THE TOXICITY OF PLANTS IN EQUINES:

A Modern Three-Point Approach to Disseminating Information

A Thesis

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ACCEPTANCE OF FINAL THESIS

This non-thesis project of Genyce F. Hanson, submitted for the degree of Master's of Science with a major in Rangeland Ecology and Management and titled "The Toxicity of Plants in Equines: A Modern Three-Point Approach to Disseminating Information," has been reviewed in final form. As indicated by the signatures and dates given below, it has now gained full and final approval.

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ABSTRACT

The problem of plant toxicity in equines is difficult and complex. When a plant toxin is suspected as the cause of equine illness or unusual behavior, it is imperative that there be a concise and complete information source that can be accessed immediately. This project's objective was to provide that accessible source of information by utilizing several dozen journal articles, books, and online resources. A literature review was written that summarizes and categorizes the available research on equines and toxic plants. A manuscript for publication was also written on the specific problem of toxic weeds and equines, a subject about which little is known. A database of toxic plants in the United States was compiled from many sources and formatted for ease of use. It contains 338 plant names (268 plants without pseudonyms) and supplies the scientific names, symptoms, organ or system affected, and plant phenotype. A website was created to permit internet users to quickly access a condensed body of information in a familiar format. The website includes the database, shortened versions of the literature review and the manuscript, and links to other resources available. Its purpose is to supply well-organized, integrated information not available elsewhere for concerned horse owners and others involved with equines.

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No road to a graduate degree is an easy or short one, and mine has been no exception. In fact, I would venture to say that it has been a good bit longer, rockier, and dustier than many. The redeeming part of the trip is the satisfaction in having traveled that road to the end.

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An Introduction to the Problem of Plant Toxicity in Equines

The issue of livestock poisoning by toxic plants is more than a nuisance - it creates tremendous difficulties for livestock and land managers. Toxic plants have a considerable economic impact on livestock production. The research on toxic plants has primarily targeted economically significant livestock species including cattle, sheep, and goats. This research has largely overlooked one group of grazing animals - the equines.

The interactions between equines (including horses, ponies, miniature horses, mules, and donkeys) and toxic plants are not well defined, though some information is available on the numbers of equines poisoned annually by toxic plants. One of the reasons that little is known about the magnitude of toxic events in equines is that these occurrences are largely unreported by veterinarians or horse owners. No comprehensive figures are available on the actual numbers of equines poisoned by plants annually.

The extent of the problem of plant poisoning in equines has not yet been adequately documented, but clearly it is a problem with significant impacts and disastrous and expensive outcomes. Impacts and costs not generally accounted for include loss of the horse, decreased use, subsequent replacement costs, carcass disposal costs, veterinary care (including long-term medications and increased number of examinations), and special diets and supplements that may be required for the life of the animal. There are a number of factors specific to

equines and the relationship between equines and toxic plants that warrant further examination.

Because horses are monogastric, they often are more sensitive to toxic plants than are other livestock, and are less able to digest and tolerate many toxins. The digestive system of equines differs from other large livestock such as cattle, sheep, and goats because equids are monogastric rather than ruminants. The particular grazing habits of equines may influence severity and frequency of toxic events. Horses are highly efficient grazers with strong incisors, capable of eating forages down to the soil surface, often including root material (Freeman et al. 2003).

A number of toxic plants are highly palatable, appearing at times when other forage is limited, and are therefore attractive to horses. Equines are notorious for searching for new, green forage in the spring, which may result in the consumption of toxic plants that emerge early in the growing season (Bailey 1916, Pfister et al. 2001, James et al. 2005). After the desirable young forage is depleted, mature grasses and other less palatable forage, including poisonous plants, may become more acceptable and more likely to be eaten (Putnam et al. 1991). Horses may consume normal amounts of forage of poor nutritional quality, including toxic plants, to avoid hunger. In addition, selective spot grazing, particularly common for equines, creates spaces for the establishment of less desirable species, including toxic plants (Freeman and Redfearn 2006).

There is often great difficulty in recognizing or diagnosing plant poisoning. Symptoms resulting from the ingestion of toxic plants occur with varying speeds and levels of intensity. The speed with which many toxic reactions occur in equines makes it problematic for both owner and veterinarian. There are physical, behavioral, and physiological differences among equines that impact the individual susceptibility to poisoning (Bailey 1916). Other factors that may affect a horse's individual ability to handle toxins are age, body condition, and existing or prior health issues. There are additional factors that may influence the occurrence and/or severity of toxic events.

There is no single set of characteristics that applies to all poisonous plants, including appearance, phenology, preferred soils, method of propagation, or growth type; nor do toxic plants possess common traits such as color, taste, or odor. For instance, physically similar plants or plants that thrive in similar areas may be very dissimilar in toxicity. There is no particular compound or single factor that creates a poisonous plant, though many do contain similar toxic chemical compounds.

Upon review of the published literature on equines and toxic plants, the impression remains that all weeds are toxic or dangerous. However, this is not necessarily the case. Weeds are no more toxic than native species, ornamentals, or garden plants. The word 'weed' is often used interchangeably with the term 'problem plant', thus a plant may be called a weed even if it is neither invasive nor noxious.

The emphasis on weeds as the only dangerous plants to equines may result in a significant potential risk: a disregard for the presence of other seemingly harmless but highly toxic plants. For instance, some ornamental or garden plants that appear innocuous can be critically toxic to equines. Each group of plants has its toxic members. For example, most native plant communities contain a number of toxic plants. There are also several common pasture grasses that can be toxic to equines, though for the most part, grasses are generally the safest forage for equines. If horse owners work only toward eradicating pastures of 'weeds' while ignoring other toxic plants, they may find that the plants they disregarded as benign are the very ones that should have been cause for concern.

There are a number of factors that can reduce the occurrence of plant poisonings in equines. The most common approach among range and livestock managers to reduce losses from plant toxicity is to change the plant community, change the grazing animal, or change grazing management strategies. Understanding grazing behavior of equines and knowledge of good forage management are the keys to providing healthy horse pastures and ranges with adequate forage.

Forage of decreased or minimal nutritional quality very frequently results in some type of problem in the resident livestock, whether the outcome is lowered physical condition and body weight, susceptibility to toxins in plants, or other health or behavioral issues. Low quality forage significantly affects body

condition, which can impact the toxicity of ingested plants on a grazing animal. Lowered nutritional states of animals may result in an increase of toxic effects and decreased rates of detoxification. If allowed to select a varied diet, animals have a lower chance of experiencing a toxic event, because eating a variety of plants spreads the toxins they contain over several detoxification systems, and because of interactions among allelochemicals that reduces toxicity.

A prime management goal for horse owners should be to maintain a balance between toxic plant presence and over-abundance. All toxic plant populations, including those of toxic weeds, need to be monitored and then treated if their numbers reach a point that may endanger equine nutrition or health. A growing contemporary challenge to management of toxic and invasive plants on rangeland and pastureland is the phenomenal increase in ex-urban development creating small parcels, many unskilled land owner/managers, and fragmented landscapes. A large number of these smaller acreages are in use by new landowners, and have been converted to horse pastures.

Many of these new landowners have little experience living outside the city, and therefore little to no experience with weed toxicity. Even the seasoned horse owner, while familiar with the plants that have been around for decades, must deal with many newly introduced species that may create new problems and risks for horse health and production. The problem of plant toxicity in equines is difficult and complex. When a plant toxin is suspected as the cause of

equine illness or unusual behavior, it is imperative that there be a concise and complete information source that can be accessed immediately.

There are compelling reasons for using the Web as an outreach tool to disseminate important information. According to the Stanford Institute for the Quantitative Study of Society (SIQSS), the most widespread use of the internet today is as an information search utility (SIQSS 2008). In a recent SIQSS research study, virtually all internet users interviewed responded that they engaged in information gathering activities. The study found that information gathering was the primary or most frequent activity of 77% of users. The study concluded that the Internet today is "a giant public library." Statistics published by the website Internet World Stats: Usage and Population Statistics show that 71.4% of the U.S population, or more than 215 million people, regularly use the internet. Internet usage in the U.S. has grown by 126% in the last eight years.

Unfortunately, many publications and websites result in significant misinformation reaching horse owners. One fairly new book quoted in some websites claims to be the "only complete guide available on plants that poison horses", but claims that horses possess gall bladders and lists only a little over 100 potentially toxic plants. Many websites and web articles are not written by rangeland managers or animal scientists, resulting in a significant amount of misinformation and false 'facts'. A quick Web search revealed that seven out of the first ten websites visited had misleading, inaccurate, or incomplete

information. These come from a variety of sources, even traditional extension sources including universities and governmental agencies.

One obstacle to the gathering of accurate and available information is that much of it is not in a format with which anyone but a scientist is comfortable. Another drawback to having such a diverse body of knowledge is that it may take someone hours or days of searching to locate the facts they need.

The primary goal of this thesis was to locate as much information as possible and to create a compilation of those resources to facilitate the use of them for a broad audience. To reach the largest audience possible, this thesis was created in three parts: a literature review of toxic plants and equines, a manuscript on toxic weeds and equines for a professional journal, and a website for horse owners and professionals. The website offers an easily used and condensed yet complete version of research-based information on how toxic plants affect equines. In this way, several audiences will be reached including researchers, extension personnel, veterinarians, land managers, and horse owners and horse professionals.

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CHAPTER 2

THE TOXICITY OF PLANTS IN EQUINES: A INTEGRATED LOOK AT A COMPLEX PROBLEM

The Magnitude of the Problem of Equine Poisoning by Plants

The issue of livestock poisoning by toxic plants is more than an annoyance; it creates tremendous difficulties for livestock and land managers. Toxic plants have considerable economic impacts on livestock production. Research on toxic plants has primarily targeted economically significant livestock species including cattle, sheep, and goats. This research has largely overlooked one group of grazing animals - the equines.

The interactions between equines (a group that includes horses, ponies, miniature horses, mules, and donkeys) and toxic plants are not well defined, though some information is available on the numbers of equines poisoned annually by toxic plants. One of the reasons that little is known about the magnitude of toxic events in equines is that these occurrences are largely unreported by veterinarians or horse owners.

There are currently no generally accessible centers for the reporting of toxic events in equines. The National Animal Poison Control Center, run by the ASPCA (American Society for the Prevention of Cruelty to Animals), accepts data on equine toxic events from veterinarian members, but this information is not reported to the public (NAPCC 2006). The National Animal Health Monitoring

System (NAHMS) of the United States Department of Agriculture (USDA) collects information on equine deaths and illnesses, but those statistics are released only once every several years, and toxic events are not recorded as separate from other deaths or illnesses (USDA NAHMS 1998).

In addition, equine death or illness due to the ingestion of toxic plants may be attributed to other causes. When death occurs, tests are seldom conducted to identify the cause, and if tests are performed, they may not identify toxic agents or specific plant matter in the digestive tract (Woods et al. 2004). In many cases, no specific evidence of plant poisoning is found during necropsy (Knight 2001). Furthermore, it is also possible for poisoning by plants to mimic equine colic or other illnesses, depending on the plant involved (Bamka and Barbour 2003). Colic encompasses several types of severe gastric distress and is frequently fatal in equines. It is often attributed to a change in pasture or diet but not to a toxin, and its cause is extremely difficult to identify (Moore 1999). It may be assumed that an animal with a history of colic is simply having another episode, when actuality poisoning may be occurring. The Equine 98 study states that the cause of owner-reported colic was reported as “unknown” (USDA NAHMS 1998). Furthermore, the symptoms of poisoning may closely resemble other diseases or disorders, such as arthritis or tumors. Toxins can physically damage or change the normal environment of the digestive tract, as with endotoxemia, gastritis, and peritonitis (Freeman and MacAllister 2006). Death

from plant poisoning may also be attributed to an animal's advanced age, in which case it may not be questioned or investigated.

Livestock losses attributed to poisonous plants have been estimated at $340 million annually in the 17 Western states (Allen and Segarra 2001). Each year, 3 to 5 percent of the cattle, sheep, and horses in western ranges are negatively affected by the ingestion of poisonous plants (James et al. 1980). The USDA's Equine 98 study estimated the direct cost of colic in equines to be $115 million in 1998, regardless of cause or outcome (USDA NAHMS 1998).

Other impacts and costs not generally included or accounted for in reports may include loss, decreased use, subsequent replacement costs, carcass disposal costs, veterinary care (including long-term medications and increased number of examinations), and special diets and supplements that may be required for the life of the animal. In extreme cases of toxic plant infestations, there may be loss of pasture productivity or utility; some pastures may become unusable for grazing without extensive treatment (DiTomaso 2000). In addition, there may be peripheral costs such as consultation fees, adding fencing or re-fencing to exclude trouble spots, planting of desirable forage plants, removing toxic plants, applying herbicides to infested areas, applying fertilizers to stimulate growth of desired forage, or moving the water source to discourage animals from gathering in areas with toxic plants (Knight 2001).

The extent of the problem of plant poisoning in equines has not yet been adequately documented, but clearly it is a problem with significant impacts and

distressing or expensive outcomes. There are a number of factors specific to equines and the relationship between equines and toxic plants that warrant further examination.

Equine Physiology that Contributes to Toxic Events

The digestive system of equines differs from other large livestock such as cattle, sheep, and goats because equids are monogastric and not ruminant. Ruminants possess a large four-chambered digestive organ that allows for microbial degradation of plant compounds, resulting in the detoxification of matter before it moves on to the small intestine, where absorption occurs (Oehme and Barrett 1986). Unlike ruminants, equines have a large single-chambered fermentation organ located past the small intestines. The toxins that an equine ingests in plant matter enter the intestine to be absorbed into the blood before detoxification via fermentation can occur (Kline et al. 2000)

This type of digestive morphology may result in some advantages and disadvantages relative to the effects toxic plant ingestion. For instance, because of microbial action in the rumen, some compounds, such as nitrates, are less toxic to monogastrics than ruminants (Thomas et al. 2001). Ruminants are generally more susceptible to nitrate poisoning because of the greater efficiency of rumen microorganisms in converting nitrate to nitrite, its toxic form. The equine is much less susceptible, because the hindgut is less efficient in converting nitrate, though poisoning is still possible (Noon 2001).

Another possibly advantageous process protecting equines is that food often passes through the gut quickly, resulting in some plant materials, including those with toxic content, being passed before digestion fully occurs (UVM 2006). A disadvantage of the equine gastric system is the reluctance or inability of horses to vomit and thereby rid themselves of toxic agents quickly (Oehme and Barrett 1986, Kiley-Worthington 1997). The most common symptom of gastric distress in equines is diarrhea, which may also serve to quickly eliminate toxins from the digestive tract (Launchbaugh et al. 2001). In the ruminant system, food cannot pass out of the rumen before degradation and particle breakdown occur. Therefore, a ruminants' intake is limited until the feed in the rumen is passed into the lower digestive tract (Janis 1976). However, in monogastrics, feed can be continually ingested as long as some is being excreted, which may result in greater intake of toxic material, though this material may pass more quickly through the digestive tract (Janis 1976). The tolerance for toxins may also vary between individual horses, depending upon physiological, physical, and medical factors such as size or weight, age, body condition, pregnancy, and pre-existing health issues or disorders (Knight 2001).

Equine Behaviors as Possible Contributors to Toxic Events

The particular grazing habits of equines may influence severity and frequency of toxic events. Instinct, unfortunately, does not protect equines from poisoning by plants, regardless of widespread belief to the contrary (Bamka and

Barbour 2003). Horses raised on a particular forage will continue to accept that forage if it is offered (Freeman and Redfearn 2006), even if it is toxic. Horses are highly efficient grazers, capable of eating forages down to the soil surface, often including root material (Freeman et al. 2003). They also are notorious for searching for new, green forage in the spring, which may result in the consumption of toxic plants such as locoweed (*Astragalus* and *Oxytropis* spp.), hemlocks (*Cicuta* and *Conium* spp.), and death camas (*Zigadenus* spp.) that emerge early in the growing season (Bailey 1916, Pfister et al. 2001, James et al. 2005). "Chasing green", as it's called, can result in an early spring diet low in fiber and high in soluble carbohydrates containing high concentrates of potentially toxic plant material (Brendemuehl 2005).

Equines possess varying plant preferences in diet selection. For instance, research has shown that even within a species, breeds may differ in the diets they prefer (Provenza et al. 2001). Social facilitation, in which animals tend to mimic the eating selections of others (Thorhallsdottir et al. 1990), may also create problems if a horse that selects toxic plants influences other horses (Pfister et al. 2001, Sweeting et al. 1985). Horses may choose patches that most recently yielded favored forage, even if the average quality of the remaining forage is now lower due to previous selective grazing (Devenport and Patterson 2005).

Alternatively, food neophobia, the reluctance to eat new forages, may protect equines from over-consuming novel toxic plants when they are placed

into an unfamiliar pasture (Harris 1999). Herd characteristics of equines, such as grouping and the common tendency to defecate in certain areas, may lead to areas that are avoided as well as areas that receive a much heavier level of use (Freeman et al. 2003). Animals characteristically select plants and plant parts containing a lower concentration of toxins, if these are available (Launchbaugh et al. 2001).

Plants vary in palatability and the temptation they present to grazing animals. There is a sequence of palatability in pastures that changes with the seasons. Horses will selectively graze for less mature, or more tender, forages (Freeman and Redfearn 2006). After the desirable young forage is depleted, mature grasses and other less palatable forage, including poisonous plants, may become more acceptable and more likely to be eaten (Putnam et al. 1991). A complicating factor is that some plants may become more toxic as conditions become drier. Furthermore, if a monoculture of forage has been created by selective grazing, the likelihood of poisoning may increase (Hart et al. 2003). The forage intake of horses does not appear to be affected by the quality of the diet (Duncan 1992). Horses may therefore consume normal amounts of forage of poor nutritional quality, including toxic plants, to avoid hunger. In addition, selective spot grazing, particularly common for equines, creates spaces for the establishment of less desirable species, including toxic plants (Freeman and Redfearn 2006).

