Important Poisonous Plants on Rangelands

Management strategies based on toxin level in the plant, animal susceptibility, and grazing behavior can reduce the risk of poisoning.

By Lynn F. James, Dale R. Gardner, Stephen T. Lee, Kip E. Panter, James A. Pfister, Michael H. Ralphs, and Brian L. Stegelmeier

Introduction

All plants have secondary compounds, some of which can be toxic to livestock if consumed in sufficient quantities. A few plants accumulate toxins at high levels and yet are relatively palatable to livestock. These pose a high risk of poisoning. Most native range communities contain a few toxic plant species that create risks to grazing livestock.

Historically, poisonous plants have caused significant problems for the livestock industry on rangelands and to a lesser degree on pastures. The estimated livestock loss from deaths and abortions exceeds $340 million in the 17 western states.[1] In addition, these plants cause weight loss, unthrifty animals, and altered management strategies.

USDA-Agricultural Research Poisonous Plant Laboratory, Logan, Utah.

The USDA became concerned about livestock poisoning in 1894 when V.K. Chestnut was hired to investigate plant poisonings on western rangelands. Formal research on poisonous plants began in 1905 when C. D. Marsh was sent to Hugo, Colorado, to establish a research station to study locoweed poisoning. This research continued in various departments and locations in the West and is now a component of the USDA Agriculture Research Service at Logan, Utah.

The mission of the Poisonous Plant Lab is to solve poisonous plant problems: identify plants that are toxic to livestock; identify, isolate, and quantify the toxin and determine its mechanism of action; determine how the toxin is metabolized and cleared from the body; develop diagnostic and prognostic procedures; ascertain the conditions when livestock graze these plants and are poisoned; and develop management strategies to reduce the risk of poisoning. A truly integrated team of scientists work cooperatively on all aspects of poisoning: veterinary medicine, pathology, toxicology, reproductive physiology, natural products and synthetic chemistry, animal nutrition and behavior, range management, and ecology. Knowledge generated from research is used to develop recommendations and management strategies to reduce or eliminate livestock poisoning. Examples of some of the important poisonous plants are listed here.

Halogeton.

Halogeton

Halogeton (*Halogeton glomeratus*) is an invasive alien noxious weed from Russia that was first collected in Wells, Nevada, in 1934. Within 4 decades, it rapidly spread throughout the cold desert and infested over 11.2 million acres in the Great Basin, Snake River Plains, Colorado Plateau, and Red Desert of Wyoming. Catastrophic livestock losses occurred in the 1940s and 1950s when entire sheep bands died overnight. *Life* magazine called it the "stock killing plant of the west." The Halogeton Act of 1952 provided funds for its eradication and control. Reallocation of federal research from Forest Service Experiment Stations to the Bureau of Plant Industries created a Range Research unit, which was placed within the Agriculture Research Service when it was established in 1953.

Halogeton poisoning of sheep was a symptom of a larger problem—the overgrazed and depleted condition of desert range.[2] Research showed that if range conditions could be improved, competition from perennial species would reduce halogeton and provide alternative nutritious forage. As a result, thousands of acres of depleted sagebrush range were seeded to crested wheatgrass. Other research projects showed that rumen microflora could be adapted to halogeton by feeding low levels of oxalate[3] such as those in shadscale. The major preventative measure was to keep hungry sheep from grazing dense patches of halogeton.[4] Poisoning occurred when sheep were stressed and hungry from hauling or trailing, then released to graze in areas where halogeton proliferated, such as disturbed areas around water sources or loading docks. Once this knowledge was available, losses generally ceased.

Veratrum

Western false hellebore (*Veratrum californicum*) caused birth defects in sheep in mountain valleys in Idaho. However, it was of great scientific interest because it was the first identified case of a dietary factor causing birth defects.[5] Chemists isolated the teratogen, cyclopamine,[6] and found it was dangerous for only a short part of the reproductive cycle. If a ewe consumes it on the 14th day of gestation, cell differentiation and migration is disrupted, causing a cyclopic or monkey-faced lamb. Limb deformities occur if it is consumed on days 27–32, and collapsed trachea on days 31–33.[7,8] This knowledge resulted in the simple management strategy of avoiding grazing Veratrum for 30 days after breeding. This was accomplished by simply reversing the grazing pattern so that the pregnant ewes were not in the Veratrum patches during early pregnancy (Lynn James, personal observation).

