

PLANT POISONING FOR HORSES

by Domenico Bergero and Cynthia Préfontaine ■ photos by TuttoArabi Archive

The horse is an herbivore that exclusively feeds himself, in nature, with diverse parts of plants, mainly herbs. Therefore, the plant kingdom also has their own survival defense. In fact, many plants contain harmful substances that could advise again their daredevil users qualitatively or/and quantitatively. Then, among herbs available for horses, there are not only essential nutrients, but also non nutritional factors that can clearly be damaging. There are however not that much poisonous plants that can be dangerous for horses. Nevertheless, in those particular cases, it's unavoidable to know them, at least insure their control in the paddocks and in field used to produce hay.

GENERAL APPROACH

Which part of the plants and when?

*First of all, in term of plant toxicity, many questions need reflection: which part of the plant is dangerous and which shape it has, or what is the lethal or toxic dose for horses? It then become of essential concern for every poisonous plant to carefully identify the plant itself, her life cycle and then, its dangerous part. For example, the ivy (*Toxicodendron radicans*) from hederia plant genus, is entirely toxic but above all for their fruits. The tamaro (*Tamus communis*) has an exclusive toxic location situated in the red fruits.*

*Yew (*Taxus baccata*), conifer native to western, central and southern Europe has toxic seed only.*

*Verat (*Veratrum* spp.) and ferns (*Pteridophyta* division) have mainly poisonous rhizoid.*



**SOLANUM
DULCAMARA**



**DATURA
STRAMONIUM**



**ATROPA
BELLADONNA**



**HYOSCYAMUS
NIGER**

The snowdrops (species of *Galanthus* genus), that form white carpets in the landscape at spring time, have toxic bulbs.

The Common Hawthorn bloom (*Crataegus monogyna*) has specific toxic flower that bloom in late spring.

That being so, realizing that your horse shows abnormal symptoms grazing on an infested field during a critic period (plant cycle of life) of the year should take your attention. Be aware of the reactions of your horse and/or manage him in an other way in case of doubts to avoid troubles.

In which condition the poison of the plants could be active?

Another aspect to consider; many essences will get their venomous action when they are fresh and lose their toxicity, mainly because of the heat and the deshydration status, when desiccated. It is the case of the Ranunculaceae family where the plants will normally lose their potential toxicity with a warm desiccation condition and will not be of major concern if the hay is well dried. Therefore, hay containing Monkshood (*Aconitum napellus*) or Hellebores (*Helleborus* genus), part of this family, will be consider save only when dry and mature in term of fermentation also.

The same thing is valuable for the Water Hemlock or Cowbane (*Conium* genus) as it contains a venom that evaporate slowly and absolutely request a complete fermentation process to be consider out of danger, while 2,5-5 kg of fresh plant is fatal for horses.

At which dosage the plants are toxic?

Another important aspect to look upon is the dosage because: "every substances is toxic and no one is perfectly harmful; only the dosage will determine the toxicity", according to Paracelsus. This mean that some venoms can be used, in particular dose, as medication (like the small amount use



TAXUS BACCATA



VERATRUM



PTERIDOPHYTA



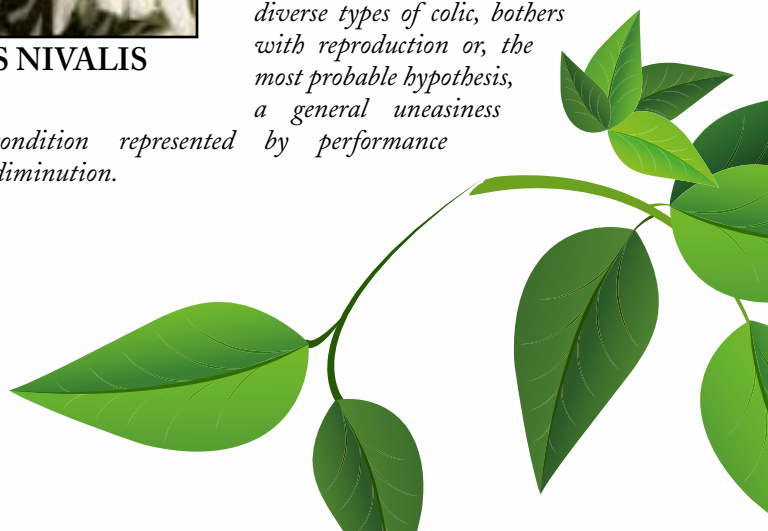
GALANTHUS NIVALIS

of *Digitalis purpurea* extract containing cardiac glycosides for the treatment of some heart diseases), while some other theoricly save can be toxic at high dosage.

