Larkspur Poisoning in Livestock: Myths and Misconceptions

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Larkspurs (*Delphinium* spp.) are major causes of cattle deaths on mountain ranges during the summer grazing season in the western U.S. For descriptive convenience, larkspurs are grouped into tall and low species based upon their growth characteristics. Tall larkspurs are generally found growing at high altitude and reaching 4 to 6 feet in height. Tall larkspurs are most commonly represented by *Delphinium barbeyi*, *D. glaucescens*, *D. glaucum,* and *D. occidentale*. Low larkspurs (*D. nuttallianum*, *D. virescens*, *D. andersonii*) seldom grow above 2 feet in height, while plains larkspur (*D. geyeri*) is intermediate in growth habit.

As the livestock industry moved westward beyond the Mississippi River into the Rocky Mountains in the 1830's, livestock began to die of plant poisoning. Larkspurs were eventually recognized as a serious problem, and in 1885 the USDA's Bureau of Animal Industry began investigating larkspur and other poisonous plants. Larkspur research centered in Colorado because of the magnitude of the losses in that state. C.D. Marsh and coworkers fed tall and low larkspur to more than 100 head of cattle from 1909 to 1911 in Colorado (near Crested Butte) and Montana (near Big Timber), and described the toxic dose, symptoms, and experimental therapies (Marsh et al. 1916). Research on tall larkspur toxicity has continued to the present day, with significant progress occurring in the last 10 years or so (Pfister et al. 1993).

Because of the lengthy history of larkspur poisoning, many myths and misconceptions have arisen in the livestock industry about various aspects of the problem. In this paper, we outline some myths that prevail about larkspur toxicity, and attempt to debunk these myths and misconceptions so that livestock producers can more realistically deal with larkspur poisoning. By necessity, most comments will pertain to tall larkspurs as most research has been directed at these species. When appropriate, we also have made comments about low larkspurs.

Myth #1: Consumption of larkspur roots causes many cattle deaths.

Tall larkspurs are sometimes eaten avidly by cattle during or just after summer storms, and cattle deaths have been attributed to consumption of tall larkspur roots when the soil is wet. Tall larkspurs have a very fibrous root crown that is impossible to completely extricate from the ground without a shovel or similar tool. At times, such as when the soil is wet, one can pull on tall larkspur stems, and a small portion of root will be uprooted with the stem. Most of the time when pulling on tall larkspur stems, however, the hollow stems break above ground level. Literally hundreds of hours have been spent observing cattle eat tall larkspur in wet and dry conditions, and never has a grazing animal been seen eating roots. Marsh et al. (1916) attempted to poison a sheep with tall larkspur roots: the sheep ate about 33 lb. of root material over 11 days with no indication of toxicity. The roots of tall larkspur do contain a small amount of toxic alkaloids (D. Gardner, personal communication), but it is unlikely that tall larkspur roots have ever poisoned cattle. Immature tall larkspur plants are the most toxic; leaves and pods contain the highest concentration of toxic alkaloids, whereas stems are low in toxicity.

Low larkspurs (e.g., *D. nuttallianum*) are shallow-rooted perennials with a relatively small root containing low concentrations of toxic alkaloids. We have seen cattle pull the entire low larkspur plant (including roots) from the ground on a few occasions when the ground was wet, but generally cattle eat only the aboveground portions of low larkspur. Even though cattle may ingest an occasional root, it is doubtful if cattle could consume enough root material for toxicity to occur. Marsh et al. (1916) fed 2.4 lb. of low larkspur roots alone to a cow without effect; mixtures of roots and plant tops were also fed to cattle (up to about 20 lb./day) without effect. In collecting samples to determine the toxicity of tall and low larkspurs, livestock managers need to sample only aboveground portions of the plant.

Myth #2: Cattle eat tall larkspur because they crave salt or other minerals.

