoms in all but the latest stages of poisoning.

The sesquiterpene glycoside ptaquiloside causes a number of symptoms. It is more concentrated in the fronds. Young tissue contains 5 times as much of this toxin as do the mature fronds, and it is retained in dried bracken. Sheep are only poisoned occasionally, and non-ruminants like horses are resistant to this bracken toxin. Problems are most often seen in cattle, resulting from high consumption of fiddleheads. About 10% of ingested ptaquiloside is excreted in cows' milk. Ptaquiloside depresses bone marrow activity, producing effects similar to radiation damage, causing a severe decrease in white blood cells and platelets which leads to bleeding. This complex is called "bracken poisoning" or acute hemorrhagic disease. Typical symptoms are blood in feces, bleeding from the nose, vagina, and membranes of the eyes and mouth (note that sweet clover hay can also reduce blood clotting efficiency). A high fever develops in the latest stages. Ptaquiloside also causes bladder cancer in ruminants, resulting in blood in the urine, termed clostridium hemolyticum disease or enzootic hematuria. In sheep retina degeneration and "bright blindness" has occurred after prolonged consumption of bracken. The name is derived from the increased reflectivity of the tapetum of the eyes. Ptaquiloside is thought to be the cause, and recovery is possible if sheep are removed from the pasture soon enough. Symptoms induced by this toxin are generally considered untreatable.

A large amount of bracken is required to produce symptoms in livestock. It has been successfully used as forage when diluted to less than 50% of the diet and/or alternated with other forage every few weeks (17). It is important to note that thiaminase and ptaquiloside are both retained in hay. In ruminants, diagnosis of bracken-induced poisoning can be difficult because symptoms may not appear until several weeks after removal of the animals from feed containing bracken.

Golden corydalis, (*Corydalis aurea* Willd.) and **Pink corydalis** (*C. sempervirens* (L.) Pers.) Fumitory family, Fumariaceae

Golden corydalis is a sprawling annual or biennial standing about 30 cm above the ground. It has smooth, often silver-tinged, much-dissected fern-like leaves (twice or more-divided), extending above the loose racemes of golden yellow flowers. One upper petal is inflated into a rounded spur at the stem end of the flower then arches up to form a hood at the opposite end. It closely resembles bleeding heart (*Dicentra* sp.). It is widely

distributed throughout the Prairie Provinces, but in BC it is mainly east of the Cascade Range. It occurs commonly in moist, open forests, and sub alpine areas.

5 mm

Yellow flower

pink flower with yellow tips

5 mm

5 cm

long petioles

5 cm

sprawling growth habit

Golden corydalis

(Corydalis aurea)

Pink corydalis

(corydalis sempervirens)
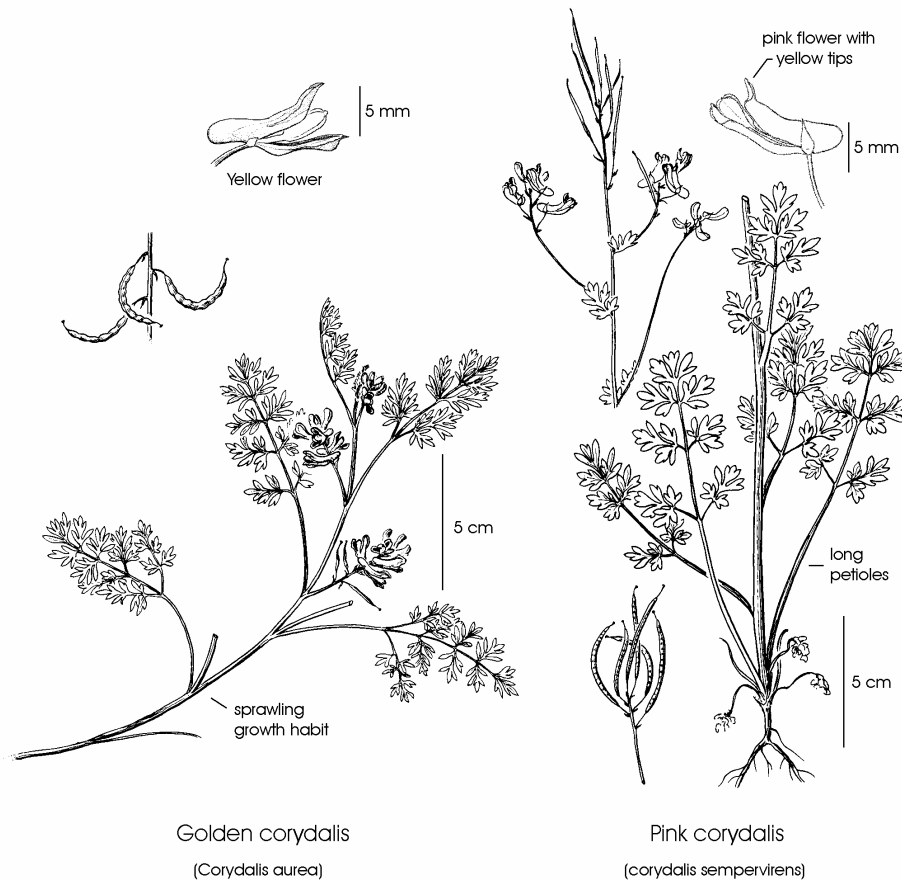
Flowers of pink corydalis are pink with yellow tips. Also a biennial or annual, this plant is similar but stands more erectly than golden corydalis, reaching up to 80 cm. The leaves have longer petioles and are only twice-divided. This species is also widely distributed but less frequent than golden corydalis in BC; it is not found on the Queen Charlotte Islands. It occurs in northern forests in the Prairie Provinces. It is found in disturbed soils, typically in burned clearings, clearcut-logged areas, and along roadsides.

Neither of these two native species has been proven to be responsible for livestock losses here but anecdotal reports indicate there may be reason for caution. Golden corydalis is widely distributed on the mountain rangelands in western USA and has a reputation for periodic episodes of sudden death losses in cattle and sheep (17). Its toxic principle is a group of isoquinoline alkaloids. Symptoms of poisoning from these toxins are restlessness and twitching of the facial muscles, followed later by convulsive spasms, and even death. Analysis of specimens of pink corydalis from Smithers, BC found the same alkaloids as in golden corydalis, at concentrations in the whole plant of over 0.3% of the dry matter (41).

These plants reproduce by seed which are spread over a large area when the pods split open explosively. Populations usually are small, but when a good seedbed is produced by soil disturbance there may be many plants. One such site was blamed for some losses of sheep used for weed control on a clearcut-logged site near Smithers (S. Simms pers commun).

Heath family, Ericaceae

Several shrubs of the heath family are poisonous to livestock. They are grouped together because they have somewhat similar growth habits, distributions and toxic principles. Given these similarities and the fact that names vary with the reference used, variety names are not included here. The reader is referred to Brayshaw (15) for further detail on identification and distribution.

Western minniebush,	
Rusty Menziesia	*Menziesia ferruginea* Sm.
White rose-bay,	
White rhododendron	*Rhododendron albiflorum* Hook
Trapper's-tea	*Ledum glandulosum* Nutt.,
Labrador-tea	*Ledum palustre* L.,
	L. decumbens (Ait.)Lodd. *ex* Steud.
Bog-rosemary	*Andromeda polifolia* L.
Bog-laurel	*Kalmia polifolia* Wang.
	K. microphylla (Hook.) Heller

Western minniebush is a medium-sized branching shrub, 1-2 m high, with thin alternate leaves that have scattered, rusty hairs on the upper surface. The flowers are greenish purple and rather small, and are borne in terminal clusters. **White rose-bay** is also a medium-sized shrub with thin, clustered leaves. The flowers, one to three in a cluster, are showy, pale yellow, and bell-shaped, about 3 cm across**. Trapper's-tea** is a low shrub with fairly thick leaves that are resin-dotted underneath; the resin makes the foliage fragrant when bruised. The flowers are small and yellowish white, and are borne in terminal clusters. **Labrador-tea** is very much like trapper's-tea, except that its leaves are densely hairy below instead of glandular. **Bog-rosemary** grows to about 50 cm tall. The leaves are small, linear-oblong, and white felty-hairy below, with the margins rolled in; the leaves are alternate on the stem. The flowers, on nodding terminal umbels, are in clusters of 2-6. **Bog-laurel** is a somewhat branching evergreen shrub, 25-60 cm high, with oblong, leathery, opposite leaves that are dark glossy green above and whitish beneath. The leaf margins are rolled under. The flowers are small and lilac-coloured, and are borne in terminal clusters.

Western minniebush and trapper's-tea occur in moist coniferous forests, wet meadows and bogs in BC and in Alberta in the southern Rocky Mountains. White rose-bay occurs in the same areas, but in drier situations. Labrador-tea, bog-rosemary, and bog-laurel are found in muskeg areas and damp forests throughout Western Canada. Ornamental

cultivars of this group are also toxic, with deciduous-leaved azaleas being less dangerous than evergreen rhododendrons.

These shrubs produce neurotoxic diterpenoids, which have an assortment of terms, including andromedotoxin and grayanotoxin. These toxins occur throughout the plant, including the nectar, and can be found in honey. They are toxic to bees (17). In livestock, symptoms of poisoning are vomiting, general depression, hypotension, irregular heartbeat, colic, convulsions, inability to coordinate voluntary muscles and recumbency. The leaves of most of these species are leathery or bitter, so their palatability is rather low. Most animal species are susceptible, including cattle, horses, llamas and goats (17), but instances of intoxication are rare. Cattle seldom graze these plants unless other feed is very short. Sheep are the most commonly affected. Animals should be removed from the infested areas and given access to hay and water. No specific antidote is known but subcutaneous injection of morphine has been used successfully in goats.

Greasewood (*Sarcobatus vermiculatus* (Hook.) Torr.), Goosefoot Family, Chenopodiaceae

This shrub is densely branched, with many of the twigs tipped with small spines. Greasewood may grow to 1 m in Canada, but a height of 50-80 cm is more common. The deciduous leaves are pale yellowish-green and fleshy, about 25-35 mm long. The tiny flowers are borne in small spikes, with the many staminate flowers at the tip hidden under small scales, giving the appearance of a catkin. Female flowers have no petals and are borne in leaf axils lower on the spike. A broad membranous wing surrounds the seed. Greasewood is found only in the southeastern part of Alberta and southern Saskatchewan on strongly saline flats. Confusion may occur because the common name "greasewood" is often applied to *Purshia tridentata* (Pursh) DC., which is also called antelope-brush or bitter-brush, a shrub of the Rose family native to some arid areas of southern BC.

The buds and young leaves of greasewood contain soluble sodium salts of oxalic acid which are rapidly absorbed by the animal. In the blood the sodium is replaced by calcium to form insoluble calcium oxalate. Crystals of the calcium oxalate are deposited in soft tissue, like kidney tubules and the rumen wall, causing swelling and impaired function. The reduction of blood calcium by the formation of the insoluble oxalate is also considered to be responsible for animal mortality. Intake of greasewood causes depression, weakness, shallow pulse and breathing, and collapse. This plant can be detoxified in the rumen but an adaptation period is required to permit significant increases in the populations of bacteria capable of oxalate degradation.

Lambs are the most susceptible to greasewood poisoning. The losses occur soon after flocks are moved to summer range, when the animals are hungry and are in unfamiliar surroundings. Calves and mature sheep have also been poisoned by greasewood. The oxalate content increases with maturity of the leaves, and losses have occurred when sheep consumed the fallen leaves. Watering immediately after animals have ingested a large amount of greasewood has been implicated in losses. It is risky to introduce

livestock into dense stands of greasewood, unless it can be done gradually. When animals are properly managed, greasewood is considered adequate forage for sheep.

Dogbanes (*Apocynum* L. spp.), Dogbane Family, Apocynaceae

Dogbanes are creeping-rooted, erect and branching perennials. They have oblong or lanceolate opposite leaves and clusters of small, pink, bell-shaped flowers which produce long, slender pods containing numerous seeds with tufts of silky hairs. The stems and leaves contain a milky latex. Dogbanes are commonly found on abandoned fields, road cuts, dry meadows and open wooded areas. They generally occur on sandy or gravelly soils, especially in pine forests. Dogbanes contain several toxic cardiac glycosides, including cymarin, which affect the excitability of the cardiac muscle. Animals may die of cardiac failure. The plants are unpalatable, and ingestion is unlikely except when animals are starving.

Green false hellebore (*Veratrum viride* var. *escholtzii* (R. & S.) Breitung), Lily Family, Liliaceae

Green false hellebore is a tall (1-2 m) perennial with broad coarsely-veined clasping leaves. It has a 30-70 cm long open panicle with widely-spaced drooping branches of white to greenish bells. It prefers moist habitats, typically at high elevation, in BC and Alberta. It contains neurotoxic steroidal alkaloids including jermanitrine, jervine, and veratrosine. Symptoms of acute poisoning include salivation, prostration and difficult respiration. In Western Canada hellebore is not considered palatable to cattle or sheep, only being consumed when other forage is lacking. However, heavy use has been observed in clearcut-logged areas where sheep have been confined overnight (D. Brooke pers commun). In the north-western USA, a related species, **corn lily** (*V. californicum* (Durand)) causes birth defects in sheep when ewes consume the plant on the fourteenth day of gestation.
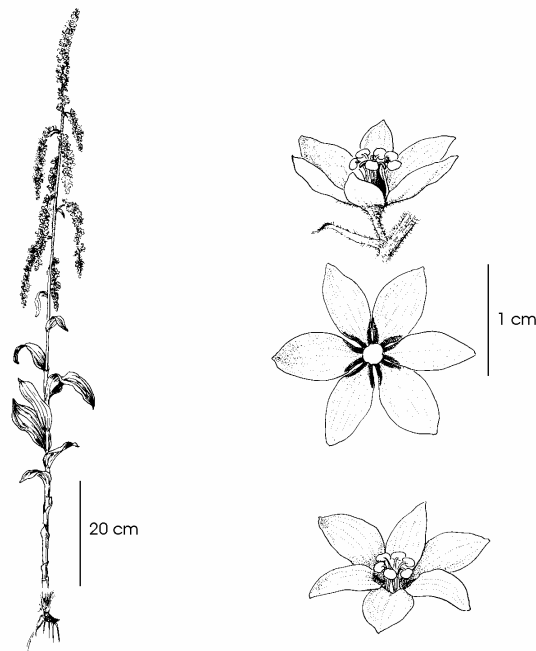
Green false hellebore

(Veratrum viride)

Horsetails (*Equisetum* spp.), Horsetail Family, Equisetaceae

Horsetails are well-known, non-flowering perennial herbs. The aerial stems are 20-60 cm tall, rush like, jointed, and generally hollow and single, ending in a cone or with whorls of four-angled, fine, green branches. They contain a rather large amount of silica, which makes them very harsh. This texture made them useful for scrubbing pots hence their alternate name, scouring-rush. The leaves are very small and scale-like, and they grow from a cylindrical sheath at the nodes or joints of the stems. Horsetails are common on roadsides in moist climates, in moist fields, swales, and meadows and around lakes and sloughs throughout Western Canada, especially in flood plains and sandy soils. They are a common component of native meadow hays and appear to be more palatable in hay than when fresh.

Poisoning is most frequent in horses and appears to be caused by thiaminase, an enzyme that destroys thiamine, and which is retained in hay. The vitamin deficiency can be remedied by administering thiamine. Horsetail poisoning is rare in ruminants. Clinical signs of poisoning are a general poor appearance, loss of weight, and gradual weakening of the animal. The symptoms may be slow to develop, as the speed of development depends upon the age of the animal. Younger horses are more susceptible. After an animal has been fed hay containing 20% or more horsetail for 2-5 weeks, it loses muscular control, falls down, and struggles violently to get up. The animal is usually willing to eat but is unable to rise and finally dies of exhaustion. It is important to immediately stop feeding the toxic hay. Large doses of thiamine are effective in horses as long as animals are still able to get up.

Locoweed, Pea Family, Fabaceae (Leguminosae)

 "Loco" is Spanish for crazy, referring to the behaviour of animals affected by the indolizidine alkaloid swainsonine which is found in some species of the Pea Family. In some botanical literature (34) the term "locoweed" is only used for the *Oxytropis* genus and we chose to follow this lead. However confusion may arise because in current usage in the USA the term is applied to all species containing the toxin, and this includes some *Astragalus* species. *Oxytropis* plants are usually stemless so leaves are in a loose basal clump and the flower stems rising from the base are leafless and unbranched. *Astragalus* flower stems are typically branched.

Silky locoweed or **early yellow locoweed** *(Oxytropis sericea* Nutt. var. *spicata* (Hook.) Barn.) is a tap-rooted perennial, growing as a dense, low tuft of grey-pubescent leaves, with conspicuous flowering stems rising to a height of up to 25 cm. It is very noticeable in early spring. The leaves are pinnate, having 2 rows of oval leaflets along a central stalk, with a membranous stipule at its base10-30 mm long. There may be 7-21 leaflets, 10-35 mm long, and densely hairy on both sides. The flowers are 15-27 mm long, whitish to pale yellow, sometimes pinkish-tinged, with 5-35 in a dense spike-like cluster, borne above the leaves. The spike elongates considerably as the pods mature. The pods are 15-25 mm long, 5-7mm in diameter, with a short beak. The pod wall is fleshy when green, but hardened and nearly 1 mm thick when dried, and has been described as "leathery" or "bony". It is covered with short hairs that are mostly white, but with a few black. Silky

locoweed is common on grasslands east of the Rockies but less common in the Parklands. It has been reported infrequently in BC, typically on edges of sub alpine talus slopes. There are several plants that can easily be confused with this locoweed.

very hairy
stipule

hairless
stipule

10 cm

stipule on
leaf stock

Silky locoweed,
Early yellow locoweed

(Oxytropis sericea
var. spicata)

toxic when infested with
a specific fungus

Field locoweed,
Late yellow locoweed

(Oxytropis campestris)
(var. cusickii)   (var. gracilis)

not toxic
may be confused with silky locoweed
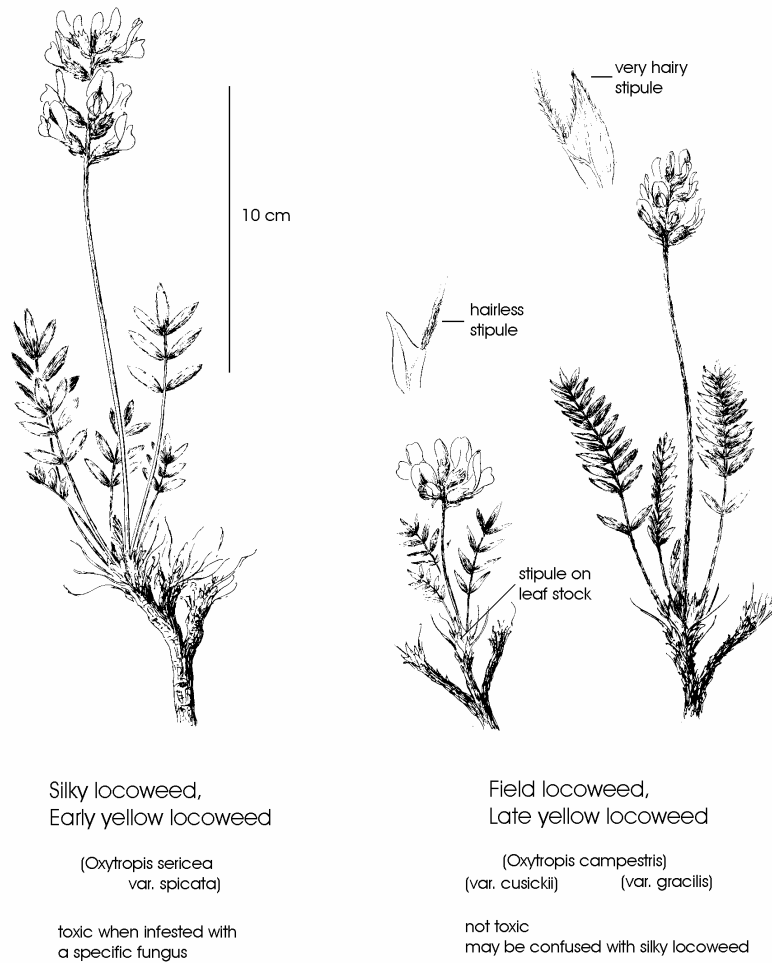
The major toxic principle of locoweed is the indolizidine alkaloid swainsonine. All species of livestock may be affected. Much research has been done on "locoism" in the USA, where swainsonine-containing plants are more abundant than in Western Canada. Swainsonine is produced by endophytic fungi (14), and the usually-toxic legumes may lose toxicity when grown without the fungi (39). The threshold dosage for toxicity is estimated to be about 0.3 mg swainsonine per kg body weight (78). This toxin is rapidly absorbed and excreted in the urine (17). Half of the toxin intake is eliminated in less than a day in cattle and sheep and is cleared after 5 days (17). However, during continuous consumption of locoweed, swainsonine is widely distributed in the body, causing damage to the nervous system, heart, and reproductive system (17). Clinical signs of poisoning in cattle include depression, a slow awkward gait, rough hair coat, dull and staring eyes, incoordination, nervousness and difficulty eating and drinking. Failure of the right side of the heart is typically seen when cattle, particularly calves, are grazing infested ranges at

high-elevation. Abortions and deformed or weak offspring may occur. Swainsonine is found in milk (17).