It is anyway good to mention that there is a lot of plants toxic at a low dosage. For example, lethal dose of *Digitalis* and *Oleander* (*Nerium oleander*) for horses are about 100 g; 250-500 g for *Hellebores*; 500 g for the *Conium* (*Conium maculatum* and *Cicuta virosa*); 500 g for some hedge trees like the *Common Laburnum* (*Laburnum anagyroides*) and the *Alpine Laburnum* (*Laburnum alpinum*), 1 kg for the *Box* (*Buxus* spp.) and 500g - 5 kg for the *Yew* (*Taxus baccata*).

In general, cases of lethal poisonings are relatively rare and above all, link to toxic plant ingestion by inexperienced and bored horses or by individuals put in a degraded pasture considerably infested by poisonous flora. On the other hand, the chronic poisonings, link with the continuous ingestion of small quantities of toxic plants are harder to recognize because of the symptom subtleties. In this case, evidences will normally be observed through the liver and the kidneys, decreasing the horse general form and performance and increase the probability to bring the animal some other relative problems. Among those damage are included a low resistance to infective agent, chronic enteritis, diverse types of colic, bothers with reproduction or, the most probable hypothesis, a general uneasiness

condition represented by performance diminution.





ACONITUM NAPELLUS



HELLEBORUS
FOETIDUS



HELLOBURUS VIRIDIS



NERIUM OLEANDER



CICUTA VIROSA



LABURNUM ANAGYROIDES



QUERCUS
ROBUR



ERODIUM
CICUTARIUM



SENECIO
JACOBAEA



PTERIDIUM
AQUILINUM

Is the toxic potentiality of a poisonous plant the same for every species of animals?

An other important point to consider is the specie-specificity: not all the poisoning plants the horses are damageable for other species of animals or at least, at the same dosage and vice versa. For example: the digitalis strongly toxic for all the animals, will be of less concern for horses compare to bovines; the oaks (Quercus) leaves and acorns, eaten by deer and peccaries, will lead to foal intoxication if ingested in the pasture, especially after a hot summer where grass is run out.

The symptoms of poisoning, not always easy to identify, will be characterized by a progressive weakening of the animal that can: becomes drowsy in term of behavior and general aspect, has a lower temperature compare to the normal standard, develops mouth ulcers, shows head compression

signs, has red-brown urine are make convulsions.

How to recognized an intoxication?

The time between the toxic plant ingestion and the symptomatic onset is also to take in account. For example, the ingestion of a Conium sp. or Erodium cicutarium horse lethal dose, will gives little hours to the horse life and we will then observe a rapid symptom insurgence.

On the contrary, the senecio (Dendrosenecio kilimanjari) or the brackens (Pteridium aquilinum) can create severe symptoms even one month after continuous ingestion. In the case of the brackens (Pteridium aquilinum) specifically, the symptoms will normally strictly appear after 30 days of hay administration containing this essence or grazing fresh in an infested pasture.

The age of the horse is also a factor to observe in term of



CONIUM MACULATUM



DIGITALIS PURPUREA



PRUNUS LAUROCERASUS



**RHODODENDRON
PONTICUM**



LABURNUM ALPINUM



**ROBINIA
PSEUDOACACIA**



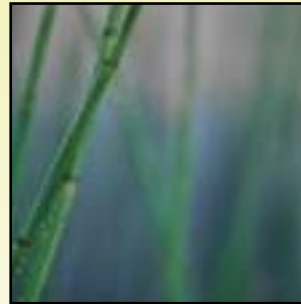
**CYTISUS
LABURNUM**



BUXUS SEMPERVIRENS



**RHODODENDRON
LUTEUM**

**T. HYBRIDUM****T. REPENS****T. INCARNATUM****AETHUSA
CYNAPIUM****OENANTHE
CROCATATA****EQUISETUM SP****SINAPIS
ARVENSIS**

problematic plant intoxication. Young horses got less enzymes to resist the action of toxic plants, obviously beyond weakened and recovering subjects, who ask major attention. In which concrete case should we then consider a plant intoxication? First of all, the horse has a natural born instinct to normally chose in the pasture, the best herbs leaving apart the others. This is moreover true for horses, that since the beginning of their life, had been habituated to stay in wide paddocks. The problem will generally observe with young foals, still inexperienced, or in some "too tight" stallion paddocks. In fact, we need to pay high attention when we face a situation where the paddock dimension is smaller then necessary, overused by the animals and where horses have eaten all the good hay and bored, start to fed with unwanted flora.