Some ranchers believe that cattle may be attracted to tall larkspur because of a craving for salt or other minerals such as phosphorus. This belief has led to widespread claims for feeding mineral salt supplements to reduce tall larkspur losses. Broadleaf plants (i.e., forbs), including larkspur, usually contain higher concentrations of minerals than do grasses. Forbs, besides tall larkspur, are always a major component of cattle diets during summer on mountain ranges, and it is unlikely that cattle grazing on most mountain ranges will be mineral deficient if provided with common salt, unless the forage in general is lacking a specific nutrient (e.g., copper). Grazing studies have shown that cattle do not eat more tall larkspur when deprived of all mineral salt supplement than cattle which have been supplemented (Pfister and Manners 1991; Pfister and Manners 1995). During one year (1990 at Yampa, Colo.) cattle ate no larkspur although the tall larkspur contained a high concentration of sodium compared to other forage. Tall larkspur may contain higher calcium concentrations than phosphorus (1.5 to 3.0% vs. 0.30%), leading some to suggest that this imbalance may increase palatability to animals. There is no indication, however, that the Ca:P ratio is of any significance in ruminant

diets, as long as neither mineral is deficient (Ternouth 1991). It is possible that cattle that are deficient in a specific mineral may eat more larkspur under some circumstances, but no experimental evidence exists to support feeding expensive, custom-formulated mineral salt supplements to cattle grazing larkspur ranges. Excess mineral salt supplementation seems unlikely to alter the susceptibility of individual animals since the larkspur toxin(s) binds at receptor sites at the neuromuscular junction, and mineral status probably would not alter binding affinity. It is possible that supplementing magnesium might, under some circumstances, help to reduce death loss from larkspur-induced bloat. It is unlikely that the question of mineral salt supplementation will ever be adequately resolved, given the number of minerals involved and the possible combinations and permutations of mineral salt supplements, forage mineral concentrations, and variable cattle mineral intake. We recommend that livestock producers consult with a local veterinarian or nutritional consultant to determine if their ranges are deficient in specific minerals, and that mineral salt supplements be fed as necessary to meet nutritional needs, but not to protect livestock against effects of tall larkspur.

Myth #3: Tall larkspur is most dangerous before flowering.

Tall larkspur is most toxic when immature, and toxicity generally decreases with maturity, except with the seed pods (Ralphs et al. 1997). Numerous grazing studies have shown that cattle eat little or no tall larkspur before stems elongate flowering racemes (Pfister et al. 1997). Since tall larkspur is only dangerous when consumed, the danger to cattle before flowering is not as great as after flowering. There is a primary toxic window during the flower and early pod stages of growth when toxicity is declining but palatability is increasing, resulting in the most dangerous grazing period. Usually most cattle deaths occur after animals have been in a pasture for a couple of weeks, often coinciding with the transition from preflower to the flowering stage of growth.

Myth #4: Monkshood causes many cattle deaths attributed to tall larkspurs.

Monkshood (*Aconitum* spp.) is also a member of the Ranunculaceae (buttercup) family, and is often confused with tall larkspur. Though the growth form and leaves are similar in appearance, it is easy to distinguish monkshood from tall larkspurs if the plants are flowering. Monkshood lacks the characteristic spur on the back of the flower that gives larkspur its name (Photo 1). Monkshood has a helmet-like hood (hence the common name) rising over the flower (Photo 2). Tall larkspurs characteristically have a hollow stem, whereas monkshood stems are not hollow, containing a pithy center.

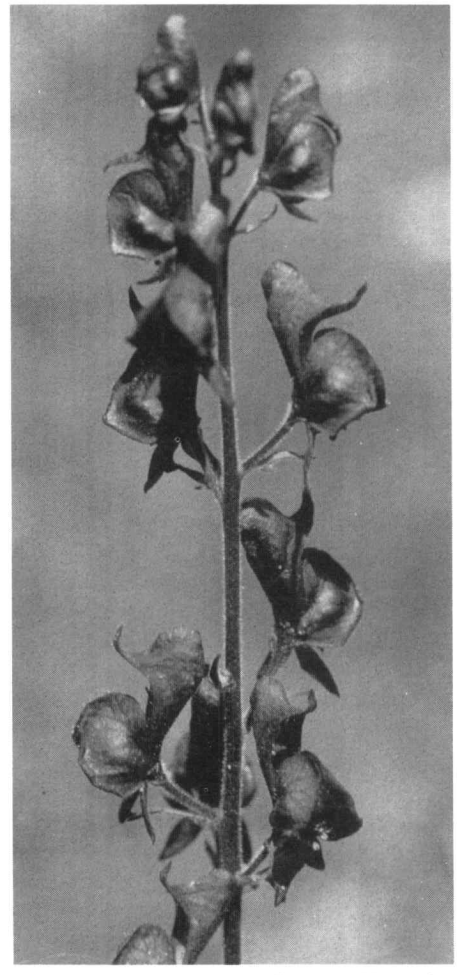

Photo 1. *Monkshood flower. Note the characteristic helmet-like hood rising over the flower, and the lack of a spur.*