Horses are the most susceptible and show the most distinctive clinical signs. These signs may include depression, with head and neck extended down as if asleep, vacant staring, and an awkward stiff gait. When handled, otherwise-docile horses may react violently. Death or injury may result from accidents precipitated by this behaviour. Horses may be permanently unsound for riding, but remain useful for breeding (17).

A prolonged period of consumption is required to cause significant signs, and removing the animal from the offending feed can allow recovery, with only a few chronic effects in most cases. Because this legume may provide high quality forage, cattle gains may be good for the first few weeks, until the effects of the toxin prevail. It has been proposed that a management alternative is to allow short-term intensive grazing of this species, then to move the herd to a non-infested site.

Field locoweed or **late yellow locoweed** (*Oxytropis campestris* (L.) DC.) is not toxic but can easily be confused with silky locoweed. It blooms slightly later in the spring than silky (early yellow) locoweed. This plant is very widely distributed, and is extremely variable in morphology. For instance, on the prairies there are 7 varieties (37), 2 of which occur in BC, while an 8th variety occurs only in BC (34). The figure shows two important varieties in our region; *cusickii* occurs in the Rocky Mountains and east to the Cascades in BC, while *gracilis* is common on well-drained sites in moist grasslands and open forest (37) throughout the Prairies. It is apparent here that the difference within the species *campestris* is great, a problem when separating it from the toxic species, *sericea.* The flowers of field locoweeds are light yellow to creamy white on the strains in BC and throughout most of the Prairies. The keel petal may be purple. The pods of field locoweed are similar to those of silky locoweed, but are typically thinner walled, being papery rather than hard (0.5 mm thick in comparison with nearly 1 mm thick). This difference may be useful for separating these two locoweeds, because the previous year's pods can remain on ungrazed plants. However, the variable nature of this species complicates the separation; some populations of field locoweed which have small flowers (<13 mm long) have thick-walled pods like those of silky locoweed.

Other species of this family may contain toxic levels of swainsonine, but it is not yet clear whether a fungus is involved in their toxicity (as is the case with silky locoweed). **Two-grooved milkvetch**, (*Astragalus bisulcatus* (Hook.) A. Gray) is a stout, many-stemmed, erect plant 30-80 cm high. It prefers soils high in selenium and has a distinct, unpleasant odor. Its ability to accumulate selenium is addressed under "Selenium poisoning" and it is illustrated in that section.

Purple locoweed (*Oxytropis lambertii* Pursh), found on the Prairie grasslands and southeastern Parklands, is similar in size to silky locoweed, but the flowers are a dark bluish purple. It can be distinguished from other purple-flowered species by its hairs: some of the hairs are shaped like a pick, or attached near the middle.

Freckled milk-vetch (*Astragalus lentiginosus* Dougl. ex Hook. s*ensu lato* L.), which also contains swainsonine, is a problem in the USA. It has been observed in BC on the dry grasslands near Kamloops, but plants from that site have not been chemically analysed. Also a tap-rooted perennial, it is a very variable species. Ours has several spreading stems 10-40 cm long. The leaves are a broad round-tipped oval narrowing at the base, light green and almost hairless. Flowers are elongate, white pinkish or pale purple-tipped. The pod is distinctive, an inflated smooth papery bladder, some with plum-coloured blotches, about 15 mm long, including an upward-curving beak.

Lupine, Pea Family, Fabaceae (Leguminosae)

pubescent banner petal

5 mm

7 - 9 leaflets

dense covering of fine hairs on stems, leaves, and pods

5 cm

9 - 17 leaflets

palmate leaves with a few hairs on lower surface; almost hairless on upper surface

5 cm

hollow stem with very few reddish hairs

Silky lupine
(Lupinus sericeus var. sericeus)
toxic

Many-leaf lupine
(Lupinus polyphyllus var. polyphyllus)
toxic
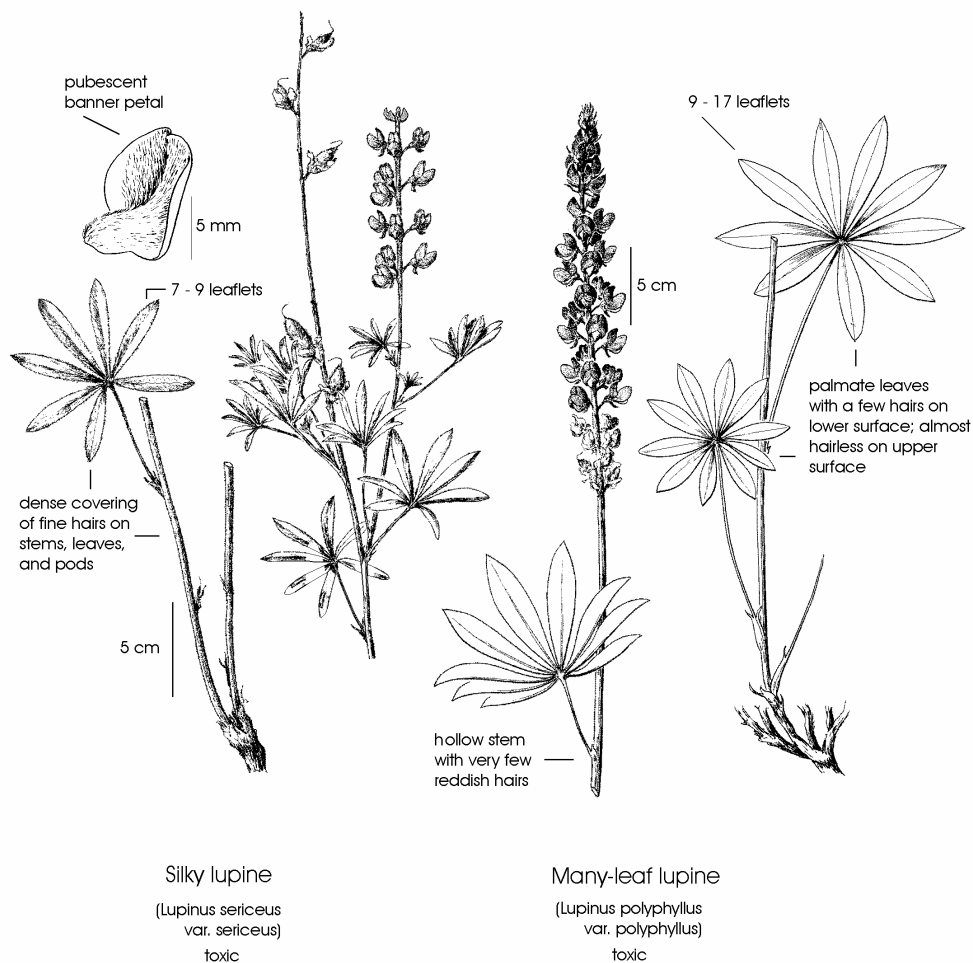
Lupines are easily recognized because of their spikes of short-stalked pea-like flowers and their palmate leaves, with a group of narrow leaflets radiating from the top of the leaf stem. There are numerous species in Western Canada; their differences developed when ice masses kept geographic regions separated. After the ice retreated the discrete groups spread out and then hybridized freely where ranges overlapped – to a greater extent than most other native plants. Consequently there is often nearly as much variation within a group as between groups so identification by botanists has been controversial (34). The

native ones are perennial. Lupines produce high quality forage, may represent a large proportion of the available pasture and under normal management they are useful range plants. In the USA however, several lupine species have caused significant, well-documented losses in range livestock. The situation in Western Canada is less clear, and anecdotal reports of losses have not been satisfactorily substantiated.

Silky lupine (*Lupinus sericeus* Pursh) occurs in fescue prairie in southwestern Alberta, and in the dry sagebrush and ponderosa pine regions of south central and southeast BC. It is found on open grassy hillsides, generally in rich, light, well-drained soils. It is silvery green because of the dense covering of fine hairs on the stems and leaves. It grows as high as 60 cm and is somewhat bushy in appearance. The leaf has five to nine long (up to 5 cm) very narrow leaflets. The many flowers are blue with two white markings, about 6-15 mm across, and are arranged in dense clusters along the spike-like stem. The pod is about 3 cm long and densely covered with short, silky hairs. The long, thick taproot allows the plant to remain green throughout the growing season. It forms a crown, as in alfalfa. The plant blooms in June and July and the seeds mature in July and August. It is common to abundant and is scattered over large areas.

The toxic principle is a group of neurotoxic alkaloids including sparteine, a quinolizidine alkaloid. The seeds and pods are the most toxic parts, so the plant is most dangerous in the fall. The alkaloids remain present after drying, so hay containing large amounts of lupines may be toxic. Most poisonings have occurred when hungry animals, usually sheep, have been turned into infested areas. Symptoms of acute poisoning are labored breathing, which is followed by coma or frenzy, frothing at the mouth, and violent spasms. When poisoned by lupine, sheep may butt anything in their way.

Two other compounds found in this species can cause problems with cattle: the quinolizidine alkaloid, anagyrine, and the piperidine alkaloid, ammodendrine. They are termed "teratogenic", sometimes causing birth defects termed "crooked calf disease" if a pregnant cow grazes the lupine between the 40th and 70th day of gestation. The risk may continue up to the 120th day in cattle, but sheep are not susceptible (69). This plant can induce defects ranging from severe limb and spinal deformities to cleft palate. Often there will be no clinical signs of illness in a cow carrying an affected fetus. The observed deformities are due to a sedative effect of the toxins, which results in cessation of fetal movement during a period when the fetus normally would be frequently changing position in the uterus. For instance the head of the sedated fetus, resting on the soft bones of the rib cage over a long period, causes a rib cage depression and spinal curvature (35). A prolonged period of repeated ingestion of toxic lupine is believed to be necessary to cause significant deformity, because the alkaloids are rapidly eliminated from the body (17). One proposal under consideration in the USA for safe use of lupine ranges is intermittent grazing of lupine, to allow the fetus to regain activity when the toxin clears the dam's system (65).

Reports of isolated cases of crooked calf disease in a herd are difficult to explain. The fact that the abnormal calf is born months after the 70th day of gestation makes it hard to be sure of the dam's diet at the critical time. There are several reasons for deformity of the fetus, including genetic abnormalities, with a few specific symptoms attributable to

lupine (72); it is advisable to consult a veterinarian for clarification of each case. Variation in incidence of crooked calf disease in range cattle may reflect differences in the diet choices of individual cows. A difference in toxin content between individual plants within a species has been observed (45, 20) as has year to year variation on a site (30a). A number of reports of deformed calves in central BC and in northern Ontario precipitated a survey of these areas in 1986 (55). In all cases lupines, and other teratogenic and otherwise poisonous plants, were ruled out as the cause of the anomalies in the calves. Although a current survey of crooked calf losses in Western Canada is not available, it appears that this disease is not a widespread problem here.

Silvery lupine (*Lupinus argenteus* Pursh.) is similar to silky lupine, but since the hairs on the stems and leaves lie flat, the plants appear less woolly. Flowers are light violet or purplish to almost white. Silvery lupine is plentiful in the Cypress Hills-Wood Mountain area and the foothills of the Rocky Mountains and is less common throughout southern Alberta along the Milk River Ridge. It contains high levels of piperidine alkaloids and has been implicated in acute poisoning of cattle on overgrazed range.

Many-leaf lupine (*Lupinus polyphyllus* Lindl.var *polyphyllus*) contains high levels of alkaloids. It is found at lower elevations, in damp meadows, stream sides and along roads throughout BC, and on grasslands and open forests of the southern Rocky Mountains in Alberta. This is the largest of our native lupines, up to 1.5 m tall, with hollow stems. It has 9-11 leaflets, forming an almost-perfect circle up 15 cm across. It is much less hairy than the above species, with sparse pubescence on the stems and lower surfaces of the leaflets. Hairs are amber colored. Flowers are bluish to violet. This species is a parent of several of the taller perennial horticultural varieties (34), so escapes from gardens may be toxic. The alkaloid content in this lupine is variable: ammodendrine content ranged from 0.34 to 0.93 g/kg dry matter, and anagyrine ranged from 2.09 to 13.58 g/kg (45). Keeler and Panter (35) force-fed ground dried lupine (*L. formosus*) at 4.5 g/kg body weight per day to Hereford heifers for 30 days and found no fetal damage when the content of ammodendrine was 0.50 g/kg air-dried lupine but symptoms occurred when the content was 0.95 g/kg. While this particular study showed a threshold toxin content over which fetal damage may occur, it did not clarify the minimum days of ingestion required.

Bigleaf lupine or Burke's lupine (*L. burkei* S. Wats.) occurs in moist sites in south central and southeastern BC. It has an unbranched fairly smooth stem like that of many-leaf lupine, but is less than 60 cm tall. In a survey of plants collected in the USA (20) this species was found to have significant alkaloid content, but most plants had anagyrine content below the threshold level for teratogenesis, 1.44 g/kg dry matter (34a).

Arctic lupine (*Lupinus arcticus* S. Wats, or *L. latifolius* Agardh) occurs throughout BC in mid to high elevation forest ecosystems. It is often in very dense stands, and is a major constituent of the ground cover on some clear-cut logged areas, becoming established from long-dormant seeds soon after soil disturbance. It is an important forage species, often being used after the plants have matured or after late summer frosts (68). Arctic lupine has blue flowers similar to those of many-leaf lupine, (with white spots similar to those on silky lupine), with rare pink or white-flowered plants. It is smaller than many-

leaf (20-60cm), has woody stem bases, and is more likely to be branched. Stems appear smooth, but have fine hairs pressed against them. There are fewer leaflets, 6-8, usually with a smooth upper surface, and a few silvery (rather than amber) hairs on the lower surface. There are two varieties of this species in BC. The variety *subalpinus* (Piper & B.L. Robins) Dunn, found north of 55°N, has leaves mostly along the stem, with leaf stalks 4-8 cm long. The variety *arcticus* on which the longest leaf stalks are 9-15 cm long is found south of 55°N. It has mostly basal leaves. The overlapping ranges of the lupines in BC have allowed them to interbreed and affect the alkaloid composition of the species. Consequently anagyrine is found in the *arcticus* variety of Arctic lupine when it interbreeds with many-leaf lupine (45). Outside of areas of overlap with many-leaf lupine, the *arcticus* variety is not considered a problem for cattle but it does contain high levels of sparteine (45). Many other lupines are found in Western Canada, but generally they are not significant components of the range resource here.

Showy milkweed (*Asclepias speciosa* Torr.), Milkweed Family, Asclepiadaceae

Showy milkweed, the most common of several milkweeds in the region, is an erect, perennial native herb up to 1.2 m tall, growing in clusters from a rhizome. The finely-hairy leaves are fleshy and somewhat leathery, up to 10 cm wide. Stems and leaves contain milky latex. The flowers are pinkish mauve, grouped in round heads at the stem tips. The fruits are large, with thick coats enclosing numerous reddish brown seeds, each with tufts of long, silky hairs attached. The plants may be common in moist locations, in pastures, abandoned fields and roadsides in south central and southeastern BC and throughout the Prairies and Parklands. The toxic principle consists of cardiac glycosides. Weakness, staggering, and deep depression are symptoms of poisoning by milkweed, which appear within a few hours after ingestion. Violent seizures occur repeatedly after an animal falls down, and death follows within 24-48 h. Milkweeds are eaten only when livestock is forced by starvation.

Onion (*Allium* L. spp), Lily Family, Liliaceae

There are a number of onion species native to Western Canada that can be toxic to livestock but records of problems are lacking. The potential for toxicity on range land appears to be limited to cases where the animals seek out the onions.

Poisoning most often occurs in onion-growing areas when culling of **domestic onions** makes large amounts of bulbs available. S-alkyl cysteine sulfoxides including S-methyl, S-propyl, and S-propenyl cysteine sulfoxides are characteristic of the onion genus. When tissue is chewed these compounds decompose to a number of disulfides including dipropyl disulfide and dipropenyl disulfide, which are oxidizing agents. They cause damage to red cell membranes leading to Heinz body haemolytic anemia (Cabbage Family). Sheep and goats can usually be fed domestic onions free choice without problems. For cattle the maximum safe level is 25% of the diet (19), but when given free choice they may prefer the onions to other feeds and ingest toxic amounts. Horses have intermediate susceptibility. The concentration of the toxins appears to increase with the

pungency of the onion. Some clinical signs may be overlooked because cattle fed onions maintain their appetite until they are severely anaemic, but reddish-brown coloured urine is a clinical sign. Treatment consists of removing animals from onions, and giving blood transfusions in severe cases.

INTRODUCED SPECIES

DOMESTIC CROPS

Perennial ryegrass (*Lolium perenne* L.), Grass Family, Poaceae (Gramineae)

Perennial ryegrass, a short-lived cool-season perennial, is an important forage species. It is also an important turf species, and the seed production industry makes forage available as straw, screenings and fall grazing of post-harvest regrowth. However, endophytic fungi in turf varieties, which create greater vigor in lawns (see Tall fescue), produce tremorgens (28), complex alkaloids that cause "ryegrass staggers". In this species of ryegrass the fungus *Acremonium lolii* produces lolitrem B, an inhibitor of neurotransmitters. Clinical signs are incoordination, head shaking, stumbling, collapse and severe muscle spasms. Symptoms are reversible and animals can recover completely on endophyte-free feed (17). Toxin threshold levels have been determined for cattle and sheep. Consult your veterinarian for advice on lab testing services to determine rate of dilution with uninfected feed. In cattle the threshold concentration of lolitrem-B is 1.68 mg/kg (28). Endophyte-free seed will produce an endophyte-free stand. The toxin is most concentrated in the lower leaf sheaths so avoidance of overgrazing reduces incidence of staggers (76). **Annual ryegrass** (*Lolium multiflorum* Lam.), another important forage and turf species, is not reported to be toxic in Western Canada.

Brassicas, Cabbage Family, Brassicaceae (Cruciferae)

This is a group of high-yielding, nutritious, and fast-growing crops that have a long history of use as annual pasture in Europe. In Canada they are less popular, but interest is growing for use as both late-season pasture and as stored feed. The brassica ("crucifer") group includes **forage kale** (*Brassica oleracea* var. *acephala* L.), **rutabaga** or Swede turnip (*B. napus* L. var. *napobrassica*), **turnip** (*B. rapa* L) and an assortment of hybrids with other species, **rape** (*B. napus* L.) and its oil-seed descendant, **canola**, from which several antiquality constituents have been removed by genetic selection. The waste from processing and the stubble of related food crops, like cauliflower and Brussels sprouts, may also be used as ruminant feed.

Although brassicas can be high in crude protein and digestible dry matter, they are low in several minerals, so that mineral supplementation is advisable. Brassicas contain high levels of sulfur, which can interfere with the dietary availability of copper. An amino acid in brassicas, S-methylcysteine sulphoxide (SMCO) can cause brassica anaemia

(haemolytic anaemia, see Onion). In the rumen is converted to dimethyl disulfide, which is an oxidant (24). SMCO is believed to be the constituent most responsible for livestock gains on pasture of this crop being lower than predicted from nutritional content and intake. Cattle are more sensitive to SMCO than are sheep and goats, but non-ruminants are unaffected. Glucosinolates (see Stinkweed) are also thought to be involved in reduced gains on brassica forage. Research with forage rape (24) showed that making it into silage does not appreciably reduce the SMCO content, although it does greatly reduce the glucosinolate content. The wilting of forage rape, as in hay making, was found to have very little effect on its content of either SMCO or glucosinolates.

Brassica pasture may consist entirely of leaves, because the plant is harvested at an early stage of growth, before any fibrous flower stems develop. Consequently, this crop can have very low fibre content, which may cause diarrhea. When grazed at less than 25 cm in height for instance, canola can cause pasture bloat. Turnips can cause free-gas bloat in cattle. There is also a risk of respiratory distress if animals are changed abruptly from dry summer pasture to lush brassica pasture (see Acute Bovine Pulmonary Emphysema). Infrequently an abrupt change to brassica pasture can cause injury to the nervous system (see Polioencephalomalacia). To avoid these problems, introduce grazing animals slowly to such pastures. Some prescriptions say over 3-4 days, but more conservative sources suggest grazing for only 1-2 hours per day, and taking 7-10 days for the transition. It is critical to limit intake to less than 75% of the diet, by supplying hay or access to grass pasture. The supplemental feed can compensate for the brassica's low fibre content. Canola has been increasingly used for forage on the Prairies, particularly when feed supplies have been short. A stand damaged by hail can be salvaged as silage or hay, usually at the flowering stage. **Mustards**, brown and oriental (*B. juncea* Coss), and yellow (*Sinapsis alba* L.) can also be utilized in this way. High moisture content in these crops makes it difficult to avoid mould problems in stored feed. Nutritional value and toxic potential can vary greatly depending on plant maturity, harvest date, and handling so feed testing is advisable. Nitrate levels in canola can be hazardous after stress from drought, heat or freezing. Canola silage, as with canola pasture, may have high digestible-carbohydrate content and consequently there is some risk of acidosis and frothy bloat. In young animals, particularly when there is insufficient long fibre in the diet, canola silage can cause Polioencephalomalacia. The best approach is to make a gradual introduction of this silage to livestock, with canola representing a maximum of 60% of the dry matter in the ration. Something about the taste of canola silage often results in significantly reduced consumption, in comparison with that of silage made from other crops.

Reed canarygrass (*Phalaris arundinacea* L.), Grass Family, Poaceae (Gramineae)

This native grass is an important forage crop on moist sites and commercial seedlots have been established on wet meadows in BC. The species contains low levels of tryptamines, gramine (indole alkaloids) and hordenine (48a). Animal performance on reed canarygrass has been greatly improved owing to the selection of low-alkaloid cultivars. Remaining old stands of reed canarygrass may reduce animal performance and increase the incidence

of diarrhea. Alkaloid levels in the grass can increase in response to soil moisture stress and nitrogen fertilizer.