Lupine

Historically, lupine (*Lupinus* spp.) has been one of the largest causes of sheep poisoning in Montana, Idaho, and Wyoming. Quinolizidine alkaloids cause neurologic problems in sheep and acute death. Lupine seeds and pods contain high levels of alkaloids and are succulent and relatively palatable. A management strategy was developed to avoid grazing dense patches of lupine in the late summer and fall as the pods develop and ripen.

More recently, lupine has been linked to "crooked calf disease" in cattle in which calves are born with crooked necks,

Veratrum.

Lupine.

White locoweed.

spines, and legs. Research determined that crooked calves were born when their mothers grazed lupine during days 40–70 of gestation.[9] The teratogenic alkaloid, anagyrine, was identified[10] and was shown to inhibit fetal movement, causing the limbs and spine to develop in abnormal positions.[11] A simple management solution was proposed to stagger grazing so that the susceptible period of gestation (40–70 days) does not overlap the flower and pod stage of lupine growth when anagyrine is highest.[12]

Current research is refining the management recommendation by defining when and why cattle graze lupine. In the scablands of eastern Washington, cattle begin eating lupine in July, when annual grasses dry up and weedy forbs mature (Ralphs et al, in review). Lupine is a deep-rooted perennial that remains green and succulent later into the summer. The traditional breeding season begins May 1 and runs through July. This puts the susceptible period of gestation from June 10 to October 8. Since lupine consumption occurs during the susceptible period of gestation, producers should either alter the breeding season or deny cows access to lupine from July to September.

Locoweed

Locoweed remains the most widespread poisonous plant problem on western US rangelands. Species of *Astragalus* and *Oxytropis* occur in every major plant community. Locoweed causes chronic poisoning similar to wasting disease, and reproduction is compromised.[13]

The toxin has been identified as the indolizidine alkaloid swainsonine.[14] It is a small sugar-like molecule that inhibits glycoprotein metabolism and lysosomal function.[15] This inhibition causes a storage disease from accumulation of abnormal and partially metabolized sugars and proteins within the cell vacuoles. Eventually, the cells die.[16] Locoweed affects all body systems. Damage occurs to the nervous system, causing depression and/or aggression, impaired locomotion, and difficulty prehending food and water. If left unattended, the animals will die of starvation. Significant weight loss occurs in stocker cattle,[17] and subsequent feedlot gains are compromised (Glen Duff, unpublished data).

The greatest economic loss comes from reproductive problems.[18] In the male, spermatogensis stops, libido is reduced, and mating behavior is disrupted. In females, estrus is altered, and ovarian dysfunction occurs. Abortions and early embryonic death are common. Offspring that are carried to term are weak and retarded. Intoxication is exacerbated in nursing offspring when they receive swainsonine in the milk when their mothers graze locoweed.

Management strategies have been developed that will reduce the risk of poisoning from white locoweed on shortgrass prairies.[19] Producers should restrict access to locoweeds during critical periods in the spring and fall, when the green-growing locoweed is more palatable than dry-dormant warm-season grasses. White locoweed is restricted to shallow rocky soils; therefore, fencing along soil boundaries can create locoweed-free pastures on the deeper, more productive soils. Heavy grazing should be avoided during the summer; if cattle run short of green grass in the summer, they will switch to grazing locoweed. Progressive ranchers in locoweed areas ride through their cattle regularly and remove animals that start eating locoweed (David Graham, personal observation). This prevents further intoxication and prevents the loco eaters from influencing others to eat it. Other ranchers have started testing their stocker cattle at the beginning of the grazing season for their propensity to eat locoweed and removing those that eat it (David Graham, unpublished data). Animals can be trained to avoid eating loco through conditioned food aversion. The taste of the plant is paired with an induced illness, causing the animals to associate the taste with the illness, and they subsequently avoid eating the plant in the field.[20]

Most locoweeds can be controlled by common rangeland herbicides.[21] However, their seeds remain in the soil to germinate and reestablish when environmental conditions are favorable.[22] Drought also affects locoweed populations; they decline and die out during extended droughts but increase dramatically during wet periods.