Animals tend to select for plants of lower toxicity if given a choice (Knight 2001, Provenza 2001). Mammals develop taste aversions that aid them in avoiding the ingestion of toxins (Garcia 1989). However, grazing animals will readily graze less palatable and possibly toxic forage if forced by hunger (Freeman and Redfearn 1996). Most forage grasses, however, have been found to be toxin-free and of little concern (Thompson et al. 2001). If a pasture is dominated largely by forage grasses and is kept in relatively healthy condition, there likely will be few, if any, toxic events (Bamka and Barbour 2003).

Some toxic plants are, however, highly palatable, and horses will seek them out in preference to other forages. These preferences, over time, may sometimes create apparent addictions to abnormal or unusual foods, called picas. Potentially addictive plants to which equines may develop a preference include yellow starthistle (*Centaurea solstitialis)*, locoweeds, and creeping indigo (Bailey 1916, Pfister et al. 2001). These preferences may develop in as little as four days (Pfister et al. 2001). The wilting of leaves of some plant species, including *Prunus* spp., as a result of cutting or frost, may result in increased sweetness, due to the conversion of glucosides to hydrocyanic acid and sugar, which contributes to palatability (Kline et al. 2000). The spraying of certain herbicides, such as 2,4-D, may raise the plants' sugar content, resulting in an increase in palatability to equines (Logue 1998, Thomas et al. 2001, Doll 2004).

Poisoning Problems Specific to Equines

"Any plant known to cause problems in other livestock species will probably affect horses" according to Redfearn and colleagues (1989). This is true, with the possible exception of nitrate toxicosis, but in addition there are also physiological characteristics specific to equines that influence susceptibility to plant toxicity.

There is often great difficulty in recognizing or diagnosing plant poisoning. Plant toxicity may mimic true colic through three potential actions; acting as a direct irritant to the gastric system, acting upon the nervous system to stimulate the gastric system, and causing obstruction or impaction (Knight 2001). A survey published in 1998 found that colic was second only to advanced age as cause of death (USDA NAHMS '98). Add to that the mortality figures from digestive problems and unknown causes, and the total is 25 percent of all equine deaths (USDA NAHMS 1998). That number is significant when one considers that a large portion of those deaths may have been attributable to the ingestion of toxic plants if further testing had been performed. In-depth examination and complicated testing are seldom used, due to expense and limited availability.

Symptoms resulting from the ingestion of toxic plants occur with varying speeds and levels of intensity. The speed with which many toxic reactions occur in equines makes it problematic for both owner and veterinarian. An ornamental plant, the Japanese yew (*Taxus cuspidata*), is one plant highly cited for its ability to fatally poison with only a few ounces, with death occurring within hours

(Purdue ADDL 2006). With other plants, by the time symptoms are present, it may be too late for treatment. Some toxic compounds affect vital organs without any symptomatic display until the organ is largely compromised, as in the case of severe liver disease (Merck 2006). Severe gaseous gastric distension may be the major symptom and prevalent necropsy finding linked to toxicosis (Woods et al. 2004).

Toxins may also affect more than one organ or physiological system, presenting somewhat minor symptoms in one system, which are then tested for and treated. Meanwhile the true and fundamental problem is severe organ disease or failure that isn't apparent until well after treatment for the initial disorder (Knight 2001). The plant or toxin causing the disorder may therefore be very difficult, if not impossible to identify. It may also be very dangerous if the owner or practitioner relies on symptoms alone. For instance, nitrate and prussic acid toxicity may be easily confused, and the treatment for nitrate toxicosis can be deadly if the disorder is in fact caused by prussic acid (Thomas and Schneider 2001). Therefore, it is imperative that blood analyses are completed when toxins are suspected.

Equine Factors Contributing to Toxic Events

There are physical, behavioral, and physiological differences among equines that impact the individual susceptibility to poisoning (Bailey 1916). Body condition is one element that can have a direct effect on an animal's response to

toxins and recovery from poisoning (Freeland and Janzen 1974, Launchbaugh et al. 2001). It is perhaps the single attribute of a horse over which the horse owner has control, and the factor that is easiest to modify. Weight is critical because it affects the amount of toxin the animal can ingest before it becomes a critical or lethal dose, as toxicity is generally based on percentage of body weight (Allison 2007). Improving an animal's condition through diet and nutrition often leads to more rapid detoxification, lessening of toxic effects and damage from toxic compounds (Boyd and Campbell 1983, Launchbaugh et al. 2001).

Individual animals vary in their ability to handle varying amounts of minerals and vitamins. However, it is accepted that either excessive or deficient levels of these can impact livestock health and production which could subsequently affect susceptibility to toxic plants (Mayland and Showmaker 2001). Deficiencies may result in severe illness, while excesses may lead to serious side effects. All equines' health and ability to cope with illness and plant toxicity depend on proper diet and nutrition (Russell and Bauer 1995).

Other factors that may affect a horse's individual ability to handle toxins are age and existing or prior health issues. Age is generally a concern only if the animal is very young or quite old. Young animals, for instance, are far more susceptible to the toxins in locoweeds (Knight 2001). Existing health issues may make the horse more susceptible to toxins, may alter the course of treatment, and must be considered on an individual basis. The breed of horse has been named a risk factor in some studies for colic (USDA NAHMS 1998), which may

indicate that it also affects other gastric problems including plant poisonings, though this has not yet been demonstrated. The color of a horse has a recognized correlation to susceptibility to some toxins; in particular, toxin-induced photosensitization is far more likely in animals with light-colored skin or skin patches (Knight 2001, Brendemuehl 2005).

An individual animal's risk for toxicosis may reflect its grazing efficiency, which varies among animals. Selective grazing patterns determine plant ingestion, and may be related to horses' vulnerability to plant-induced toxicosis (Marinier et al. 1992). Immunities to toxins may be a result of long-term exposure to certain poisonous plants (Freeland and Janzen 1974). As a group, equines are less immune and therefore more highly susceptible to some toxins, including those found in tansy ragwort (*Senecio jacobea*), locoweeds, and members of the nightshade family (*Solaceae* spp.; Knight 2001, Pfister et al. 2001, DiTomaso 2002).

Plant Factors Creating or Contributing to Toxic Events

No common denominators exist in every plant poisoning of an equine. Rather, there are many factors that may influence the occurrence and severity of these events. There is no one particular compound or single factor that creates a poisonous plant. Toxic plants do not possess common characteristics such as color, growth form, taste, or odor. "The degree of danger a poisonous weed represents is a function of the plant's prevalence, toxicity, and desirability"

(Bamka 2003). The general toxicity of a plant is directly related to the toxin or toxins it contains, the organ or system it affects, the amount consumed, and the individual animal consuming it.

The amount of toxic plant matter consumed has a direct effect on the duration and severity of plant poisoning. The intake level most often cited as potentially lethal is five to ten percent of the animal's body weight over a period of two to ten weeks (Knight 2001). However, this is only a general guideline, and deadly or critical poisoning may occur in as little as a few hours or as long as several months after ingestion of the toxic plant material. Toxins may accumulate in an animal's organs and tissue (Launchbaugh et al. 2001), which can result in symptoms appearing in winter when the animal is on uncontaminated hay rather than the previous summer when the green plant was actually grazed (Knight 2001). There is a toxic threshold for all toxic compounds, which is when clinical symptoms initially appear (Pfister et al. 1997). Toxic amounts may range from as little as a few ounces, as for Japanese yew or water hemlock (*Cicuta douglasii*), to as much as 86 to 200% of the animal's body weight, as has been estimated for yellow starthistle (Knight 2001). Small doses of some compounds are medicinal, while larger doses may be toxic, as in the cases of belladonna (*Solanum* spp.) and aconite (*Ranunculus* spp.; Bailey 1916). In theory, nearly anything can be toxic to an animal if a sufficient quantity of the material is consumed (Hintz and Brown 2001). Fortunately, most equines will seldom gorge themselves on unpalatable or less tender plants or plant parts,

choosing instead to expend the energy to seek out preferred foods (Devenport and Patterson 2005).

The specific part of the plant consumed may also significantly impact the amount of toxin ingested, as plant parts may vary widely in toxicity (Bailey 1916). For instance, seeds, such as those of the fiddleneck (*Amsinckia intermedia*), creeping indigo, and rattlebox or rattlepod (*Crotalaria* spp.) contain the highest concentrations within the plant of an alkaloid toxic to horses (Knight 2001). Therefore, small amounts of these seeds are sufficient to cause toxicity. The leaves of the red maple (*Acer rubrum*) are toxic in the fall, but the stems and bark are not (ADDL 2007).

The growth stage of plants often has a significant effect on the amount of toxins an equine will ingest when grazing the plant. In some plants, the immature, new growth may be the most poisonous, while in others that stage may be the least toxic (Bailey 1916). Many plants such as larkspur (*Delphinium* spp.) and death camas are far more toxic when young, which is also when they are most succulent and likely to be selected (Knight 2001). The leaves of the red maple, however, are not toxic when green, but are poisonous when dried or shed (Knight 2001). The toxic effects of one tree, the black walnut (*Juglans nigra*), do not even require ingestion, but are the result of contact with its tissues or inhalation of its oils or pollen (Knight 2001).

Environmental Factors That Contribute to Plant Toxicity

Defoliation, heat, drought, frost, and other physical influences can all affect the plant's toxicity (Shulaw 1999, Pfister et al. 2001, Thomas and Schneider 2001). Climatic events that vary among geographic areas, seasons, and even years in the same area can all affect plant toxicity, depending on the volatility of the compound involved (Bailey 1916). Drought may significantly affect the toxicity of an individual plant (Thomas et al. 2001), and the composition of entire plant communities, as in the case of Senecio species (Pfister et al. 2001) and nitrate-containing plants (Allison 2007), which may become more prevalent during drought. Some plants in which toxin levels are increased by drought include nightshades, sorghum grasses (*Sorghum* spp.), wild cherry or chokecherry (*Prunus* spp.), and pigweeds (*Amaranthus* spp.; Shulaw 1999). On the other hand, excessive moisture can also alter toxicity. In the case of certain sweet clovers (*Melilotus officinalis* and *M. alba*), hay or haylage containing these plants may be toxic if put up under wet conditions or not properly cured, due to the presence of coumarin (Knight 2001).

A killing frost or hard freeze is sufficient to convert certain glycosides in plants to prussic acid, also known as hydrogen cyanide (Stanton and Whittier 1992, Sulc 2006). This is most often observed in forage Sudan grasses (*Sorghum vulgare*; Sulc 2000, Maas 2001) but the potential also exists in white clover, vetches (*Astragalus* spp.) and chokecherry (*Prunus virginiana*; Stanton et al.

1992). The lush regrowth of sorghum species after frost also may accumulate high levels of prussic acid (Selk 2006).

Soil mineral content and balance may in certain cases have an effect on plant toxicity. Selenium is one mineral that livestock producers and equine owners in many areas have difficulty regulating in pastures and hay. Soils high in selenium readily supply plants with the mineral, though not all selenium in soil is available to plants (Davis et al. 2006). There are two types of plants that accumulate selenium: those that grow only on soils high in selenium are called obligate accumulators, and those that do not need soil with high selenium content for tissues of high selenium concentration are called facultative accumulators (Ruyle 1993). Obligate accumulators are generally unpalatable and possess an odor of garlic and sulfur (Knight 2001). Examples of these are milkvetches, prince's plume (*Stanleya pinnata*), woody asters (*Xylorrhiza glabriscula*), and goldenweeds (*Haplopappus engelmannii*). Facultative accumulators are usually more palatable and readily eaten by horses but they become less so with higher soil selenium levels (Davis et al. 2006). These include saltbush (*Atriplex* spp.), curlycup gumweed (*Grindelia squarrosa*), and broom snakeweed (*Gutierrezia sarothrae*; Davis et al. 2006). Other potentially toxic minerals include aluminum, cadmium, mercury, molybdenum, and arsenic, though poisonings due to these are quite rare (Ammerman et al. 1977). Soils that are deficient in phosphorus or sulfur may also contribute to the accumulation of nitrates by plants (Shulaw 1999).

Application of herbicides and fertilizers may also impact the toxicity levels in plants (Shulaw 1999). For example, herbicide application can increase levels of cyanogenic glycosides in plants that contain these compounds (Knight 2001). The herbicide metsulfuron has also been found to increase the toxicity of larkspur (Pfister et al. 2001). Fertilizers may also raise the toxic levels of cyanogenic glycosides in some plants and plant families (Knight 2001).

Drying of plant material, either in the pasture or as hay, can affect the toxicity and palatability of certain plants. Some palatable toxic plants, such as white snakeroot (*Eupatorium rugosum*), are more toxic as very mature plants compared to younger plants, a condition that persists when dried, resulting in possible poisoning problems if included in hay (Knight 2001). There are yet other plants, including houndstongue (*Cynoglossum officinale*), that are highly palatable and toxic in hay, but not generally palatable or eaten as green plants in a pasture (Pfister et al. 2001). These include buckwheat (*Fagopyron esculentum*), St. Johnswort (*Hypericum perforatum*), brackenfern (*Pteridium aquilinum*), and houndstongue (Pfister et al. 2001). Some poisonous plants become less dangerous if the plant material is consumed as dry hay. Examples of this are plants containing cyanogenic glycosides such as oleander (*Nerium oleander*) and buttercups (*Ranunculus* spp.), and others containing certain alkaloids (Knight 2001, James et al. 2005). Most plants containing pyrrolizidine alkaloids are generally unpalatable and not selected by horses when green, but

become palatable and may be eaten when dried in hay (James et al. 2005, Knight 2001).

Primary Types of Toxins Affecting Horses

To a large degree, the toxic effects that equines experience from the ingestion of poisonous plants are due to the specific toxic compound present in the plant. Some toxic agents are more dangerous than others, even in small amounts, while others may rarely affect equines or have fairly mild effects. Every plant toxin has what is termed a 'toxic margin', however, the significance of which is that even mild toxins will have deleterious effects if consumed in sufficient quantities (Knight 2001).

Plant toxins can be grouped based on their chemical components. The groups of compounds most toxic to equines are alkaloids, nitrates, organic acids, and glycosides. In addition, some molds are highly toxic if consumed in sufficient quantities.

Alkaloids poison more livestock than any other group of toxic compounds (Pfister et al. 2001). Pyrrolizidine alkaloids are a specific type of alkaloid, and are the largest plant-poisoning problem for livestock worldwide (James et al. 2005). Horses are more sensitive to both swainsonine and solanine than are any other grazing livestock (Knight 2001, Pfister et al. 2002). Alkaloids are responsible for causing hepatotoxicosis and liver failure, sudden death, neurological problems, and photodermatitis, among other disorders (James et al. 1980, Ruyle 1993,

Knight 2001, Launchbaugh et al. 2001, James et al. 2005). The reactions caused by alkaloids may be acute, which are very swift and lethal, or chronic, which commonly causes irreversible liver disease (Knight 2001). Some examples of alkaloid-containing plants are tansy ragwort, horsebrush (*Tetradymia glabrata*), locoweed, larkspur, lupine (*Lupinus* spp.), houndstongue, death camas, and poison hemlock.

Some forage grasses contain an endophytic fungus that is capable of producing ergot alkaloids toxic to grazing animals (Thompson et al. 2001). The grass of greatest concern is tall fescue (*Festuca arundinacea*), which frequently causes toxicity in broodmares. This often results in foal mortality, decreased milk production, and inability to conceive (APHIS 1999, Redfearn et al. 1989).

Nitrate poisoning is much less likely in horses than in cattle (Ruyle 1993, Knight 2001, Noon 2001). However, equines are still somewhat susceptible because nitrate can be converted to toxic nitrite in the cecum (Thomas et al. 2001). Frost, drought, or fertilization may increase the nitrate levels in Sudan grasses and hybrids (Maas 2001, Allison 2007). Some plants that may contain toxic levels of nitrates include kochia (*Kochia scoparia*), Johnsongrass (*Sorghum halapense*), nightshades, Russian thistle (*Salsola iberica*), and field bindweed (*Convolvulus arvensis*). Plants with over 1.5% nitrate are problematic; a lethal dose of nitrates may be as low as 0.05% of body weight (Allison 2007). Nitrate poisoning normally results in sudden death, but can also reveal itself as hypoxia, weakness, severe trembling, breathing difficulties, and coma (Allison 1998).

The most common poisoning problem in the organic acid group is from oxalic acid or oxalates (Ruyle 1993). Colic may be one of the primary symptoms of oxalate poisoning. Oxalic acid binds up calcium and may result in hyperparathyroidism resulting in “Big Head” disease and other skeletal problems (Freestone and Seahorn 1993). Plants containing these include Russian thistle, greasewood (*Sarcobatus vermiculatus*), and halogeton (*Halogeton glomeratus*). Tannins are compounds in a related organic acid group, are present in some oak varieties (*Quercus* spp.), and can cause poisoning (Ruyle 1993, Knight 2001).

The glycoside group contains a number of compounds that are related but cause different types of toxic reactions. These include cardiac glycosides, cyanogenic glycosides, coumarins, and saponins. The glycosides are responsible for many of the sudden deaths from plant toxins in equines. Sudden death is usually the first sign of cyanogenic glycoside poisoning, while other symptoms of cardiac glycoside toxicity may be colic, diarrhea, and cardiac irregularities (Knight 2001). Commonly ingested plants containing these compounds include serviceberry (*Amelanchier alnifolia*), chokecherry), milkweeds (*Asclepias* spp.), oleander, hemlocks, yews, and wild blue flax (*Linum* spp.). Prussic acid (hydrogen cyanide or hydrocyanic acid) poisoning may be a problem after environmental stress such as drought or frost for grasses including Sudan grass and Johnsongrass (Stanton et al. 1992, Sulc 2000, Redfearn and Freeman 2006). This is generally responsible for chronic cyanide poisoning, which results in neurological diseases (Knight 2001).

Some varieties of white and yellow sweet clovers contain coumarin, which can become toxic under specific conditions. High levels of coumarin, can be converted by molds to dicoumarol, a strong anticoagulant that may cause bleeding disorders. The amount that must be ingested before symptoms appear is rather large, but mortality is high if untreated (Knight 2001).