Obviously, horses always pent in a box are at "risk subjects" because receiving rarely green diet, they haven't develop enough their selective instinct for herbs and are simply attracted by any fresh herbs when they are suddenly at disposition.

Specific distinctions between plants

A particular distinction in the toxic plant description can be done between toxic trees for horses, offhanded and cultivated herbs. In the first category, beyond Yew (*Taxus*), we find many essences cultivates or used as hedge, that will be ingested only by inexperienced horses often hold in the surrounding of the city, like the Cherry Laurel (*Prunus laurocerasus*), the *Buxus* sp., the Black Locust (*Robinia pseudoacacia*),

the *Rhododendron* sp. (*Rhododendron ponticum* and *Rhododendron luteum*) and *Cytisus laburnum*.

The most important offhand herbaceous toxic plants will be described in the following lines bellow. Concerning intoxication done with cultivated plant we can mention the one done: with clover (*Trifolium hybridum*, *Trifolium repens*, *Trifolium incarnatum*), related to the rising spring and those links with beet consummation that particularly have leaves rich in oxalates.

Among poisoning offhand plants present in Europe, we can find almost all the *Ranunculaceae* family (*Ranunculus acris*, *repens*, *flammula*; *Caltha palustris*) which have diverse toxicity level and appear to be save when included in a well prepared hay. Poisoning symptoms are: general inflammation, skin irritation and narcosis. As mention before, two species of this family are especially dangerous by maintaining their toxicity in the hay which are: *Hellebores* (*Helleborus* genus) and *Monkshead* (*Aconitum napellus*). Specifically for them, the ingestion will lead the horse to: depression, convulsions, paralysis and short term death (some hours for *Aconitum napellus* and some weeks for *Helleborus* genus).

An other plant problematic family for horses in Europe are the *Apiaceae* or *Umbelliferae* referring for example to *Cicuta virosa*, *Conium maculatum*, *Aethusa cynapium* and *Oenanthe crocata*.

Acknowledged to attack the nervous system with their diverse

damaging components, this is of major concern for Arabian horses often involved in dressage. The intoxication level for horse is situated between 0,5 and 2,5 kg of plant depending on the species and will cause narcosis and paralysis.

Some families of toxic plants

Composite family containing among others the *Senecio iacobaea*, known to be toxic fresh or dry due to different active principles. In this case of plant ingestion, we observe a long term and cumulative toxic effects described by a particular degeneration status of the liver and damages on blood vessels. The "target organ" in this situation is the liver, but can also affected the kidneys and lungs. Horses will develop symptoms some weeks and even some months after the ingestion; some of those can suddenly die, while some others will manifested a long term sickness related with hepatic impairment and subsequently show liver and nervous systems problems. Among *Polypodiaceae* family, bracken (*Pteridium aquilinum*) will induce a poisoning status similar to the one

create by the ingestion of *Equisetum* sp., commonly known as horsetails and scouring rushes.

From the *Scrophulariaceae* or the figwort family, the Common Foxglove or Purple Foxglove (*Digitalis purpurea*,) still toxic in the hay at small dosage, will create hallucinations, cardiac rhythm alterations and convulsion.

The most dangerous plant for horse among *Brassicaceae* family, also called *Cruciferae*, is wild mustard or charlock (*Sinapis arvensis*). Native from Europe and well profuse on the territory, the plant contains seeds that induce episodes of acute gastroenteritis and colic even taken in hay.

Finally, in the *Solanaceae* family we have to quote: the Bittersweet (*Solanum dulcamara*), the black nightshade, sunberry, or wonderberry (*Solanum nigrum*), the Jimson Weed (*Datura stramonium*), the deadly nightshade or belladonna (*Atropa belladonna*) and the henbane (*Hyoscyamus niger*) that all have high toxicity even in hay. □

CLINICAL SIGNS & RELATED POISONOUS PLANTS SALIVATION-INDUCING PLANTS

The excess of saliva will be involved by the organism to prevent the swallowing of saliva, liquid and food in case of: mouth injuries (traumatic, chemical or infectious), obstruction to the oesophagus, sharp point on teeth, inappropriate use of bits, vesicular stomatitis (which cause buccal ulcers) and membrane traumatism. The following table 1 contains plants that will often lead to oral lesions resulting in excess of saliva, difficulty of feeding, diminution of feed intake. Sometime, plants with thorns, bristles, stinging hairs or sharp awns may cause skin trauma on all the digestion system membranes. Eye injury are also observed, specifically with the burdock for example.