Table 1. Scientific and common names of plants and type of poisoning.		
Family / Genus / Species	**Common name**	**Type of poison**
Chenopodiaceae *Halogeton glomeratus*	Halogeton	Kidney damage and acute death
Liliaceae *Veratrum californicum*	Western false hellebore	Deformed fetus
Leguminosae *Lupinus spp.* *Astragalus spp.* *Oxytropis spp.*	 Lupine Locoweed or milkvetch Locoweed	 Birth defects, crooked calf disease Wasting disease Wasting disease
Ranunculaceae *Delphinium spp.*	Larkspur	Acute death from respiratory failure
Plants containing pyrrolizidine alkaloids		Liver damage, chronic poisoning
Compositae *Senecio jacobaea* *S. longilobus* *S. riddellii* *S. vulgaris*	 Tansy ragwort Woolly or threadleaf groundsel Riddell's groundsel Common groundsel	
Boraginaceae *Cynoglossum officinale* *Amsinckia intermedia* *Symphytum officinale* *Heliotropium europaeum* *Echium plantagineum*	 Hounds tongue Tarweed or fiddle neck Comfrey Heliotrope Paterson's curse	
Leguminosae *Crotalaria spectabilis* *C. retusa*	 Showy crotalaria Rattlebox	

Larkspur

Larkspurs (*Delphinium* spp.) kill more cattle on mountain summer range than any other plant or disease. It is acutely toxic; cattle that eat a lethal dose die within 5 hours. There are over 40 alkaloids in tall larkspur, but the toxic class of alkaloids contain the *N*-(methyl-succinimido)-anthranilic ester group.[23] The mechanism of action of these toxic alkaloids is to block the nerve–muscular junction, resulting in muscular paralysis. This causes fatigue, collapse, and rapid death from respiratory paralysis.

Some drugs can reverse toxicity if the animals are down but not dead. Physostigmine and neostigmine (not yet approved for use in cattle) can reverse the nerve blockage,[24] but they can be toxic if the effective dose is exceeded.

Research has defined a toxic window when cattle are likely to be poisoned.[25] Toxic alkaloid levels are very high in the early new growth but decline over the growing season. Cattle will not graze larkspur early in the season but begin eating it as it begins to flower. Consumption increases as the plant matures, but there is not enough alkaloid in the plant after the pod stage to kill a cow. The management recommenda-

Tall larkspur.

tion was to graze early in the season when larkspur is unpalatable, remove the cows during the flowering period, then graze late in the season after the pod matures.[26]

There are several other management strategies that can reduce the risk of poisoning.[27] Graze sheep before cattle; sheep are 4–6 times more resistant to larkspur alkaloids, and larkspur is considered good sheep feed. Aversion conditioning can train cattle to avoid eating larkspur. Aversions are likely to last indefinitely if conditioned cattle are grazed separately from unconditioned cattle.[28] Herbicides can control larkspur patches where poisoning occurs, and losses can be reduced.[29] The larkspur mirid is host specific to tall larkspur and can be used as a biological tool to damage the larkspur plant. Livestock will not graze severely damaged plants.[30] If the mirid populations can be maintained at high levels, the risk of cattle poisoning can be reduced.

A decision-making handbook is available[31] to assist ranchers in determining their risk of poisoning. It includes measuring alkaloid levels and determining when cattle graze larkspur to define the toxic window, then avoid grazing at that time. Other management recommendations are also presented.

Ponderosa Pine and Broom Snakeweed

Ponderosa pine needles and broom snakeweed cause late-term abortion in cattle. Over several years, a major effort was made to isolate the abortificent compound in pine needles using a large animal bioassay (cow). Isocuppressic acid (ICA) was found to be the parent compound in the needles that induced abortions.[32] ICA has subsequently been identified in Monterey Cyprus, lodgepole pine, and common juniper.[33]

Cattle graze pine needles in cold winters and when deep snow covers the other vegetation.[34] Cattle in low body condition eat more needles than fat cows. The management recommendation is to keep cattle out of pines during the last trimester of gestation.