There are also a number of more minor groups of plant compounds that cause problems for equines, though on a lesser scale than the toxins mentioned above. Chemical compounds in these plants include alcohols, ketones, and terpenes. Blister beetles must also be mentioned: though not a toxin, they feed on plants including alfalfa (*Medicago sativa*) and goldenrod (*Solidago* spp.) and subsequently produce toxic deposits to which horses are highly susceptible (Campbell 2001).

Body Systems and Organs Damaged by Toxic Plants

Plant toxins can have varied, dangerous, and complex effects when ingested by equines. The major and most common clinical signs of plant poisoning in equines are most easily grouped by the body region, organ, or system affected. Symptoms of plant toxicosis can be revealed as physical damage, colic and diarrhea, teratogenic effects, photodermatitis and photosensitization, hepatotoxicosis and liver disease or failure, sudden death, anemia, neurological disease and disorders, and muscle weakness and lameness.

Physically injurious plants are a large and varied group. Physical damage from plants may result in blistering, ulceration, cuts or lesions to the mouth, tongue, gums, cheeks, nose, eyes, throat, and esophagus, and may progress into the digestive tract. Examples of plants causing physical damage or trauma to the skin include puncture vine (*Tribulis terrestris*), burdock (*Arctium minus*), sandbur (*Cenchrus longispinus*), foxtail barley (*Hordeum jubatum*), cheatgrass (*Bromus tectorum*), cockleburs (*Xanthium* spp.), medusahead rye (*Taentherum asperum*), thistles (*Cirsium* spp.), and cactus (*Opuntia* spp.).

There are several types of colic, the development of which are dependent upon the action of the plant toxin. Colic may be caused by a direct irritant, spasms caused by the nervous system, impaction or obstruction, excessive fermentation, and displacement in which portions of the intestine become displaced or twisted (Freeman and MacAllister 2006, Knight 2001, Keen et al. 1996). Effects of colic include abdominal pain/distention, blockage in lower digestive tract, diarrhea, muscle pain or weakness, and anxiety. Plants causing colic include leafy spurge (*Euphorbia esula*), iris (*Iris missouriensis*), horsetail (*Equisetum* spp.), bitterweed (*Hymenoxys odorata*), mustards (*Sinapis* spp.), nightshades, field bindweed, and buttercups.

Possible teratogenic (i.e., resulting in deformation of foals) and gestational (i.e., during pregnancy) effects of toxins are spontaneous abortion, defects in the fetus or foal, decreased milk production, temporary or permanent infertility, prolonged pregnancy, and the need for cessation of the pregnancy. These are

much more likely and problematic if the plant toxin is consumed within the first trimester of pregnancy (Keeler 1984). Teratogenic plants include tall fescue, milkvetches, locoweeds, lupine, Sudan grass, and western false hellebore (*Veratrum californicum*).

There are two types of photosensitization, primary and secondary (Clare 1955). Equines with lighter colored hair, including light or white patches, often have higher susceptibility to primary photosensitization from toxins. However, they are no more susceptible to secondary photosensitization than dark-haired animals. In primary photosensitization, the plants are eaten and the toxin is absorbed, and the effects may be cumulative over time. Dermatitis may result from photosensitization, and may be severe enough to cause loss of the entire skin, extreme pain, and prohibit the animal from eating and/or drinking, potentially resulting in death. Primary photo-sensitizing plants include St. Johnswort, buckwheat, some ryegrasses (*Lolium* spp.), and burr trefoil (*Medicago polymorpha*). In secondary photosensitization, liver disease is the first and underlying cause of the cellular damage, after which photosensitivity appears (Ivie 1982). A severely damaged liver is unable to eliminate by-products of chlorophyll that accumulate in the blood and cause cellular damage, which then results in photosensitization. Secondary photosensitizing plants include alsike clover (*Trifolium hybridum*), fiddleneck, houndstongue, stickseed (*Hackelia* spp.), Kleingrass (*Panicum coloratum*), indigos, and rattlepod or rattlebox.

Liver disease is a consequence of poisoning by relatively few plant toxins, though many plants contain these compounds. Symptoms may occur only after the majority of the liver is destroyed. Acute liver disease is irreversible, and often results in death in a short time. Species causing hepatotoxicosis include the species listed above for secondary photosensitization and several species of ragwort, butterweed (*Senecio glabellus*), and groundsels (*Senecio* spp.).

Sudden death results from ingestion of relatively few plant compounds. Three basic types of compounds are known to cause sudden death: cyanogenic glycosides, cardiac glycosides such as digitoxin, and certain alkaloids (Knight 2001). Very small amounts of these toxins may be enough to cause sudden death, which often is the first clinical sign or symptom. Plants containing these compounds include serviceberry, death camas, larkspur, yews, hemlocks, lily of the valley (*Convallaria majalis*), oleander, milkweeds, dogbane (*Apocynum cannabinum*), and Sudan grasses, when severely stressed.

Anemia resulting from ingestion of plant toxins is due to changes in blood chemistry or physiology, with ensuing hemolysis or hemorrhaging (Pierce et al. 1972). There are three well-known plants known to cause anemia: domestic and wild onions (*Allium* spp.), red maple, and moldy sweet white or yellow clovers.

Neurological disorders are normally fairly observable, as the clinical signs usually include significant changes in behavior (James andVan Kampen 1971). Some symptoms may be reduced vision, lack of coordination, inability to chew, swallow, or recognize food, and severe physical manifestations including

convulsions, falling , circling, severe change in temperament, and abnormal reaction to stimuli such as movement, odors, sounds, or light. Chemicals causing neurotoxicosis include terpenes, alkaloids, nitroglycosides, thiaminase, tremetol, and cyanogenic glycosides (Knight 2001). Plants responsible for neurological disease include yellow starthistle, Russian knapweed (*Centaurea repens*), locoweeds, milkvetches, brackenfern, horsetails, white snakeroot, and Sudan grasses.

Two common causes of muscle weakness or lameness are a deficiency in calcium or an excess of selenium. Selenium toxicosis may be either chronic or acute. The chronic form results from the ingestion of selenium accumulators for several months and is often called staggers, blind staggers, or Alkali disease. Acute selenium poisoning usually results in death before clinical signs are evident.

Another cause of lameness is contact with the shavings, sawdust, or pollen of the black walnut. Laminitis may also be due to a severe episode of colic. Plants that may cause muscle weakness or lameness include hoary alyssum (*Berteroa incana*) and day-blooming jessamine (*Cestrum diurnum*). Plants causing selenium toxicosis are milkvetches, woody asters, saltbush, curlycup gumweed, bastard toadflax (*Comandra pallida*), snakeweed or broomweed, and golden weeds (*Haplopappus* spp.).

Toxic Symptoms and Diagnosis

The foremost indicator of a toxic event is any significant change in an animal's behavior. Any inconsistency or unusual behavior should be cause for concern (Sestric and Coates-Markel 2005). It is possible for behavioral changes to result from other disorders, such as problem teeth, but many plant toxins can result in mortality fairly quickly. Some of the more common physical and behavioral symptoms include changes in appetite, observable physical trauma, digestive changes, neurological symptoms, and muscle weakness or lameness. Signals that may be observed as an indication of ingestion of a toxic compound or contact with a toxic or physically injurious plant are:

- Decreased appetite, refusal to eat customary foods, evidence that the animal is eating unusual plants or materials, lack of ability to recognize food when it is presented, chewing when food is absent, rapid weight loss, refusal to drink or excessive consumption of water, and frequent lipping of or splashing in water.
- Symptoms of liver disease include weight loss, abnormal behavior, anemia, and depression.
- Physical symptoms such as drooling, excessive salivation, blisters, ulcerations, cuts, lesions, skin conditions appearing severe or painful, sunburn to light or white areas, and loss of hair coat.
- Digestive problems or changes such as diarrhea, hard, dark, or bloody feces, lack of or painful elimination, abdominal distention, urine that is

unusual or dark in color, flatulence, unusual odors on the animal's breath, restlessness, increased pulse or temperature, sweating, and lying down and/or attempting to roll.

- Neurological signs such as decreased vision, peculiar gait, lack of coordination, poor balance, convulsions, circling, abnormal reaction to common stimuli, tremors, fearfulness or anxiety, rolling eyes, and excessive or unusual friendliness or aggression.
- Lameness or inability to stand, weakness, refusal to put weight on a leg or legs, joint tenderness, refusal to move, stiffness, and leg or limb swelling or edema.

Treatment of Plant Toxicosis

Removal of the animal from the pasture containing the possible toxicant is always a necessary first action. In some cases, removal from pasture can clear the toxin from the animal's system within days (Brendemuehl 2005). Plant matter can clear the stomach in 24 hours and the entire digestive system in as little as 48 hours (Kline et al. 2006). However, in the case of some toxins such as pyrrolizidine alkaloids, chronic toxicity occurs with the ingestion of small amounts over weeks or months, and clinical signs may occur months or even years after ingestion of the plants (Knight 2001).

Treatments do exist that can alleviate some or all of the symptoms or discomfort of plant poisoning by certain toxins. Common therapies may include

special diets based on alfalfa and cereal grains, supplementation with proteins, vitamins or minerals, intravenous fluids therapy, blood transfusions, surgery, medications such as domperidone, sedation, intravenous vitamin K, injections of sodium nitrite or sodium thiosulfate, activated charcoal, potassium therapy, and respiratory stimulants, among others (Knight 2001, Thompson et al. 2001).

Antidotes or preventive medications are occasionally discovered that aid grazing animals in survival of toxic events (Launchbaugh et al. 2001). One of these is the use of Ivermectin, a medication normally used as a wormer to treat internal parasites, for the treatment for alkaloid toxicity (Thompson et al. 2001), though the efficacy of this has not yet been documented for equines. Alkaloid toxicity can also be counteracted with physostigmine injections or a certain dopamine antagonist (Aldrich et al. 1993, Pfister et al. 1994), although, again, this has not been documented in horses. Special diets or supplementation with grains or molasses may decrease nitrate toxicosis (Thomas et al. 2001). Laboratories such as the USDA-ARS Poisonous Plant Laboratory in Logan, Utah are also conducting research on toxins with the target of developing vaccines for livestock, which may also prove to be useful for horses (James et al. 2005). However, in many cases, there is no available antidote or treatment, even if the signs are recognized in the early stages of toxicity (Knight 2001).

The Impact of Grazing and Pasture Management on the Severity of Toxic Events

There are a many actions that can reduce the occurrence of plant poisonings in equines. The most common approach to diminish losses from plant toxicity is to change the plant community, the grazing animal, or grazing management strategy (Launchbaugh et al. 2001). Changing the grazing animal may be the best approach in some situations, as in the case of infestations of tansy ragwort, which sheep can ingest without toxic effect (DiTomaso 2002). However, for most horse owners, changing the grazing animal is not an option. In other cases the of changing the plant community may be effective but cost prohibitive. Therefore, changing grazing management is most frequently the appropriate option. Most range and pasture communities contain a few toxic plants (James et al. 2005) but with appropriate grazing and pasture management, these plants are not necessarily a danger to equines. Understanding grazing behavior of equines and principles of good forage management are the keys to providing healthy horse pastures and ranges with adequate forage.

- The first factor must be the overall condition of the pasture: forage of low nutritional quality very frequently results in some type of problem for livestock, including lowered physical condition and body weight, susceptibility to toxins in plants, and other health or behavioral issues (Boyd and Campbell 1983). Low quality forage significantly affects body

condition, which may have an impact on the toxicity of ingested plants (Launchbaugh et al. 2001). Lowered nutritional states of animals may result in an increase of toxic effects and decreased rates of detoxification (Freeland and Janzen 1974). Therefore, toxic plants are potentially more dangerous to equines in poor body condition. In addition, proper nutritional condition can result in higher intake of toxic foods without negative repercussions (Wang and Provenza 1996). If allowed to select a varied diet, animals have a lower chance of experiencing a toxic event, because eating a variety of plants spreads the toxins they contain over several detoxification systems (Freeland and Janzen 1974), and because of interactions among allelochemicals that may reduce toxicity (Launchbaugh 1996).

- Selective grazing is the first line of defense that an animal has against toxic plants (Launchbaugh 1996). While there is a natural tendency to select diets composed of several plant species (Launchbaugh et al. 2001), the actual forage consumed often depends on the nutritional state of the animal (Provenza 1995). Normally, grazing animals avoid toxicity by limiting consumption of a toxic plant, allowing time for detoxification (Provenza et al. 2001, Foley et al. 1995). Animals also limit toxin consumption by selecting parts of plants that contain lower concentrations of toxins (Provenza 1995, Pfister 1999). However, in depleted pastures, hungry horses may eat plants they would not if provided with ample,

preferred forage (Redfearn and Freeman 1989). If pastures contain inadequate or inferior forage, selectivity may result in stands with monocultures of less palatable and more toxic plants.

- When toxic plants are permitted to thrive in pastures, they may have either direct or indirect effects on the health of the grazing animals. Direct ingestion of toxic plant matter is the predominant method by which toxins affect animals negatively. However, certain toxins also have a tendency to reduce the digestibility of other forages eaten along with the toxic plant. These compounds may tie up nutrients, kill digestive microbes, inhibit enzymes, and reduce preference (Provenza et al. 2001). This results in reduced nutritional value of the total forage to animals. Thus, even if the compound itself does not generate a toxic reaction, it may indirectly cause immediate or eventual systemic or digestive damage. Often the affects of many toxins are cumulative, with no appearance of clinical signs for months or years (Knight 2001).

- Stocking rates determine the amount of forage available per animal. The total forage supply is affected by soil moisture and type, season and weather, pasture composition, and forage species. Forage demand in a pasture is influenced by the size of horses and the length of grazing season (Freeman and Redfearn 2006). Knowledge of pasture type and size and awareness of the needs of individual horses are necessary to maintain appropriate stocking rates.

- The provision of essential nutrients is imperative; for instance, making salt available so that animals do not develop a preference for and seek out higher sodium forage (Villalba and Provenza 1990).
- Ensuring proper water quality and quantity is vital, because limited water availability can lead to greater toxic effects. Some toxic events may be avoided entirely with an adequate supply of drinking water (Knight 2001).
- Weed management is necessary, as overgrazing can result in higher populations of toxic invasive weeds, such as milkweed (Knight 2001). Plant diversity often increases following weed management, increasing the levels of nutritional forage (DeLoach 1991). Pastures should be examined for weeds in late summer, since frequently broadleaf weeds remain green long after other, more nutritious, forages have been depleted (DiTomaso 2000). Presence of weeds in late season may be indicative of the necessity of mowing or for the application of herbicides.
- Attention must be paid to all pasture conditions: soil condition, plant communities, water, and forage availability, and to the physical and nutritional condition of the equines grazing it, if poisoning by toxic plants is to be avoided.

In Summary

The problem of poisoning of livestock by plants is known to be a considerable one, and the issue of plant toxicity in equines is of comparable significance, though the body of research regarding equines and toxic plants is much smaller. This may be a result of several factors: horses are not generally regarded as economically significant when compared with other livestock; other livestock industries have significant budgets for research studies; equines are not often found in, and therefore poisoned in, large numbers within a pasture or range; the huge variance in sizes and weights of grazing equines results in the difficulty of application of known data to all animals, and the fact that plant toxicity in equines is often not recognized as such but instead attributed to other causes.

There is an obvious need to establish reporting methods for toxic events in equines and centers for dealing with that data. In addition, clearer and more concise knowledge is needed about equine physiology and its specific response to plant toxins, the place of plant toxins in the equine diet and equines' tolerance for them, and less expensive and more reliable testing for common toxic plant compounds, in order that horse owners and land managers may develop the appropriate management strategies to avoid or circumvent plant toxicity in equines.

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Chapter 3

Invasive Weeds, Poisonous Plants, and Horses:

A New Look at an Old Problem

Introducing the Problem

Invasive weeds are becoming an increasingly serious problem in North American forests, rangelands and pasturelands. A growing contemporary challenge to the management of invasive plants on rangeland and pastureland is the phenomenal increase in ex-urban development creating small parcels, many unskilled land owner/managers, and fragmented landscapes. A large number of these smaller acreages are in use by new landowners, and have been converted to horse pastures.

Many of these new landowners have little experience living outside the city, and therefore little to no experience with invasive and/or toxic plants. Even the seasoned horse owner, while familiar with the weeds that have been around for decades, such as Canada thistle, must deal with many newly introduced species, such as houndstongue, that may create new problems and risks for horse health and production.

The target of most existing publications on weeds and pastures has been the professional rancher or farmer, and there are few that focus directly on the toxicology of invasive weeds to horses. Knowledge of weed toxicology and weed

management for the horse owner, especially novice landowners, would have a positive impact on the health of pasturelands and the equines that inhabit them.

Invasive weeds have highly detrimental impacts on grazing lands and pastures, including loss of preferred forages and reduced productivity. These changes to grazing lands may result in poor body condition or nutritional states of livestock. The subset of weeds that contain toxic compounds may add to these problems, because they can have immediate and significant negative impacts on animal health and husbandry, where condition and nutrition are issues. In addition, veterinary care may be extremely costly, and may not be effective when plant toxins are involved. Of course, the greatest cost to many horse owners is simply the emotional cost from the loss of or disability to a beloved and useful horse.

Poisonous Plant Impacts on Equines

Because horses are monogastric, they often are more sensitive to plant toxins than are ruminant livestock like cattle and sheep, that are more able to digest and tolerate many toxins. A number of toxic plants are highly palatable, appearing at times when other forage is limited, and are therefore attractive to horses. No comprehensive figures are available on the actual numbers of equines poisoned by plants annually. Therefore, it is impossible to know the differences in numbers of wild vs. domestic horses affected by plant toxicity, but such information could give us enormous clues to the severity of this problem

and the necessary changes to weed management, grazing management, and feeding programs of equines. The limited size of pastures reduces choices and prohibits migration and pursuit of preferred forage; therefore, what we know about horses and plant toxicity was gained under rather controlled conditions. The practice of feeding hay means little choice for most equines for a sizeable part of the year. Horse owners make decisions that determine what forages are available to domestic equines throughout the year. Are unnatural conditions created for domestic horses that promote poisoning by toxic weeds, even if the weeds themselves would rarely, in normal conditions, create severe problems?