Table 1 Mechanically injurious plant

Common name	Scientific name
Burdock bristle	<i>Arctium</i> spp.
Oat awns	<i>Avena sativa</i>
Thistles	<i>Cirsium</i> spp.
Barley awns	<i>Hordeum vulgare</i>
Prickly pear cactus	<i>Opuntia (littoralis)</i> spp.
Rye awns	<i>Secale cereale</i>
Bristle grasses, foxtails millet	<i>Setaria (italica)</i> spp
Needle, spear, or porcupine grass	<i>Stipa</i> spp.
Wheat awns	<i>Triticum aestivum</i>
Puncture vine, goat head	<i>Tribulus terrestris</i>
Stinging nettle	<i>Urtica</i> spp.

COLIC AND DIARRHEA-INDUCING PLANTS

Diagnosing plants-induced causes of colic or diarrhea is not that easy for many reasons. First of all, there is normally no apparent lesions in the gastrointestinal tract. Then, post-mortem analysis are not efficient to identify a poison-

ing plant in a proper way when they have been chew and taken by the digestive enzymes. In case of doubts or troubles, pay strong attention to your pasture to identify problematic plants in this case (Table 2).

Table 2 Colic and Diarrhea-Inducing Plants

Common name	Scientific name	Plant toxin	Symptoms
<i>Foxglove</i>	<i>Digitalis purpurea</i>	Cardiac glycosides	Diarrhea, barf, shock, arrhythmia & death in less the 24 hours.
<i>Oleander</i>	<i>Nerium oleander</i>		
<i>Yellow oleander</i>	<i>Thevetia peruviana</i>		
<i>Halogeton</i>	<i>Halogeton glomeratus</i>	Oxalates	Diarrhea, rarely renal disease. Prolonged intake of small quantity leads to calcium deficiency.
<i>Shamrock, soursob, sorrel</i>	<i>Oxalis spp.</i>		
<i>Horse chestnut, buckeye</i>	<i>Aesculus spp.</i>	Aesculin Saponins	Muscle tremors and ataxia.
<i>Corn cockle</i>	<i>Agrostemma githago</i>		
<i>Pokeweed</i>	<i>Phytolacca americana</i>	Saponins & oxalates	Diarrhea
<i>Coffee or senna weed</i>	<i>Cassia spp.</i>	Anthraquinone	
<i>Oak</i>	<i>Quercus spp.</i>	Tannins in leaves, bark or acorns, especially when green	Hard, dark feces; later bloody diarrhea. Anorexia, depression. May have oral ulcers & choke signs. Liver and kidney damage. Plasma calcium increased & phosphor decreased.
<i>Field bindweed or morning glory</i>	<i>Convolvulus arvensis</i>	Tropane alkaloids	Bradycardia & dilated pupils.
<i>Laurel</i>	<i>Kalmia spp. (angustifolia)</i>	Grayanotoxins & arbutin	Salivation, defecation, depression and ataxia.
<i>Azaleas</i>	<i>Rhododendron spp.</i>	Glycosides	Salivation & diarrhea
<i>Mountain pieris</i>	<i>Pieris spp.</i>		
<i>Maleberry</i>	<i>Lyonia spp.</i>		
<i>Privets</i>	<i>Ligustrum vulgare</i>		
<i>Buttercup & anemone</i>	<i>Ranunculus spp.</i>	Protoanemonin	
<i>Hellebore</i>	<i>Helleborus spp.</i>		
<i>Marsh marigold</i>	<i>Caltha palustris</i>		
<i>Clematis, Traveller's Joy, Anemone Clematis</i>	<i>Clematis spp. (C. vitalba, C. Montana)</i>		
<i>Castor beans, wonderboon</i>	<i>Ricinus communis</i>	Lectins	Trembling, ataxia & diarrhea.
<i>Rosary peas</i>	<i>Abrus precatorius</i>		
<i>Black locust</i>	<i>Robinia pseudoacacia</i>		
<i>Nightshade & potato, Jimson weed (thorn apple)</i>	<i>Solanum spp.</i>	Hyoscyamine, solamine & hyocine with atropine . effects	Excitement the depression. Diarrhea & weakness.
<i>Tomato</i>	<i>Lycopersicon spp.</i>		
<i>Avocado (Guatemalan, not Mexican smooth-skin fruit variety)</i>	<i>Persea Americana</i>	Unknown toxin. Flesh of ripe fruit not toxic.	Diarrhea, congestive heart failure, oedema of abdomen, head & lung. Death in less then 2 days.
<i>Persimmon</i>	<i>Diospyros virginiana</i>	Not toxic but may cause impaction.	Impaction colic.
<i>Mesquite</i>	<i>Prosopis glandulosa</i>		