Broom snakeweed contains several toxic diterpene acids similar to ICA, but the specific abortificient compound has not been identified. Snakeweed is not palatable,[35] but livestock will graze it if all other forage has been depleted.[36] The management recommendation is to always provide adequate forage.

Plants Containing Pyrrolizidine Alkaloids

Pyrrolizidine alkaloids (PA) cause chronic liver damage (cirrhosis), and animals die weeks to months later. They cause the largest problem of plant poisoning worldwide in both livestock and humans. Research has determined the toxicity of the principal PAs in plants[37] and the damage they do as pyrroles in the liver.[38] Many of the invasive noxious weeds contain PAs (Table 1). Their seeds contaminate grains, and their foliage contaminates hay and fodder. They are usually not palatable when green. Management recommendations include identifying and controlling weeds and avoiding grazing when PA concentrations are high in late summer.[39]

Vaccines

We are also conducting research on immunologic assays of the toxins in larkspur,[40, 41] pine needles,[42] pyrrolizidine alkaloids,[43] and Veratrum.[44] These toxins are not large enough to be immunogenic or to stimulate the immune system by themselves. The approach is to attach these toxins to large-molecular-weight proteins that the body's immune system will recognize and to which it will generate an antibody response. If successful, this can be developed into ELISA diagnostic tools and perhaps vaccines.

General Management Recommendations

Most plant poisonings can be avoided by knowing when a particular plant is most toxic, understanding when livestock are likely to eat it, and avoiding grazing in infested areas at those times. Specific recommendations for important poisonous plants in the western United States can be found in USDA Bulletin 415, "Poisonous Plants of Western US,"[45] on our Web site (http://www.pprl.usda.gov).

Authors are Research Leader (James), Chemists (Gardner and Lee), Reproductive Toxicologist (Panter), Rangeland Scientists (Pfister and Ralphs), and Veterinary Pathologist (Stegelmeier) at the USDA-ARS Poisonous Plant Lab, Logan, UT 84341.

References

1. Nielsen, D. B., and James, L. F. 1992. The economic impacts of livestock poisoning by plants. *In:* L. F. James, R. F. Keeler, E. M. Bailey Jr., P. R. Cheeke, and M. P. Hegarty [eds.]. Poisonous plants. Ames, IA: Iowa State University Press. p 3–10.
2. Young, J. A. 1999. Halogeton. USDA/ARS Miscellaneous Publication 1553.
3. James, L. F., J .C. Street, and J. E. Butcher. 1967. In vitro degredation of oxalate and cellulose by rumen ingesta from sheep fed Halogeton glomeratus. *Journal of Animal Science* 45:1438–1444.
4. James, L. F., and E. H. Cronin. 1974. Management practices to minimize death losses of sheep grazing on Halogeton-infested range. *Journal of Range Management* 27:424–426.
5. Binns, W., L. F. James, J. L. Shupe, and G. Everett. 1963. A congenital cyclopian-type malformation in lambs induced by maternal ingestion of a range plant, *Veratrum californicum. American Journal of Veterinary Research* 24:1164–1175.
6. Keeler, R. F. 1969. "Teratogenic compounds of *Veratrum californicum* VI. The structure of cyclopamine. *Phytochemistry* 8:223.
7. Binns, W., J. L. Shupe, R. F. Keeler, and L. F. James. 1965. Chronologic evaluation of teratogenicity in sheep fed *Veratrum californicum. Journal of the American Veterinary Medical Association* 147:839–842.
8. Keeler, R. F., S. Young, and R. Smart. 1985. Congenital tracheal stenosis in lambs induced by maternal ingestion of *Veratrum californicum. Teratology* 31:83–88.