Weeds as Toxic Plants: Fact or Fiction?

Upon review of the published literature on equines and toxic plants, the impression remains that all weeds are toxic or dangerous. However, this is not necessarily the case. Weeds are no more toxic than native species, ornamentals, or garden plants. The word 'weed' is often used interchangeably with the term 'problem plant', thus a plant may be called a weed even if it is neither invasive nor noxious.

Section 403 of the Federal Plant Protection Act (PPA) defines Noxious Weeds as: "any plant or plant product that can directly or indirectly injure or cause damage to crops (including nursery stock or plant products), livestock, poultry, or other interests of agriculture, irrigation, navigation, the natural

resources of the United States, the public health, or the environment." (APHIS 2008)

An 'invasive species' is defined as a species that is 1) non-native (or alien) to the ecosystem under consideration, and 2) whose introduction causes or is likely to cause economic or environmental harm or harm to human health. Invasive species can be plants, animals, and other organisms (e.g., microbes). Human actions are the primary means of invasive species introductions (NISIC 2008).

There is a lack of education about and awareness of weeds, particularly in the novice horse owner. 'Noxious' appears to mean 'toxic' to many people. Upon reflection, one wonders if it could be that the two terms are similar enough so that they are confused by the non-scientist. The terms 'noxious' and 'invasive' are also used interchangeably in some places.

There is a distinct difference between invasive weeds, noxious weeds, and problem plants in horse pastures, though problem plants may also be either noxious or invasive. Overstocked pastures or those with inadequate forage tend to eventually contain an overabundance of unpalatable plants, which remain uneaten until they are the only choices for forage. Palatable plants are the preferred diet for livestock, and therefore receive the most grazing pressure. Poisonous, unpalatable plants in horse pastures are most often weeds by definition, as they are unwanted plants in those places.

Weeds can be signs of infertile soils, misused pastures, or drainage problems. These problems are most often due to mismanagement of pastures, so most invasive weeds themselves are not usually the initial cause of ingestion of plant toxins by equines. With a few exceptions, individual toxic weeds are not problematic; however, communities of plants with high numbers of toxic plants and low availability of good forage plants are a considerable problem.

Publications and websites result in significant amounts of misinformation reaching horse owners. Many sources of information discuss non-weeds while the title refers only to “weeds”. All unwanted plants in pastures are often labeled weeds, and many readers do not understand that while an unwanted plant may be a ‘weed’, it is not necessarily a noxious or invasive weed. One of the reasons horse owners have problems finding accurate information is that there is an abundance of misinformation online on the subject of horses and toxic plants.

One fairly new book claims to be the “only complete guide available on plants that poison horses”, but claims that horses possess gall bladders and lists only a little over 100 plants. A bulletin from the Weed Science Department at the University of Wisconsin is entitled “Poisonous Weeds of Pastures & Forages”, yet names chokecherry and oak among the list of toxic “weeds”. This type of publication leads to the misconceptions that all weeds are poisonous, or that all poisonous plants are weeds. Many websites and articles are not written by rangeland managers or scientists, which results in a significant amount of

misinformation and false 'facts'. A quick Web search revealed that seven out of the first ten websites visited had misleading, inaccurate, or incomplete information. These come from a variety of sources, even traditional extension sources including universities and governmental agencies. Statements such as "Most poisonings occur in hay, so check hay closely for weeds" lead to a false sense of security for horse owners: if hay is weed-free then it must be toxin-free.

In a comparison of plants lists, weeds show an average toxicity of 17.8%; the averaged toxicity of abundant rangeland, native, and ornamental plant categories is 20.1% (Table 3.1). Weeds are apparently not the most lethal of plants, as is a common assumption.

Table 3.1 Comparisons of toxic plant database with lists of rangeland plants, native plants, ornamentals, and weeds.

Plants Lists by Each Of Four Categories Specific Sources Listed In Resources Section	**Total Number of Plants on List**	**Total Number Toxic**	**Total Percent Toxic**
Range Plants			
SRM: Master List of 200 Range Plants	200	43	21.5
USU Extension: Utah Range Plants	165	31	18.8
Average toxic percent			**20.2**
Native Plants			
BLM: Northern Intermountain Native Plants List	101	16	15.8
UMN Extension: Minnesota Native Plants List	144	22	15.3
Average toxic percent			**15.6**
Ornamentals			
OSU Department of Horticulture: Ornamentals List	79	11	13.9
WSU Extension: Horticulture: Ornamentals List	54	19	35.2
Average toxic percent			**24.6**

Weeds			
CIPM: Worst Weeds of US List	75	25	33.3
APHIS/USDA Federal Noxious Weeds	98	3	3.1
NRCS/USDA Federal Noxious Weeds	96	3	3.1
Noxious Lists Case Study: 5 States: CA, ID, NE, NM, UT (USDA)	246	55	31.7
Average toxic percent			**17.8**

This is not to say that weeds are always benign. Some noxious or invasive weeds are highly toxic to equines and can cause tremendous problems if allowed to invade horse pastures (Table 3.2).

Table 3.2 Known toxic weeds in the United States.

Common Name	Scientific Name
Bermuda grass	*Cynodon dactylon*
Black henbane	*Hyoscyamus niger*
Bristlegrass	*Setaria* spp
Buffalo burr nightshade	*Solanum rostratum*
Buffalobur	*Solanum rostratum*
Bull nettle	*Solanum carolinense*
Bull thistle	*Cirsium vulgare*
Burdock	*Arctium minus*
Butter and eggs	*Linarea vulgaris*
Canada thistle	*Cirsium arvense*
Cheatgrass	*Bromus tectorum*
Crofton weed	*Ageratina adenophora*
Downy brome	*Bromus tectorum*
European hemlock	*Conium maculatum*
Field bindweed	*Convolvulus arvensis*
Green bristlegrass	*Setaria* spp.
Halogeton	*Halogeton glomeratus*
Hooked bristlegrass	*Setaria spp*
Horse nettle	*Solanum carolinense*
Houndstongue	*Cynoglossum officinale*
Johnsongrass	*Sorghum halepense*
Klamath weed	*Hypericum perforatum*
Leafy spurge	*Euphorbia esula*
Longspine sandbur	*Cenchrus longispinus*
Medusahead rye	*Taentherum asperum*
Morning glory	*Convolvulus arvensis*
Poison hemlock	*Conium maculatum*
Prickly pear	*Opuntia* spp.
Puncture vine	*Tribulus terrestris*
Rattlebox	*Crotalaria spp*
Rattlepod	*Crotalaria spp*
Russian knapweed	*Acroptilon repens*
Russian thistle	*Salsola iberica*
Sandbur	*Cenchrus longispinus*
Scotchbroom	*Cytisus scoparius*
Silverleaf nightshade	*Solanum elaeagnifolium*
Smartweed	*Polygonum* spp
St. Johnswort	*Hypericum perforatum*
Tanglehead	*Heteropogon contortus*

Common Name	Scientific Name
Tansy ragwort	*Senecio jacobaea*
Wild iris	*Iris missouriensis*
Yellow bristlegrass	*Setaria pumila*
Yellow starthistle	*Centaurea solstitialis*
Yellow toadflax	*Linarea vulgaris*

Some Facts About Toxic Weeds

The growth habits of a number of toxic weeds can make them attractive to horses. For instance, the growth cycle of some weeds results in their prevalence in pastures early in spring. The consequence of this is that horses are often “chasing green” at that time, and find nearly anything green attractive, including toxic weeds such as death camas, some locoweeds, and poison hemlock.

The extensive taproot in many broadleaf weeds allows them to remain green longer into the dry season, thereby becoming potentially attractive to grazing horses in poor pastures. A short list of these includes tansy ragwort, yellow starthistle, Russian knapweed, Canada thistle, poison hemlock, field bindweed, houndstongue, Scotchbroom, horsetails, leafy spurge, black henbane, Klamath weed or St. Johnswort, kochia or fireweed, yellow toadflax, silverleaf nightshade, and puncture vine.

There are a number of types of compounds that cause a plant to be toxic, the presence of which is dependent upon the specific plant or its family. Some of the toxins involved include cyanide and prussic acid, nitrates, cardiac glycosides, saponins, enzymes such as thiaminase, allergenic compounds, tannins, alkaloids,

oxalates or oxalic acid, terpenes, less common compounds such as ricin and phytotoxins, and at times endophytes and fungal vectors.

There is no single set of characteristics that applies to all poisonous plants, including appearance, phenology, preferred soils, method of propagation, or growth type. All poisonous plants do not share a list of common factors, though many do contain similar toxic chemical compounds. For instance, physically similar plants or plants that thrive in similar areas may be very dissimilar in toxicity (Table 3.3).

Table 3.3 Comparisons of toxic and non-toxic plants by four groupings

	Toxic Plant	**Non-toxic Plant**	
Growth Type			
Grass	Cheatgrass	Smooth brome	*Bromus inermis*
Forb	Lupine	Western dandelion	*Taraxacum officinale*
Woody	Sand sage	Wild or Woods' rose	*Rosa woodsii*
Tree	Red maple	Narrowleaf willow	*Salix exigua*
Propagation Type			
Seeds	Yellow starthistle	Arrowleaf balsamroot	*Balsamorhiza sagittata*
Spores	Brackenfern	Western sword fern	*Polystichum munitum*
Rhizomes	False hellebore	Western yarrow	*Achillea millefolium*
Habitat Type			
Arid	Mesquite	Buckbrush	*Ceanothus cuneatus*
Prairie	Locoweed	Mule-ears wyethia	*Wyethia amplexicaulis*
Marsh	Horsetails	Meadow foxtail	*Alopecurus pratensis*
Root System			
Deep taproot	Houndstongue	Tapertip hawksbeard	*Crepis acuminata*
Shallow roots	White snakeroot	Big-head clover	*Trifolium macrocephalum*

Some weeds, such as foxtail barley, may have physical characteristics that cause digestive problems in horses. These are considered 'toxic' plants even though the damage they cause is not because of toxic compounds in the plant. Some weeds, including St. Johnswort and buttercups, are harmful even if not consumed, as their compounds may cause severe dermatitis on contact.

The emphasis on weeds as the only dangerous plants to equines may result in a significant potential risk - a disregard for the presence of other seemingly harmless but highly toxic plants. For instance, some ornamental or garden plants that appear to be inoffensive can be critically toxic to equines. These include onions, Japanese yew, oleander, lily of the valley, larkspur or delphinium, persimmon, avocado, indigo, Indian paintbrush, Easter lily, daffodil, tomato, potato, English ivy, and pincherry. Many problem plants in pastures are ornamental and produce escapees from landscaping and gardens.

Each community of plants has its toxic members. Most native plant communities contain a number of toxic plants. A few of these are big sagebrush, white prairie aster, hawthorns, elderberry, goldenrods, and mahogany species. Common pasture and range forbs known to be toxic include buttercups, wild mustard, wild onion, horsetails and scouring rush, wild iris or blue flag, and wild blue flax.

There are a number of common pasture grasses that can be toxic to equines, though for the most part, grasses are generally the safest forage for equines. The list of possibly toxic grasses includes Sudan grass, Bermuda grass,

Sorghum, German millet, and tall fescue. There is also a potential risk by ignoring toxic trees and shrubs in pastures. Included in this list are ponderosa pine, black walnut, red maple and some maple hybrids, cherry species, and serviceberry.

Other common toxic pasture plants include some clovers such as alsike clover, yellow sweet clover, white sweet clover, and other clovers of the *Trifolium* spp. If horse owners work only toward eradicating pastures of 'weeds' while ignoring specifically toxic plants, they may find that the plants they disregarded as benign are the very ones that should have been cause for concern.

While invasive and noxious weeds can be highly problematic in pastures and rangelands, it is not appropriate to automatically label all of them as dangerous to equines. There are a number of so-called weeds that are beneficial to grazing animals for forage value. Cheatgrass and common dandelion, for example, supply necessary nutrition in early spring before many other forage plants have emerged. Some weeds are actually higher in nutrients, sugars, and proteins than some forage grasses, providing often-needed sustenance in fall when grass populations are depleted.

The Impact of Grazing and Pasture Management on the Severity of Toxic Events

There are a number of factors that can reduce the occurrence of plant poisonings in equines. The most common approach among range and livestock managers to reduce losses from plant toxicity is to change the plant community, change the grazing animal, or change grazing management strategies. Changing the grazing animal may be the best approach in some situations, as in the case of infestations of tansy ragwort, which sheep can ingest without toxic effect, while it is highly toxic to cattle and horses. However, for many horse owners, changing the grazing animal is not an option. The approach of changing the plant community may be cost-prohibitive. Therefore, changing grazing management is most frequently the appropriate option. Most range and pasture communities contain a few toxic plants but with appropriate grazing and pasture management, these plants are not necessarily a danger to equines. Understanding grazing behavior of equines and knowledge of good forage management are the keys to providing healthy horse pastures and ranges with adequate forage.

The first factor to consider must be the overall condition of the pasture: forage of decreased or minimal nutritional quality very frequently results in some type of problem in the resident livestock, whether the outcome is lowered body condition and weight, susceptibility to toxins in plants, or other health or behavioral issues. Low quality forage significantly affects body condition, which has an impact on the toxicity of ingested plants on a grazing animal. Lowered nutritional states of animals may result in an increase of toxic effects and

decreased rates of detoxification. Therefore toxic plants are potentially more dangerous to equines in poor body condition. In addition, improved nutritional state can result in higher intake of toxic foods without negative repercussions and can also improve behaviors including forage selection. If allowed to select a varied diet, animals have a lower chance of experiencing a toxic event, because eating a variety of plants spreads the toxins they contain over several detoxification systems, and because of interactions among allelochemicals that can reduce toxicity.

Selective grazing is the first line of defense that an animal has against toxic plants. While there is a natural tendency to select diets composed of several plant species, grazing preferences often depend on the nutritional state of the animal. Normally, grazing animals avoid toxicity by limiting consumption of a toxic plant. Animals also limit toxin consumption by selecting parts of plants that contain lower concentrations of toxins. There is a subset of toxic plants that are not normally grazed, because of their extreme unpalatability due to the toxic compounds they contain.

In depleted pastures, hungry horses may eat plants they would normally avoid if provided with ample, preferred forage. If pastures contain inadequate or inferior forage, selectivity may result in stands with monocultures of less palatable and more toxic plants. When toxic plants are permitted to thrive in pastures, they may have either direct or indirect effects on the health of the grazing animals.

Direct ingestion of toxic plant matter is the predominant method by which toxins negatively affect animals. However, certain toxins also have a tendency to reduce the digestibility of other forages eaten along with the toxic plant. These compounds may tie up nutrients, kill digestive microbes or enzymes, or reduce preference. This results in reduced nutritional value of the overall diet. Thus, even if a specific compound does not generate a toxic reaction, it may indirectly cause immediate or eventual systemic or digestive damage. Low availability of nutritious forages can result in problems with toxicosis as lowered physical condition and weight of horses increases susceptibility to diseases and disorders from plant toxins.

Proper stocking rates set the amount of forage available per animal in a pasture. The total forage supply is set by factors such as soil moisture and type and forage species. The appropriate stocking rate takes into account differences between seasons, size of horses, and length of grazing season. Lack of attention to stocking rate may result in decreased forage and the increased presence of unwanted plants.

The provision of essential salt and minerals is imperative so that animals will not develop cravings that will lead to consumption of poisonous plants. Ensuring proper water quality and quantity is vital because limited water availability can lead to greater effects of toxins. Some cases of plant poisoning may be entirely avoided with an adequate water supply as a result of water's

dilutive properties and ability to facilitate movement of nutrients and minerals through the digestive system more quickly.

Weed management is necessary because heavy grazing can often results in higher populations of invasive weeds, including toxic weeds such as milkweed. Plant diversity often increases following grazing management. Pastures should be examined for undesirable plants in late summer, since frequently broadleaf weeds often remain green long after other, more nutritious, forages have been depleted. When those plants have been identified, control measures may be taken.

In Summary

With horse husbandry in mind, a prime management goal for horse owners should be to maintain a balance between The mere presence and over-abundance of toxic plants. All toxic plant populations, including those of toxic weeds, need to be monitored and then treated if their relative numbers reach a point that may endanger equine nutrition or health. With land management in mind, weed populations should be monitored, with eradication occurring when weeds reach a point that compromises the ecological integrity of the pasture or rangeland. The practice of sustainable management in horse pastures, as with all grazing lands, is a necessary goal for the health of both the land and the animals it supports. Attention must be paid to all pasture conditions: soil condition, plant communities, water, and forage availability, as well as to the

physical and nutritional condition of the equines grazing it, if poisoning by toxic plants, including weeds, is to be avoided.

In addition to the references listed below, the reader may want to refer to the website companion of this publication at http://www.cnr.uidaho.edu/range/toxicplants_horses/.

Table 3.4 List of plants mentioned in text with common and scientific names.