Meadow foxtail (*Alopecurus pratensis* L.), Grass Family, Poaceae (Gramineae)

This introduced perennial is adapted to moist sites. It yields less than timothy (*Phleum pretense* L.), another introduced grass that looks very much like foxtail and also performs well on moist sites. However foxtail begins growth earlier in the spring and is more nutritious. An unknown antiquality factor in foxtail significantly reduces growth rate in cattle (74). The reduction in performance cannot be attributed to intake level and continues after animals are removed from foxtail pasture. The factor is thought to persist in foxtail silage.

Sweet clover (*Melilotus* spp. L.) Pea Family, Fabaceae (Leguminosae)

Sweet Clover

white flowers

yellow flowers

serrations along most of the leaf margin

5 mm

2 cm

2 cm

2 cm

(Melilotus alba)

(Melilotus officinalis)

Alfalfa

may be confused with Sweet Clover

yellow flowers

blue flowers

serrations only halfway down from leaf tip

2 cm

5 mm

2 cm

5 mm

2 cm

(Medicago falcata)

(Medicago sativa)
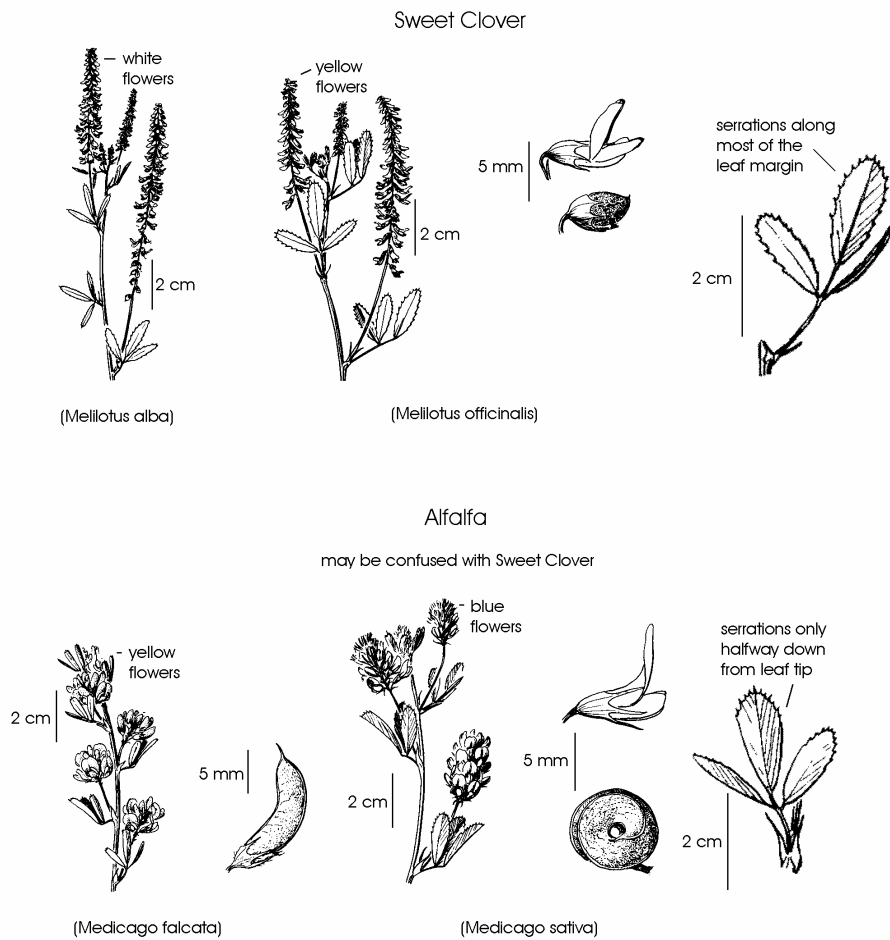
Sweet clover is an introduced annual or biennial crop reaching up to 2 m in height. Long considered a weed, it occurs widely in disturbed areas, particularly along roads, spreading

by seed. Selective breeding has improved the plant's usefulness, and for over a century it has been cultivated as a forage crop, for weed control, erosion control, bee pasture and green manure. There are two major species in Western Canada, yellow flowered sweet clover (*Melilotus officinalis* (L.) Lam.) and white flowered (*M. alba* Desr.). In the vegetative stages both can be difficult to distinguish from alfalfa (*Medicago falcata* L. and *M. sativa* L). Leaves of both plants are trifoliate, with the centre leaflet having a significant stalk. In sweet clover the leaflet is serrated along most of its edge, while in alfalfa the leaflet is serrated less than halfway back from the tip. Sweet clover contains the glycoside melilotoside, which releases the fragrant phenolic compound coumarin when the plant tissue is crushed. This sweet fragrance can be used to distinguish the plant from alfalfa, even in the seedling stage.

Flowers of alfalfa are quite similar to those of the sweet clovers, with an elongate-sweet-pea shape, but there are useful differences. In sweet clover they are either yellow or white, and always held in long slender sprays. In contrast, alfalfa flowers are in compact clusters. Most alfalfa flowers are in various shades of purple and blue (*Medicago sativa* L.), but some are yellow (*M. falcalta* L.)

Sweet clover is not toxic as pasture. Toxicity problems only occur when improper feed preservation allows conversion of the non-toxic coumarin in the harvested tissue to the anticoagulant dicoumarol. Various fungi are responsible for the production of this mycotoxin (67). Dicoumarol inhibits vitamin K_1 function, so that there is an inadequate supply of clotting factors in the blood, and internal haemorrhaging results. Sheep and horses are not nearly as susceptible as are cattle (67). External clinical signs include swollen joints, bloody faeces, pale mucous membranes, swellings under the skin, and bleeding from the vagina. Some animals may only exhibit weakness and pallor caused by the internal bleeding. Handling of the animals or surgical incisions may cause haemorrhaging, and castration or de-horning of affected animals may lead to death. Typically several weeks on contaminated feed are required before symptoms are seen. This lag is because of the pre-existing clotting capacity of the blood. The suspect feed should be removed immediately. Treatment in very severe cases may require a blood transfusion, which can be effective within hours. In less serious cases intramuscular or intravenous administration of vitamin K_1 is effective within 24 hours, but it must be repeated for several days, and is expensive. Supplying a natural source of vitamin K, such as fresh alfalfa hay, may suffice.

Selective breeding has produced low-coumarin varieties for Canada: white-flowered Polara and yellow-flowered Norgold. However, the toxicity problem has not been eliminated, because of high-coumarin sweet clover populations growing in the region, and the availability of "common" seed with unknown coumarin content. Hay may appear to be well dried, but inside the large stems there may be enough moisture to allow decay organisms to grow. Incomplete packing at the margins of a silage pit may cause insufficient fermentation, enhancing growth of such organisms. Oxygen-excluding silage-making methods can reduce the risk of sweet clover poisoning. Sweet clover hay can remain toxic for 3 or 4 years. One option is to alternate the feed by offering the suspect hay for 2 weeks, then supplying uncontaminated hay for 1 week. Another is to dilute the

suspect feed 1:3 with uncontaminated feed. It is wise to take cattle off suspect sweet clover 3 weeks prior to surgery or 1 month prior to calving.

Tall fescue (*Festuca arundinacea* L) Grass Family, Poaceae (Gramineae)

This is a hay and pasture species long used in the USA, but only recently common in Western Canada. Tall fescue toxicosis was a serious and perplexing issue in the USA until the discovery of the infection by the fungus *Neotyphodium coenophialum*, formerly *Acremonium coenophialum*, (17). Forage varieties of tall fescue now registered in Canada are safe for all livestock.

The fungus is an "endophyte", meaning that it grows entirely within the plant and does not alter the appearance of the plant. The infection cannot be cured with a foliar application of fungicide. The toxic principle is mainly the ergopeptide alkaloid ergovaline. It causes blood vessel constriction and suppression of prolactin secretion. There are numerous clinical signs, including gangrene of the extremities ("fescue foot"), failure to shed the winter coat, susceptibility to heat stress, and impaired reproduction and milk production. In horses, problems are primarily limited to poor reproduction. When consuming tall fescue in late gestation, mares have a longer gestation (resulting in abortion or large weak foals) and have poor milk production.

This fungus gives the host plant such advantages as greater seedling vigor, drought tolerance, and resistance to insect damage, so turf growers exploit it in their varieties of tall fescue. This situation has led to some serious poisoning incidents in Canada. Part of the problem may have arisen because other non-toxic turf fescues were grown for seed here before tall fescue was, and the cattle industry successfully used seed screenings as feed. Toxin concentration is high in tall fescue seeds, and screenings from seed cleaning should be kept out of the feed supply. Likewise, other crop residues from tall fescue turf varieties should be avoided or used with caution. Laboratory determination of endophyte content in feed is available to producers. A small proportion of infected plants in a stand does not constitute a problem. The toxin is not reduced by making hay or silage, but ammoniation does destroy it. Feed supplies containing infected fescue can be diluted with other forage. Infected fescue pasture can be less toxic if grazed and harvested before seed is produced.

Endophyte-freedom is not the result of selective breeding. Seed certification in Canada, through which genetic purity and weed seed content are assured, currently does not imply freedom from this toxin-producing fungus. (The bag of seed may well be free of the fungus, but for this quality the purchaser must rely on the seed seller.) Although the toxin is translocated throughout the plant, the endophyte organism is limited to certain structures. This situation has made it possible to use tissue culture methods to completely remove the fungus from a tall fescue clone. Plants produced in a laboratory from a small slice of fungus-free tissue have been used to produce endophyte-free seed, with the same genetic makeup ("genotype") as the original plant. Research is underway on use of non-toxic endophytes in forage tall fescue.

WEEDS

Common groundsel (*Senecio vulgaris* L.), Sunflower Family, Asteraceae (Compositae)

Common groundsel is an annual weed in cropland and barnyards. It is usually avoided by grazing animals but has caused losses when animals ate contaminated hay. It reaches 60 cm, but without competition for light it can be much shorter. Stems are hollow, and the alternate leaves are somewhat fleshy, hairless, and wavy-margined to deeply lobed. The flowers are inconspicuous 5-8 mm long heads of yellow disc florets, each enclosed in a tube of green bracts.
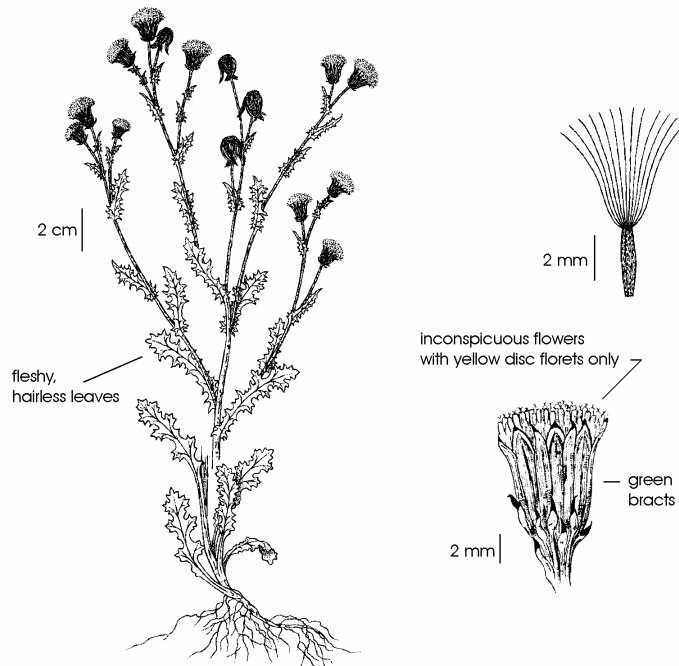
Common groundsel

(Senecio vulgaris)

The toxic principle is a group of pyrrolizidine alkaloids including senecionine, and alkaloids of this type are present in significant amounts in several species of the genus S*enecio*. Cattle and horses develop liver cirrhosis, which can be reflected in jaundice and, in the case of horses, staggering, walking in a straight line regardless of obstruction, and continual pressing of the head against objects. Young animals are most susceptible. The liver disease is irreversible, chronic, and progressive. A high dose can cause fairly rapid death, but animals have recovered from limited exposure to a low dose. However, in general, the effects of alkaloid poisoning are cumulative, sometimes resulting in the death of animals many months after consumption of the plant (36). A veterinarian may be able to evaluate liver function with a blood test, and so predict an animal's chance for recovery. Pyrrolizidine alkaloids can also be carcinogenic. In common groundsel, few or no symptoms may be visible until 2 or 3 days before death. Sheep and goats are quite resistant to these alkaloids.

Tansy ragwort (*Senecio jacobaea* L.), Sunflower Family, Asteraceae (Compositae)

This toxic weed, introduced from the British Isles, is a problem in southwestern BC. It contains pyrrolizidine alkaloids that cause liver damage (see Common groundsel). It

grows to about 1 m, typically in a dense clump of somewhat woody stems, with alternate pinnate leaves twice-divided into irregular segments (ragged appearance, hence the common name, "ragwort"), and showy flat-topped clusters of small yellow daisy-like

daisy-like flowers with disc and ray petals

button-like flowers without ray petals

5 cm

ragged edged leaves

flat fern-like leaves

Tansy Ragwort

(Senecio jacobaea)

toxic

Common Tansy

(Tanacetum vulgare)

not toxic but may be confused with tansy ragwort
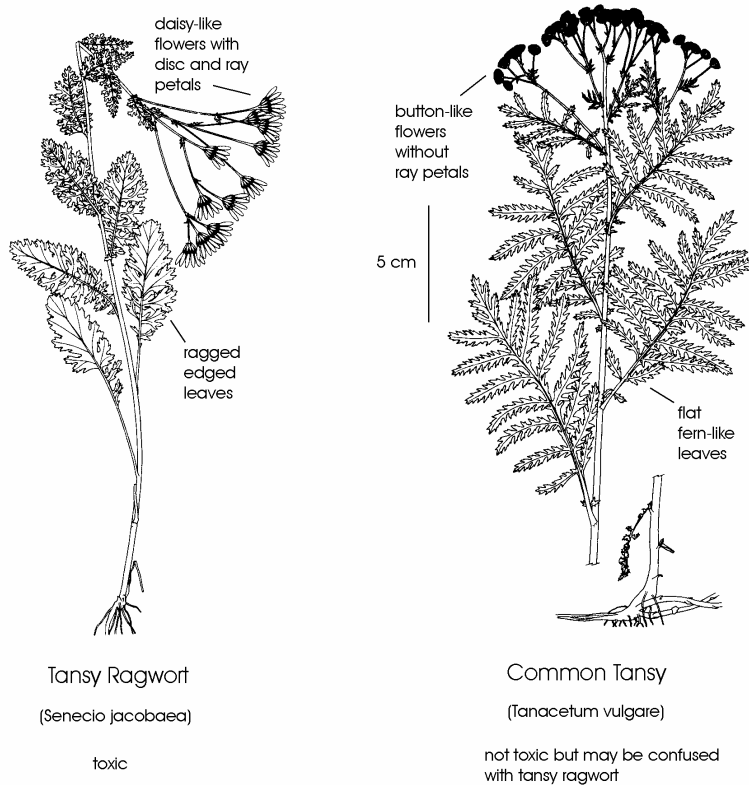
flowers. It is a rhizomatous biennial, becoming perennial if mowing prevents seed production, but is controlled by cultivation. It has very low palatability to livestock. The toxins can be found in honey (22). This weed is easily confused with **common tansy** (*Tanacetum vulgare* L.) which may occur in the same area. The confusion is compounded because the term "tansy" has been used for both plants. Common tansy was introduced from Europe as a medicinal herb and is not considered toxic to livestock. Some accounts in the popular press, of it being toxic, are thought to have arisen because some product sold as common tansy was actually ragwort (19). Common tansy has more finely divided and flatter, more fernlike, foliage. Its flowers are button-like heads of entirely disc florets, lacking ray petals. Only tansy ragwort has a "pappus" of white hair-like bristles on its seeds. Three native species of *Senecio* collected in BC were also found to contain the alkaloids but the levels were relatively low (7): streambank **butterweed** (*S. pseudaureus* Rydb.), **Rocky Mountain butterweed** (*S. streptanthifolius* Greene) and **arrow-leaved groundsel** (*S. triangularis* Hook). **Cut-leaved ragwort** (*S. eremophilus* Rich ssp. *eremophilus*), native to the Peace River area of BC, and in the boreal forest and parklands of the Prairies, also contains pyrrolizidine alkaloids.

Common hound's-tongue (*Cynoglossum officinale* L.), Forget-Me-Not Family, Boraginaceae

A noxious weed originating in Europe, it is common east of the Cascade Mountains in BC, and is found periodically in pastures and uncultivated areas on the Prairies. It contains pyrrolizidine alkaloids, differing from those in *Senecio* but with a similar type of liver toxicity. It is a biennial with a rosette of large, softly hairy, pointed oval leaves (said to resemble a dog's tongue) in the first season. The second year it produces a coarse single stem, reaching up to 120 cm, leafed to the top, with curved flower stems, each bearing red-purple 5-petalled flowers along one side. The seed is an oval disc or nutlet covered with hooked bristles, which readily stick to animals' hair. Removal of these very visible seeds from cattle before shipping to market, to make the animals more attractive to buyers, can be a costly problem for ranchers.
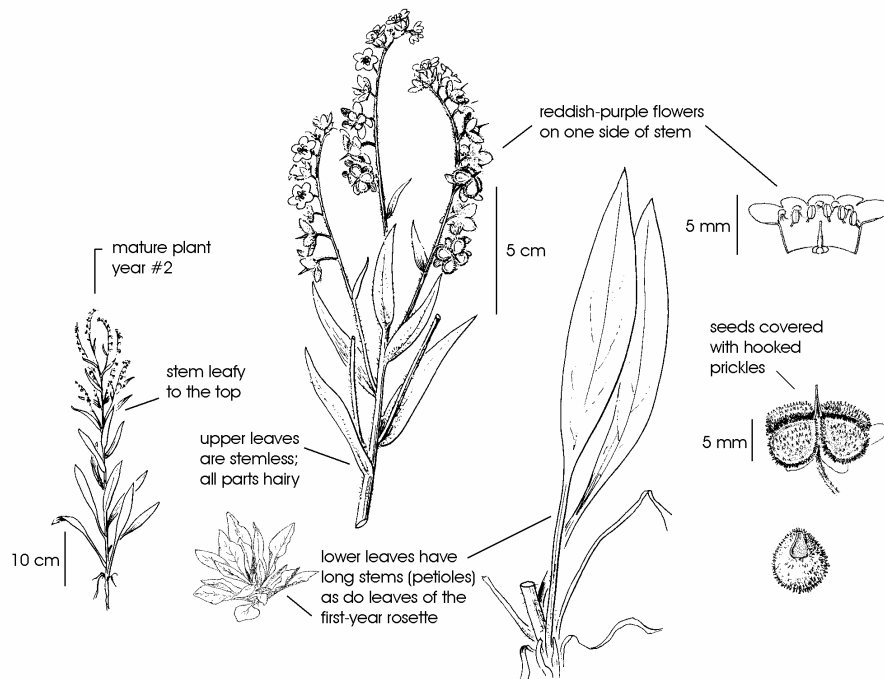
Common hound's - tongue

(Cynoglossum officinale)

In BC this weed is mainly restricted to rangeland, but it does not cause poisoning there because the distinctive odor of the green plant discourages grazing. It can be a concern if allowed to contaminate hayfields, because when dried it becomes palatable and remains toxic. The highest alkaloid concentration occurs in young tissue and, as with the alkaloids in *Senecio*; liver damage is the main problem. Diagnosing hound's-tongue poisoning may be difficult because death can occur some time after the animal has been taken off the contaminated feed. For horses, the toxic dose has been estimated to be 2% of daily dry matter intake, about one plant per day, for two weeks (77). This species may be confused with other large, but blue-flowered, members of the Forget-Me-Not family. A noxious weed of south central BC, **blueweed** (*Echium vulgare* L.) has bulbous-based bristles on its stems while **northern hound's-tongue** (*Cynoglossum boreale* Fern.), a native of northern forests, has leafless flower stems.

Common comfrey (*Symphytum officinale* L.), Forget-me-not Family, Boraginaceae

This relative of hound's-tongue contains significant amounts of pyrrolizidine alkaloids. It is a vigorous-rooted perennial up to 1.5 m tall, with large, coarse, pointed-oblong bristly leaves, up to 12 cm wide. Flowers are cream to purplish bells, less than 2 cm long, in drooping coiled clusters. It is found as an escape from cultivation in southern BC. Despite its toxic constituents, this plant has long been used as a medicinal herb, and has even been promoted as livestock forage. The lack of reports of its toxicity may be due to the cumulative effects and delayed action of the toxins (19). Two other similar comfrey species have been introduced into BC: **rough comfrey** (*S. asperum* Lepech.) and **hybrid comfrey** (S*ymphytum* x *uplandicum* Nyman.).