9. Shupe, J. L., W. Binns, L. F. James, and R. F. Keeler. 1967. Lupine, a cause of crooked calf disease. *Journal of the American Veterinary Medical Association* 151:198–203.
10. Keeler, R. F. 1976. Lupin alkaloids from teratogenic and nonteratogenic lupins. III. identification of anagyrine as the probable teratogen by feeding trials. *Journal of Toxicology and Environmental Health* 1:887–898.
11. Panter, K. E., T. D. Bunch, R. F. Keeler, D. V. Sisson, and R. J. Callan. 1990. Multiple congenital contractures (MCC) and cleft palate induced in goats by ingestion of piperidine alkaloid-containing plants: reduction in fetal movement as the probable cause. *Clinical Toxicology* 28:69–83.
12. Keeler, R. F., L. F. James, J. L. Shupe, and K. R. Van Kampen. 1977. Lupine-induced crooked calf disease and a management method to reduce incidence. *Journal of Range Management* 30:97–102.
13. James, L. F., W. J. Hartley, and K. R. Van Kampen. 1981. Syndromes of Astragalus poisoning in livestock. *Journal of the American Veterinary Medical Association* 178:146–150.
14. Molyneux, R. J., and L. F. James. 1982. Loco intoxication: indolizidine alkaloids of spotted locoweed (*Astragalus lentiginosus*). *Science* 216:190–196.
15. Stegelmeier, B. L., L. F. James, K. E. Panter, M. H. Ralphs, D. R. Gardner, R. J. Molyneux, and J. A. Pfister. 1999. The pathogenesis and toxicokinetics of locoweed (*Astragalus and Oxytropis*) poisoning in livestock. *Journal of Natural Toxins* 8:35–45.
16. Van Kampen, K. R., and L. F. James. 1972. Sequential development of the lesions in locoweed poisoning. *Clinical Toxicology* 5:575–580.
17. Torrell, L. A., L. P. Owen, K. C. McDaniel, and D. Graham. 2000. Perceptions and economic losses from locoweed in northeastern New Mexico. *Journal of Range Management* 53:376–383.
18. Panter, K. E., L. F. James, B. L. Stegelmeier, M. H. Ralphs, and J. A. Pfister. 1999. Locoweeds: effects on reproduction in livestock. *Journal of Natural Toxins* 8:53–62.
19. Ralphs, M. H., J. D. Graham, and L. F. James. 2002. A close look at locoweed poisoning on shortgrass prairies. *Rangelands* 24:30–34.
20. Ralphs, M. H., F. D. Provenza, J. A. Pfister, D. Graham, G. C. Duff, and G. Greathouse. 2001. Conditioned food aversion: from theory to practice. *Rangelands* 23:14–18.
21. McDaniel, K. C. 1999. Controlling locoweed with herbicides. *In:* T. M. Sterling and D. C. Thompson [eds.]. Locoweed research updates and highlights. Las Cruces, New Mexico State University. New Mexico Agricultural Experimental Station Research Report 730. p 52–53.
22. Ralphs, M. H., and E. H. Cronin. 1987. Locoweed seed in soil: density longevity, germination and viability. *Weed Science* 35:792–795.
23. Manners, G. D., K. E. Panter, and S. W. Pelletier. 1995. Structure-activity relationships of norditerpenoid alkaloids occurring in toxic larkspur (*Delphinium*) species. *Journal of Natural Products* 58:863–869.
24. Pfister, J. A., K. E. Panter, G. D. Manners, and C. D. Cheney. 1994. Reversal of tall larkspur (*Delphinium barbeyi*) toxicity with physostigmine. *Veterinary and Human Toxicology* 36:511–514.
25. Pfister, J. A., G. D. Manners, M. H. Ralphs, Z. X. Hong, and M. A. Lane. 1988. Effects of phenology, site, and rumen fill on tall larkspur consumption by cattle. *Journal of Range Management* 41:509–514.
26. Pfister, J. A., M. H. Ralphs, G. D. Manners, K. W. Price, and L. F. James. 1997. Early season grazing by cattle of tall larkspur-infested rangeland. *Journal of Range Management* 50:391–398.
27. Pfister, J. A., M. H. Ralphs, G. D. Manners, K. E. Panter, L. F. James, B. L. Stegelmeier, and D. R. Gardner. 1993. Tall larkspur poisoning in cattle: current research and recommendations. *Rangelands* 15:157–160.
28. Ralphs, M. H. 1997. Persistence of aversions to larkspur in naive and native cattle. *Journal of Range Management* 50:367–370.
29. Nielsen, D. B., M. H. Ralphs, J. O. Evans, and C. A. Call. 1994. Economic feasibility of controlling tall larkspur on rangeland. *Journal of Range Management* 47:369–372.
30. Ralphs, M. H., W. A. Jones, and J. A. Pfister. 1997. Damage from the larkspur mirid deters cattle grazing of larkspur. *Journal of Range Management* 50:371–373.
31. Pfister, J. A. 1998. Grazing tall larkspur ranges: a livestock producer's decision-making handbook. Logan, UT. USDA/ Poisonous Plant Research Lab Bulletin.
32. Gardner, D. R., R. J. Molyneux, L. F. James, K. E. Panter, and B. L. Stegelmeier. 1994. Ponderosa pine needle-induced abortion in beef cattle: identification if isocupressic acid as the principal active compound. *Journal of Agricultural and Food Chemistry* 42:756–761.
33. Gardner, D. R., and L. F. James. 1999. Pine needle abortion in cattle: analysis of isocupressic acid in North American gymnosperms. *Phytochemical Analysis* 10:132–136.
34. Pfister, J. A., and D. C. Adams. 1993. Factors influencing pine needle consumption by grazing cattle during winter. *Journal of Range Management* 46:394–398.
35. Pieper, R. D. 1989. Broom snakeweed content in herbivore diets. *In:* E. W. Huddleston and R. D. Pieper [eds.]. Snakeweed: Problems and perspectives. Las Cruces, NM: New Mexico State University. New Mexico Agricultural Experimental Station Bulletin 751. p 203–210.
36. Ralphs, M. H., and R. D. Wiedmier. 2004. Conditioning cattle to graze broom snakeweed (*Gutierrezia sarothrae*). *Journal of Animal Science* 82:3100–3106.
37. Johnson, A. E., R. J. Molyneux, and G. B. Merrill. 1985. Chemistry of toxic range plants: variation in pyrrolizidine alkaloid content of *Senecio, Amsinckia,* and *Crotalaria* species. *Journal of Agricultural and Food Chemistry* 33:50–55.
38. Stegelmeier, B. L., D. R. Gardner, L. F. James, and R. J. Molyneux. 1996. Pyrrole detection and the pathologic progression of *Cynoglossum officinale* (hound's tongue) poisoning in horses. *Journal of Veterinary Diagnostic Investigation* 8:81–90.