Common Name	Scientific Name
Alsike clover	*Trifolium hybridum*
Avocado	*Persea americana*
Bermuda grass	*Cynodon dactylon*
Big tall sagebrush	*Artemesia tridentata*
Black henbane	*Hyoscyamis niger*
Black walnut	*Juglans nigra*
Buttercups	*Ranunculus spp.*
Canada thistle	*Cirsium arvense*
Cheatgrass	*Bromus tectorum*
Chokecherry	*Prunus virginiana*
Clovers	*Trifolium spp.*
Daffodil	*Narcissus spp.*
Dandelion	*Taraxacum spp.,*
Death camas	*Zigadenus spp.*
Easter lily	*Lilium longiforum*
Elderberry	*Sambucus canadensis*
English ivy	*Hedera helix*
Field bindweed	*Convolvulus arvensis*
Foxtail barley	*Hordeum jubatum*
German millet	*Setaria italica*
Goldenrods	*Solidago spp.*
Hawthorns	*Crataegus spp.*
Horsetails	*Equisetum spp.*
Horsetails and scouring rush	*Equisetum spp.*
Houndstongue	*Cynoglossum officinale*
Indian paintbrush	*Castilleja linariaefolia*
Indigo	*Indigofera spp.*
Japanese yew	*Taxus cuspidata*
Klamath weed or St. Johnswort	*Hypericum perforatum*
Kochia	*Kochia scoparia*
Larkspur or delphinium	*Delphinium spp.*
Leafy spurge	*Euphorbia esula*
Lily of the valley	*Convallaria majalis*
Locoweeds	*Oxytropis and Astragalus spp.*
Mahogany	*Cercocarpus spp.*
Maple hybrids	*Acer spp.*
Oleander	*Nerium oleander*
Onions	*Allium spp.*
Persimmon	*Diospyros virginiana*
Pincherry	*Prunus pensylvanica*
Poison hemlock	*Conium maculatum*
Ponderosa pine	*Pinus ponderosa*
Potato	*Solanum spp.*
Puncture vine	*Tribulis terrestris*
Red maple	*Acer rubrum*
Russian knapweed	*Acroptilon repens*
Scotchbroom	*Cytisus scoparius*
Serviceberry	*Amelanchier alnifolia*
Silverleaf nightshade	*Solanum elaeagnifolium*
Sorghum	*Sorghum spp.*

Common Name	Scientific Name
Sudan grass	*Sorghum bicolor*
Tall fescue	*Festuca arundinacea*
Tomato	*Lycopersicon spp.*
White prairie aster	*Aster falcatus*
White sweet clover	*Melilotus alba*
Wild blue flax	*Linum lewisii.*
Wild iris or blue flag	*Iris missouriensis*
Wild mustard	*Sinapsis arvense*
Wild onion	*Allium spp.*
Yellow starthistle	*Centaurea solstitialis*
Yellow sweet clover	*Melilotus officinalis*
Yellow toadflax	*Linarea vulgaris*

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Chapter 4

Utility and Composition of a Website on the Effect of Toxic Plants on Equines

There are compelling reasons for using the Internet as an outreach tool to disseminate important information. According to the Stanford Institute for the Quantitative Study of Society (SIQSS), the most widespread use of the Internet today is as an information search utility (SIQSS 2008). In a recent SIQSS research study, virtually all internet users interviewed responded that they engaged in information gathering activities on the Internet. The study found that the primary or most frequent activity of 77% of users was information gathering. The study concluded that the internet today is "a giant public library."

Statistics published by the website Internet World Stats: Usage and Population Statistics show that over 71% of the U.S population, or more than 215 million people, regularly use the internet. Internet usage in the U.S. has grown at a rate of over 126 percent in the last eight years. (Internet usage information comes from data published by Nielsen//NetRatings, by the International Telecommunications Union, by local NIC, and other reliable sources.)

Livestock losses due to poisonous plants have been estimated at $340 million annually in the 17 Western states alone (Allen and Segarra 2001). Each year, 3 to 5% of the cattle, sheep, and horses in western ranges are negatively

affected by the ingestion of poisonous plants. Due to physiological, physical, and behavioral characteristics, horses and other equines may be much more susceptible to the toxins in many plants than most horse owners realize.

There is a shortage of comprehensive and concise resources on the subject of equines and toxic plants, and therefore this website was created. Its goal is to assist in educating horse owners and others in the equine fields on the prevalence of toxic plants in pastures and the effects that ingestion of or contact with them may have on equines.

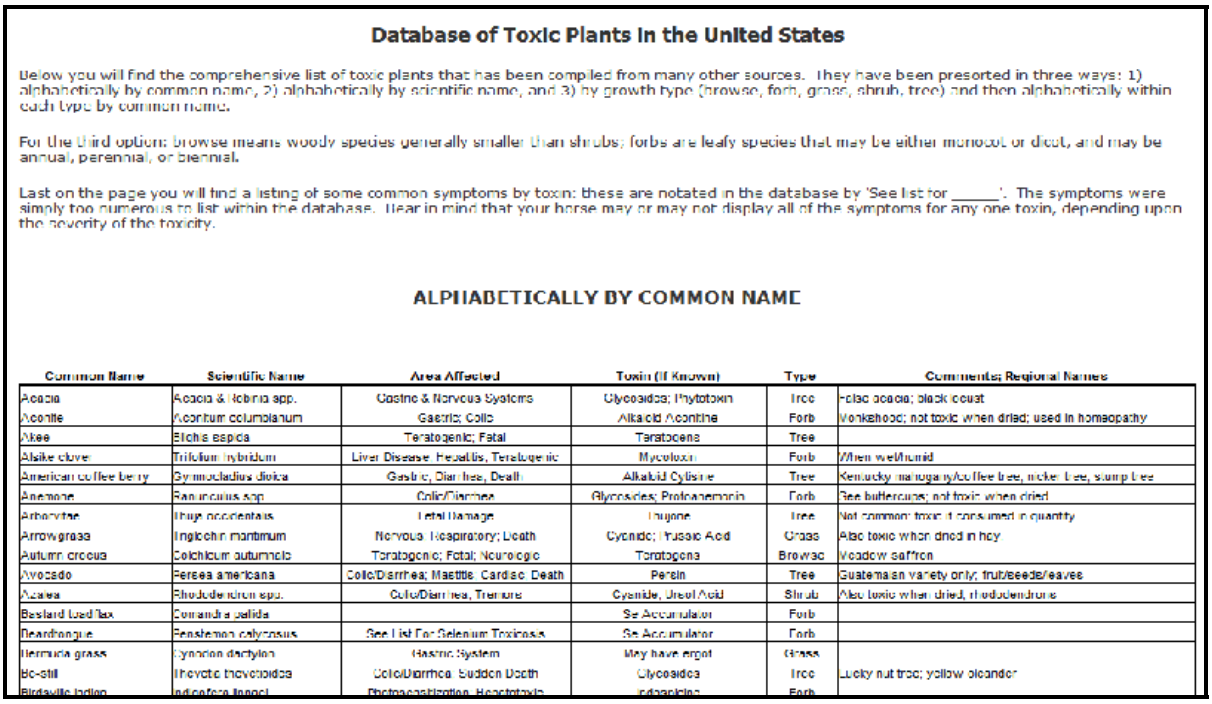
To reach the largest audience possible, the website offers a condensed yet complete version of research-based information on how toxic plants affect equines at: http://www.uidaho.edu/range/toxicplants_horses/. In this way, several audiences will be reached including researchers, extension personnel, veterinarians, land managers, and horse owners and horse professionals.

The design was created with utility and flow in mind. A FrontPage template with an attractive equine theme was purchased from DJM Web Development, Inc. (http://www.prothemes.com) and then expanded with information and specifics. The main body of the website includes six sections offering:

- an overview of toxic plants and equines;
- a database with nearly 350 toxic plants with common and scientific names, symptoms, organ or body system affected, growth type, and nicknames (Figure 4.1);

- a section on frequently asked questions (Figure 4.2);
- a problem synopsis that categorizes and summarizes the general areas related to plant poisoning of equines and has ten additional linked pages of specific information (Figure 4.3);
- a section of other resources available and published literature used in the website (Figure 4.4); and
- a short section on the author, contacts, and a link to the entire thesis on the University of Idaho – Rangeland Ecology and Management website (Figure 4.5).

Figure 4.1 A sample of information listed in a database of toxic plants presented in a webpage on horses and toxic plants: http://www.cnr.uidaho.edu/range/toxicplants_horses/Toxic Plant Database.html



Database of Toxic Plants in the United States

Below you will find the comprehensive list of toxic plants that has been compiled from many other sources. They have been presorted in three ways: 1) alphabetically by common name, 2) alphabetically by scientific name, and 3) by growth type (browse, forb, grass, shrub, tree) and then alphabetically within each type by common name.

For the third option: browse means woody species generally smaller than shrubs; forbs are leafy species that may be either monocot or dicot, and may be annual, perennial, or biennial.

Last on the page you will find a listing of some common symptoms by toxin: these are notated in the database by 'See list for _____'. The symptoms were simply too numerous to list within the database. Bear in mind that your horse may or may not display all of the symptoms for any one toxin, depending upon the severity of the toxicity.

ALPHABETICALLY BY COMMON NAME

Common Name	Scientific Name	Area Affected	Toxin (If Known)	Type	Comments; Regional Names
Acacia	Acacia & Robinia spp.	Gastric & Nervous Systems	Glycosides; Phytotoxin	Tree	False acacia; black locust
Aconite	Aconitum columbianum	Gastric; Colic	Alkaloid Aconitine	Forb	Monkshood; not toxic when dried; used in homeopathy
Akee	Blighia sapida	Teratogenic; Fatal	Teratogens	Tree	
Alsike clover	Trifolium hybridum	Liver Disease, Hepatitis, Teratogenic	Mycotoxin	Forb	When wet/mould
American coffee berry	Gymnocladus dioica	Gastric, Diarrhea, Death	Alkaloid Cytisine	Tree	Kentucky mahogany/coffee tree, nicker tree, stump tree
Anemone	Ranunculus spp.	Colic/Diarrhea	Glycosides; Protoanemonin	Forb	See buttercups; not toxic when dried
Arborvitae	Thuja occidentalis	Lethal Damage	Thujone	Tree	Not common; toxic if consumed in quantity
Arrowgrass	Triglochin maritimum	Nervous, Respiratory; Death	Cyanide; Prussic Acid	Grass	Also toxic when dried in hay
Autumn crocus	Colchicum autumnale	Teratogenic; Fetal; Neurologic	Teratogens	Browse	Meadow saffron
Avocado	Persea americana	Colic/Diarrhea; Mastitis, Cardiac Death	Persin	Tree	Guatemalan variety only; fruit/seeds/leaves
Azalea	Rhododendron spp.	Colic/Diarrhea, Tremors	Cyanide, Oxalic Acid	Shrub	Also toxic when dried, rhododendrons
Bastard toadflax	Comandra pallida		Se Accumulator	Forb	
Beardtongue	Penstemon calycosus	See List For Selenium Toxicosis	Se Accumulator	Forb	
Bermuda grass	Cynodon dactylon	Gastric System	May have ergot	Grass	
Bo-still	Thevetia thevetioides	Colic/Diarrhea, Sudden Death	Glycosides	Tree	Lucky nut tree; yellow oleander
Birdsville Indigo	Indigofera linnaei	Photosensitization, Hepatotoxic	Indospicine	Forb	

Figure 4.2 A section of the "Frequently Asked Questions" page on a website about toxic plants and horses at: http://www.cnr.uidaho.edu/range/toxicplants_horses/FAQ.html. On this web page, answers are given to six frequently asked questions with opportunities to expand to additional questions in the future.

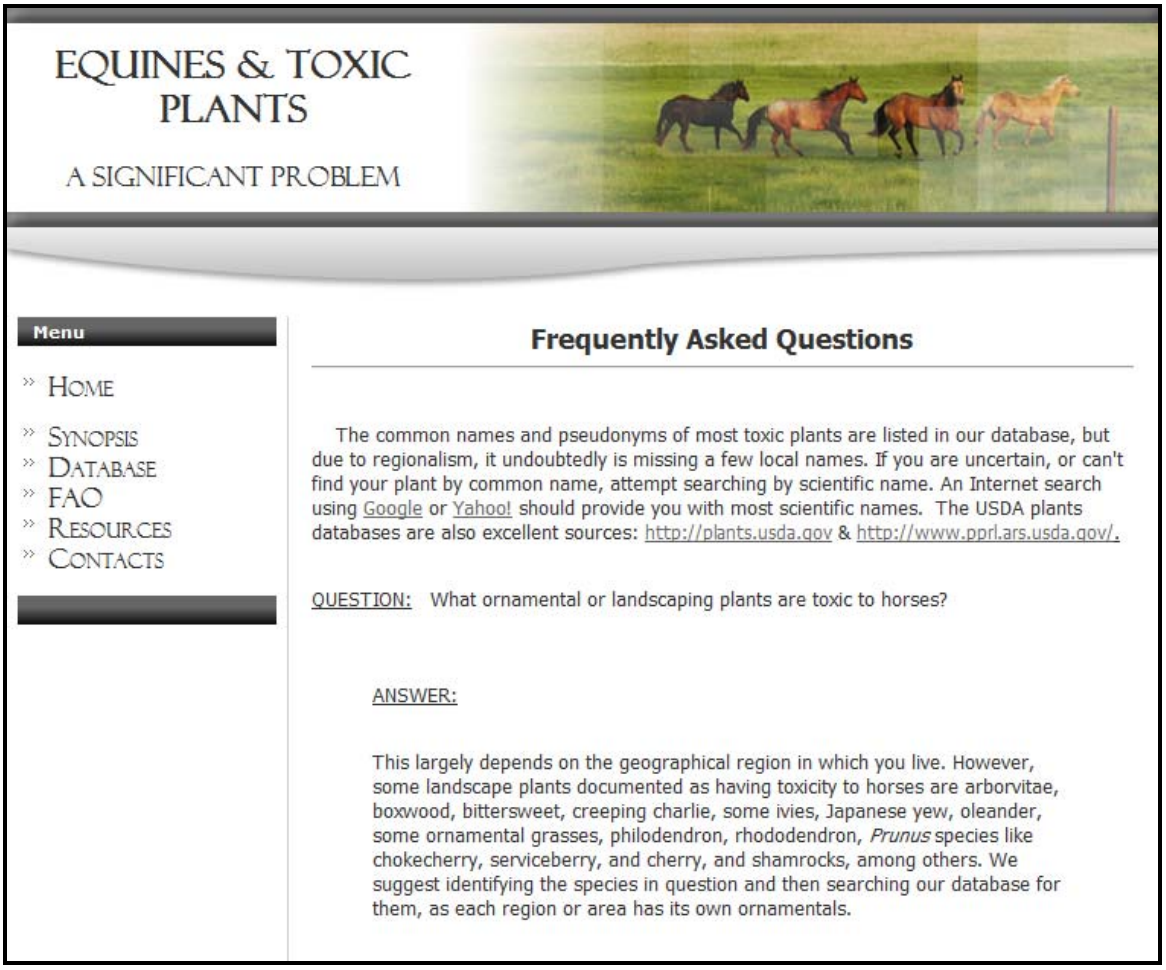


Figure 4.3 A portion of the "problem synopsis" web page on toxic plants and horses available at: http://www.cnr.uidaho.edu/range/toxicplants_horses/Problem Synopsis.html. This section of the website offers detailed information on ten important topics on the manner in which toxic plants affect horses.

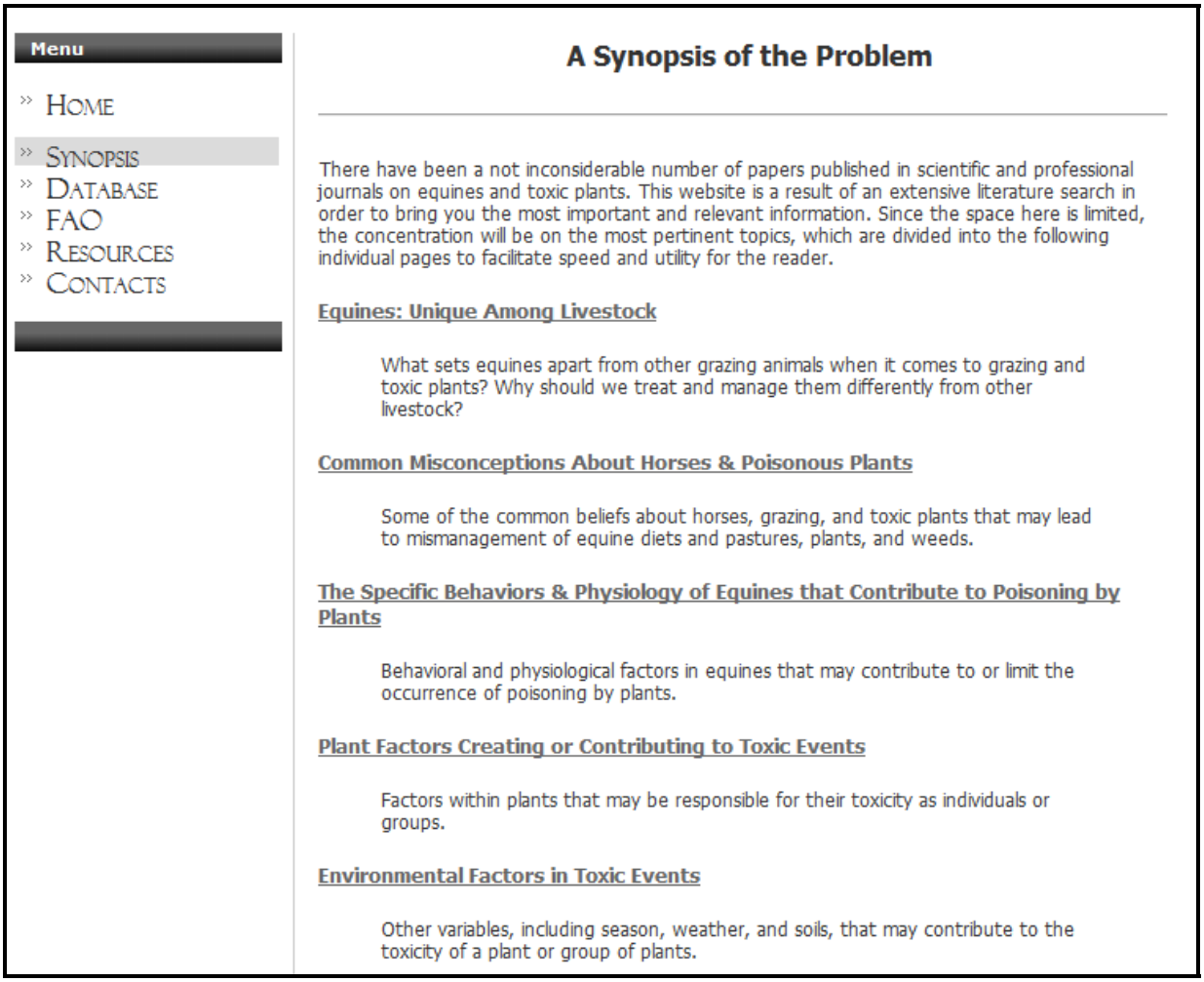


Figure 4.4. A portion of the "Resources" section of a website on equines and toxic plants available at: http://www.cnr.uidaho.edu/range/toxicplants_horses/Citations & Resources.html. This section of the web site offers links to several websites related to toxic plants, additional equine resources, and a list of published resources on topics related to horses and toxic plants.