Kochia (*Kochia scoparia* (L.) Schrad.), Goosefoot Family, Chenopodiaceae

2 mm

winged flowers

5 cm

JRJ

lower surface
of leaf softly hairy

Kochia
(Kochia scoparia)
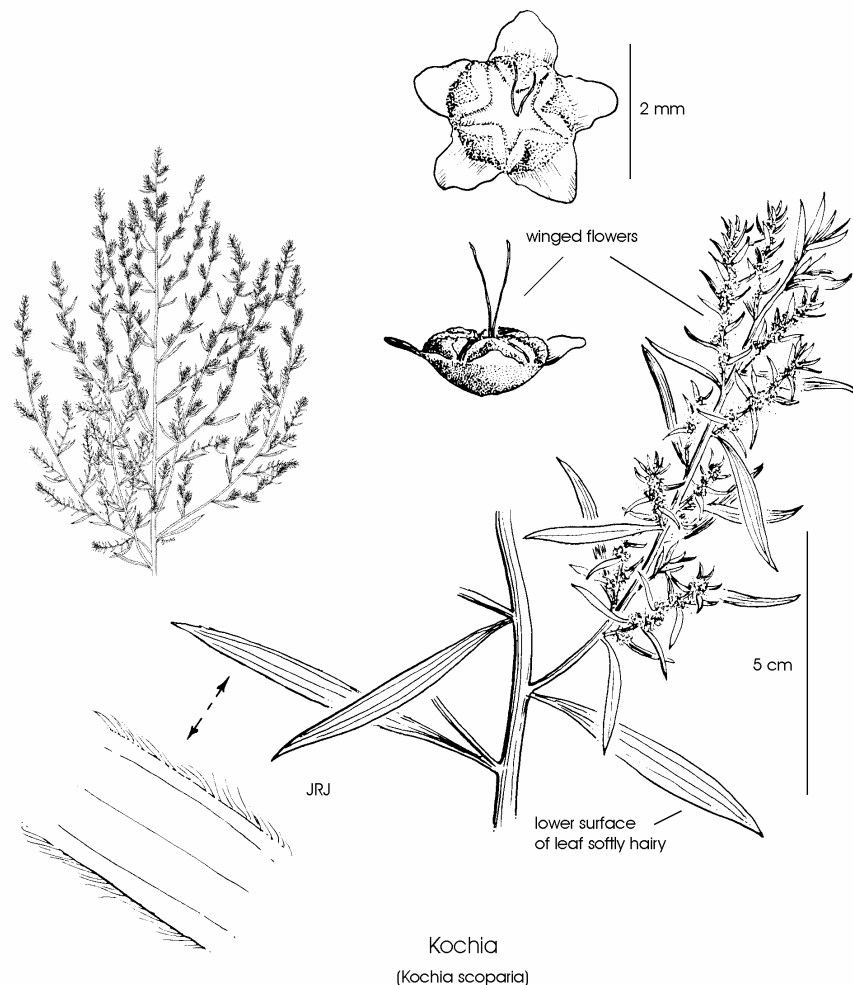
This plant was originally introduced from Europe for use in gardens because of its symmetrical shape and red color in the fall (37) but it has escaped to become a serious weed. It is a salt-tolerant annual which is common in cropland and roadsides. It typically

has a multi-branched Christmas-tree shape up to 1.5 m tall, with slim, often hairy leaves and inconspicuous flowers. During feed shortage, as in a drought, it may be necessary to use it for emergency forage, both as pasture or stored feed. At early bloom stage it has the nutritional value of 20% bloom alfalfa, but frequently it produces low weight gains. The sources of this problem are not clearly understood, but the plant is not considered toxic unless it constitutes a high proportion of the diet. Grazing of kochia for 2 or more weeks has caused such clinical signs as depressed appetite, swollen eyelids, crusty areas on the nose and sloughing of areas of non-pigmented skin (see Photosensitization). Both ruminants and horses are susceptible, and continued consumption may lead to death. Kochia can accumulate oxalates and they are retained in stored feed (see Greasewood). Losses have occurred when animals have been introduced suddenly to a diet high in Kochia. However, gradual introduction allows ruminants to safely adapt to oxalate.

Nitrates are periodically accumulated to a dangerous level in kochia (see Nitrate poisoning). On very saline soils kochia can accumulate high concentrations of sulfur and may cause brain lesions (see Polioencephalomalacia). Liver damage has been reported, with resultant photosensitization reflected in skin lesions. Kidney damage may also occur. Saponins and alkaloids have been found in kochia but a direct link to symptoms is not conclusive. A survey of Alberta farmers reported that kochia became a problem only when it made up more that 50% of the animal's diet. Toxicity varies with growth stage. Kochia should be diluted with other forage, to a maximum of 30% of the diet when plants are young, and 40% for later stages of growth.

Distinguish this annual weed from **immigrant forage kochia** or **prostrate kochia** (*K. prostrata* (L.) Schrade.). This is a semi-evergreen perennial which was introduced from central Eurasia and is widely used in the western USA for fall/winter grazing and as a green fire break. Although unrelated to kochia, leaves of **rhubarb** (*Rheum rhaponticum* L., Polygonaceae) also contain high levels of oxalate and are extremely dangerous. However they are unpalatable.

Poison-hemlock (*Conium maculatum* L.), Carrot Family, Apiaceae (Umbelliferae)

Poison-hemlock is a large biennial species introduced as an ornamental from Eurasia because of its attractive fern or carrot-like foliage. It is not common in Western Canada, occurring mainly in southwestern BC, and rarely in south central BC. Its large umbrella-shaped cluster of small white flowers leads to confusion with other members of the carrot family; related species are described with Douglas' water-hemlock. It produces large amounts of seed, and can encroach on cropland. In the second year it grows from an over-wintered rosette, with a smooth, stout stem up to 3 m tall, clearly purple spotted. Its leaves are large, smooth, and much divided. The large, white, unbranched taproot is easily distinguished from the root of the water-hemlocks because the base of the stem and the root are not chambered. Although it requires ample moisture, it is not semi aquatic so it has a broader range of habitat than do the water-hemlocks.

This plant is acutely toxic to all classes of livestock. Fresh plant material consumed at 5 and 10 g/kg body wt can be a lethal dose in cattle and sheep respectively (64). The toxic principle in poison-hemlock consists of a group of piperidine alkaloids. These alkaloids are also teratogenic, causing skeletal deformations in the fetus (64). Leaves are especially dangerous in spring if forage is scarce, but toxicity increases with maturity, and roots only become toxic late in the season. Seeds can be very toxic and can be a problem if they fall into a poultry pen or are harvested with grain. Symptoms include muscular weakness, loss of coordination, trembling, knuckling of the fetlock joints, excessive salivation, cyanotic membranes, central nervous stimulation followed by depression, dilated pupils and frequent urination and defecation. Treatments include administration of stimulants and large doses of mineral oil to save animals that have not eaten an excessive amount of poison-hemlock.
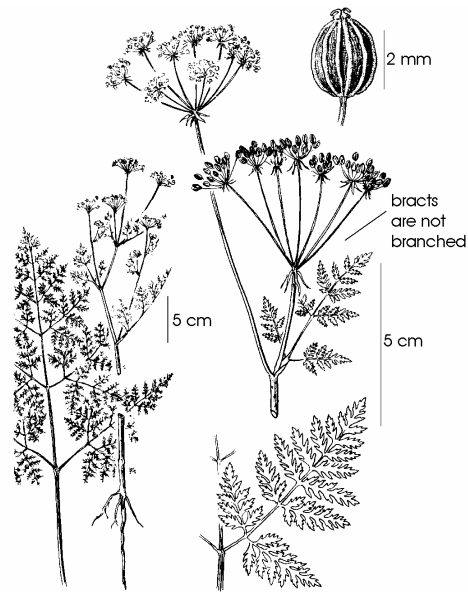
Poison Hemlock
(Conium maculatum)

Toxic

Stinkweed (*Thlaspi arvense* L.), Cabbage Family, Brassicaceae (Cruciferae)

Stinkweed is a native of Europe and is a common annual weed of croplands and disturbed sites. Often over wintering as a low rosette, it reaches up to 0.6 m, has white flowers and distinctive round flat seedpods with a broad winged border. Cattle rarely graze it, but may consume it in hay. The seeds may be present as a contaminant of grain or screenings, but usually at a safe level. Stinkweed seeds contain high levels of glucosinolates (sinigrin) and digestion releases the irritant oil allyl thiocyanate. The oil can cause profuse swelling of the fore stomachs, mucosal necrosis and bleeding of the cecum and colon. The animals become colicky, develop bloody diarrhea and will die if the seed source is not removed. The oil is stable in the rumen (40) so it can be transferred to milk. Making silage of glucosinolate-containing forage can reduce the glycoside content by 90% (24). Heat treatment of stinkweed-containing grain can render the feed safe. A related weed of grain fields, **brown mustard** (*Brassica juncea* (L.) Cosson) also contains sinigrin. In this mustard, the breakdown product in ruminants is allyl isothiocyanate (42) which causes similar symptoms in the animal but does not taint milk. Glucosinolates are present in many weeds and forages of the Cabbage family but their content is usually too low to be a health hazard.

Hoary alyssum (*Berteroa incana* (L.) DC.), Cabbage Family, Brassicaceae (Cruciferae)

Hoary alyssum is an annual or short-lived perennial weed, introduced from Eurasia, and is found across the region. Common in dry fields and on roadsides, it grows from a slender tap root, erect and branched to 1.1 m. The whole plant is covered with star-shaped hairs. White flowers are in clusters along and at the ends of slender stems, and flattened oval seedpods are held close to the stems along most of their length. A wide range of symptoms have been reported in the USA (17), including increased foot temperature, swelling of lower legs and stiffness of joints. Symptoms occur in horses eating the seeds in heavily infested pastures or in contaminated hay; the plant is usually not consumed by grazing animals unless they are very hungry. This plant contains glucosinolates but the symptoms reported are not typical of those caused by these glycosides. Most animals recover quickly if exposure to the plant is stopped.

Black nightshade (*Solanum nigrum* L.) Nightshade family, Solanaceae

The nightshade family is also called the potato family and it includes many members of economic importance including the common (Irish) potato (*Solanum tuberosum* L.), tomatoes, peppers, ornamentals such as petunias and drug plants such as tobacco. The flower is quite distinctive, making it easy to recognize many members of the family. However, some members have long been in use by humans for medicinal, hallucinogenic and poisonous properties, so that the folk history of the Solanaceae family, combined with frequent misidentification of plants used in earlier toxicology studies, have complicated assessment of their potential risk to livestock in Western Canada.

Black nightshade
(Solanum nigrum)

Hairy nightshade
(Solanum sarrachoides)
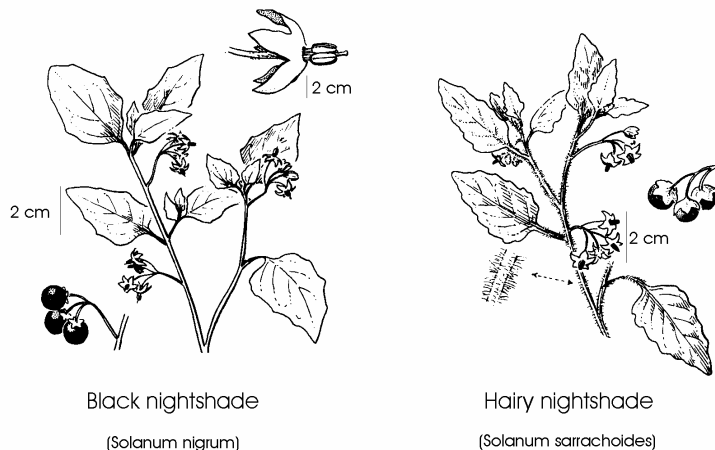
Black nightshade is an annual Eurasian weed found primarily on disturbed soil and therefore common in cultivated fields. It may be erect and up to 50 cm tall or prostrate, with relatively hairless and odourless ovate leaves and clusters of white flowers. Its morphology is extremely variable, so that it is known under many common names around the world. (Use of the name "deadly nightshade" for this species is incorrect and misleading; see "Belladonna" below.) Several subspecies are recognized; our plant is the subspecies *nigrum* because the few hairs which are present lack glands (11). Ripe fruit is a dull black berry with a short green calyx or cap like that on an eggplant. The primary toxic principle is the glycoalkaloid, solanine. The major disorders are irritation of the

gastrointestinal tract and impairment of the nervous system. The toxicity of this alkaloid is much greater when injected than when given orally. Ingested solanine is poorly absorbed and in the digestive tract most of it is metabolized to a less-toxic component, solanidine, and rapidly excreted. Despite the potential toxicity of the glycoside, and the widespread occurrence of the plants, solanine poisoning is uncommon (61).

For black nightshade, and for many close relatives, the record on toxicity is unclear. Environmental factors and other variables contribute to wide variations in toxicity. While several animal feeding trials have been conducted, a large proportion of the research on this weed's toxicity in North America was done before it became widespread, so the work was probably done on other *Solanum* species that were incorrectly identified (17).   Most reports of livestock problems are likely the result of large doses of the plant. In some crops like corn silage or first-year alfalfa the contamination by this weed may be heavy, warranting caution in using the feed. The level of solanine is reduced if forage is made into silage (71) presumably by formation of the less-toxic solanidine. The weed readily absorbs nitrogen and can contain up to 2.5% nitrate (82). A level of 0.4% nitrate (NO_3^-, not "nitrate-N") is considered safe so nitrate may account for some reported poisonings. Making silage of a crop also reduces nitrate content.

Several species of this genus are common in our region, and their toxic principals are mainly glycolalkaloids. There is general agreement that most can, under some circumstances, cause digestive upsets and even less frequently neurologic problems in livestock. Ruminants are considered to be less susceptible than other animals, with sheep and goats being less susceptible than cattle (17). The native species, *S. americanum* Mill., which is also called **black nightshade**, is not found in Western Canada.

Two similar white-flowered and weedy species of this genus are found in parts of this region but, in the absence of research evidence, their toxicity should be viewed with caution. The first of these is **hairy nightshade** (S. *sarrachoides* Sendt.) which is from S. America. It often has a spreading growth habit, reaches 80 cm, and is densely covered with short glandular hairs on leaves and stems, with calyx cupping over half of the yellowish or greenish berry. Trials with feeding this fruit to hamsters did not demonstrate toxicity (10).

Wild tomato or **cutleaf nightshade** (*S. triflorum* Nutt) is a low-growing native annual on disturbed areas, mainly on the southern Prairies. The leaves are sparsely to moderately hairy, while the fruit is greenish. Although some popular press articles call this plant highly toxic this could not be verified with scientific literature.
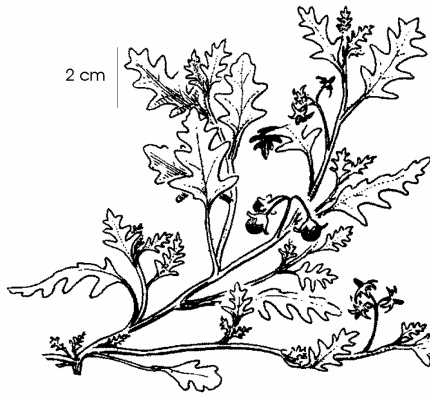
Wild tomato, Cutleaf nightshade

(Solanum triflorum)

Buffalobur

(Solanum rostratum)

JRJ

Buffalobur (*S. rostratum* Dunal) is a striking yellow-flowered native annual widely found on disturbed sites and sandy soils in the western USA. It has been reported in our region in gardens and on reseeded rangeland, having been introduced as a contaminant in seed mixes. It is low growing, with stems up to 60 cm long. The entire plant, except the petals, is covered with straight spines and the leaves are covered with star-like hairs, which reduce its palatability so that it is not considered a toxicity problem. The fruit is a spiny bur which can become caught in sheep's wool. Accounts of its toxicity may have all originated from a 1943 account of an isolated case of mismanagement which resulted in pigs consuming toxic amounts of this plant (17).

purple flowers

red berries
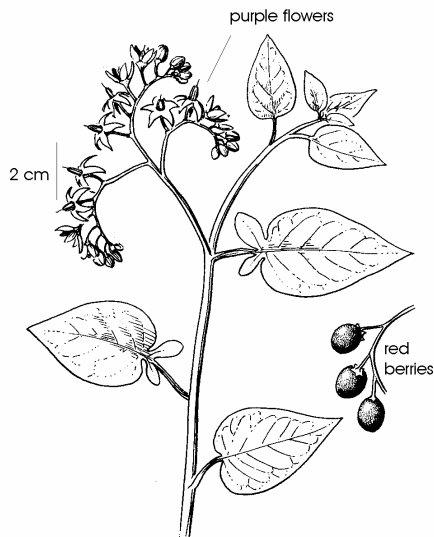
Climbing nightshade, Bittersweet

(Solanum dulcamara)
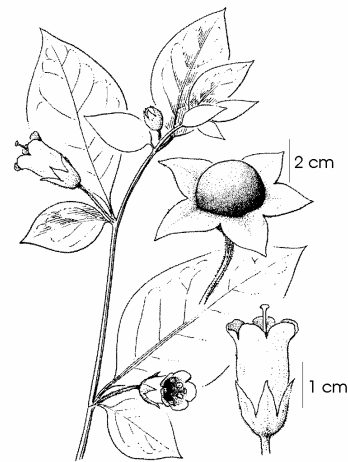
Deadly nightshade

(Atropa belladonna)

Climbing nightshade or bittersweet (*S. du1camara* L.) is a European weed that is often erroneously called deadly nightshade. It is a rhizomatous perennial, with long stems (without tendrils) which sprawl over other vegetation to a height of 3 m in moist areas. It

has blue to violet flowers with prominent yellow anthers, but is most conspicuous for its clusters of bright red egg-shaped berries 8-11 mm long. The main toxic principles are glycoalkaloids, but toxicity in feeding trials with livestock is not documented. As with other species of this genus, animals should not ingest a large quantity of the plant. If clinical signs, such as salivation, indicate nervous system disorder atropine or physostigmine may be administered (17). **Deadly nightshade** (*Atropa belladonna* L.) is a European perennial which may occur as a weed or as an escape, but is uncommon. It has been grown as a source of the drug atropine and for its hallucinogenic properties. Its toxic principle is a group of tropane alkaloids. Its significance here is mainly that its common name has been applied erroneously to black nightshade and climbing nightshade.

The common **potato** plant is widely reported as being toxic, primarily due to the glycoalkaloids chaconine and solanine, (both of which degrade to the less-toxic aglycone solaninidine during digestion). Low levels of these toxins are considered necessary for flavour, but too much causes bitterness. Alkaloid content is heritable so there is a maximum level used in screening new potato varieties (19). Potato tubers are stem rather than root tissue. Alkaloid content increases when exposure to sunlight promotes greening of the tubers, or when the tubers sprout new stems. Greening of potatoes is a major reason for culling tubers, and culls are frequently used as cattle feed. Dilution with other feed is recommended when green tuber content is high. Consideration must be given to preventing animals from choking on whole tubers. Because of its high energy content potato waste is similar to moist feed grain in the diet, although the starch has lower digestibility. Surplus cooked potato products are successfully used as cattle feed. Cooking increases digestibility of the starch and destroys several inhibitors of digestive enzymes, to the extent that this material can be safely fed to pigs (70). Potato vines contain higher amounts of alkaloids than do tubers. The content of berries is somewhat greater than that of tubers (27). There are anecdotal reports of problems occurring when cattle have been allowed to graze fields from which potatoes have recently been harvested and when cattle have been hand-fed fresh tops. Potato vines are a mechanical problem for harvesting equipment, so are typically desiccated before harvest by killing with a non-translocated herbicide. Because this practice is perceived as environmentally unsound, there is recurrent interest in harvesting vines for feed before potatoes are dug. Nicholson et al. (60) used laboratory silos to evaluate potato top silage, adding about 2/3 other feed to absorb water, and found the product palatable to sheep and much lower in solanine than predicted from the content of the fresh plant. Currently the main impediments to the use of potato tops as silage appear to be pesticide residues and high moisture content, not toxicity. The green fruit and vines of **tomato** (*Lycopersicon esculentum* L.) also contain glycoalkaloids but an extensive feeding trial with dried tomato vines failed to show any adverse effects in cattle other that weight loss (75). There are anecdotal reports of tomato vine toxicity (17) so it may be prudent not to allow livestock to eat large amounts.
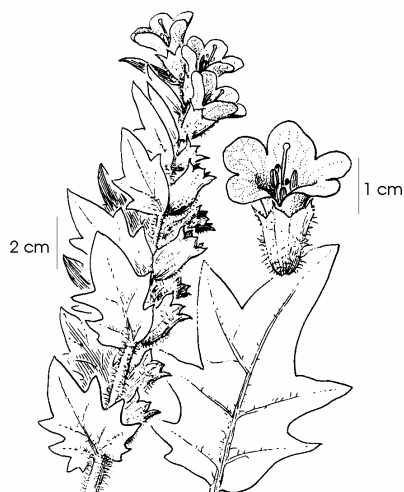
Black henbane

(Hyoscyamus niger)

toxic
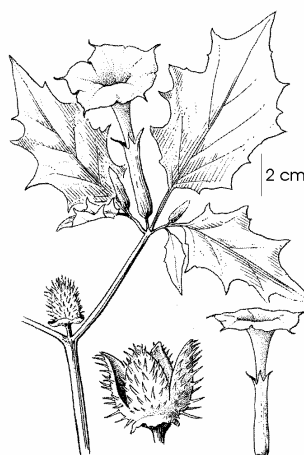
Jimsonweed , Datura

(Datura stramonium)

toxic

Black henbane (*Hyocyamus niger* L.) was introduced for its medicinal properties and now is a weed of roadsides and pastures on the southern Prairies, but is rarely found in cultivated fields. It may grow as an annual or biennial, the latter producing only a low rosette of lobed leaves in the first year. The second year it reaches up to 1 m tall, a simple or slightly branched erect stem with clasping leaves, all covered with long sticky hairs. Flowers are bell-shaped, 25-35 mm wide, yellowish green with purple veins, concentrated on one side of the stem. Symptoms from ingestion by humans include hallucination. Its historical association with sorcery may be responsible for causing many more problems now with ill-advised use by humans than with consumption by livestock. The plant has a strong odour and is not consumed by animals unless other forage is lacking. The main toxic principle is a group of tropane alkaloids. The toxins are retained in hay and have been reported in honey. Clinical signs include restlessness, pupil dilation, increased heart rate, labored breathing, bloat and, on occasion, seizures. Typically there are no lesions, and victims usually recover completely, with deaths most likely due to misadventure while impaired by a high dose. Treatment is not usually necessary, but (hourly) administration of physostigmine may be helpful (17). Death from henbane has been observed in cattle confined to a corral in Alberta (M. H. Benn pers commun).