39. Johnson, A. E., R. J. Molyneux, and M. H. Ralphs. 1989. Senecio: a dangerous plant for man and beast. *Rangelands* 11:261–264.
40. Lee, S. T., T. H. Schoch, D. R. Gardner, and B. L. Stegelmeier. 2000. Development of enzyme-linked immunosorbent assays for toxic larkspur (*Delphinium* spp.) alkaloids. *Journal of Agricultural and Food Chemistry* 48:4520–4528.
41. Lee, S. T., B. L. Stegelmeier, K. E. Panter, J. A. Pfister, D. R. Gardner, T. K. Schoch, and L. F. James. 2003. Evaluation of vaccination against methyllycaconitine toxicity in mice. *Journal of Animal Science* 81:2332–2238.
42. Lee, S. T., D. R. Gardner, M. Garrosian, K. E. Panter, A. N. Serrequi, T. K. Schoch, and B. L. Stegelmeier. 2003. Development of enzyme linked immunosorbent assays for isocupressic acid and serum metabolilts of isocupressic acid. *Journal of Agricultural and Food Chemistry* 51:3228–3233.
43. Lee, S. T., T. H. Schoch, B. L. Stegelmeier, D. R. Gardner, K. A. Than, and R. J. Molyneux. 2001. Development of enzyme-linked immunosorbent assays for the hepatotoxic alkaloids riddelliine and riddelliine-N-oxide. *Journal of Agricultural and Food Chemistry* 49:4144–4151.
44. Lee, S. T., K. E. Panter, W. Gaffield, and B. L. Stegelmeier. 2003. Development of enzyme-linked immunosorbent assay for Veratrum plant teratogens: cyclopamine and jervine. *Journal of Agricultural and Food Chemistry* 51:582–586.
45. James, L. F., R. F. Keeler, A. E. Johnson, M. C. Williams, E. H. Cronin, and J. D. Olsen. 1980. Plants poisonous to livestock in the western states. USDA/SEA Agricultural Information Bulletin 415.