PRIMARY PHOTODERMATITIS-INDUCING PLANTS

The ingestion of plants shown in Table 3 will involve photosensitization dermatitis resulting of photodynamic compounds accumulation in the skin. When the skin is then exposed to the sun, those compounds release a radiant energy which causes cellular necrosis that will call dermatitis. Arabian horses that are often incompletely pigmented are more subject and less protected to this kind of troubles. There is two types of photodermatitis depending the on the way the toxin will act in system. In the primary photodermatitis case, the toxins (photosensitive pigments) are absorbed and

accumulate in the skin and will then be photo reactive. The secondary or hepatogenous photodermatitis, more common then the other, will not be located in the skin, but in the liver where they will cause damages. The diagnosis of this second one is often done to late as the photoreaction in this case is due to the accumulation of the phylloerythrin (chlorophyll by-product) in the blood. The phylloerythrin is a product that the liver is not able to eliminate; when it appear in the blood in concentration high enough to create photoreaction and skin necrosis, the liver disease is often irreversible.

Table 3 Primary Photodermatitis-Inducing Plants

Common name	Scientific name	Toxin	Symptoms
<i>St. John's wort</i>	<i>Hypericum perforatum</i>	Hypericin	Primary photodermatitis
<i>Buckwheat</i>	<i>Fagopyrum esculentum</i>	Fagopyrin	Primary photodermatitis
<i>Spring parsley</i>	<i>Cymopterus watsonii</i>	Furocoumarins	Primary photodermatitis
<i>Bishop's weed</i>	<i>Ammi majus</i>		
See Table 4	See Table 4	Hepatotoxins	Secondary or hepatogenous photodermatitis. Liver disease.
Hepatotoxic plants	Hepatotoxic plants		

Table 4 Hepatotoxic plants (Liver disease-Inducing Plants)

Common name	Scientific name	Toxin
<i>Ragwort, stinking willie, tansy ragwort</i>	<i>Senecio spp. (ex. jacobaea)</i>	Pyrrolizidine alkaloids
<i>Fiddleneck, tarweed</i>	<i>Amsinckia spp.</i>	Pyrrolizidine alkaloids
<i>Rattlepod, rattlebox</i>	<i>Crotalaria spp.</i>	Pyrrolizidine alkaloids
<i>Hound's tongue</i>	<i>Cynoglossum officinale</i>	Pyrrolizidine alkaloids
<i>Salvation Jane</i>	<i>Echium lycopsis</i>	Pyrrolizidine alkaloids
<i>Heliotrope, stickseed</i>	<i>Heliotropium spp.</i>	Pyrrolizidine alkaloids
<i>Creeping indigo</i>	<i>Indigofera spicata</i>	Indospicine
<i>Birdsville indigo</i>	<i>Indigofera dominii</i>	Indospicine
<i>Alsike clover pasture</i>	<i>Trifolium hybridum</i>	Probably a micotoxin
<i>Kleingrass pasture</i>	<i>Panicum coloratum</i>	Probably a micotoxin

NEUROLOGICAL DISEASE-INDUCING PLANTS

Dressage competition, where are really often Arabian horses, is an activity that is strongly dependent on the nervous system. It is then of major concern to consider in table 5 that will give you an idea of the neurological disease-in-

ducing plants. Those diseases are normally characterized by behavioural alterations, inability to take and chew food, ataxia, depression, convulsions and other physical abnormalities.