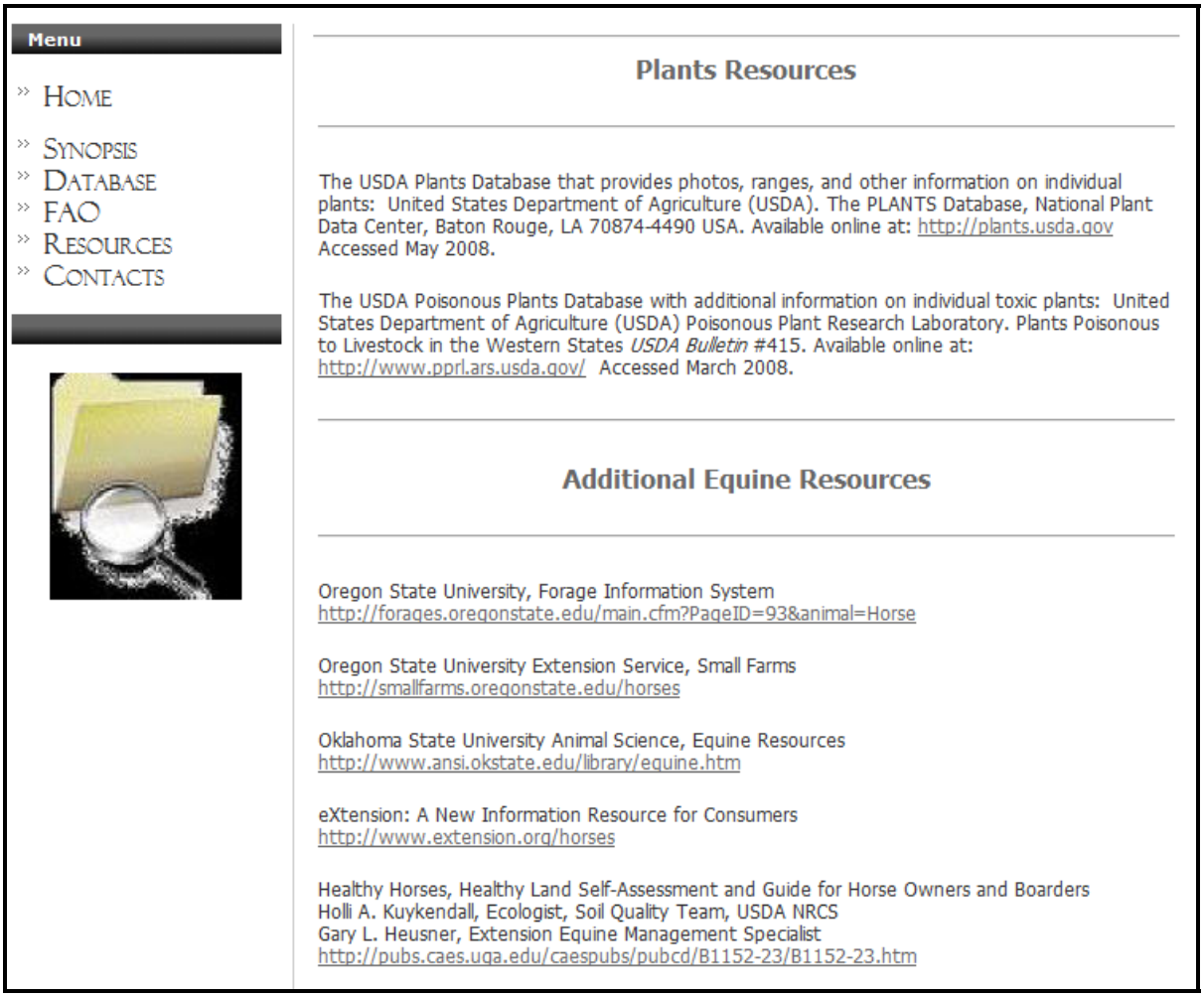
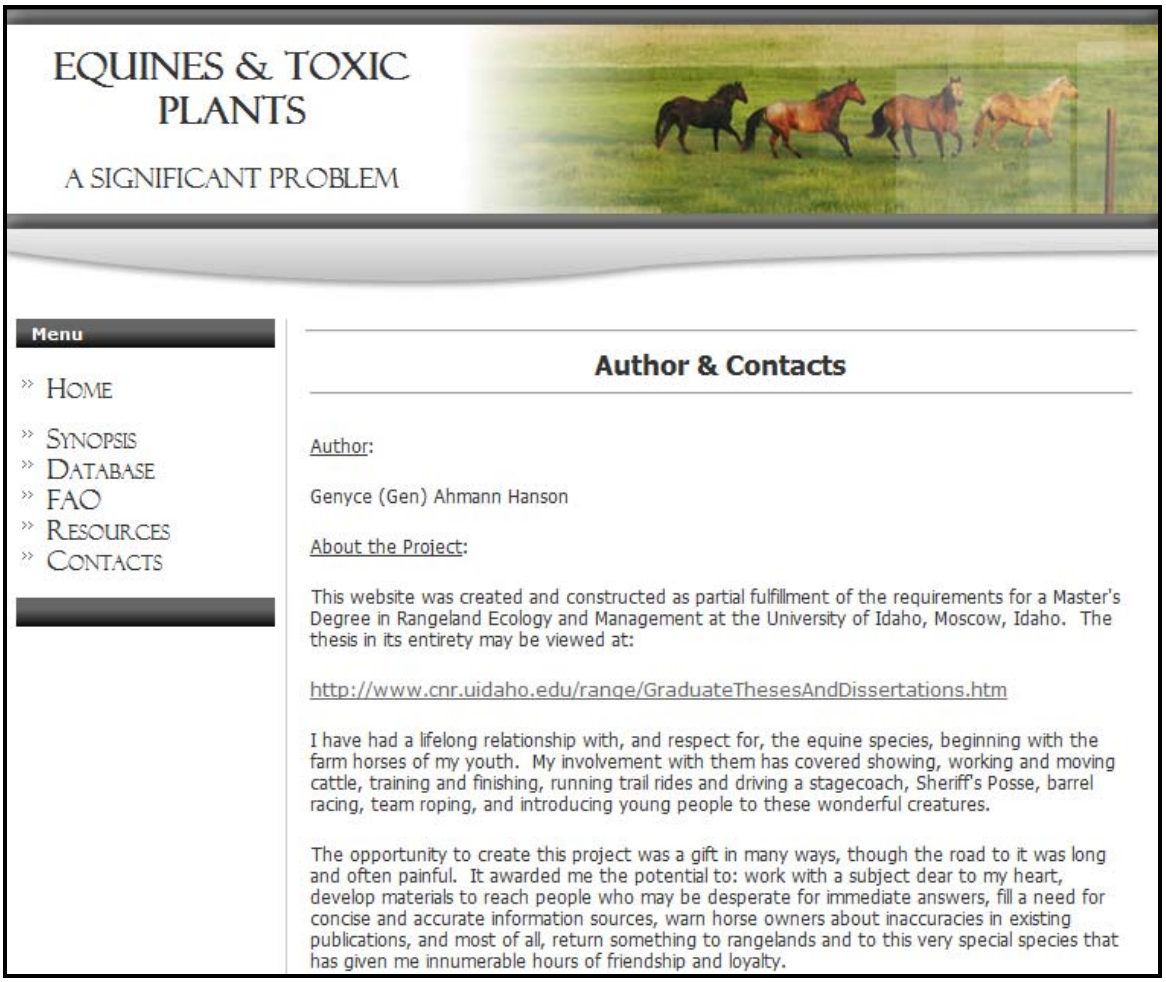


Figure 4.5 A section of the website: http://www.cnr.uidaho.edu/range/toxicplants_horses/Authors & Contacts.html provides information on the author, contacts for additional information, and link to a thesis created on the topic of toxic plants and equines.



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APPENDICES

Appendix I - Toxic Plants Database

Common Name	Scientific Name	Area Affected	Toxin (If Known)	Type	Comments; Regional Names
Acacia	*Acacia & Robinia spp.*	Gastric & Nervous Systems	Glycosides; Phytotoxin	Tree	False acacia; black locust
Aconite	*Aconitum columbianum*	Gastric; Colic	Alkaloid Aconitine	Forb	Monkshood; not toxic when dried; used in homeopathy
Akee	*Blighia sapida*	Teratogenic; Fetal	Teratogens	Tree	
Alsike clover	*Trifolium hybridum*	Liver Disease; Hepatitis; Teratogenic	Mycotoxin	Forb	When wet/humid
American coffee berry	*Gymnocladius dioica*	Gastric; Diarrhea; Death	Alkaloid Cytisine	Tree	Kentucky mahogany/coffee tree; nicker tree; stump tree
Anemone	*Ranunculus spp*	Colic/Diarrhea	Glycosides; Protoanemonin	Forb	See buttercups; not toxic when dried
Arborvitae	*Thuja occidentalis*	Fetal Damage	Thujone	Tree	Not common: toxic if consumed in quantity
Arrowgrass	*Triglochin maritimum*	Nervous; Respiratory; Death	Cyanide; Prussic Acid	Grass	Also toxic when dried in hay.
Autumn crocus	*Colchicum autumnale*	Teratogenic; Fetal; Neurologic	Teratogens	Browse	Meadow saffron
Avocado	*Persea americana*	Colic/Diarrhea; Mastitis; Cardiac; Death	Persin	Tree	Guatemalan variety only; fruit/seeds/leaves
Azalea	*Rhododendron spp.*	Colic/Diarrhea; Tremors	Cyanide; Ursol Acid	Shrub	Also toxic when dried; rhododendrons
Bastard toadflax	*Comandra pallida*	See List For Selenium Toxicosis	Se Accumulator	Forb	
Beardtongue	*Penstemon calycosus*	See List For Selenium Toxicosis	Se Accumulator	Forb	
Bermuda grass	*Cynodon dactylon*	Gastric System	May have ergot	Grass	
Be-still	*Thevetia thevetioides*	Colic/Diarrhea; Sudden Death	Glycosides	Tree	Lucky nut tree; yellow oleander
Birdsville indigo	*Indigofera linnaei*	Photosensitization; Hepatotoxic	Indospicine	Forb	
Bishop's weed	*Ammi majus*	Photodermatitis	Coumarin	Forb	Large bullwort
Bitter nightshade	*Solanum dulcamara*	Colic/Diarrhea	Alkaloids; Solanine	Forb	European bittersweet; climbing nightshade
Bitter sneezeweed	*Helenium hoopesii*	GI Irritant	Sesquiterpene Lactone	Forb	Sneezeweed
Bitterweed	*Hymenoxys odorata*	Mouth/GI Irritant	Hymenoxen	Forb	
Black henbane	*Hyoscyamus niger*	Convulsions; Coma; Death	Glycoalkaloids	Forb	
Black locust	*Robinia pseudoacacia*	Ataxia; Colic/Diarrhea; Paralysis	Lectin	Tree	Bark is most toxic
Black nightshade	*Solanum nigrum*	Colic/Diarrhea	Alkaloids; solanine	Forb	Deadly nightshade
Black walnut	*Juglans nigra*	Weakness; Laminitis; Dermatitis	Unknown	Tree	
Bladderpod	*Lesquerella gordonii*	Diarrhea; Weakness; Death	Saponins	Shrub	
Bleeding heart	*Dicentra spectabilis; formosa*	Convulsions; weakness; staggering	Alkaloids; Isoquinoline	Forb	Dutchman's breeches
Bloodroot	*Sanguinaria canadensis*	Gastric; Muscular; Respiratory	Alkaloids	Forb	
Blue mustard	*Chorispora tenella*	Deformed Foals	Glucosinolates	Forb	
Bouncing Bet	*Saponaria officinalis*	Gastric/Diarrhea; Depression	Saponin	Forb	Soapwort
Boxwood	*Buxus spp.*	Neurologic; Colic; Respiratory	Cyclobuxin (Alkaloid)	Shrub	All plant parts toxic
Bracken fern	*Pteridium aquilinum*	Thiamin Deficiency; Ataxia	Thiaminase; Filicin	Browse	Western brackenfern; all parts poisonous
Bristlegrass	*Setaria spp*	Physical; Mouth/GI; Foam/Drool	Physical	Grass	Green bristlegrass; hooked bristlegrass
Broom groundsel	*Senecio spartioides*	Hepatotoxic; Weakness	Alkaloids	Forb	
Broom snakeweed	*Gutierrezia sarothrae*	Se List; Abort/Repro	Se Accumulator; Terpenes	Shrub	Turpentine weed; snakeweed; matchweed
Broomweed	*Gutierrezia microcephala*	Abortion; See List For Se Toxicosis	Se Accumulator	Forb	
Buckeye	*Aesculus hippocastanum*	Neurologic; Weakness; Trembling	Saponin	Tree	All parts toxic; horse chestnut
Buckwheat	*Fagopyrum esculentum*	Photosensitization	Fagopyrin	Forb	
Budsage	*Artemesia spinescens*	Neurological	Possibly monoterpenes	Shrub	
Buffalo burr nightshade	*Solanum rostratum*	Colic/Diarrhea	Alkaloids; Solanine	Forb	Buffalobur
Buffalobur	*Solanum rostratum*	Colic/Diarrhea	Alkaloids; Solanine	Forb	Buffalo burr nightshade
Bull nettle	*Solanum carolinense*	Colic/Diarrhea	Alkaloids; Solanine	Forb	
Bull thistle	*Cirsium vulgare*	See List For Nitrate Toxicosis	Nitrates	Forb	
Burdock	*Arctium minus*	Mouth/GI	Physical	Forb	
Burr trefoil	*Medicago polymorpha*	Photosensitization From Liver Failure	Mimosine	Forb	
Burrow weed	*Haplopappus tenuisecta*	Neurological; Ataxia; Muscular	Tremetol	Forb	Trembles
Butter and eggs	*Linarea vulgaris*	Gastric System	Glycosides	Forb	Yellow toadflax
Buttercup	*Ranunculus spp*	Mouth/GI Irritant/Colic	Protoanemonin (Alkaloid)	Forb	All buttercups; loss of milk production

Common Name	Scientific Name	Area Affected	Toxin (If Known)	Type	Comments; Regional Names
Butterweed	*Senecio glabellus*	Hepatotoxic	Alkaloids	Forb	
Button bush	*Cephalanthus occidentalis*	Gastrointestinal	Glycosides	Shrub	
California buckeye	*Aesculus californica*	GI Irritant	Saponin; Aesculen	Tree	New growth; leaves; nuts
Canada thistle	*Cirsium arvense*	See List For Nitrate Toxicosis	Nitrates	Forb	
Carolina jessamine	*Gelsemium sempervirens*	Gastric; Weakness; Respiratory	Alkaloids	Shrub	Evening trumpetflower, false jessamine
Castor bean	*Ricinus communis*	Colic/Diarrhea; Ataxia	Ricin (Toxalbumin); Lectin	Forb	Seeds in particular; highly toxic; see rosary pea
Cat's ears	*Hypochaeris radicata*	Possible stringhalt; Neurological	Alkaloids	Forb	Flatweed, false dandelion
Cheatgrass	*Bromus tectorum*	Mouth/Digestive Tract	Physical	Grass	Downy brome
Chinaberry	*Melia azedarach*	Gastric; Cardiac; Diarrhea	Terpenes; unidentified	Tree	
Chokecherry	*Prunus virginiana*	Sudden Death	Cyanide/Glycosides	Tree	Drought & freeze increase toxicity
Christplant	*Euphorbia milii*	Skin Irritations; Mouth/GI	Latex	Shrub	Crown of thorns
Clematis	*Clematis spp.*	Colic/Diarrhea; Neurotoxic	Protoanemonin (Alkaloid)	Browse	All plant parts
Climbing bittersweet	*Celastrus scandens*	Neurological; Gastric	Solanine	Browse	Nightshades
Climbing nightshade	*Solanum dulcamara*	Colic/Diarrhea	Alkaloids; Solanine	Forb	European bittersweet; bitter nightshade
Clovers	*Trifolium spp*	Photosensitization; Hepatotoxic	Slaframine	Forb	
Cockle burrs	*Xanthium spp.*	Mouth/GI tract	Physical	Forb	Cockleburs; 2 spp; seedlings/seeds toxic
Cockleburs	*Xanthium spp.*	Mouth/GI tract	Physical	Forb	Cockle burrs; 2 spp; seedlings/seeds toxic
Cockspur	*Crataegus crus-galli*	Colic/Diarrhea	Physical impaction	Tree	Hawthorn; cockspur hawthorn; fruits toxic
Coffee weed	*Cassia occidentalis*	Lameness; Muscle Weakness	Anthraquinone	Forb	Sienna weed
Cohosh	*Actaea rubra*	Respiratory; Cardiac	Glycoside	Forb	Red baneberry
Colorado rubberweed	*Hymenoxys richardsonii*	GI Irritant; Photosensitization	Sesquiterpene	Forb	Rubberweed; pingue
Comfrey	*Symphytum officinale*	Chronic Poisoning; Liver	PA/Alkaloids	Forb	
Common groundsel	*Senecio vulgaris*	See List For PA	PA/Alkaloids	Forb	
Common vetch	*Vicia sativa*	Blindness; Convulsions	Cyanide; Neurotoxin	Forb	
Copperweed	*Oxytenia acerosa*	GI Irritant; Weakness; Coma	Possibly sesquiterpenes	Shrub	
Corn cockle	*Agrostemma githago*	Muscles; ataxia	Saponin	Forb	Seeds in particular
Corn lily	*Veratrum californicum*	Colic/Diarrhea; Teratogenic	Steroid (Alkaloids)	Forb	Western false hellebore; false hellebore; chasing green
Creeping charlie	*Glechoma hederacea*	Respiratory; Sweating	Glechomin	Forb	Ground ivy; all parts when fresh; not in stored hay
Creeping/trailing indigo	*Indigofera spicata*	Teratogenic; Ataxia; Hepatotoxic	Indospicine	Forb	Picas
Crofton weed	*Ageratina adenophora*	Ataxia; Muscular	Tremetol	Forb	Eupatorium adenophorum
Crown of thorns	*Euphorbia milii*	Skin Irritations; Mouth/GI	Latex	Shrub	Christplant
Crown vetch	*Coronella varia*	Weight loss; Abnormal behavior	Neurotoxin	Forb	
Curly dock	*Rumex crispus*	Kidney; Tremors; Coma; Death	Oxalates	Forb	
Curlycup gumweed	*Grindelia squarrosa*	See List For Selenium Toxicosis	Se Accumulator	Forb	Resinweed; gumweed
Cutleaf nightshade	*Solanum*	Colic/Diarrhea	Alkaloids/Glycosides	Forb	
Daffodil	*Narcissus spp.*	Gastric/mouth, Dermatitis	Alkaloids; Narcissine	Forb	Bulbs in particular; narcissus
Deadly nightshade	*Solanum nigrum*	Colic/Diarrhea; Collapse	Alkaloids/Solanine	Forb	Black nightshade; drought increases toxicity
Deathcamas	*Zigadenus spp*	Neurotoxin; Colic; Death	Alkaloids	Forb	15 spp deadly within days; 8-10 lbs
Delphinium	*Delphinium spp*	Paralysis; Respiratory	Aconitin (Alkaloid); Nitrates	Forb	Larkspur
Dogbane	*Apocynum cannabinum*	Cardiac; Sudden Death	Cardiac Glycosides	Forb	Hemp dogbane; Indian hemp; under 24 hours
Downy brome	*Bromus tectorum*	Mouth/Digestive Tract	Physical	Grass	Cheatgrass
Dutchman's breeches	*Dicentra cucullaria*	Trembling; Diarrhea; Convulsions	Alkaloids	Forb	Squirrel corn; bleeding heart family
Easter lily	*Lilium longiflorum*	Kidney; Gastric; Weakness	Unidentified	Forb	
Elderberry	*Sambucus canadensis*	Cardiac/Sudden Death; Ataxia	Nitrates; Glycosides	Tree	See list for nitrate toxicosis; death within minutes
English ivy	*Hedera helix*	Gastric; Respiratory; Coma; Death	Saponin	Browse	Entire plant
European bittersweet	*Solanum dulcamara*	Colic/Diarrhea	Alkaloids	Forb	Bitter nightshade; climbing nightshade
European hemlock	*Conium maculatum*	Death; Teratogenic	Teratogens; Alkaloids	Forb	Spotted/poison hemlocks; 2-3 hours
European milkvetch	*Astragalus spp*	Fetal Damage	Teratogens	Forb	
Evening trumpetflower	*Gelsemium sempervirens*	Gastric; Weakness; Respiratory	Alkaloids	Shrub	Carolina jessamine, false jessamine

