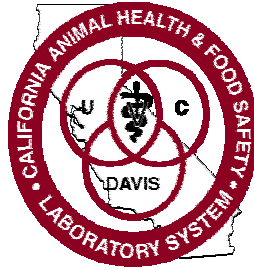


Poisonous Plant Threats to Cattle and Horses: Tansy Ragwort, Common Groundsel and Fiddleneck



Summary

Tansy ragwort, common groundsel and fiddleneck, weeds commonly found in California, are extremely toxic to sensitive species such as cattle and horses due to their content of toxins called pyrrolizidine alkaloids (PAs) and can cause significant economic loss to cattle producers and horse owners. Repeated exposure to PA-containing plants in hay, alfalfa pellets or silage results in chronic liver failure, poor-doing and non-productive animals, and, eventually, death. Clinical signs often do not occur until many months after cessation of exposure to PAs and diagnosis of poisoning can be difficult. There is no effective treatment for affected animals, so preventing exposure is essential. Based upon the insidious and cumulative nature of the poisoning, there is no safe consumption level. Due to the potential for PAs to contaminate milk, any forage contaminated with PA-containing plants should not be fed to lactating dairy cattle.

Frequently Asked Questions

What are pyrrolizidine alkaloids?

Pyrrolizidine alkaloids (PAs) are potent liver toxins that have been identified in over 6000 plants worldwide. Most PAs occur in 3 different plant families: the Boraginaceae, (examples include *Amsinckia menziesii* var. *intermedia* or common fiddleneck and *Cynoglossum officinalis* or hound's tongue), the Compositae (primarily *Senecio* species included *S. jacobaea* or tansy ragwort and *S. vulgaris* or common groundsel) and the Leguminosae (especially *Crotalaria* spp. or "rattlebox").

What PA-containing plants are of most concern in California?

The most commonly found PA-containing plants in California associated with poisoning are fiddleneck, tansy ragwort and common groundsel (see below). The PA content of these plants ranges from less than 0.5% up to 1.2% dry weight. Plant parts ranked in decreasing concentration of PA's are: flowers and seeds > leaves > stems > roots. PA's are present at all stages of growth. While there is some degradation of PAs in silage, the PA content of hay remains constant over many months. Also, PAs are stable to high temperatures.

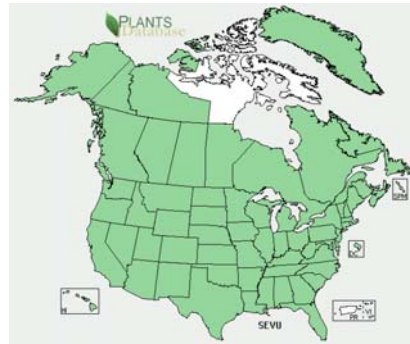
Most PA-containing plants are not considered to be highly palatable when fresh, but incorporation into hay, pellets or silage decreases the ability of an animal to selectively avoid them. Thus, most cases of PA intoxication documented by the California Animal Health and Food Safety Laboratory System have involved ingestion of one of the aforementioned types of feed.

Plant Appearance and Distribution

Senecio vulgaris or common groundsel



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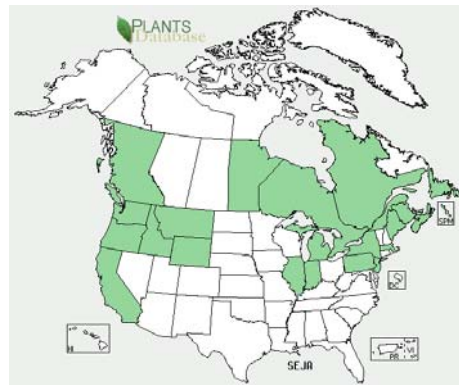


USDA United States Department of Agriculture
Natural Resources Conservation Service

Senecio jacobaea or tansy ragwort



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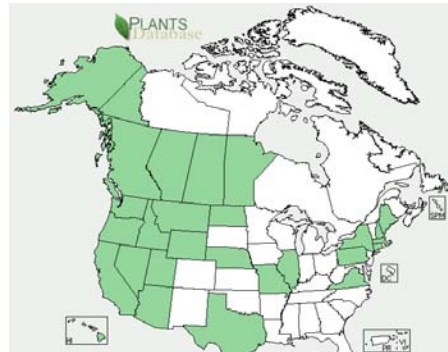


USDA United States Department of Agriculture
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Amsinckia menziesii var. *intermedia* or common fiddleneck



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What species of livestock are affected by PAs?