Table 5 Neurological disease-Inducing Plants

Common name	Scientific name	Plant Neuro-toxin	Food intake (FI) Salivation (S) Muscle tremor (MT)	Gait, Abnormal behaviour (Ab), Depression or weakness (D), Excitation (E)	Notes and Recovery (R)
Sagebrush	<i>Artemisia spp.</i>	Monoterpenoids	Normal FI, no S, no MT	Forelimb ataxia & falling, small Ab, no D, no E	Sage smell on breath & feces, R: 1-2 weeks
Locoweed	<i>Oxytropis and Astragalus spp.</i>	Indolizidine alkaloids (IA)	Decreased FI, no S, no MT	Ataxia, falling, high steps, head bobbing, high Ab, moderate D, high E	Lymphocyte vacuoles, R: partial only
Milkvetch	<i>Astragalus spp.</i>	Nitroglycosides & IA	Can't eat, lot of S, No MT	Ataxia, posterior weakness, no Ab, low D, no E	Dyspnea, R: partial
Yellow star thistle & Russian Knapweed	<i>Centaurea solstitialis</i> C. or <i>Acroptilon repens</i>	Sesquiterpene lactone?	Decreased FI, no S, no MT	Possilbe circling & head tossing, small Ab, no D, no E	Abrupt onset of open mouth, tongue out, inability to prehend or chew feed, R: no
Horsetail, maretail, horsebrush, or snake grass	<i>Equisetum spp.</i>	Thiaminase	Normal FI, no S, $\pm$ MT	Posterior ataxia, reluctance to move, no Ab, low D, no E	Possible blindness, diarrhea, constipation
Bracken fern	<i>Pteridium aquilinum</i>	Thiaminase	Decreased FI, no S, no MT	Posterior ataxia, no Ab, moderate D, no E	Serum thiamin low & pyruvate high. R: yes with vitamin B1 injections
Sensitive fern	<i>Onoclea sensibilis</i>				
White snakeroot	<i>Eupatorium rugosae</i>	Tremetol	Decreased FI; difficulty swallowing & choking appearance, lot of S, high MT	Ataxia, no Ab, low D, no E	Patchy sweating, myocardial degeneration. R: recovery or death within a few days.
Crofton weed, Jimmyweed or rayless goldenrod	<i>Eupatorium adenophorum</i>				
Burrow weed	<i>Haplopappus spp.</i>				
	<i>Haplopappus tenuisectus</i>				
Johnson grass	<i>Sorghum halepense</i>	Cyanogenic glycosides	Normal FI, no S, no MT	Posterior ataxia; and sitting or falling when backed, no Ab, low D, no E.	Cystitis & dribble urine; bladder & possibly vulva, rectum & tail paralysis, rarely sudden death. R: yes early; later partial.
Sudan grass	<i>Sorghum sudanense</i>				

LAMENESS AND MUSCLE WEAKNESS-INDUCING PLANTS

If lameness or muscle faintness are establish to be the major clinical signs, plants from Table 6 should be strongly considered.

Table 6 Lameness and muscle Weakness-Inducing Plants

Common name	Scientific name	Toxin	Predominant Clinical Effects
Black walnut	Juglans nigra	In shaving & sawdust.	Laminitis, leg edema, colic, anorexia, depression and sometime dysnea.
Hoary alyssum	Berteroa incana	Unknown	Limb edema, fever, laminitis
Coffee weed or coffee sema	Cassia occidentalis	High in seeds	Ataxia and sudden death
Day-blooming jessamine	Cestrum diurnum	Vitamin D-like	Chronic weight loss, generalized stiffness to important lameness and recumbency; hypercalcemia and calcinosis
Golden oat grass	Trisetum flavescens	Vitamin D-like	
	Solanum malacoxylon		
Milkvetches	Astragalus (24 spp.)	Selenium accumulator plants	Mane & tail hair brake off, stiff & tender gait, hoof rings & cracks, sometimes emaciation, anemia & cirrhosis.
Golden weeds	Haplopappus spp.		
Woody asters	Xylorrhiza glabriuscula		
Prince's plume	Stanleya pinnata		
Many cultivated fields, alfalfa amd grasses grown on high selenium soils			
Five hooked bassia	Bassia hyssopifolia	Oxalate-induced Calcium deficiency	Shifting leg lameness, bone tenderness & possible emaciation, loose teeth, respiratory noise & “big head”.
Halogeton	Halogeton glomeratus		
Greasewood	Sarcobatus vermiculatus		
Shamrock, soursob, sorrel	Oxalis spp.		
Red-rooted pigweed	Amaranthus spp.		
Purslane	Portulaca oleraceae		
Russian thistle, tumbleweed	Salsola spp.		
Sorrel, dock	Rumex spp.		
Rhubarb	Rheum rhaponticum		
Sugar beet	Beta Vulgaris		
Lambsquarter	Chenopodium spp.		
Bristle, foxtail grass	Setaria spp.		
Panic grasses	Panicum spp.		
Paspalum, Argentine & Dallis grasses	Paspalum spp.		
	Sporobolus spp.		
Buffel grass	Cenchrus ciliaris		
Signal grass, para grass	Brachiaria spp.		
Pangola grass	Digitaria recumbens		
Napier, mission grass	Pennisetum spp.		
Seteria grass	Setaria sphacelata		
Foxtail millet	Setaria italica		

ANEMIA-INDUCING PLANTS

Two different kinds of anemia can occur in horses eating plants written in Table 10 which are the ones caused by red cells haemolysis or the one by haemorrhaging. The ingestion of onions (*Alliums* spp.), red maple (*Acer rubrum*) leaves or phenothiazine toxicosis will be particularly associated

with the anemia due to haemolysis and will be accompanied by haemoglobinuria and icterus. On the other hand, haemorrhaging anemia may occur with if the horse eat spoiled or moldy sweet clover hay.