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False dandelion	*Hypochaeris radicata*	Lameness	Alkaloids	Forb	Cat's ears, flatweed
False hellebore	*Veratrum californicum*	Colic/Diarrhea; Teratogenic	Steroid (Alkaloids)	Forb	Western false hellebore; corn lily; chasing green
Fetterbush	*Pieris spp.*	Colic/Diarrhea	Arbutin	Shrub	
Fiddleneck	*Amsinckia menziesii*	Hepatotoxic; Weight Loss	PA; Nitrates	Forb	Tarweed; see list for nitrate toxicosis
Field bindweed	*Convolvulus arvensis*	Colic/Diarrhea; Cardiac	Alkaloids; Nitrates	Forb	Morning glory; see list for nitrate toxicosis
Field mustard	*Brassica rapa*	Gastric/Diarrhea; Anorexia	Glucosinolates	Forb	
Fireweed	*Kochia scoparia*	Photosensitization; Hepatotoxic; Nitrate	Oxalates; Nitrates	Forb	Kochia; summer cypress
Flatweed	*Hypochaeris radicata*	Lameness	Alkaloids	Forb	Cat's ears, false dandelion
Flixweed	*Descurainia sophia*	Deformed Foals	Teratogens	Forb	Sophia, tansy mustard
Foxglove	*Digitalis purpurea*	Cardiac; Gastric; Sudden Death	Glycosides; saponins	Forb	Purple Foxglove; under 24 hours
Foxtail barley	*Hordeum jubatum*	Mouth/GI	Physical	Grass	
Freckled milkvetch	*Astragalus lentiginosus*	Neurological	Neurotoxin	Forb	
Fringed sage	*Artemisia frigida*	Neurological	Neurotoxin	Shrub	Prairie sagewort
Fumewort	*Corydalis spp.*	Convulsions; Staggers; Collapse	Alkaloids	Forb	Several species are included.
Gambel's oak	*Quercus gambelii*	Colic/Diarrhea	Tannic Acid; Gallotannins	Tree	Tannins in young leaves; bark; acorns
German millet	*Setaria italica*	Kidney; Joint; Bone		Grass	Crop/pasture grass
Golden chain tree	*Laburnum anagryroides*	GI; Neuromuscular	Alkaloids; Cytisine	Tree	Entire plant; seeds in particular
Golden oatgrass	*Trisetum flavescens*	Weakness; Weight loss; Calcinosis	Unknown	Grass	
Golden weed	*Haplopappus engelmannii*	See List For Selenium Toxicosis	Selenium	Shrub	
Goldenrods	*Solidago spp*	See List For Nitrate Toxicosis	Nitrates	Forb	
Goose grass	*Triglochin spp.*	Ataxia; Death	Cyanogenic Glycosides	Grass	Arrow/pod grasses; within minutes
Goosegrass	*Eleusine indica*	Gastric Distress	Alkaloids	Grass	Wiregrass
Greasewood	*Sarcobatus vermiculatus*	Ca Deficiency; Colic/Diarrhea	Oxalates	Shrub	
Green bristlegrass	*Setaria spp*	Mouth/Throat/Gastric	Physical	Grass	Bristlegrass; hooked bristlegrass
Green false hellebore	*Veratrum eschscholtzii*	Fetal Damage; Colic/Diarrhea	Protoanemonin; Alkaloids	Forb	Cyclops foals
Ground ivy	*Glechoma hederacea*	Respiratory; Sweating	Glechomin	Forb	Creeping charlie; all parts when fresh; not in stored hay
Groundsel	*Senecio plattensis*	Hepatotoxic; Teratogenic; Weakness	PA; Teratogens	Forb	Ragwort.
Gumweed	*Grindelia squarrosa*	See List For Selenium Toxicosis	Se Accumulator	Shrub	Curlycup gumweed; resinweed
Hairy nightshade	*Solanum sarrachoides*	Colic/Diarrhea	Alkaloids	Forb	
Halogeton	*Halogeton glomeratus*	Kidney; Colic/Diarrhea	Oxalates	Forb	
Hard maple	*Acer spp.*	Possibly Anemia	Unknown Oxidant	Tree	Possibly toxic, as with other maple spp.
Hawthorn	*Crataegus crusgalli*	Colic/Diarrhea; Impaction	Physical	Tree	Cockspur; fruits
Heliotrope	*Heliotropium arborescens*	Weight Loss; hepatotoxic	Pyrrolizidin	Forb	Seeds in particular
Hemp	*Cannabis sativa*	Neurotoxin	60 Compounds	Forb	Marijuana
Hemp dogbane	*Apocynum cannabinum*	Cardiac; Sudden Death	Cardiac/Cyanogenic Glycosides	Forb	Dogbane; Indian hemp; all parts toxic
Hoary alyssum	*Berteroa incana*	GI; Founder; Laminitis; Edema	Unknown	Forb	
Honey mesquite	*Prosopis glandulosa*	Colic/Diarrhea; Impaction	Physical	Tree	Fruits only; mesquite
Honeysuckle	*Ionicera periclymenum*	Diarrhea; Lethargy		Shrub	Plants and berries
Hooked bristlegrass	*Setaria spp*	Mouth/Throat/Gastric	Physical	Grass	Green bristlegrass; bristlegrass
Horse chestnut	*Aesculus hippocastanum*	Muscles; Ataxia	Saponin; Aesculen	Tree	All parts toxic; buckeye
Horse nettle	*Solanum carolinense*	Colic/Diarrhea; Paralysis; Death	Alkaloids	Forb	Nightshades
Horsetails	*Equisetum spp.*	Weakness; Stumbling; Arrhythmia	Aconitic Acid	Forb	Snakegrass; scouring rush; all parts toxic; B1 may help
Houndstongue	*Cynoglossum officinale*	Liver; Weight Loss	Hepatotoxin/PA	Forb	All stages of plant; liver failure
Hyacinth	*Hyacinthus orientalis*	Diarrhea; Colic	Alkaloids	Forb	Garden hyacinth; foliage may cause dermatitis
Hydrangea	*Hydrangea spp.*	Gastric/Diarrhea; Respiratory	Cyanogenic Glycosides	Shrub	Several spp; entire plant toxic
Indian hemp	*Apocynum cannabinum*	Cardiac; Sudden Death; Colic	Cardiac Glycosides	Forb	Dogbane; hemp dogbane; under 24 hours
Indian paintbrush	*Castilleja linariaefolia*	See List For Selenium Toxicosis	Se Accumulator	Forb	
Ironweed	*Sideranthus grindelioides*	See List For Selenium Toxicosis	Se Accumulator	Shrub	
Jack-in-the-Pulpit	*Arisaema triphyllum*	Lips/Mouth/Tongue; Salivation	Oxalates	Forb	Several spp.

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Jamestown weed	*Datura stramonium*	Cardiac; Coma; Death	Hyoscyamine (Alkaloid)	Forb	Stinkweed; mad apple; jimson weed; thornapple
Japanese yew	*Taxus cuspidata*	Cardiac; Sudden Death	Taxine (Alkaloid)	Shrub	All parts except berries; death within hours
Jerusalem cherry	*Solanum pseudocapsicum*	Colic/Diarrhea; See Nitrate List	Alkaloids; Nitrates	Shrub	
Jessamine, day blooming	*Cestrum diurnum*	Calcinosis; Lameness/Weakness	Hyoscine	Tree	Wild jasmine
Jessamine, false	*Gelsemium sempervirens*	Gastric; Weakness; Respiratory	Alkaloids	Shrub	
Jessamine, night blooming	*Cestrum nocturnum*	Lameness/Muscle Weakness	Atropine-like Alkaloids	Tree	
Jimmy weed	*Haplopappus heterophyllus*	Neurological; Muscle; Ataxia	Tremetol	Shrub	Southern goldenbush; trembles
Jimson weed	*Datura stramonium*	Teratogenic; Neurological; See Nitrates	Alkaloids; Nitrates	Forb	Stinkweed; mad apple; jamestown weed; thornapple
Johnsongrass	*Sorghum halepense*	Ataxia; Cardiac	Cyanogenic Glycosides	Grass	Within minutes
Kentucky coffee tree	*Gymnocladius dioica*	Gastric/Diarrhea, Respiratory	Cytosine	Tree	Kentucky mahogany, nicker, stump, American coffee tree
Kentucky mahogany tree	*Gymnocladius dioica*	Gastric/Diarrhea, Respiratory	Cytosine	Tree	Kentucky/American coffee tree; nicker tree; stump tree
Klamath weed	*Hypericum perforatum*	Skin; Photosensitize	Naphthodianthron; Hypericin	Forb	St. Johnswort; goatweed?
Kleingrass	*Panicum coloratum*	Liver Disease; Hepatitis	Mycotoxin	Grass	Wet/humid conditions; not in hay; dew poisoning
Kochia	*Kochia scoparia*	Photosensitization; Hepatotoxic; Nitrate	Oxalates; Nitrates	Forb	Fireweed, summer cypress
Lamb's tongue groundsel	*Senecio integerrimus*	Hepatotoxic	PA	Forb	
Lambsquarters	*Chenopodium album*	See List For Nitrate Toxicosis	Nitrates	Forb	
Lantana	*Lantana camara*	Liver damage; depression; neurological	Lantadene	Shrub	Several spp.; entire plant
Large bullwort	*Ammi majus*	Photodermatitis	Coumarin	Forb	Bishop's weed
Larkspur	*Delphinium spp*	Colic; Respiratory Paralysis; Death	Aconitin; Esters; Nitrates	Forb	Delphinium; poison weed; 80 spp?; young plants especially
Laurel	*Kalmia spp.*	Colic/Diarrhea; Depression; Ataxia	Alkaloids; Arbutin	Shrub	Several spp.; leaves especially toxic
Leafy spurge	*Euphorbia esula*	Photosensitivity; Colic/Diarrhea	Latex	Forb	Especially light-colored horses
Lily of the valley	*Convallaria majalis*	Sudden Death; Cardiac; Gastric	Glycosides	Forb	Under 24 hours
Littleleaf horsebrush	*Tetradymia glabrata*	Hepatotoxic	Alkaloids	Shrub	
Locoweed	*Astragalus; Oxytropis spp*	Neurological; Teratogen; Wasting	Alkaloids; Swainsonine	Forb	Early spring emergence; addictive; equines susceptible
Longspine sandbur	*Cenchrus longispinus*	Mouth/GI	Physical	Forb	Sandbur
Lucky nut tree	*Thevetia thevetiodes*	Colic/Diarrhea; Sudden Death	Glycosides	Tree	Be-still; yellow oleander
Lupine	*Lupinus spp*	Teratogenic; Fetal	Alkaloids; Slaframine	Forb	Young & bolting more toxic
Mad apple	*Datura stramonium*	Cardiac; Coma; Death	Hyoscyamine (Alkaloid)	Forb	Stinkweed; jimsonweed; Jamestown weed; thornapple
Maleberry	*Lyonia ligustrina*	Colic/Diarrhea; Ataxia; Depression	Arbutin	Shrub	
Mallows	*Malvaceae spp*	See List For Nitrate Toxicosis	Nitrates	Forb	Several spp.
Marestail	*Equisetum spp*	Neurological	Thiaminase; Neurotoxin	Forb	
Marijuana	*Cannabis sativa*	Neurotoxin	60 Compounds; THC	Forb	Hemp
Marsh marigold	*Caltha palustris*	GI Irritant; Colic/Diarrhea	Protoanemonin	Forb	
Matchweed	*Gutierrezia sarothrae*	See List For Selenium Toxicosis	Se Accumulator	Shrub	Broomweed; turpentine weed; snakeweed; broom snakeweed
Mayapple	*Podophyllum peltatum*	Gastric/Diarrhea, Respiratory; Neuro	Podophyllotoxin	Forb	
Meadow saffron	*Colchicum autumnale*	Teratogenic; Fetal	Teratogens	Forb	Autumn crocus
Medusahead rye	*Taentherum asperum*	Mouth/GI	Physical	Grass	
Mesquite	*Prosopis glandulosa*	Colic/Diarrhea; Impaction	Physical Impaction	Tree	Fruits only; honey mesquite
Milkvetch	*Astragalus spp*	Teratogenic; ataxia; neuro; weakness	Neurotoxin; Se	Forb	Hoof abnormalities; wasting disease; several spp.
Milkweeds	*Asclepias spp*	Cardiac; Sudden Death; Colic	Glycosides	Forb	Under 24 hours; several spp.
Mistletoe	*Phoradendron & Viscum spp.*	Colic, Diarrhea, Sudden Death	Glycoproteins	Shrub	Several spp.
Monkshood	*Aconitum columbianum*	See Nitrate List; Gastric; Neurological	Nitrates; Alkaloids	Forb	Aconite
Morning glory	*Convolvulus arvensis*	Colic/Diarrhea; Cardiac	Alkaloids; Nitrates	Forb	Field bindweed
Mountain fetterbush	*Pieris spp.*	Colic/Diarrhea	Acetylandromedol; Grayanotoxins	Shrub	Needles in particular
Mountain laurel	*Kalmia latifolia*	Colic/Diarrhea; Cardiac	Andromedotoxin	Shrub	
Mountain mahogany	*Cercocarpus spp.*	Cardiac	Cyanide	Tree	
Narcissus	*NarcIssus spp.*	Gastric/mouth, Dermatitis	Alkaloids; Narcissine	Forb	Daffodils
Needlegrass	*Stipa spp*	Mouth/Gastric System	Physical	Grass	
Nicker tree	*Gymnocladius dioica*	Gastric/Diarrhea, Respiratory	Cytosine	Tree	Kentucky coffee/mahogany, American coffee, stump trees