Pigs and chickens are considered to be the most sensitive livestock species; cattle and horses, while still considered to be sensitive species, are less sensitive. Sheep and goats are the least sensitive livestock species due to detoxification of PAs by rumen microbes and the liver. Under some conditions, sheep can consume up to 20 times the dose of some PA-containing plants known to kill cattle. Although sheep and goats have been used to control stands of PA-containing plants, the long term consequences to their health are not well known. Young animals of all species are more sensitive than adult animals.

How do PAs damage the liver?

PAs themselves are not toxic but become toxic following bioactivation or conversion in the liver to the toxic form known as pyrroles. In tissues, pyrroles are highly reactive and bind to adjacent nucleic acids or proteins. The resulting pyrrole-tissue complexes (called adducts) result in loss of liver cell function. Chronic damage to the liver results in significant scarring (termed fibrosis) and ultimately liver failure.

What are the clinical signs associated with poisoning?

While ingestion of very high concentrations of PAs can result in rapid and significant liver damage, the more typical problem in livestock is exposure to lower concentrations of PAs over time which results in chronic and insidious liver damage. Typically, clinical signs do not become apparent until months after an animal has begun eating a PA-containing plant and, in many cases, up to a year after exposure ceases.

In horses, the onset of clinical signs is often sudden and is associated with a build-up of waste products normally eliminated by the liver. Signs are typically neurologic and

include head pressing, aimless walking or pacing, persistent chewing, yawning, drowsiness, rectal straining and incoordination. Other signs include fluid build-up in the abdomen, diarrhea or constipation. In cattle, early clinical signs associated with poisoning are generally more subtle and often include loss of appetite, decreased milk production, weight loss, rectal straining and weakness. Nervous system signs similar to those reported in horses can occur prior to death. Affected animals are often noted to “do poorly”. Pregnancy, lactation, transport, poor nutrition or other forms of stress can precipitate onset of disease-related signs. Unfortunately, animals that “recover” often have residual effects such as exercise intolerance or a permanent decrease in productivity.

How is PA-poisoning diagnosed?

Identification of PA-containing weeds and detection of PAs in forage are important for preventing exposure or establishing a diagnosis of poisoning. However, due to the prolonged delay in onset of clinical signs the affected forage that was eaten weeks or months before may no longer be available for evaluation. Unfortunately, there is no good way to detect PAs or their breakdown products in tissues. However, liver damage generally results in a characteristic appearance to the liver when examined microscopically. Thus, when there is concern about PA poisoning, it is critical that a postmortem examination be conducted on any dead animal. A liver biopsy can be obtained from live animals to demonstrate the presence of characteristic liver damage.

Can affected animals be treated?

Although a number of treatments have been tried, to date, none are effective once an animal develops clinical signs. After the onset of clinical signs, the prognosis is poor. Thus, prevention is the best approach.

Is there a “safe” level of PAs that can be fed to sensitive species such as horses and cattle?

Safe PA concentrations have not been determined for sensitive species such as cattle and horses. Little is known about what levels and durations of exposure are damaging or what the long term effects of “sub-clinical” exposures (exposures not causing obvious clinical signs) are on long term growth and productivity. Because of the variation in PA content of plants and in animal sensitivity, almost any daily exposure must be considered a risk to health if continued for a sufficient period of time.

Is there a potential for PA residues in the milk of lactating animals?

Low-level exposure may occur in people through the presence of PAs in foods, such as honey and milk, but no reports of human poisoning caused through ingestion of these foods are available. Even at levels of exposure that cause clinical signs in animals, resulting concentrations of PAs in milk are relatively low (reportedly 1 ppm or less). However, in view of the established ability of some PAs to cause cancer in rats, human

exposure to PAs should be minimized. Thus, any PA-contaminated forage should not be fed to lactating dairy cattle or goats.

Additional Information:

Livestock Poisoning with Pyrrolizidine Alkaloid-Containing Plants (Senecio, Crotalaria, Cynoglossum, Amsinckia, Heliotropium, and Echium spp.) by Bryan Stegelmeier, Dale R. Gardner and T. Zane Davis, <http://www.bioone.org/doi/pdf/10.2111/1551-501X-31.1.35>.

IPCS International Programme on Chemical Safety, Health and Safety Guide No. 26, Pyrrolizidine Alkaloids Health and Safety Guide, <http://www.inchem.org/documents/hsg/hsg/hsg026.htm#SectionNumber:4.1>.

Pyrrolizidine Alkaloid Toxicity by John R. Elliott, Perry J. Bain, and Kenneth S. Latimer, <http://www.vet.uga.edu/VPP/clerk/elliott/index.php>.

University of California, Cooperative Extension, Environmental Toxicology Newsletter, Vol. 1 No. 4 April 27, 1981, Toxic Plants II by Art Craigmill, http://extoxnet.orst.edu/newsletters/n14_81.htm.

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