Jimsonweed (*Datura spp.* L.) is a tropical genus of which several species are used here as ornamental annuals. Some species have naturalized as weeds of roadsides and other disturbed soil. They are large coarse erect plants with strong-smelling leaves and large upward-facing trumpet-shaped and highly fragrant flowers. The large seed pods are covered with prickles, hence the alternate common name, **thorn apple.** The toxic principles are tropane alkaloids (85). Toxins are present in all parts of the plant, increasing with plant maturity, and remaining after drying. Seeds have the highest concentration. Fresh plants are very unpalatable, but dried material may be eaten. Since these compounds are relatively insensitive to heat, the seeds are a concern in cereal grains, particularly if the seeds are ground. In pigs 1-2% of ground seed by weight is considered toxic (85) and in horses 0.5% for 10 days produced symptoms (83). In cattle

5% ground seed reduced intake and caused some intoxication (59), but 1% is thought to be sufficient for concern. In chickens 3% of ground seeds only decreased intake, and there was no effect at 1% (17).

TREES

Black walnut (*Juglans nigra* L.), Walnut family, Juglandaceae,
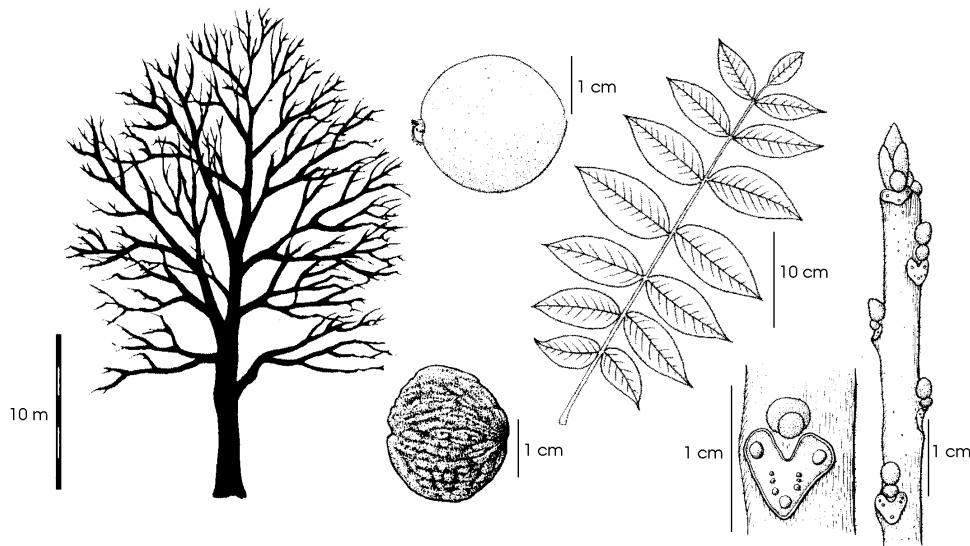
Black walnut
(Juglans nigra)
toxic

Black walnut is a native of eastern North America which is infrequently planted in southern BC. Black walnut and **butternut** (*Juglans cinerea* L.) are harvested for lumber in the USA, where the use of their shavings for bedding has been found to rapidly cause reversible laminitis in horses. As little as 20% contamination, with shavings less than one month old, can be a problem (17). Black walnut contamination is indicated by the presence of black shavings strips. The toxic principal is not known, but the compound juglone, a napthoquinone that is toxic to many other plants within the tree's root zone (17), has been virtually eliminated as a possibility. The tree poses no problem when used for shade in horse pastures, except in spring when pollen from the flowers reportedly has induced an allergic reaction or laminitis.

Horse chestnut (*Aesculus hippocastanum* L.), Horse chestnut Family, Hippocastanaceae

This is a medium sized tree, native to south eastern Europe, used as an ornamental in the warmer regions of south and coastal BC. Flowers are in large showy erect clusters, and fruits are large, greenish spiny capsules containing shiny brown nuts with a large pale spot. Extracts of the nuts have long been used as herbal medicine, and considerable research has been done on the effects on humans. The coumarin glycoside aesculin was originally implicated as the toxic principle because its hydrolysis product resembled coumarin (see Sweet clover). More recently the toxic effects have been attributed to a group of 30 or more saponins (17). All classes of livestock can be affected. The first clinical sign is a sawhorse stance with reluctance to move. If required to move the animal will be uncoordinated and have abnormal posture. Seizures and collapse may follow, but death rarely occurs. Typically the incapacitation, though dramatic, is temporary, and recovery occurs in a day or two. The nuts and young shoots are the most toxic. Young foliage is palatable so there may be a problem in spring but mature leaves are considered less of a risk. The onset of symptoms decreases intake so the disease is considered self-limiting.

10 cm

10 m

2 cm

full-size tree
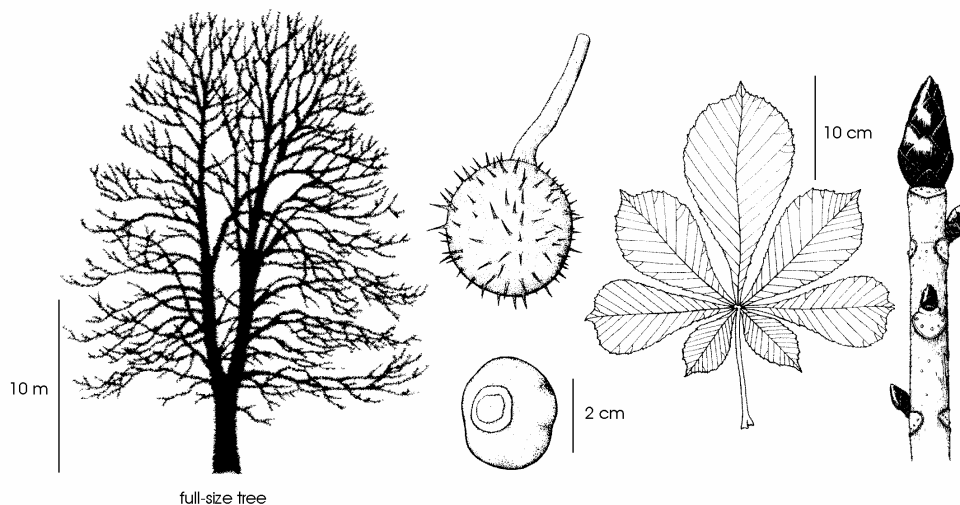
Horse chestnut

(Aesculus hippocastanum)

toxic

Red maple (*Acer rubrum* L.), Maple Family, Aceraceae

Red maple is a medium sized tree whose leaves can be toxic to horses. Native to eastern North America, it is used as an ornamental in our region. Clusters of red tassel-like flowers appear in late winter, long before the leaves (25). The leaves have 3-5 shallow notches, and in the summer are a glossy light green on the upper surface, with a white underside. They turn bright red (carmine to orange) only in autumn.

Maple poisoning only occurs when the leaves are dry or wilted, and they remain toxic for about 30 days after dropping from the tree. An unknown plant constituent is apparently converted to an oxidant during drying.

Red maple
(A. rubrum)
toxic

Silver Maple
(A. saccharinum)

Sugar maple
(A. saccharum)

Norway maple
(A. platanoides)

Douglas maple
(A. glabrum var. douglasii)

Amur maple
(A. ginnala)
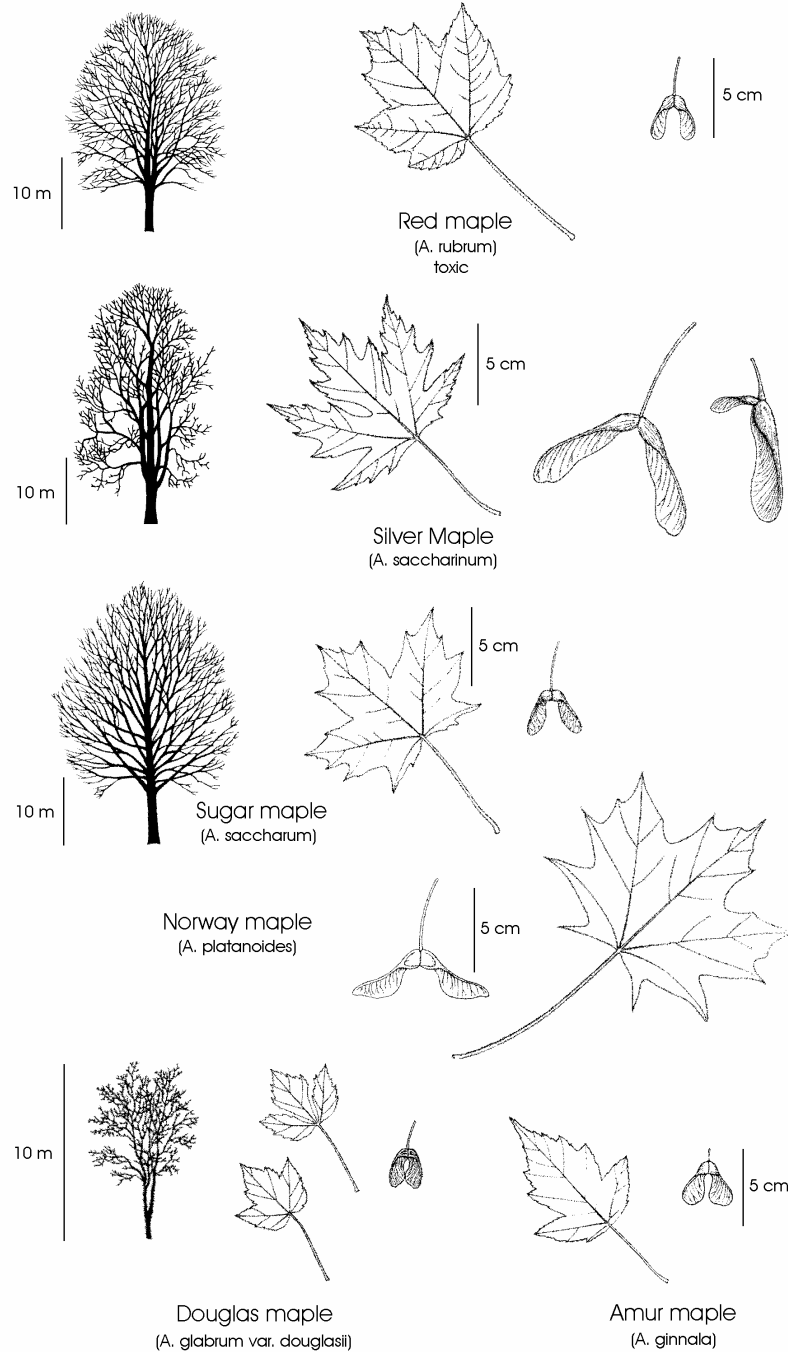
There are two syndromes involved in the toxicity. The oxidant combines with blood hemoglobin to form methemoglobin, thus destroying the blood's oxygen-carrying

capacity (see Nitrate poisoning). Less than 2 kg of leaves is sufficient to kill a horse, and often the damage has been done by the time the problem is noticed. Treatment is usually unsuccessful, but intravenous administration of large doses of ascorbic acid has been effective in some cases. Cattle, sheep and goats are not susceptible.

Another syndrome in horses caused by red maple is Heinz body haemolytic anaemia (see Onion). Mares can abort without symptoms being noted. This species is known to hybridize with another eastern native, **silver maple** (*A. saccharinum* L.) (25). It is not clear if the toxicity is retained in crosses with silver maple, or with other maple species. Although most research has used leaves of red maple, there have been cases of poisoning where the only *Acer* present was identified as silver maple. Therefore this species is believed to be a potential problem (M. Murphy pers commun). The widely-planted and apparently non-toxic introduced maple that has burgundy leaves in both summer and fall is **Norway maple** (*A. platanoides* L.). The figure gives some characteristics to use in distinguishing red maple from several other species, including the native Douglas maple (*A. glabrum* var. *dougalsii* (Hook.) Dippel) and the introduced Amur maple (*A. ginnala* Maxim). Scale bars (25) show the maximum height attainable, but most specimens will be much shorter at maturity here.

If horse pastures cannot be planned to exclude maple trees, care should be taken to quickly remove red maple branches that have fallen, even in summer. Similarly, if it is not possible to avoid incorporating red maple leaves in hay bales, it is prudent to store any suspect bales for a month to allow the toxin to dissipate, before feeding to horses.

Yew, (*Taxus* L.) Yew Family, Taxaceae

5 cm

fleshy
red fruit

1 cm
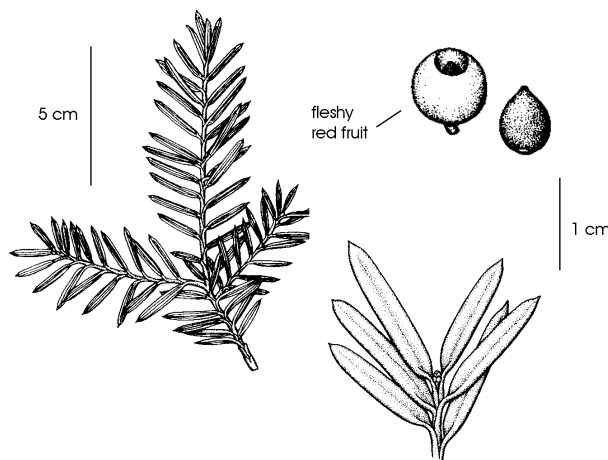
Western yew
(Taxus brevifolia)
toxic

This genus of very toxic evergreen conifers contains species ranging in size from small shrubs to trees. They are important because some of them are popular ornamentals in

temperate parts of BC. Yews are dioecious; female trees bear a distinctive fruit, a red, fleshy cup-shaped "aril" surrounding the dark greenish seed, which is toxic to humans.

English yew (*Taxus baccatta* L.) has long been grown here, and is particularly dangerous. In Europe it is a tree up to 25 m tall but the varieties used here are typically shorter, particularly when grown in cooler zones. It has ascending to erect branches, with dense dark green foliage. The toxic dose for cattle has been estimated at 0.36-0.7 g of fresh English yew per kg of body wt (66). The toxic dose for horses is even lower. **Japanese yew** (*T. cuspidata* Seibold & Zucc.), is less toxic but has been blamed for many livestock deaths (17). A species native to BC, **western yew** or Pacific yew (*Taxus brevifolia* Nuttall), is found in the understory of moist coastal forests and in interior cedar-hemlock forests. It is a bushy, untidy tree up to 9 tall, with drooping branchlets. Two other species are native to North America and other species are imported for gardens; likely the winter hardiness will be improved so the geographic range for yews may be increased in the future. Note that there are now many horticultural selections with differing physical characteristics; some even have golden foliage. Although yews are dangerous for domestic livestock and poultry, wild ruminants such as moose and deer have been observed to safely browse several of the species (17), and birds eat the seeds (15).

This genus contains many biologically active compounds but the primary toxic principle is a group of taxines, pseudo-alkaloids which cause heart or respiratory failure. It is easy for an animal to eat a lethal quantity. Although the foliage contains an aromatic oil which reduces its palatability, the green foliage can attract animals, particularly in winter. The toxin remains active in dried plant tissue for several months. Many poisonings have occurred because animals ate trimmings that were discarded in their pasture.

OTHER FORAGE PROBLEMS

Acute Bovine Pulmonary Emphysema (ABPE)

Seasonal losses of range cattle associated with non-infectious, acute respiratory distress have long been known in the interior of BC. Some other terms for the poorly understood syndrome are "Fog Fever", "Skyline Disease" and "Atypical Interstitial Pneumonia". The USA term is "Acute Respiratory Distress Syndrome".

Animals mildly affected with ABPE are alert but breathe quickly and normally recover on their own. They stand apart from the herd, are reluctant to move, and may seek shade. Severely affected animals stand with their mouths wide open gasping for air. Coughing is rare, but there may be a fever. Death can occur within 24 hours of initial signs, especially after exertion. The condition can occur very quickly, so cows may be found dead without any warning signs.

Typically the clinical signs appear within 2-4 days following a drastic change from a dry, low energy diet to a relatively lush, rapidly-growing pasture. In BC, the low-energy pasture is typical of the late summer forage in the dry interior forest range and the lush

pasture is typical of the fall re-growth on grassland range or home pastures, especially when there is adequate moisture during September and October. However, an obvious contrast in pasture quality may not be necessary for the symptoms to appear, so careful observation of the herd is prudent whenever cattle are moved between pastures, particularly in the fall. As cattle choose to graze the best part of a pasture first they may be at risk, even if most of the crop appears safe. Sudden introduction to pasture of members of the brassica or cabbage family (rape, turnip tops, kale) has also been implicated.
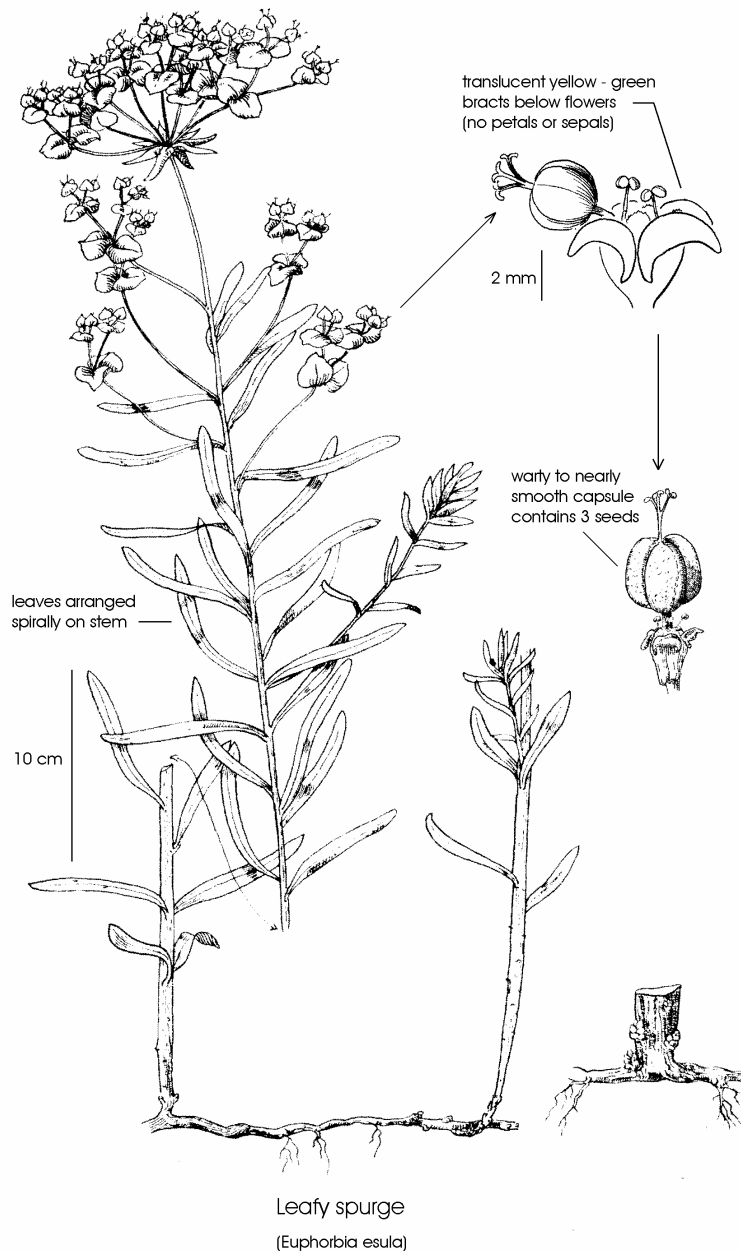
On pasture the cause is believed to be derived from tryptophan, a naturally-occurring amino acid. Apparently the concentration of this compound is high in rapidly-growing tissue, particularly in the fall. It is not clear why the component is dangerous in fresh forage but apparently not in wilted or stored feed of the same origin. The tryptophan in pasture is believed to be converted to another compound in the rumen and carried by the blood to the lungs, where a further metabolite binds to cell walls and causes damage. In response, lung passages become filled with viscous fluid and air-filled "blisters". The critical result is that the lung capacity is suddenly greatly reduced. At post-mortem, affected lungs are large, heavy and fail to collapse. The syndrome is reported as being largely restricted to mature cattle, while nursing calves are unaffected. A sudden onset of pneumonia-like symptoms in range cows, with no cases among their nursing calves, points to ABPE. In theory, the calves would be more susceptible than their mothers to an infectious disease, so having only cows affected is an important indicator that diet is a factor. In beef-cattle grazing studies near Kamloops the condition was frequently observed in animals as young as one year old (49). It is the "lushness" of the pasture that is important and not the forage species. An *in vitro* test was conducted on preferred legume and grass species from emphysema-inciting pastures near Kamloops. Toxicity of plant samples to lung cell cultures was determined during a typical fall disease outbreak and in samples collected in spring. Black medic *(Medicago lupulina* L.) had no effect on lung cells and Kentucky bluegrass *(Poa pratensis* L.) was somewhat toxic, but no more so in fall than in spring (G. S. Yost pers commun).