Table 10 Anemia-Inducing Plants

Common name	Scientific name	Toxin	Major Effects
Garlic or Onions, wild & domestic	<i>Allium</i> spp.	<i>N</i> -propyl disulfide in plants & bulbs.	There is a slow development of onion effects; smell of onion in breath of either onion or red maple; hematocrit 10–15%; hemoglobinuria, icterus, Heinz bodies, weakness; increased heart and respiratory rates.
Red maple	<i>Acer rubrum</i>	In bark & dry or wilted, but not green leaves.	Rapid development of red maple effects, increase AST, SDH & bilirubin.
Sweet clover (if moldy)	<i>Melilotus</i> spp.	Dicoumarol anticoagulant in moldy hay.	Haematomas; normal appetite, temperature & until terminal, pulse and respiratory rate; haemorrhaging; increased prothrombin & partial thromboplastin times.

Table 11 Teratogenic Plants

Known teratogenic plants for horses

Common name	Scientific name
Milkvetch, locoweed European or spotted	<i>Astragalus</i> spp.
Hemlock	<i>Conium maculatum</i>
Lupine	<i>Lupinus</i> spp.
Wild tree tobacco	<i>Nicotiana glauca</i>
Tobacco	<i>Nicotiana tabacum</i>
Hellebore	<i>Veratrum eschscholtzii</i>
Sudan grass	<i>Sorghum Sudanese</i>

Suspected teratogenic plants for horses

Common name	Scientific name
Akee	<i>Blighia sapida</i>
Autumn crocus	<i>Colchicum autumnale</i>
Cycad fern	<i>Cycadaceae</i> spp.
Jimson weed	<i>Datura stramonium</i>
Creeping indigo	<i>Indigofera spicata</i>
Wild pea	<i>Lathyrus</i> spp.
Mimosa	<i>Leucaena leucocephala</i>
Locoweed	<i>Oxytropis</i> spp.
Poppies	<i>Papaveraceae</i>
Wild black cherry	<i>Prunus serotina</i>
Groudsel	<i>Senecio</i> spp.
Periwinkle	<i>Vinca rosea</i>

TERATOGENIC PLANTS

We call teratogenic plants that will involve problems in the physical development of a foetus. The teratogenic damage effect have been observed to be stronger it happens in first trimester pregnancy period. Chemical substances contains in those plants will easily cross the placenta and lead to foetal resorption, abortion, stillbirth and deformations. Table 11 will give you the basic teratogenic official plants for horses; while many other plants have been suspected like Sudan grass (*Sorghum Sudanese*) hay and *Sorghum* hybrids.



Atropa belladonna



Hyoscyamus niger

SUDDEN DEATH-INDUCING PLANTS

Recognizing the first clinical signs and rapidly and then associated them with a poisoning plant founded in our area is the only small way to get out of it. The only chance we really get in this kind of situation is related with the fact that a horse will eat this kind of plant only in some specific cases. For example, if the horse have been posted in an

overgrazed pasture with no other reminding food, it could be relatively easy to identify to poisoning plant. Some other reasons mentioned before are still valuable here to describe the situation, but the "take home message" is **ACT QUICKLY** in case of doubts and be aware of your pasture management and components