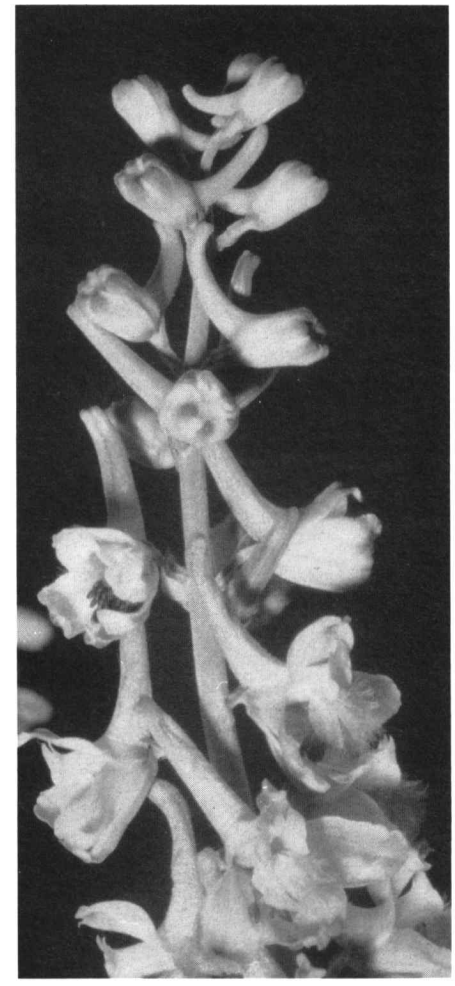

Photo 2. *Larkspur flower. Note the characteristic spur on the flower.*

Monkshood is not eaten to any great extent by grazing cattle. Grazing studies for several years in areas with substantial populations of both tall larkspur and monkshood showed that cattle rarely ate more than a few bites of monkshood, yet ate large quantities of tall larkspur (Pfister, unpublished data). In our opinion, if other forage is available, grazing cattle will rarely consume a toxic dose of monkshood, especially since the quantity of toxic alkaloid in monkshood is considerably less than that found in larkspurs. We believe that nearly all deaths in areas with tall larkspur and monkshood growing together can be attributed to tall larkspur. Monkshood poisoning is reportedly more of a problem in Europe than in North America.

However, since monkshood contains similar alkaloids as those in larkspur, it can produce similar signs of paralysis and death if eaten in sufficient quantity.

Myth #5: All larkspurs are equally toxic.

The toxicity of larkspurs varies depending upon the species, and the conditions in which the plants are growing. The species that have the highest concentrations of toxic alkaloids include *D. glaucum*, *D. barbeyi*, *D. glaucescens*, *D. geyeri*, and *D. occidentale* (Ralphs et al. 1997). Even within a population of the same species of larkspur there is variability in the alkaloid content (Pfister et al. 1994). At times, plants growing in full sun and under drought conditions may be more toxic. It is wise to assume that all species of larkspur, including horticultural varieties, are potentially poisonous until proven otherwise.

Myth #6: Delphinine is the major toxic alkaloid.

Early efforts to identify the larkspur toxin characterized delphinine as the toxic alkaloid. In reality, there is no alkaloid called delphinine in North American tall larkspurs (Pelletier et al. 1989), as *Delphinium staphisagria* (a European plant) is the only larkspur species found to contain delphinine. At least 150 alkaloids have been identified in larkspurs but the primary toxins are two related alkaloids: methyllycaconitine (MLA) and 14-deacetylnudicauline (DAN; Manners et al. 1995). Deltaline occurs in greater quantity than any other alkaloid in tall larkspurs, but is relatively nontoxic. The alkaloids MLA and DAN, plus the highly toxic alkaloid nudicauline, are the major toxins in low and plains larkspurs.

Myth #7: Cattle, sheep, and horses are equally susceptible to larkspur poisoning.

Larkspur poisoning has been recognized as a serious problem in cattle, sheep, and horses in the mountainous areas of the western United States since the beginning of the century. There is, however, considerable species variation in susceptibility to the larkspur alkaloids with cattle being most susceptible and sheep being able to tolerate 4 times the dose of larkspur that would be fatal to cattle. Horses are intermediate in their susceptibility to larkspur poisoning. Horses are rarely poisoned by larkspur and will not normally eat the plant unless other forages are unavailable. Toxicity studies on the susceptibility of goats, llamas, and alpacas have not been conducted, but it is logical to assume the potent alkaloids in larkspur will have similar effects in these species as in cattle and sheep.