 There is no prescribed treatment for animals with ABPE. Drugs such as the ionophore monensin (an antibiotic that suppresses the conversion of tryptophan in the rumen) have been effective in the USA for preventing or reducing the severity of this disorder. However, this approach may not be as effective here: at Kamloops a monensin (Rumensin CRC$^®$) bolus was administered to cattle 10 days before turnout on emphysema-causing pasture, but it did not prevent the onset of clinical signs (49). Affected cattle should be kept off the offending pasture and be fed only hay until they recover strength – usually in a few days. This must be done very carefully because the animals may not have sufficient lung capacity to survive any exertion. New cases may be observed in the days following observation of the first clinical signs in animals.

Prevention by pasture management involves avoiding a sudden dietary change, from a relatively poor pasture to a lush one. The condition is not like a food allergy, in which a relatively small amount of the allergen can trigger a reaction. It is more like an overload of tryptophan in the animal's digestive system, so management constitutes a temporary dilution of this dietary component. Allowing more time, as much as 10 days of gradual exposure is advised by some authorities, for adaptation of rumen microflora to the new,

lush diet. In the USA recommendations include: 1. cutting the forage and allowing it to wilt before it is consumed, 2. limiting grazing time on the lush pasture, 3. feeding hay in a drylot before each day's grazing or 4. delaying the use of the lush pasture until after a hard frost that kills the aerial growth.

Contact irritants

translucent yellow - green
bracts below flowers
(no petals or sepals)

2 mm

warty to nearly
smooth capsule
contains 3 seeds

leaves arranged
spirally on stem

10 cm

Leafy spurge
(Euphorbia esula)

Certain plants cause skin irritations, rashes and gastric inflammations by primary contact without activation by sunlight. The milky latex of **leafy spurge** (*Euphorbia esula* L.) of the spurge family, Euphorbiaceae, causes severe dermatitis and irritation of the mouth

and digestive tract. The causative agents are phorbal esters (19). Sheep and goats will graze this plant but cattle usually avoid leafy spurge. Cattle also tend to avoid grazing members of the buttercup family, Ranunculaceae.

An example is **tall buttercup** (*Ranunculus acris* L.) which typically remains untouched in a heavily-grazed pasture. When chewed, the leaves of many plants of the family produce an intense burning sensation, after a short lag time. The plant contains the glycoside ranunculin as well as an enzyme that, when combined after cell disruption, produce an irritant oil. In BC, seven members of the buttercup family were found to produce significant quantities of ranunculin including species of *Ranunculus, Anemone, Clematis* and *Trollius* (8). Ingestion of the living plants can cause gastric distress including irritation of the digestive tract, abdominal pain and diarrhea. When plants such as tall buttercup are harvested for hay or silage, the resulting cell damage allows enzymatic release of most of the toxin. Therefore ranunculin content in stored feed is much reduced and usually does not cause problems.
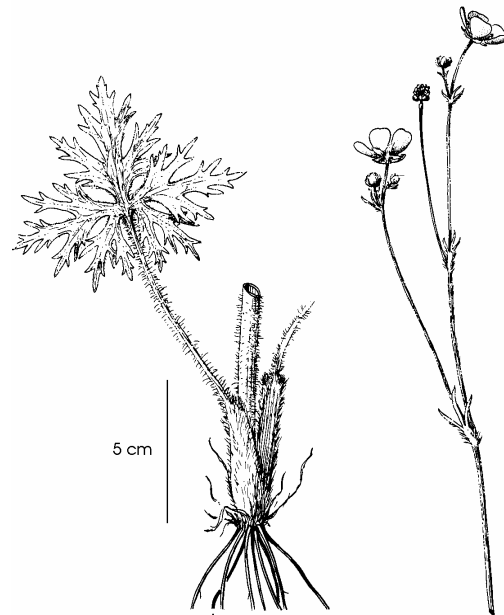
Tall buttercup
(Ranunculus acris)

toxic

Baneberry (*Actaea rubra* (Ait.) Willd., or *A. arguta* Nutt.), Buttercup Family, Ranunculaceae

This is a native member of the buttercup family to which we have addressed particular attention because of some alarming accounts in the literature. An herbaceous perennial, it is erect, 30-100 cm tall with one to several branched stems. Leaves are 2 or 3 times divided into threes, with coarsely toothed leaflets. The inflorescence is a terminal or axillary cluster of small white flowers, with conspicuous long white stamens. Fruit is a smooth glossy bright red or, less frequently, white berry. It is widely distributed across Western Canada, in rich woodlands and on stream bank beaches and open slopes. Authorities on North American poisonous plants do not list this species as being toxic (36, 19). However, some recent lists of toxic plants include *Actaea rubra*, and even incorrectly state that it contains ranunculin. Typically these accounts refer to an anecdotal report of a taste-test of some berries. However, they fail to mention the editor's note, included with the 1903 report. The editor pointed out that the *A. spicata* plant, from which the supposedly-toxic berries were taken, had been transplanted from England, so was in fact not our baneberry species.

In a survey of the occurrence of ranunculin in BC species of the buttercup family (8), this glycoside was not detected in *A. rubra*. During collections of ripe baneberries near Kamloops in 2003 and 2004, it was observed that deer had browsed many of the flower stems.

Glossy red or white berries

3 cm

flower clusters look like fuzzy white balls because of many protruding stamens

5 cm

2 mm

Baneberry
(Actaea rubra)
not proven to be toxic
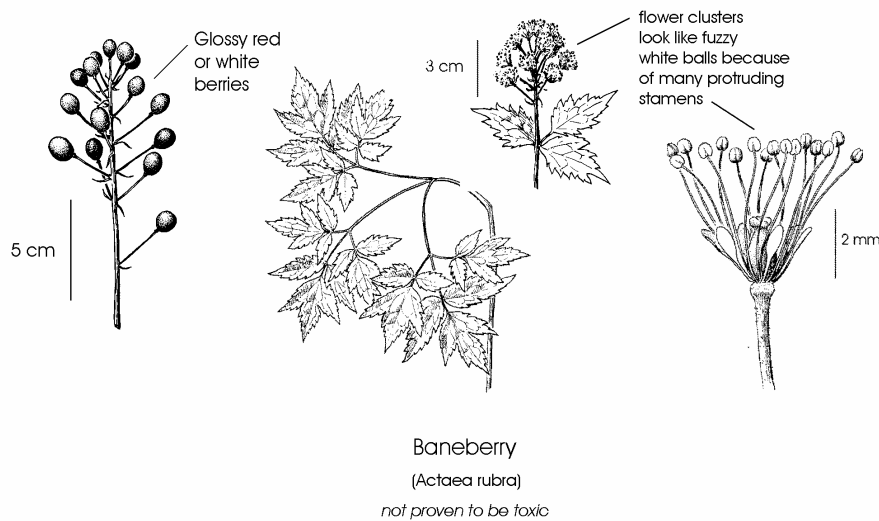
Note that a recent revision of taxonomic nomenclature has assigned all species of *Cimifuga* to the genus *Actaea*. **Black cohosh** (*C. racemosa* Nutt), the most widely known American *Cimifuga* species, has long been used as an herbal treatment for variety of conditions in humans. Our studies on *A. rubra* berries and those of other *Cimifuga spp.* have yielded a complex mixture of triterpene glycosides (W. Majak unpubl data) which may have pharmacological properties but are not considered acutely toxic.

Fungal toxins (Mycotoxins)

 Mushrooms

Mushroom poisoning in livestock has not been documented in Western Canada, even though some highly toxic species are native to the region. For instance, several toxic members of the genus *Amanita* occur in forests of southwestern BC, particularly on Vancouver Island (81). It is unclear whether animals avoid toxic species during grazing but cattle have been observed to seek out large non-toxic mushrooms for consumption.

 Endophytic fungi

Some species of fungi cause toxicity problems in specific plants and these are addressed elsewhere in this manual. Endophytic fungi produce toxins within the living cells of such plants as **tall fescue**, **perennial ryegrass** and **locoweed**.

External fungi

Fungi visible on the leaves of some legume species are implicated in a complex of photosensitization symptoms, which include "**alsike clover** disease" and "trifoliosis" (see Alsike clover).

Decay fungi

A group of decay fungi species produce mycotoxins from non-toxic constituents of sweet clover. Mycotoxins can also be produced when any forage is improperly harvested or stored, but a large number of fungi species are involved and the situation is not clearly understood. Animals, particularly ruminants, are able to withstand some fungal toxins. A case in point is *Fusarium* head blight (9), a grass and cereal disease that under some conditions can produce the tricothecene mycotoxin deoxynivalenol or "vomitoxin". In ruminants and poultry this toxin is poorly absorbed, metabolized and quickly excreted. However in pigs vomitoxin is well absorbed from the feed and becomes a serious problem.

All forages grown under natural conditions carry the spores of many species of fungi. Given the presence of some oxygen, and the right conditions of moisture and temperature, the spores will proliferate and produce moldy or mildewed feed. The appearance of silage is not a reliable indicator of risk as not all moldy feed contains toxins, and optimum conditions for growth of toxin-producing fungi may occur only periodically. Some mycotoxins suppress immunity, which further complicates diagnosis. Actual identification of decay fungi or their toxins by laboratory analysis of moldy stored feed is currently considered impractical for livestock managers (in contrast with the assays for endophytic fungi in fescue and ryegrass). It is believed that a significant proportion of the abortions in cattle are caused by toxins contained in moldy feed. Abortions usually occur long after the toxin is absorbed so determining cause is difficult.

As the fungi grow, they utilize the nutrients in the feed. If their growth is rapid, heating can occur, to further reduce feed quality. Moldy feed has a lower palatability which can reduce feed intake and gains. It can cause digestive disturbances. It is usually economically undesirable to destroy all such feed, but its limitations as livestock feed should be recognized and managed. It has long been recommended that moldy silage be diluted with undamaged feed to lessen its negative impact. However recent research has indicated that this course of action may not be economically sound. An uncovered silage pit can have spoilage down as far as a meter. Mixing this material from the top of the pack with the undamaged feed may cause a reduction in productivity sufficient to negate any feed savings. To minimize losses from the mycotoxins and nutrient deficiencies it is prudent to avoid feeding moldy feed to pregnant, lactating and growing livestock (69). Proper packing and covering to maximize exclusion of air during storage is essential. Once a silo or tube of baled silage is opened it is important to minimize exposure of silage to the air before feeding. Ammoniation of hay and corn has been shown to prevent

spoilage. The reader is referred to the 2004 Alberta Silage Manual (3) for detailed information.

When dry moldy hay is fed the clouds of spores that are released can be damaging to the respiratory system of all livestock (and humans). Horses are particularly prone to an allergic reaction to the spores which cause airway inflammation and reduction in breathing capacity. One of the most common diseases of horses, it is termed "chronic obstructed pulmonary disease" or "heaves" (69). In cattle, when spores are inhaled the pathogens cross into the blood stream, and can cause fungal infection of the fetus, resulting in its death. Inhalation of the spores may constitute a more important source of abortion-causing infections in cattle than does ingestion of mycotoxins in the feed.

Ergot (*Claviceps purpurea* (Fr.) Tul.)

Ergot is a naturally occurring fungus that infects grasses and produces mycotoxins. It is non-endophytic, unlike the fungi in tall fescue, living as an external parasite on the plant. Airborne spores settle in the immature grass flowers, replacing the ovary with a mycelium that enlarges, darkens and eventually hardens into a sclerotium or fruiting body. This black ergot "body" is roughly the same shape as the grass seed but is easily seen because it is usually 2-5 times larger. In Canada, rye and triticale are most susceptible because they are cross-pollinators, requiring a longer period to become pollinated, but other cereals (including perennial cereal rye) and many grasses (including tall fescue) can also become infested. Moist conditions favour infection. The ergot body contains a number of toxic ergot alkaloids, varying with the grass species. Therefore some different symptoms are seen in the USA because of different host grass species there. Ergot alkaloids have a direct stimulatory effect on smooth muscle, causing vasoconstriction and increased blood pressure in arterioles, intestines and uterus.

There are three ergot-alkaloid syndromes in Western Canada: peripheral, hyperthermic and reproductive (69):

1. Clinical signs of peripheral ergotism are most often seen in extremities, lower parts of the hind legs, tail and ears. Reduced blood flow leads to coldness, reddening, loss of hair, lack of sensation, followed by death of the tissue. Effects are more pronounced at low temperature. Severe diarrhea may be evident.
 2. The hyperthermic syndrome occurs in hot weather. Cows have elevated temperature, fail to shed the winter coat, are susceptible to heat stress and seek shade and water.
 3. Reproductive ergot toxicity is uncommon in cows but reduced fertility and milk production have been observed in mares and ewes.

 Problems can occur with both stored feed and pasture. Feed should not contain ergot at over 0.1% of dry matter intake. For a 400 kg cow this is about 120 ergot bodies in 24 hours. Pasture is safe if grazed before flowering or if the infected heads are clipped off before use. The most likely source of ergot toxicity is in grain screenings. Dilution may be possible, but pregnant stock should not be given any ergot-containing feed. Ergot alkaloids are not transferred in milk.

Grass tetany (Hypomagnesemia)

This is a complex metabolic disorder of mature cattle or sheep on spring pasture of cool-season grasses (51), which is attributed to a deficiency of magnesium (Mg) or a reduction in its absorption. Older cows in early lactation are most often affected, because of their high Mg requirements. It can occur as quickly as 1-2 weeks after animals are moved to a new pasture. Clinical signs include nervousness and muscle twitching in mild cases. This may develop to excitement, increased reaction to noise or touch, frequent urination or defecation, stiffness, lack of coordination, and sometimes aggression with head held high. Severe cases progress to collapse, struggling, convulsions, coma and death. The condition has been confused with hypocalcemia (see Milk fever), which also can result in collapse of the animal. Grass tetany differs in its symptoms of extreme stiffness. An indication with a corpse is evidence at the site of thrashing or paddling of the feet, seen in cases of tetany but not in cases of milk fever. Accounts of grass tetany disorder typically note that it is a disease of farms with better forage management, because it often occurs on pasture that has received high levels of nitrogen and potassium fertilizer or manure. Therefore, it is increasingly a concern where there is a reduction in the land available for disposal of manure from confined livestock. The condition occurs with many grass species, including cereals, most often with lush, rapidly growing pasture and in cold wet weather. Magnesium availability drops if the forage is high in certain other minerals, for instance potassium and nitrogen, but high levels of such nutrients appear to be tolerated if the Mg content is above 0.25% of diet dry matter (33).

Treatment is with intravenous solutions of Mg and calcium borogluconate. This is most effective if the animal is still standing, although this can be difficult to do on pasture. Since Mg is rapidly excreted in milk and urine, it is possible to prevent the disorder by supplementing with Mg salts while the animals are on feed which is low in Mg, but not before. Pastures can be managed to reduce the risk as follows: (a) grow legumes with the grass, (b) apply lime to decrease soil acidity and choose the high-Mg (dolomite) type, (c) avoid heavy fertilizer or manure application in the spring, and (d) supplement with grain to improve availability of Mg (73). **Winter tetany** is the term applied to this syndrome when it occurs during winter on poor pasture, or on such stored feed as cereal straw, green feed or silage.

Milk fever (Hypocalcemia)

Milk fever is a condition of cattle, sheep and goats occurring around the time of giving birth. The group most susceptible is high producing dairy cows, most frequently Jerseys, over five years old (69). It is caused by serum calcium deficiency: Calcium (Ca) leaves the blood to support milk production more quickly than it can be replaced from the diet and from body stores in skeleton and kidneys. Affected animals become weak, unable to stand and may die. A corpse will usually show no signs of a struggle, in contrast to death from magnesium deficiency. Affected animals respond quickly to intravenous administration of calcium borogluconate.

Normally cows respond to the high Ca demand by secreting parathyroid hormone to enhance absorption of Ca from body stores. If this hormone-activated process fails, milk fever can occur. Until 1984 it was believed that a high Ca diet prior to calving depressed the hormone activity during the dry period, thus rendering cows susceptible to milk fever (69). Therefore suggested management consisted of feeding a low-Ca diet prior to calving. However such a diet is difficult to formulate(31) and it may be low in other important nutrients, which is contrary to the school of thought that the late-pregnancy cow should be fed well, to improve body stores for later milk production, antibody content of the milk and fertility; important objectives in beef as well as dairy operations.

A review by Goff (31) outlines some advances in research on milk fever. The syndrome has been shown to be caused by metabolic ketosis induced by diets high in such components as potassium (K) and nitrate, which cause the blood to become more alkaline. When these minerals were not in excess, research showed that high Ca levels in the diet did not cause milk fever. However, a low-Ca diet was still able to prevent the milk fever caused by high K content. Magnesium deficiency can also affect parathyroid hormone action and lead to milk fever, so supplementation may be required. Nutritionists can add chemicals to the feed which will offset the negative effects of K during the last weeks of gestation, but the additives can be unpalatable. Goff concludes that, for the last weeks of gestation, the best strategy is to incorporate low-K forages in the diet (51).

Alfalfa typically has a higher K content than grasses, and alfalfa hay from fields receiving heavy applications of K fertilizer and manure will be very high in K. There are differences between grass species in K content. Tremblay et al. (80) compared 5 cool season grasses and found that **timothy** (*Phleum pratense* L.), particularly in the second cut, had the lowest K content so was the best suited for reducing milk fever in dairy cows. **Orchardgrass** (*Dactylis glomerata* L.) was the least suited in their test group, while **meadow bromegrass** (*Bromus riparius* Rehmann), **tall fescue** (*Festuca arundinacea* Schreb.) and **smooth bromegrass** (*B. inermis* Leyss.) were intermediate. Corn, which is warm-season grass, has a low K content, so corn silage is a good choice for late-gestation cows (51). Preparation of a pasture dedicated to cows in late pregnancy can be worthwhile, and involves changes to fertility management in addition to choice of forage species. If soil K is allowed to drop severely, alfalfa can have a K level close to that of some grasses (51). Reducing soil K levels requires avoiding use of any farm manure, reducing the K component of chemical fertilizer applications and even planting crops to absorb soil K, which will be removed when hay is harvested prior to the field being used as a pasture.

Laminitis (Founder)

This is an important cause of lameness, most commonly in horses but also seen in cattle. The term "founder" describes the mechanical result, the lameness due to laminitis. This extremely painful condition is one of the most frequent causes of death in horses, largely because animals do not respond sufficiently to treatment, and must be euthanized.

Laminitis is due to a malfunction of the blood supply to the living laminae tissue of the hoof, which supports the bones of the hoof and therefore the weight of the horse. The blood supply problem is most often caused by a grain or lush-pasture overload but may also be caused by such stresses as infection, retained placenta, plant toxins (see Black walnut), long confinement and obesity. Typically horses, and especially ponies, develop laminitis when grazing pasture with high protein and soluble carbohydrate content. Rapid growth of bacteria in the cecum and large intestine results in production of large quantities of lactic acid and endotoxin which leads to reduction in blood flow to the laminae and to eventual death of the laminae. Consequently the poorly-supported coffin bone within the hoof may rotate and sink through the bottom of the hoof. Clinical signs include evidence of pain, particularly in the front feet. The horse will attempt to shift the weight off of the front feet by tucking the hind feet as far as possible under the body while standing, with head held low and back arched. It will lie down for long periods of time. There will be excessive heat and more-easily felt pulse at the hoof. Eventually there will be deformity of the hoof, with abnormal growth rings and placement of the animal's weight on the heel to minimize pain. Most cases can be avoided by not allowing access to good feed when there is insufficient exercise. Mild cases are difficult to diagnose (slight lameness), but they can recover if feed intake is reduced immediately. Laminitis is a significant cause of lameness in cattle, particularly feedlot cattle and high-producing dairy cows on high-energy, low-roughage diets (51).

Mechanically injurious

Some common plants, although not poisonous, are mechanically injurious and occasionally cause considerable pain to livestock. They have sharp seeds, awns, or spines that may work their way into the tongue, gums, eyes, nose, or skin. This injury may result in sores that cause extreme discomfort or inflammation. Suffering animals go off feed, and develop a generally poor condition. The sores may also allow the entry of pathogenic bacteria into the tissues and circulatory system, causing local or general infections. The fibres of some plants, such as foxtail barley, may form balls that lodge in the stomach or intestines. Microscopic slivers from burdock seeds can become imbedded in an animal's eyelid, scraping the eyeball to cause a serious lesion that may be misidentified as pinkeye. The following plants present a risk of mechanical injury for livestock.

Downy brome	*Bromus tectorum* L.
Needle-and-thread	*Stipa comata* Trin . & Rupr.
Northern porcupine grass	*Stipa spartea* Too. var. *curtiseta* A.S. Hitchc.
Columbia needlegrass	*Stipa columbiana* Macoun
Porcupine grass	*Stipa spartea* Trin.
Red three-awn	*Aristida longiseta* Steud.
Foxtail barley	*Hordeum jubatum* L.
Barley (rough-awned)	*Hordeum vulgare* L.
Rye	*Secale cereale* L
Wild oats	*Avena fatua* L.
Lesser burdock	*Arctium minus* (Hill) Bernh.
Great burdock	*Arctium lappa* L.

Brittle prickly-pear cactus   *Opuntia fragilis* (Nutt.) Haw.
Plains prickly-pear cactus   *Opuntia polyacantha* Haw.