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Oak	*Quercus breviloba*	Colic/Diarrhea	Tannic Acid/Gallotannins	Tree	Young leaves; bark; acorns; seasonal; picas
Ohio buckeye	*Aesculus glabra*	GI Irritant, Hemolysis	Aesculin	Tree	New growth; leaves; nuts
Oleander	*Nerium oleander*	Sudden Cardiac Death; Colic/Diarrhea	Cardiac Glycosides	Shrub	Highly toxic
Onion, domestic	*Allium spp*	Anemia; Weakness	Alcylsulfides; Disulfides	Forb	In large quantities
Perennial ryegrass	*Lolium perenne*	Skin; Photosensitize	Photosensitizing Agent	Grass	
Periwinkle	*Vinca rosea*	Teratogenic	Teratogens	Shrub	
Persimmon	*Diospyros virginiana*	Colic/Diarrhea; Impaction	Physical	Tree	Fruits only
Philodendron	*Philodendron spp*	Mouth, GI Tract, Respiratory, Death	Oxalates	Browse	
Pigweed	*Amaranthus retroflexus*	See Lists for Nitrate Toxicosis	Nitrates; Oxalates	Forb	Redroot pigweed; drought increases toxicity; oxalate list?
Pincherry	*Prunus pensylvanica*	Sudden Death	Cyanogenic Glycosides	Tree	Drought increases toxicity
Pingue	*Hymenoxys richardsonii*	GI Irritant; Photosensitization	Sesquiterpene	Forb	Colorado rubberweed; rubberweed; bitterweed
Poinsettia	*Euphorbia pulcherrima*	Gastric; Dermatitis; See Nitrate List	Latex, Euphorbin, Nitrates	Shrub	
Poison hemlock	*Conium maculatum*	Death; Teratogenic	Alkaloids	Forb	All parts toxic; not in hay; chasing green in spring; 2-3 hours
Poison ivy	*Toxicodendron radicans*	Gastrointestinal, Dermatitis	Urushiol, Catechols	Forb	
Pokeweed	*Phytolacca americana*	Colic/Diarrhea	Phytolaccatoxin	Forb	Entire plant toxic
Ponderosa pine	*Pinus ponderosa*	Abortive	Isocuppressic acid	Tree	
Poppies	*Papaver spp.*	Teratogenic	Teratogens	Forb	
Porcupine grass	*Miscanthus sinensis*	Mouth/GI tract	Physical	Grass	
Potato	*Solanum tuberosum*	Colic/Diarrhea; Weakness	Alkaloids; Solanine	Forb	
Prairie sagewort	*Artemisia frigida*	Neurological	Neurotoxin	Shrub	Fringed sage
Prairie three-awn	*Aristida oligantha*	Mouth/GI	Physical	Grass	
Prickly pear	*Opuntia spp.*	Mouth/GI	Physical	Shrub	
Prince's plume	*Stanleya pinnata*	See List For Selenium Toxicosis	Selenium	Shrub	
Privets	*Ligustrum vulgare*	Colic/Diarrhea	Glycosides; Iridoid	Shrub	Entire plant
Puncture vine	*Tribulus terrestris*	Mouth/GI tract	Physical	Forb	
Purple foxglove	*Digitalis purpurea*	Cardiac; Sudden Death	Cyanogenic Glycosides	Forb	Foxglove
Purple locoweed	*Oxytropis lambertii*	Neurological; Teratogen; Wasting	Alkaloids; Swainsonine	Forb	Early spring emergence; addictive; equines susceptible
Purple mint	*Perilla frutescens*	Respiratory	Ketones	Forb	
Ragweed	*Ambrosia artemesiafolia*	See List For Nitrate Toxicosis	Nitrates	Forb	
Rattlebox	*Crotalaria spp*	Weight loss; hepatotoxic; chronic	PA	Shrub	Rattlepod
Rattlepod	*Crotalaria spp*	Weight loss; hepatotoxic; chronic	PA	Shrub	Rattlebox
Red baneberry	*Actaea rubra*	Respiratory; Cardiac	Glycoside	Forb	Cohosh
Red buckeye	*Aesculus pavia*	GI Irritant; Muscle Weakness; Coma	Glycosides	Tree	New growth; leaves; nuts
Red clover	*Trifolium pratense*	Skin; Liver	Mycotoxin	Forb	
Red maple	*Acer rubrum*	Anemia; Arrythmia; Weakness	Unknown Oxidant	Tree	Red X silver hybrids may be toxic; small amounts; dried only
Redroot pigweed	*Amaranthus spp*	See Lists for Nitrate Toxicosis	Nitrates; Oxalates	Forb	Pigweed; drought increases toxicity; oxalate list?
Reed canarygrass	*Phalaris arundinacea*	Muscular Weakness; Collapse	Alkaloids	Grass	
Resinweed	*Grindelia squarrosa*	See List For Selenium Toxicosis	Selenium	Shrub	Curlycup gumweed
Rhododendron	*Rhododendron spp.*	Colic/Diarrhea; Tremors; Respiratory	Cyanide; Ursol Acid	Shrub	Also toxic when dried; azalea.
Rhubarb	*Rheum rhaponticum*	Gastrointestinal; Cardiovascular	Oxalates	Forb	Leaves
Richweed	*Ageratina altissima*	Muscular; Ataxia; Swallowing	Tremetol	Forb	White snakeroot
Riddell's ragwort	*Senecio riddellii*	Hepatotoxic; Weakness; Chronic	PA	Forb	
Rock maple	*Acer spp.*	Possibly Anemia	Unknown Oxidant	Tree	Possibly toxic, as with other maple spp.
Rosary pea	*Abrus precatorius*	Colic/Diarrhea; Ataxia	Lectin	Browse	See castor bean
Rubberweed	*Hymenoxys richardsonii*	GI Irritant; Photosensitization	Sesquiterpene	Forb	Colorado rubberweed; pingue
Russian knapweed	*Acroptilon repens*	Chewing Disease	Sesquiterpene; Another Unknown	Forb	Same toxin as starthistle; no cure/recovery
Russian thistle	*Salsola iberica*	See List For Nitrate Toxicosis	Nitrates	Forb	
Sagebrush	*Artemesia spp*	Neurological	Monoterpenes	Forb	
Saltbush	*Atriplex spp*	See List For Selenium Toxicosis	Selenium	Forb	
Sand sage	*Artemesia filifolia*	Neurological; Ataxia	Volatile Oils	Forb	Sage sickness
Sandbur	*Cenchrus longispinus*	Mouth/GI	Physical	Forb	Longspine sandbur

Common Name	Scientific Name	Area Affected	Toxin (If Known)	Type	Comments; Regional Names
Scarlet pimpernel	*Anagallis arvensis*	Mouth; Staggering; Ataxia	Terpenoid; Glycoside	Forb	
Scotchbroom	*Cytisus scoparius*	Gastrointestinal; Hepatotoxic	Alkaloids	Shrub	Entire plant
Scouring rush	*Equisetum arvense*	Neurological; Ataxia; Blindness	Alkaloids; Thiaminase	Forb	Horsetails; snakegrass; all parts toxic; B1 may help
Sensitive fern	*Onoclea sensibilis*	Neurological; Ataxia	Thiaminase	Forb	
Service berry	*Amelanchier alnifolia*	Death; Ataxia; Heart	Cyanogenic Glycosides	Tree	Death within minutes is possible
Shamrock	*Oxalis spp*	KIdney; Diarrhea/Colic	Oxalates	Forb	
Shepherd's purse	*Capsella bursa-pastoris*	Teratogenic	Teratogens	Forb	Deformed foals
Shinnery oak	*Quercus havardii*	Colic/Diarrhea	Tannic Acid	Tree	Shin oak; tannins in young leaves; bark; acorns
Sienna weed	*Cassia occidentalis*	Colic/Diarrhea; Ataxia; Weakness	Anthraquinone	Forb	Coffee weed
Silver maple	*Acer spp.*	Possibly Anemia	Unknown Oxidant	Tree	Possibly toxic, as with other maple spp.
Silverleaf nightshade	*Solanum elaeagnifolium*	Colic/Diarrhea	Alkaloids; Solanine	Forb	
Skunk cabbage	*Symplocarpus foetidus*	Cardiovascular; Shock	Alkaloids	Forb	
Smartweed	*Polygonum spp*	See List For Nitrate Toxicosis	Nitrates; Saponin	Forb	Entire plant
Snakegrass	*Equisetum arvense; spp*	Ataxia; Neurological; Blindness	Alkaloids; Thiaminase	Forb	Horsetails; scouring rush; all parts toxic; B1 may help
Snakeweed	*Gutierrezia sarothrae*	See List For Selenium Toxicosis	Selenium	Shrub	Turpentine weed; broom snakeweed; matchweed
Sneezeweed	*Helenium hoopesii*	GI Irritant	Sesquiterpene Lactone	Forb	Bitter sneezeweed
Snow on the mountain	*Euphorbia marginata*	Mouth/GI; Diarrhea	Esters	Forb	
Soapwort	*Saponaria officinalis*	Gastric/Diarrhea; Depression	Saponin	Forb	Bouncing Bet
Soft maple	*Acer spp.*	Possibly Anemia	Unknown Oxidant	Tree	Possibly toxic, as with other maple spp.
Sophia	*Descurainia sophia*	Deformed Foals	Teratogens	Forb	Flixweed, tansy mustard
Sorghum	*Sorghum spp.*	Cardiac; See List For Nitrate Toxicosis	Cyanogenic Glycosides; Nitrates	Grass	20 spp.; when stressed/drought; sudan grass
Sorrel	*Oxalis & Rumes spp.*	Kidney; Colic/Diarrhea	Oxalates	Forb	
Soursob	*Oxalis spp*	Kidney; Colic/Diarrhea	Oxalates	Forb	
Southern goldenbush	*Haplopappus heterophyllus*	Neurological; Muscle; Ataxia	Tremetol	Shrub	Jimmy weed; trembles
Spineless horsebrush	*Tetradymia canescens*	Hepatotoxic	Alkaloids	Shrub	
Spiny cocklebur	*Xanthium spinosum*	Mouth/GI, Hepatotoxic	Glycosides; Physical	Forb	
Spotted cowbane	*Pastinaca sativa*	Colic/Diarrhea; Photodermatitis	Coumarin	Forb	Wild parsnip
Spotted locoweed	*Astragalus spp*	Teratogenic; Neurological; Death	Swainsonine	Forb	
Spring parsley	*Cymopterus watsonii*	Skin; Photosensitize	Coumarin	Forb	Photodermatitis
Squirrel corn	*Dicentra cucullaria*	Trembling; Diarrhea; Convulsions	Alkaloids	Forb	Dutchman's breeches; bleeding heart family
Squirreltail	*Sitanion hystrix*	Mouth/GI	Physical	Grass	Elymus elymoides
St. Johnswort	*Hypericum perforatum*	Skin; Photosensitize	Naphthodianthron; Hypericin	Forb	Klamath weed; entire skin loss
Staggergrass	*Amanthium muscaetoxicum*	Respiratory; Weakness; Death	Alkaloids	Grass	
Star of Bethlehem	*Ornithogalum umbellatum*	Gastric/Diarrhea; Depression	Saponin	Forb	Entire plant
Stickseed	*Hackelia & Trichodesma spp.*	Weight Loss; Hepatotoxic; Chronic	PA	Forb	
Stinging nettle	*Urtica dioica*	Mouth/GI; See List For Nitrate Toxicosis	Physical; Nitrates	Forb	
Stinkweed	*Datura stramonium*	Teratogenic; Cardiac; Death	Hyoscyamine (Alkaloid)	Forb	Jimsonweed; mad apple; jamestown weed; thornapple
Stump tree	*Gymnocladius dioica*	Gastric/Diarrhea, Respiratory	Cytosine	Tree	Kentucky mahogany/coffee, American coffee, nicker trees
Sudan grass	*Sorghum bicolor*	Death; Ataxia; Teratogenic; Paralysis	Cyanogenic Glycosides	Grass	Drought & freeze increase toxicity
Sugar maple	*Acer spp.*	Possibly Anemia	Unknown Oxidant	Tree	Possibly toxic, as with other maple spp.
Summer cypress	*Kochia scoparia*	Photosensitization; Hepatotoxic; Nitrate	Oxalates; Nitrates	Forb	Fireweed; kochia
Summer pheasant's eye	*Adonis aestivalis*	Cardiac; Colic	Adonidine	Forb	
Sweet bubby	*Calycanthus floridus*	Tetany; Convulsions; Neurological	Calycanthin; Alkaloids	Shrub	Sweet shrub
Sweetshrub	*Calycanthus floridus*	Tetany; Convulsions; Neurological	Calycanthin; Alkaloids	Shrub	Sweet bubby
Tall big sagebrush	*Artemisia tridentata*	Neurologic	Volatile Oils	Shrub	
Tall fescue	*Festuca arundinacea*	Abortion; Malformed Fetus	Endophytic Fungus; Alkaloids	Grass	
Tanglehead	*Heteropogon contortus*	Mouth/GI	Physical	Grass	
Tansy mustard	*Descurainia sophia*	Deformed Foals	Teratogens	Forb	Flixweed, sophia

Common Name	Scientific Name	Area Affected	Toxin (If Known)	Type	Comments; Regional Names
Tansy ragwort	*Senecio jacobaea*	Hepatotoxic; Neurological	PA	Forb	Maintains toxicity in hay; equines highly susceptible
Tarweed	*Amsinckia intermedia*	Hepatotoxic; Weight Loss	PA	Forb	Fiddleneck
Thornapple	*Datura stramonium*	Teratogenic	Hyoscyamine (Alkaloid)	Forb	Jimson weed; Jamestown weed; stinkweed; mad apple
Threadleaf groundsel	*Senecio douglasii*	Hepatotoxic; Weakness	PA	Shrub	Woolly groundsel
Three-awn grasses	*Aristida spp.*	Mouth/GI	Physical	Grass	
Tobacco	*Nicotiana glauca*	Teratogenic	Teratogens	Forb	
Tomato	*Lycopersicon spp.*	Colic/Diarrhea; Weakness	Hyoscine	Forb	
Tulip	*Tulipia spp*	Dermatitis; Weakness; Respiratory	Tuliposide	Forb	
Tumble mustard	*Sysymbrium altissimum*	Teratogenic	Teratogens	Forb	
Turpentine weed	*Gutierrezia sarothrae*	See List For Selenium Toxicosis	Selenium	Shrub	Broom snakeweed; snakeweed; matchweed
Two-grooved milkvetch	*Astragalus bisculatus*	See List For Selenium Toxicosis	Selenium	Forb	Freeze
Viper's bugloss	*Echium vulgare*	Liver Fibrosis/Failure	PA	Forb	Seeds in particular
Water hemlock	*Cicuta douglasii*	Tremors; Convulsions; Death	Cicutoxin	Forb	Western water hemlock; single mouthful; death in minutes
Western brackenfern	*Pteridium aquilinum*	Thiamin Deficiency	Thiaminase; Filicin	Browse	Bracken fern; all parts poisonous
Western chokecherry	*Prunus spp*	Sudden Death	Cyanogenic Glycosides	Tree	
Western false hellebore	*Veratrum californicum*	Fetal Damage	Steroid (Alkaloids)	Forb	False hellebore; corn lily
Western water hemlock	*Cicuta douglasii*	Tremors; Convulsions/ Death	Cicutoxin	Forb	Water hemlock; single mouthful; death in minutes
White bryony	*Bryonia cretica*	Sweating; Diuresis; Convulsions	Lectine; Polyhydroxic Acids	Browse	Berries; sap; roots; worse when dried
White locoweed	*Oxytropis sericea*	Neurological; Teratogen; Wasting	Alkaloids; Swainsonine	Forb	Early spring emergence; addictive; equines susceptible
White maple	*Acer spp.*	Possibly Anemia	Unknown Oxidant	Tree	
White prairie aster	*Aster falcatus*	See List For Selenium Toxicosis	Selenium	Forb	
White snakeroot	*Ageratina altissima*	Muscular; Ataxia; Swallowing	Tremetol	Forb	Richweed
White sweet clover	*Melilotus alba*	Anemia; Hemorrhage; Weakness	Prussic Acid; Dicoumarol	Forb	When spoiled; in haylage
Wild blue flax	*Linum lewisii*	Ataxia; Cardiac	Glycosides	Forb	Death may occur within minutes
Wild cherry	*Prunus serotina*	Sudden Death	Cyanogenic Glycosides	Tree	
Wild iris	*Iris missouriensis*	GI Irritant; Diarrhea; Dermatitis	Irisine	Forb	New growth; leaves; nuts
Wild jasmine	*Cestrum diurnum*	Calcinosis; Lameness/Weakness	Hyoscine	Tree	Jessamines
Wild mustard	*Sinapis arvensis*	Salivation; Collapse; Death	Glucosinolates	Forb	
Wild oat grass	*Avena fatua*	See List For Nitrate Toxicosis	Nitrates	Grass	Wild oats
Wild onion	*Allium spp*	Anemia; Weakness	Alcylsulfides; Disulfides	Forb	
Wild parsnip	*Pastinaca sativa*	Colic/Diarrhea; Photodermatitis	Coumarin	Forb	Spotted cowbane
Wild pea	*Lathyrus latifolius*	Teratogenic	Teratogens	Browse	
Wild tree tobacco	*Nicotiana glauca*	Fetal Damage	Teratogens	Tree	
Wiregrass	*Eleusine indica*	Gastric Distress	Alkaloids	Grass	Goosegrass
Woody aster	*Xylorhiza glabriscula*	See List For Selenium Toxicosis	Selenium	Shrub	
Woolly groundsel	*Senecio douglasii*	Hepatotoxic; Weakness; Chronic	PA	Shrub	Threadleaf groundsel
Woolly locoweed	*Astragalus mollismus*	Neurological; Teratogen; Wasting	Alkaloids; Swainsonine	Forb	Early spring emergence; addictive; equines susceptible
Yellow bristlegrass	*Setaria pumila*	Mouth/GI; Foam/Drool	Physical	Grass	
Yellow buckeye	*Aesculus octandra*	GI Irritant; Hemolysis	Aesculin	Tree	
Yellow oleander	*Thevetia peruviana*	Colic/Diarrhea; Sudden Death	Glycosides	Shrub	Be-still; lucky nut tree
Yellow starthistle	*Centaurea solstitialis*	Chewing Disease	Alkaloids; Sesquiterpenes	Forb	Russian knapweed; no recovery; addictive
Yellow sweet clover	*Melilotus officinalis*	Anemia; Hemorrhage; Weakness	Dicoumarol	Forb	Only when spoiled and in haylage
Yellow toadflax	*Linarea vulgaris*	Gastric System	Glycosides	Forb	Butter and eggs
Yew	*Taxus spp*	Sudden Death; Neurological; Cardiac	Taxin (Alkaloid); Cyanogens	Shrub	All parts except berries; single mouthful deadly; within hours

Table II - Compounds and Symptoms

Nitrates

Oxygen starvation
Weakness
Unsteady gait
Collapse
Coma
Death

PA (Pyrrolizidine alkaloids)

Weight loss
Anemia
Neurological signs
Photosensitization
Chronic liver damage
Death in weeks to months
Chronic poisoning

Selenium (In General)

Unsteady gait
Respiratory difficulty
Weight loss
Paralysis
Lameness
Deformed hooves

Se - Acute

Often death before signs/symptoms, due to organ damage
Respiratory failure
Organ failure

Se - Chronic

Loss of appetite and weight
Loss of cardiac function
Trouble with bones/joints: possibly similar to arthritis symptoms
Cirrhosis of the liver
Possible anemia

Blind staggers (rarely in equines): circling, stumbling, loss of appetite, blindness

Alkali disease: hoof deformation, hair loss, laminitis, lameness, cirrhosis, emaciation, roached mane, bob-tail appearance, stiff-leggedness