Table 12 Sudden Death-Inducing Plants

Common name	Scientific name	Toxin	Major Clinical Effects
Serviceberry or Saskatoon berry	<i>Amelanchier alnifolia</i>	Cyanogenic glycosides in all the plant and especially elevated during growth & seed periods.	Bright red poisonous blood, dark red to cyanotic membranes, fast and difficult respiration, buccal frothing and large pupils. Tremors of muscles, ataxia, convulsions and sudden mortality in the minutes following the ingestion. Positive cyanide test on stomach, liver and/or muscle.
Wild blue flax	<i>Linum spp.</i>		
Chokecherry	<i>Prunus virginiana</i>		
Elderberry	<i>Sambucus spp.</i>		
Johnson grass	<i>Sorghum halepense</i>		
Sudan grass, or broom or kafir corn	<i>Sorghum sudanense</i>		
Milkweed	See Table 13	Green plants contains highest concentration of cardiac glycosides, but dry leaves more palatable & are toxic.	Colic, diarrhea sometime with blood, chewing, dyspnea, cardiac arrhythmias & shock Tetany. Mortality within 24 hours after ingestion of small amount of plant.
Foxglove	<i>Digitalis purpurea</i>		
Oleander	<i>Nerium oleander</i>		
Yellow oleander	<i>Thevetia peruviana</i>		
Be-still or lucky nut tree	<i>T. thevetioides</i>		
Lily of the valley	<i>Convallaria majalis</i>		
Dogbane or Indian hemp	<i>Apocynum cannabinum</i>	Diterpenoid alkaloids elevated in green leaves and flowers. after ingesting the plant.	Excitable, stiff, base-wide stance, can't stand and could have colic. Death suddenly occurs in some hours
Larkspur	<i>Delphinium spp.</i>		
Monkshood	<i>Aconitum spp.</i>	Piperidine alkaloids high in high leaves and stems before fruits.	Salivation, colic, tremors, ataxia, dyspnea, cyanosis, coma and death after 2-3 hours of eating little quantity of plant.
Poison, European, or spotted hemlock	<i>Conium maculatum</i>		
Water hemlock	<i>Cicuta spp.</i>	Cicutoxin alkaloid in the entire plant, particularly in root.	Salivation, chewing, teeth grinding, large pupils, tremors, violent convulsions, respiratory paralysis & death following few hours of ingesting 0,2 kg or 1 root.
Yew	<i>Taxus spp.</i>	Taxine alkaloid in almost all the plant.	Stress, dyspnea, ataxia, diarrhea, bradycardia, convulsions & rapid mortality after eating some 0,5 kg of plant.
Death camas	<i>Zigadenus spp.</i>	Zigacine & zigadenine in all the plant, particularly the onion like bulbs.	Salivation, colic, weakness, ataxia & death following several days of ingesting 3,6- 4,6 kg of plant.
Avocado (Guatemalan and not Mexican smooth-skin fruit variety)	<i>Persea americana</i>	Unknown, but not in ripe fruit flesh.	Diarrhea, colic & congestive heart failure inducing oedema of abdomen, neck, head & lungs causing dyspnea & death in less then 2 days after the ingestion.

Table 13 Common Toxic Milkweeds

Common name	Scientific Name	Toxicity (quantity of green plant as a percent of animal's body weight that is lethal)
<i>Labriform milkweed</i>	<i>Asclepias labrifornis</i>	0.05
<i>Western whorled milkweed</i>	<i>Asclepias subverticillata</i>	0.2
<i>Easter whorled milkweed</i>	<i>Asclepias verticillata</i>	0.2
<i>Woolypod milkweed</i>	<i>Asclepias eriocarpa</i>	0.25
<i>Milkweed</i>	<i>Asclepias asperula</i>	1-2
<i>Plains or dwarf milkweed</i>	<i>Asclepias pumila</i>	1-2
<i>Swamp milkweed</i>	<i>Asclepias incarnata</i>	1-2
<i>Mexican whorled milkweed</i>	<i>Asclepias mexicana</i>	2
<i>Showy milkweed</i>	<i>Asclepias speciosa</i>	2-5
<i>Broad-leaf milkweed</i>	<i>Asclepias latifolia</i>	1
<i>Narrow-leafed milkweed</i>	<i>Asclepias stenophylla</i>	?
<i>Butterfly weed</i>	<i>Asclepias tuberosa</i>	?
<i>Milkweed</i>	<i>Asclepias hirtella</i>	?
<i>Antelope horn</i>	<i>Asclepias viridis</i>	?

The toxic plants described should be considered and managed as all the other field infested non-toxic plants; a good, rigorous and regular control, by an agronomist or a biologist, accompanied by some good works on the pasture from regular work to, in the worst case, a total renewing of the field, can limit the development of unwanted flora.

If it's true that a potential intoxication depends on the ingested dose, it is then non logical to let a horse grass on a pasture infested by poisonous plant potentially toxics. It is particularly true if you don't know with precision the quantity of leaves, seeds and fruits that can cause problems. The major danger that we have to think about is often not related to a sharp and strong poisoning, but the chronic one which is much more difficult to identify for a veterinarian in a first

clinical examination.

At the end, there is not such to need to ring the emergency bell if the pasture is sporadically infested by *Ranunculus* sp. for example, which is really common in Europe. Of course, if beyond this one, there is enough hay of good quality, to permit the horse to avoid them and choose the best forage available; thing that he will anyway do by instinct in this condition. On the contrary, if the field mainly contains *Ranunculus* sp., the risk to get bad effects with consequences then grow up with the possibility for your horse to ingest a "sufficient" dose to reach this point. To get in touch with botanist, agronomist or biologist and use botanic manual to identify the principal poisonous species could help to manage your own situation. □

*Taxus baccata**Veratrum**Pteridophyta**Senecio
jacobata**Pteridium
aquilinum**Solanum
dulcamara**Datura
stramonium**Galanthus nivalis**Crataegus monogyna*