Nitrate poisoning

Several forage crops and weeds can cause poisoning of ruminants through accumulation of large amounts of nitrate. These include **barley, oats, wheat, rye, triticale, millet, canola, corn, sunflower, bromegrass, kochia, pigweed, black nightshade, Canada thistle** and **Russian thistle**. With careful management, these plants can be safely used as feed. In ruminants the nitrate (NO_3) in forage is converted to nitrite (NO_2) and then to ammonia by bacteria in the rumen. Ammonia is absorbed into the bloodstream and utilized or excreted as urea. When forage contains high levels of nitrate, the bacteria continue to convert it to nitrite but may be unable to convert all of it to ammonia. The excess nitrite is absorbed into the bloodstream where it combines with hemoglobin, the oxygen carrier in the blood, to form methemoglobin. Because methemoglobin does not combine with oxygen, the oxygen-carrying capacity of the blood is reduced to the extent that affected animals may die from lack of oxygen. Nitrite also lowers blood pressure so that peripheral circulation becomes impaired. Acute symptoms appear within one half to 4 hours after ingestion of a toxic dose, and animals can die within the first 1 to 8 hours. Typical symptoms in cattle include staggering, drowsiness, labored breathing, excessive salivation, frequent urination, and occasional convulsions. The blood becomes dark chocolate brown as shown by a grey to brown color of the mucous membranes. Finally the animal collapses, rolls on its side, and dies quietly without a struggle. Low concentrations of nitrate can cause chronic toxicity, usually reflected in poor weight gains or milk production, abortions or weak premature calves. When silage is made from high-nitrate forage, toxic nitrogen oxide gases, "silo gas", may be produced (19). These gases are heavier than air so that in barns adjacent to silos they may collect in concentrations sufficient to kill animals.

Nitrate, and sometimes nitrite, can be a problem in pasture, stored feed, and the drinking water. A lab test can determine the nitrate concentration, but it is important to be aware of how a lab reports the level. Feeds with nitrate (NO_3) content of 0.4% on a dry matter basis are considered safe. The acceptable maximum for pregnant cows is 0.33% nitrate. Feeds with 0.5 to 1.0% nitrate are potentially harmful, and should be diluted with feed low in nitrate. Alternatively "nitrate nitrogen" (N) is safe at 0.12%, while "potassium nitrate" (KNO_3) is safe at 0.8%.

Treatment involves intravenous injection of methylene blue, which will regenerate the hemoglobin, plus an adrenaline injection, which will stimulate the blood pressure. However treatment is usually not practical because of the large number of animals affected, and the difficulty in obtaining large amounts of methylene blue. More importantly, the animals are oxygen-starved, so the stress involved in treating them could be harmful. The best approach is to remove the toxic feed immediately, and avoid moving the animals.

Plants take up nitrate from the soil and use it to form amino acids and protein. When stress reduces the rate of protein formation nitrate can accumulate in the tissues. This stress may be frost, drought, heat, hail, herbicide application, plant disease, or even periods of overcast skies. Some contributing factors are a high level of nitrogen in the soil, either from chemical fertilizers or manure, and a crop experiencing rapid growth. Legumes typically do not accumulate nitrate under these conditions. Nitrate intoxication is only a serious problem in ruminants. When a damaged crop is grazed, it is important to allow the rumen microflora adequate time to adapt to the feed; 6 days is optimum and adaptation can quickly be lost if the feed is changed (17). To determine the severity of the toxicity, the crop should be sampled 3-4 days after damage has occurred. The animals should be well fed before first grazing the crop, and taken off it after only one-half to one hour. They should be gradually allowed to graze longer each day; the process will take 5 to 7 days.

When a high nitrate crop is stored as silage, microbes can break down some of the nitrate but as much as 50-80% may be retained. When the crop is stored as hay, all excess nitrate is retained. However, because of cell damage caused by drying it is released more quickly during consumption than if it were grazed. If hay is baled at too high a water content, subsequent heating can increase toxicity 10-fold by microbial conversion of the nitrate to nitrite. Cattle that are thin, suffering from respiratory disease or otherwise in poor health have increased susceptibility to nitrate poisoning. The best way to feed high nitrate forage is to incorporate it in a mixed ration. If this is not possible, increasing the feeding frequency to 3 times per day is an option. It is the amount of nitrate in the rumen at one time that causes the problem. It is important to avoid interruptions in the regime because the time off feed may increase appetite so much that the cattle will consume the feed too quickly when they again have access to it. Healthy cattle will adapt to feeds containing a moderate amount of nitrate, over a period of 6 to 8 days. Feeding grain, a source of readily fermentable carbohydrates, helps by reducing nitrite accumulation.

Polioencephalomalacia

Polioencephalomalacia (PEM), or cerebrocortical necrosis, is a non-infectious brain disorder of ruminants. The term for the lesion is comprised of three Greek words (*polio*: grey, *encephalo*: brain and *malacia*: softening: softening of the grey matter of the brain). The condition is caused by a number of factors in feed and water that are not completely understood. The reader is referred to D.G. Gould (32) for an in-depth review. Clinical signs are a sudden onset of aimless wandering, blindness, head pressing, incoordination, muscle tremors, with severe cases showing a "star gazing" posture, lying on their side, convulsions and death. It is believed that on high-grain diets, rumen conditions can induce resident bacteria to greatly increase production of the thiamine-destroying enzyme thiaminase and thereby induce thiamine deficiency. In calves, administration of thiamine (vitamin B_1) very quickly after onset of the disease can often promote quick recovery. There is potential for PEM in brassica pasture or silage, which may have such high digestible-carbohydrate content that they can mimic high-grain diets. PEM is most often

seen in young animals, particularly when there is insufficient long fibre in the diet. Chewing this long fibre stimulates production of saliva which counteracts acidosis in the rumen.

Another possible cause of PEM is high sulfur content in feed, which is the case with both brassicas and kochia. Recommended maximum sulphur content is 0.4% in feed on a dry matter basis (58). Water is often an overlooked factor in diagnosing this cause of PEM. A feed test may show that sulfur levels are tolerable, but when the sulphate contribution of the water is considered, total sulfur is too high. Similarly, animals with sulphur-induced PEM have been misdiagnosed as having selenium poisoning, because water supplies in high-selenium regions are often high in sulphate (62). With high sulphur diets, PEM symptoms are not strictly due to a thiamine deficiency. Hydrogen sulphide, produced from sulfate by rumen bacteria, apparently plays a direct role in PEM. Sulfide is highly toxic to the nervous system, and it can be absorbed from the rumen or by the lungs after inhalation of belched rumen gases. The fact that thiamine is reported to be effective in treating some cases of sulfur-induced PEM could be due to its therapeutic benefits in brain disease. Clinical signs of PEM can be seen within two weeks, which is the interval required for bacteria to produce sufficient sulphide. In some cases it may be necessary to replace the water source.

PEM-causing forages can usually be fed safely if they are diluted with other appropriate feeds, to reduce total sulfur and increase fibre. On brassica pastures this means limiting intake to less than 75% of the diet, by supplying hay or access to grass pasture. Similarly when feeding brassica silage or hay it is advisable to dilute the toxic feed to about 60% of the diet, or preferably to a level based on a lab test for sulfur in both feed and water. Kochia should be diluted with other forage, to a maximum of 30% of the diet when plants are harvested young, and 40% for later stages of growth.

Pasture bloat

Alfalfa is recognized as one of the most nutritious forages available and is widely used as conserved forage in diets of cattle and sheep. However, the utilization of alfalfa by grazing livestock has been limited due to its potential for causing frothy bloat on pastures. Pasture bloat occurs when the eructation or burping mechanism in cattle or sheep is impaired or inhibited because the rate of gas production in the rumen exceeds the animal's ability to expel the gas. In pasture bloat the eructation mechanism is inhibited by frothy rumen contents. The gas remains trapped in the rumen fluid and the frothy contents inflate the rumen. Death is likely caused by suffocation, when the distended rumen pushes upon the diaphragm and prevents inhalation.

Stage of growth or crop maturity is the most important factor in preventing pasture bloat (79). Bloat potency is highest at the vegetative (or pre-bud) stage, decreasing as the plant matures to full flower. Swathing the alfalfa and allowing it to wilt before consumption is effective in reducing bloat. A minimum of 30% reduction in the dry matter content is necessary, and this took about 48 hr in trials under dry weather conditions at Kamloops,

BC (44). When drying conditions are poor the swath should be laid out so that it is as wide as possible, to hasten wilting. Some water-soluble surfactant products have also been found effective in bloat control (43, 46). A drawback to these products was that correct dosage required preventing cattle from drinking from any water source other than the medicated water supply. Recent studies show that feeding orchardgrass hay before grazing alfalfa can also significantly reduce the incidence of pasture bloat in cattle.

Other forage species can also cause pasture bloat, to a varying degree. Species in the "low-risk" category are generally considered risk-free, but isolated cases of pasture bloat have been observed especially when they are very lush, at early stages of growth. A manual on bloat is available (47). Bloat causing, moderate risk, and low-risk forages used as pastures in Western Canada are listed below:

Bloat causing	Moderate risk	Low-risk
Alfalfa	Berseem clover	Sainfoin
Red clover	Persian clover	Birdsfoot trefoil
White clover	Canola	Cicer milkvetch
Alsike clover	Perennial ryegrass	Fall rye
Kura clover		Most perennial grasses

Photosensitization

If "photodynamic agents" (photosensitizing substances) are present in sufficient concentration in the skin, damage occurs when the skin is exposed to sunlight (69). These agents absorb the light energy that causes them to react with the tissue of unprotected skin and mucous membranes. The photodynamic agent can be of either plant or animal origin, and typically is delivered to the skin by the blood, but only areas exposed to sunlight are damaged. Direct skin contact with an agent, such as the juice (latex) of a "phototoxic" plant, can also cause this problem. Symptoms (which initially can be difficult to distinguish from actual sunburn) are reddening of the skin, followed by itching which may cause the animal to rub the area. Then swelling occurs, and the skin may eventually die and slough off. If the skin contains dark pigments it is usually protected from the syndrome, while adjacent light patches will be affected. Therefore symptoms have been observed to be restricted to only the white patches on a spotted horse. Heavy coats of wool or hair are effective in preventing sensitization on underlying skin. Eyes may become sensitive to light and cornea may become inflamed. Photosensitization is usually divided into three main types:

Primary photosensitization results from the direct ingestion of or skin contact with a photodynamic agent occurring naturally in a phototoxic plant.

Hepatogenous or secondary photosensitization is the result of liver dysfunction, which may be caused by consumption of a toxic plant. Under normal conditions the chlorophyll pigment in plants is broken down into phylloerythrin (a "porphyrin") which is removed

from the blood and normally excreted in the bile (69). When the liver fails, the phylloerythrin levels in the blood increase and the substance is delivered to the skin where it becomes a photodynamic agent. Note that liver damage will not cause photosensitization symptoms if the animal is not in sunlight.

Porphyria and **protoporphyria** are rare diseases involving dysfunction of enzymes involved in hemoglobin (heme) synthesis resulting in the accumulation of photosensitizing porphyrin intermediates (69). Toxic plants are not involved. The diseases are heritable in some lines of livestock, so culling is done in an attempt to eliminate the flaw from breeding stock. It follows that there is value in ensuring that phototoxic plants are not the cause of symptoms before culling an animal.
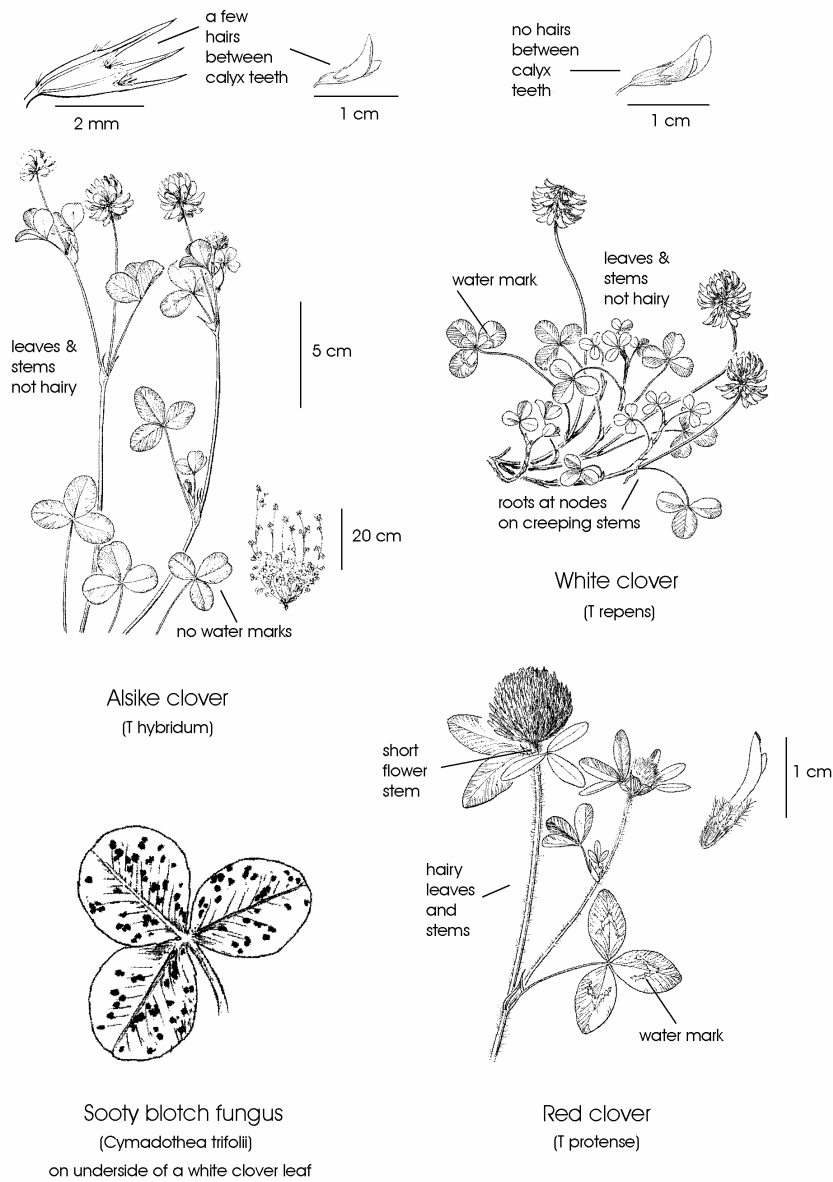
Only primary and hepatogenous photosensitization are caused by toxic plants, but there is sometimes confusion as to correct classification: whether the plant toxin causes liver damage as well as being delivered to the skin as a photodynamic agent. In most cases of primary photosensitization the plant responsible must be eaten in large amounts, and typically is in the lush green stage. It usually takes a few days for symptoms to appear. The liver damage involved in the hepatogenous type is considered a long-lasting consequence, and recovery may not be assured.

If there is no liver damage, recovery is usually rapid after changing the feed and/or keeping animals protected from direct sunlight. This means keeping them in the shade, not absolute darkness, for several days or weeks. They can be housed and allowed to graze at night. Otherwise treatment is mainly to address the symptoms: anti-inflammatory drugs, wound management and fly control. Even dramatically damaged skin will recover, but two or more months may be required for complete recovery. Note that cows treated with corticosteroids to induce calving may develop teat dermatitis which may be misdiagnosed as photosensitivity (69). Local plants that are most commonly implicated in photosensitization in animals are described below.

Alsike clover (*Trifolium hybridum* L.), Pea Family, Fabaceae (Leguminosae)

Alsike clover is an introduced short-lived perennial forage species adapted to moist soils. It has been implicated in photosensitization in horses. However the plant itself is not toxic. The toxic principal is an unidentified mycotoxin, produced by a fungus that is visible on leaves. In Canada the disease is called **"sooty blotch"** or "sooty blotch of clover", but in some locations **"black blotch"** is preferred. Alsike clover is infected with sooty blotch only in some areas and under some specific environmental conditions. Sooty blotch has been recorded on many other legume species and has been reported to also cause photosensitization in cattle and sheep (52). The reader is referred to an extensive

review of the literature by Mercer (51a).

a few hairs between calyx teeth

2 mm

1 cm

no hairs between calyx teeth

1 cm

leaves & stems not hairy

5 cm

20 cm

no water marks

Alsike clover
(T hybridum)

water mark

leaves & stems not hairy

roots at nodes on creeping stems

White clover
(T repens)

short flower stem

1 cm

hairy leaves and stems

water mark

Sooty blotch fungus
(Cymadothea trifolii)
on underside of a white clover leaf

Red clover
(T protense)

Sooty blotch is visible as small dark spots mainly on the lower surface of legume leaves. To the naked eye the colonies of this fungus appear as if one took a fine-tipped felt pen and dotted the leaf with tan to brown to black blotches (M. Murphy pers commun). There may be large numbers of blotches, often overlapping. Leaves are infected in the spring by air-borne spores (13). Very moist conditions are necessary for significant fungus growth and subsequent penetration of the leaves. The first conspicuous leaf symptoms appear in late spring as small raised, mealy olive-green spots on the lower surface. Later, shiny black blotches, up to 2 mm across, appear beside the green spots. Late in the summer a small brownish-yellow spot may appear on the upper surface directly above each blotch. Surrounding leaf tissue usually remains green, and the leaves do not fall off immediately (51a). With a severe attack, under prolonged cool and moist conditions, defoliation can

occur. However, this leaf disease is generally not considered a major economic problem for crop producers (51a). The fungus completes its life cycle on the leaf tissue and over winters on fallen leaves (51a). It is believed to survive on the soil for several years (13).

There were no reports available on scientific feeding trials, where a group of healthy horses is divided, with one group being fed infected legume forage and the other uninfected forage, to demonstrate toxic effects after an extended period of observation. However, from the existing literature, based on other types of observation, there appear to be three main disease conditions:

1. In "**dew poisoning**" only the parts of the horse in physical contact with pasture plants, for instance muzzle and fetlocks, exhibit skin symptoms. This condition can be classed as primary contact photosensitization (17). The water on the foliage is apparently a vector for the toxin and extension agencies in the USA suggest that turning the horses out after the pasture is dry will minimize dew poisoning.
2. "**Trifoliosis**" is a primary- photosensitization skin condition occurring when animals have consumed significant amounts of fungus-infected clover. It is considered to be reversible if animals are removed from the offending pasture or hay (17). Some have classified this as hepatogenous photosensitization, implying that the skin reactions are triggered by liver dysfunction. In the majority of trifoliosis cases studied, liver damage was not found (36), although in some studies liver function tests have indicated problems (52).
3. In "**alsike clover disease**" or "**big liver disease**", there is liver damage. With a high proportion of infected clover in the diet, poisoning can occur within a few weeks, but usually it takes a year or more to develop (17). The symptoms listed for alsike clover disease include jaundice, anorexia, loss of body condition, and neurological disturbances like aimless walking and head-pressing (19). Most cases with these clinical signs result in death and there is no known treatment.

In some Canadian sources (9) a fourth category, "reproductive problems" is given significance equal to that for the skin conditions. Some legumes contain "**phytoestrogens**", compounds which can affect reproduction in livestock, with sheep being more susceptible than cattle (17). The species most commonly associated with this syndrome are subterranean clover (*Trifolium subterraneum* L.) in Australia, white clover (*T. repens* L.) in Scandinavia (17) and red clover (*T. pratense* L.) (69) in eastern Canada. Leaf diseases can increase the natural levels of such compounds (51a). However natural levels of phytoestrogens are typically low in most legumes grown in North America (17) so it is unlikely that sooty blotch is causing reproductive problems in Western Canada.

This syndrome in livestock has a long history. As in "fescue foot" management of the problem has been difficult to clarify because of the fact that a mycotoxin, and not a toxic plant, is responsible. However, this fungus is also a problem for plants, so plant-disease authorities and research findings may be helpful in addressing livestock protection (51a) e.g. recognition of the fungus on forage. Canadian surveys of the geographic distribution of plant diseases have historically included this fungus (6, 12). It has also been the subject of recent laboratory research, not related to reducing its negative effects on crops

or livestock, but because of interest in its vegetative development within the host plant. There are numerous scientific names for this fungus, which goes through a series of dramatic changes in appearance in the course of its life cycle. Names have been coined by different scientists, independently observing this same species at different stages, and some of these have been retained in the literature (13, 51a). Two frequently-used Latin names are (*Cymadothea trifolii* (Pers.ex Fr.) Wolf and *Polythrincium trifolii* Kunze ex Ficinus & Schubert) (13). Confusion with the common name may arise because there is a disease of fruit which is also called "sooty blotch". A frequently-cited reference to an endophytic bacterium *Capnocytophaga* as a possible cause of the photosensitization (19) should be discounted.

There have been efforts to investigate this syndrome in Canada. A survey of horse poisonings attributed to alsike clover was derived from veterinary case records (57) but pasture details were unavailable so it was assumed that alsike clover was being consumed. The only alsike feeding trials in Canada were conducted with horses during 1928-1933. However, these trials should be discounted because they had such serious flaws in experimental design as: no horses with an alsike-free diet for comparison (56). In a preliminary trial in Minnesota (4) sooty blotch fungus was grown on agar in laboratory Petri plates, and then fed to mice for 18 days. Blood samples indicated deterioration of liver function in treated mice in comparison with samples from the control group, but there was no visible difference in liver tissue. In New Zealand in 1968-9 the systemic fungicide benomyl sprayed on infected pastures did not control this fungus (51a). There are reports of differences within legume species in resistance to sooty blotch and of differences between strains of this fungus in their ability to infect different legume species (51a).