Myth #8: Bleeding is an effective treatment for larkspur poisoning.

Many remedies have been advocated for the treatment of larkspur poisoning since the problem was first recognized. Bleeding of larkspur-poisoned animals by cutting the tail has been touted as an effective treatment along with remedies such as giving atropine, strychnine, potassium permanganate, aluminum sulfate, tobacco, and even whiskey. A great deal is understood about how larkspur kills cattle, and given this knowledge it is likely that any apparent success with these early treatments was related to the toxic dose the animal had ingested and not the treatment itself. If less than a lethal dose of larkspur alkaloids was consumed, the animal would likely recover in spite of any treatment, unless bloat or vomition occurred while the animal was down.

The primary site of action of the larkspur alkaloids is the neuromuscular junction where they reversibly bind to acetylcholine receptor sites. This blockage inhibits nerve impulse transmission and muscle paralysis develops rapidly. This is reflected in the progression of clinical signs beginning with muscle weakness, staggering gait, inability to stand, bloat, respiratory paralysis and death. Any stress placed on the affected animal invariably hastens its death. Bloating occurs because of the paralysis of the eructation mechanism, and the presence of the highly fermentable larkspur in the rumen probably plays an important role in the high mortality associated with larkspur poisoning. Some success of early remedies for larkspur poisoning such as giving bacon fat and turpentine orally may have been efficacious in that fat and turpentine can help reduce bloating.

In light of present knowledge about larkspur poisoning, treatment should be aimed at reversing the neuromuscular blockade by the intravenous administration of physostigmine sulfate. This compound maintains high concentrations of the neurotransmitter acetylcholine, thereby restoring normal neuromuscular activity. Since bloating is a significant component of larkspur poisoning it is also important to ensure the animal is kept resting on its sternum, and to administer appropriate antibloating medication. Above all, it is critical that the animal not be stressed, even to the point of not treating the animal if it requires stressing it.

Conclusions

Many anecdotal stories about tall larkspur poisoning have become accepted as fact by the livestock industry during this century. Research results have shown, however, that many myths and misconceptions about tall larkspur poisoning are not factual. Ranchers with a history of dealing with tall larkspur poisoning can be a rich source of information leading to testable research ideas, but some notions should be permanently discarded by the livestock industry so that animal and range managers can deal realistically with tall larkspur poisoning based on correct and current information.

Literature Cited

Manners, G.D., K.E. Panter, and S.W. Pelletier. 1995. Structure-activity relationships of norditerpeniod alkaloids occurring in toxic larkspur (*Delphinium*) species. J. Nat. Prod. 58:863–869.

Marsh, C.D., A.B. Clawson, and H. Marsh. 1916. Larkspur poisoning of livestock. USDA Bureau of Animal Industry Bull. 365.

Pelletier, S.W., P. Kulanthaivel, and J.D. Olsen. 1989. Alkaloids of *Delphinium barbeyi*. Phytochem. 28:1521–1525.

Pfister, J.A. and G.D. Manners. 1991. Mineral supplementation of cattle grazing larkspur-infested rangeland during drought. J. Range Manage. 44:105–111.

Pfister, J.A. and G.D. Manners. 1995. Effects of carbachol administration in cattle grazing tall larkspur-infested range. J. Range Manage. 48:343–349.

Pfister, J.A., G.D. Manners, D.R. Gardner, and M.H. Ralphs. 1994. Toxic alkaloid levels in tall larkspur (*Delphinium barbeyi*) in western Colorado. J. Range Manage. 47:355–358.

Pfister, J.A., M.H. Ralphs, G.D. Manners, D.R. Gardner, K.W. Price, and L.F. James. 1997. Early season grazing of tall larkspur- (*Delphinium* spp.) infested rangeland. J. Range Manage. 50:In press.

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.

Ralphs, M.H., G.D. Manners, J.A. Pfister, D.R. Gardner, and L.F. James. 1997. Toxic alkaloid concentration in tall larkspur species in the western U.S. J. Range Manage. In press.

Ternouth, J.H. 1991. The kinetics and requirements of phosphorus in ruminants. p. 143–152. *In*: Y.W. Ho, H.K. Wong, N. Abdullah, and Z.A. Tajuddin (eds.), Recent Advances in the Nutrition of Herbivores, Vinlin Press, Kuala Lumpur.

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