This syndrome has occurred with both pasture and hay. Sooty blotch is not a decay fungus, as is the case with sweet clover poisoning, but if the mycotoxin is present in the standing crop, it will be retained in the hay. The blotches are visible in hay although care must be taken to distinguish them from the normal pigment change which occurs with leaf drying (M. Murphy pers commun). Distinguish this organism from another fungus, *Rhizoctonia leguminicola*, which forms more diffuse bronze to black spots or rings, "black patch disease", on the upper side of the leaves of several forage legumes. It produces the mycotoxin slaframine which can cause profuse salivation, or "slobbers" in horses (17).

Sooty blotch has been reported around the world (13). In addition to alsike clover, other host plants reported for this fungus include white clover (*Trifolium repens* L.), red clover (*T. pratense* L.) and alfalfa (*Medicago falcata* L. and *M. sativa* L.). Although birdsfoot trefoil (*Lotus corniculatus* L.) has been implicated in photosensitization in Canada, this legume is not known to be a host of this fungus (L. Couture pers commun). It does not appear to be a widespread problem in our region. As this fungus prefers a moist climate, its distribution is sporadic. Note that the different mixture of forage species chosen by each individual animal in a pasture may further complicate the picture: in a pasture observed to contain infested clover plants, some horses will not exhibit disease symptoms, presumably because they do not eat the infected plants (M. Murphy pers

commun). Articles in the popular press have apparently instilled concern in horse owners in some localities. The long list of possible symptoms attributed to the syndrome has contributed to this problem. For instance, at Chilliwack, BC, a producer making grass-legume hay was observed to rake the already-dry swath repeatedly "to knock off the clover leaves" (A. M. Clegg pers commun). We questioned some forage seed retailers in BC: there was no concern about including alsike clover in a retailer's seed mix for horse pasture in the dry Interior, but a coastal dealer felt pressed to exclude all clover species from such a mix.

It is important to put this disease situation in perspective in Western Canada. Many horses here consume legumes in pasture and hay without adverse effects. The mycotoxin is much less dangerous than other hazards that livestock may encounter, so a short period of consuming infested plants appears unlikely to cause serious problems. Only plants infected with sooty blotch have the potential to cause the photosensitization and liver symptoms. Therefore it should be possible for livestock producers in Western Canada to learn to identify this fungus, inspect their feed supply and determine disease potential.

Grey horsebrush (*Tetradymia canescens* DC. and *T. glabrata* Gray)

This is a low, spreading, pale green to grey native shrub of the aster family, found infrequently on dry grasslands of southern BC. It was a serious problem with sheep in the in the USA, but is currently not an issue here because of its small population, low palatability, and the discontinuation of sheep use of native grasslands. It can be confused, and often grows with, rabbit brush (*Chrysothamnus nauseosus* (Pall) Britt.). It has soft, hairy needle-like leaves (1 cm vs. 2 - 7 cm in rabbit brush), and a small deep yellow tubular flower in clusters, beginning to bloom in June in contrast with July for rabbit brush. Horsebrush causes hepatogenous photosensitization in sheep, with swelling on the head being termed "bighead disease". A small amount of horsebrush tissue, 0.5% of the animal's weight, can cause liver damage sufficient to induce photosensitization. Larger amounts produce more severe liver damage and rapid death without symptoms of photosensitization (36).

Lady's-thumb (*Polygonum persicaria* L.), Polygonum Family, Polygonaceae

This is a Eurasian annual of the knotweed family, common in moist disturbed sites in southwestern BC, but also present on the Prairies. Stems are prostrate to erect, up to 1 m, with oval or lanceolate leaves bearing a purplish black spot, and tiny pale pink flowers in erect spikes. There is a membranous sheath at the junction of leaf and stem. If eaten when green, it can cause photosensitization in animals. **Water smartweed** (*Polygonum amphibium* L.) can also cause photosensitivity. A native, it occurs in sloughs, marshy areas, and shallow water, throughout the region.

Puncturevine (*Tribulus terrestris* L.) Caltrop Family (Zygophyllaceae)
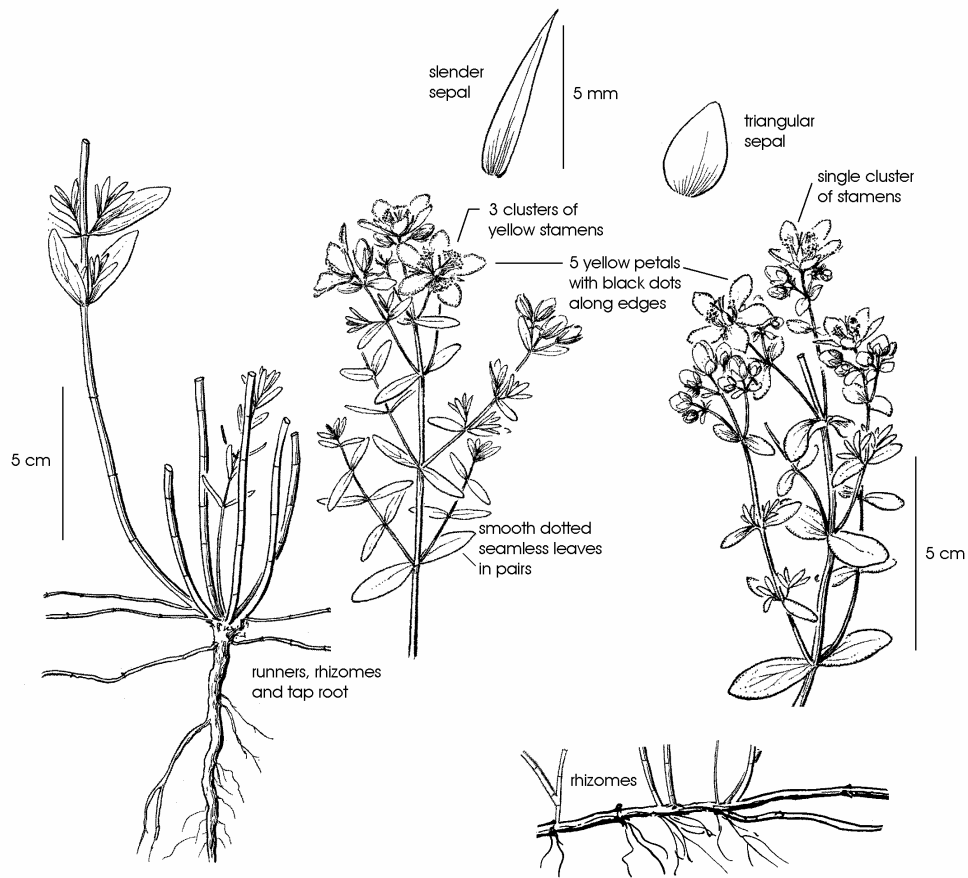
This Mediterranean weed is a very drought-tolerant annual found on roadsides and along railways in southern BC. The large spines of the fruit are notorious for breaking off and puncturing tires. The leaves are pubescent but palatable, particularly to sheep. This plant is considered a secondary or hepatogenous photosensitizer although it is not clear whether mycotoxins or saponins are responsible for causing liver damage. Records of poisoning in our region were not found, and there are only a few cases recorded in the USA, possibly due to the plants having low levels of the specific compounds implicated elsewhere (Australia, South Africa) (17).
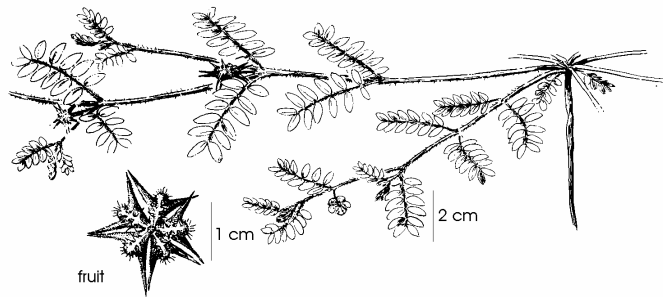
Puncturevine

(Tribulus terrestris)

Common St. John's-wort (*Hypericum perforatum* L.) St. John's-wort or Mangosteen Family, Clusiaceae (Hypericaceae)

This noxious weed was introduced from Europe where it has a long history of medicinal use. It is a perennial, 20-80 cm in height with a tap root, stolons and short rhizomes, which make it an aggressive invader of native pasture. The flowers are yellow and pods are many-seeded brown capsules. The leaves, stems and floral organs have tiny but easily visible glands embedded in their surfaces. If you hold a leaf up to the light, these glands appear as perforations, hence the species name "*perforatum*". These glands contain pigments that include the phototoxin hypericin (black glands) or its chemical predecessor, protohypericin (translucent glands); the greater the number of these glands the higher the toxicity of the plant. Hypericin is transmitted unchanged to the skin where it is activated by sunlight (17). Common St. John's-wort is limited to BC, mainly on southern Vancouver Island and in the southern Interior. In the western USA, where it is a widespread problem, it is often called "Klamath Weed". Toxin concentrations are low in young shoots, then increase as the plant matures; risk is offset by the reduction in palatability of the plant in late summer. This weed can cause skin problems for most species of livestock, although deer and goats are reported to be unaffected (17). It is not usually considered to be a problem for cattle because it is not known to be palatable to them. However, we have observed its use by cattle on forested range, and have received reports of dermatitis and mortality occurring near Kamloops. The owner of some of the

slender
sepal

5 mm

triangular
sepal

single cluster
of stamens

3 clusters of
yellow stamens

5 yellow petals
with black dots
along edges

5 cm

smooth dotted
seamless leaves
in pairs

5 cm

runners, rhizomes
and tap root

rhizomes

Common St. John's - wort

(Hypericum perforatum)

toxic

Western St. John's - wort

(Hypericum formosum)

toxic

affected cattle suggested mowing roadsides before trailing his herd through the St. John's-wort infestation. This may have been the most practical solution in his case, although it seems prudent to prevent the animals from consuming the dry material. Hypericin levels drop when the cut plants dry in the sun, but hay containing this weed has caused skin problems (17). There are several native species of this genus occurring in BC and Alberta. **Western St. John's-wort** (*H. formosum* H. B. K.) is the only one with the black glands on the petals, but its triangular sepals under the petals distinguish it from *H. perforatum*. In addition there are several other species in horticultural use in this region. Common St. John's-wort is somewhat affected by beetles that have been introduced as biological control agents.

Other plant species that have caused photosensitivity include turnip greens, oats, Sudan grass, cicer milk-vetch, and kochia. In the latter it is not clear whether it is a primary photosensitizer (17). Drinking water showing a bloom of blue-green algae (cyanobacteria) can cause hepatogenous photosensitization.

cross section
of pod showing
two grooves

1 cm

1 cm

10 cm
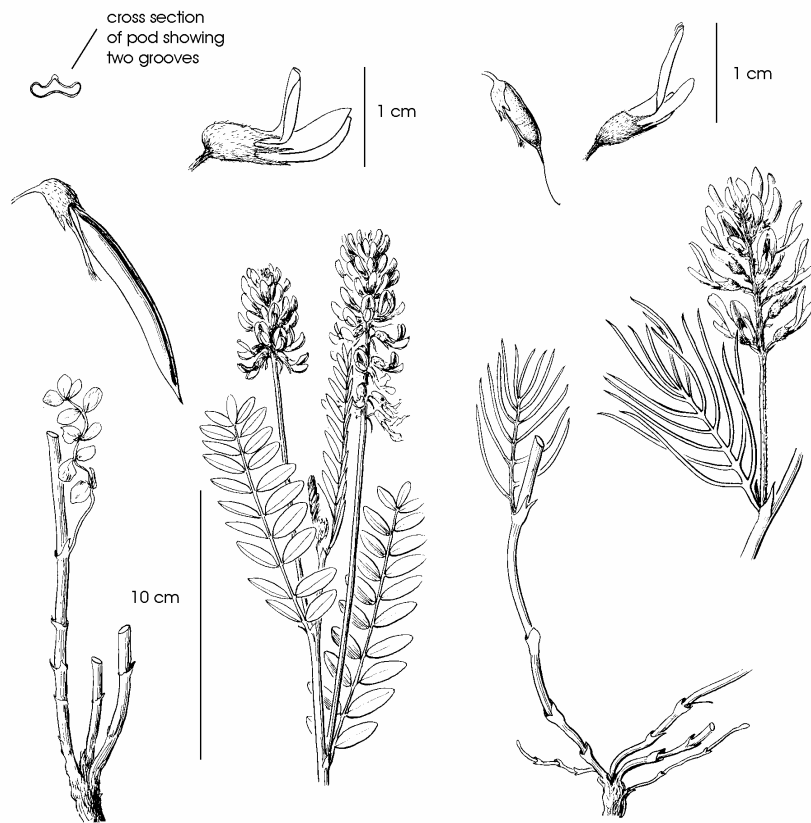
Two-grooved milkvetch

(Astragalus bisulcatus)

may contain nerve toxin and
accumulates selenium

Narrow-leaved milkvetch

(Astragalus pectinatus)

accumulates selenium

Selenium poisoning

Some plants growing on soils containing selenium (Se) accumulate this element in the form of selenoamino acids, such as selenomethionine and selenocystine. Selenium is toxic because in animals, selenium-containing amino acids compete with essential sulfur-containing amino acids with the resultant synthesis of defective proteins. Plants have been divided into two groups according to their ability to accumulate selenium. "Selenium accumulator" plants, which include two species of *Astragalus* in this region, can accumulate levels up to 3,000 ppm Se. Because they prefer selenium-rich soils, they are also called "selenium indicator" plants. They can cause acute selenium poisoning, but this is rare because their offensive odour usually makes them unpalatable, so poisoning occurs primarily on severely overgrazed pastures. Signs of acute Se poisoning may include staggering, abnormal posture, elevated temperature, diarrhea, and laboured

breathing. There is no known treatment, death usually occurs within a few hours and often before a diagnosis can be made.

Non-accumulator plants, which can be grasses, shrubs, and herbaceous plants such as alfalfa, contain very low levels of Se. When their content reaches 5 ppm Se, they may become dangerous if ingested over a prolonged period. In this chronic Se poisoning, or alkali disease, hooves become overgrown, death of primary hair follicles causes a general loss of the long hair (mane and tail of horses, switch of cattle) and reproduction is impaired. To treat affected animals, remove them from the area or provide supplemental feed known to be low in Se.

Historically another chronic condition, blind staggers, was attributed to excess Se, but recent research has questioned this connection (62). The symptoms are loss of muscular coordination, impaired vision with restless movement including bumping into fences. These symptoms are now attributed to polioencephalomalacia or to locoism. The former condition is viewed as the result of excess sulfate, mainly contained in alkaline water, a problem typical in sites with high-selenium soils. Locoism, a neurological disorder, has been attributed to the indolizidine alkaloid swainsonine (see Silky locoweed) rather than to the Se in *Astragalus*. Selenium is also an essential element and deficiencies can cause several problems, including white muscle disease in newborn calves. Two local species of milk-vetch are linked with Se poisoning. **Two-grooved milkvetch** (*Astragalus bisulcatus* (Hook.) A. Gray) is a stout, many-stemmed, erect plant 30-80 cm high. It prefers seleniferous soils and has a distinct, unpleasant odor. It also contains the indolizidine alkaloid swainsonine, which causes locoism. The pinnate leaves have 17-27 elliptic leaflets 10-20 mm long. The flowers are showy, deep purple and about 10 mm long and grow in long, dense racemes at the ends of long stems. The seedpods are 10-15 mm long and have two deep grooves along one side. This plant is very common throughout southern Saskatchewan and Alberta. It is usually found on semi-moist sites. It may be abundant in ditches along new roads with heaviest stands occurring on shallow soils covering shale. **Narrow-leaved milkvetch** (*Astragalus pectinatus* Dougl. (ex Hook.) is another Se accumulator. It is an erect or semi-erect, much-branched, and 30-50 cm tall. The leaves are pinnate with 11-21 very narrow leaflets 12-50 mm long. In June there are 5-20 very noticeable cream-colored flowers 20-25 mm long on each of several racemes. The seedpods are woody, oblong, ellipsoidal, and 10-15 mm long. This plant is found in southwestern Manitoba, southern Saskatchewan, and southern Alberta on open prairies and roadsides, usually on lighter soils. Both species are on the Canadian government's Noxious Weed Seed list, so seed cannot be imported by gardeners.

Water quality

Water quality is an extremely important aspect of livestock management. On native rangelands, and often in cultivated pastures, the sole source is a dugout or pond. When animals drink directly from these sources they may contaminate the water with feces and urine, which contribute both nutrients and pathogens. Animals are generally able to choose clean water. For instance, in an experimental situation cattle were observed to

distinguish clean water from that containing only 0.005% fresh manure by weight (84). However, in another trial they were observed to choose the water that was familiar over that which was somewhat cleaner. When water is unpalatable, animals reduce intake of both water and of forage, resulting in a reduction in gain. Cattle that had reduced their water intake because of low palatability spent more time resting and less time grazing (84). Pumping the water to a trough, and preventing livestock from fouling the water source, can result in significant improvement in animal performance. However, ranchers may be unwilling to completely fence off a pond because of risk of pump failure. Many factors of water quality influence animal performance, and a few may cause morbidity and mortality.

Blue-green algae (*Anabaenaflos-aquae* (Lyngb.) de Breb., *Microcystis aeruginosa* Kutz emend Elenkin) Cyanobacteria Family, Cyanophyceae.

These microscopic organisms of fresh and salt water have recently been judged to be more like bacteria than algae so they are now classified as Cyanobacteria. Some members of this extremely large group are economically important. For instance, the genus *Spirulina* is a blue-green widely used as a health food, and other genera can fix nitrogen in rice fields. The reader is referred to two reviews (18, 23) for detailed coverage of the toxicity of blue-greens.

There are several species native to Western Canada, but two are currently believed to present the greatest risk to livestock. *Microcystis aeruginosa* is a colonial blue-green occurring in unicellular diffuse clusters. *Anabaenaflos-aquae* is a non-branching, filamentous blue-green. Under favourable conditions, the cells multiply sufficiently to discolour the water to a blue-green colour, and occasionally even greenish-brown to red. This heavy concentration of cells is called a "water-bloom". Blue-greens may be confused with non-toxic duckweed (*Lemna* spp. L.), a green plant with a floating 3mm leaf (frond) and tiny root, often found on shallow sloughs. True macro algae that grow in filaments can also be mistaken for micro blue-greens. When macro colonies are handled, they hang together in a stringy mass, while blue-greens fall apart and disperse in the water when handled.

Every slough, pond or dugout, except those with extreme salinity, is a potential site for this problem. Blue-green poisoning has even been observed in large lakes (J. McGillivray pers commun). Low levels of blue-greens are common and problems usually only occur when populations are dense. Very rapid growth of populations can occur when water becomes polluted with fertilizers containing nitrogen and phosphorus. Under pasture conditions, the manure of animals in dugout water can be the source of this pollution, especially when precipitation is low and water levels are well below normal. The presence of blue-greens is evident both as surface water-bloom and as large clumps floating below the surface. They are able to modify their buoyancy, allowing them to move to where light and nutrient levels are best, so they can disappear and later reappear. Wind can concentrate them along a shoreline, and can be another reason for their sudden appearance or disappearance. Both wild and domestic animals will avoid drinking blue-greens, which have strong odours and flavours.

Anabaenaflos-aquae contains a neurotoxic alkaloid, anatoxin-A, which acts very rapidly on the nervous system (within 10 to 30 min) causing muscular tremors, stupor, staggering, collapse, convulsions and death by respiratory failure. The genus *Anabaena* contains another neurotoxin, an organophosphate which has been called anatoxin-a(s); the "s" because a symptom of poisoning is excess, viscous salivation. *Microcystis aeruginosa* contains a polypeptide liver toxin, microcystin-A, which acts less rapidly (within 30 min to 24 h) causing weakness, nausea, vomiting, excessive salivation, bloody faeces, diarrhea, jaundice and anaemia. In survivors, the liver damage may result in skin damage. Clinical signs in cattle include reddening, blistering, swelling and peeling of light-skinned areas of the body such as the nose, around the eyes, and on the udder. All classes of livestock are susceptible to these toxins, and there is a public health risk (21) but there is no known antidote.

When a water source is to be checked for toxic blue-greens, it is important to sample the water-bloom quickly, before it is blown away. Aeration can reduce blue-green growth in stagnant water bodies. The most effective algaecide is copper sulphate (bluestone, $CuSO4.5H_2O$) which is available in a wide range of products. The objective is to deliver 0.25 mg of elemental copper per litre (bluestone contains 25.4% copper, Cu) to the top meter of water, with an approximately 60% lower rate if fish are present (1). Applications are most effective in early summer before large algae populations develop (1).Treatment may have to be repeated, but no more than to 4 times per season (1). The toxins are released as the organisms die and they are stable in water, so the water may be dangerous for at least 14 days after treatment (1). Blue-greens can also be controlled by barley straw (59a), and by deciduous leaf litter, rotting in water. A recommended procedure is to anchor a small square bale of barley straw in the bottom of a dugout in spring before it fills up with water. Compounds released during decomposition should keep the water clear of blue-greens for at least 3 months.

Water intoxication

This is also termed "water on the brain". Cattle forced to use a poor source of water will reduce their intake. Low water quality can usually be attributed to a high natural mineral or sulfate content, or when overcrowding results in fouling with manure from the drinking animals. When such animals are given access to a good source of water, such as a stream, they may over-consume. Drinking too much water can be fatal because the excess fluid exerts pressure on the brain. This can be avoided by allowing only limited access to sources of water for the period immediately after animals are moved from the problem pasture.

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