Is Tansymustard Causing Photosensitization of Cattle in Montana?

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Photosensitization of grazing cattle has been a persistent and perplexing problem in northern and eastern Montana for the past 5 years. In some herds and years, as many as 50% of lactating cows have been affected. Affected cattle develop severe phototoxic reactions in lightly pigmented areas of skin around the head, neck, and udder, and may lose large patches of hair and skin. Udders of lactating cows become inflamed, weepy, and scabby, and calves may not be allowed to nurse. Besides reduced weaning weights, many cows have delayed estrous cycles. Although photosensitization is not usually fatal, the economic impact has been substantial in Montana.

What is Photosensitization?

Two general types of photosensitization occur in livestock: primary and secondary (hepatic). Primary photosensitization occurs when animals graze a plant containing a phototoxic compound. The toxic compound in the bloodstream reacts with sunlight to produce a phototoxic reaction, which may resemble a severe sunburn. St. Johnswort (Hypericum perforatum) and spring parsley (Cymopterus watsoni) are two common range plants that contain phototoxic compounds and cause primary photosensitization.

Secondary photosensitization occurs when a plant toxin causes liver damage. The damaged liver is not able to normally excrete phylloerythrin, a breakdown product of chlorophyll. With the normal mode of excretion blocked, phylloerythrin then begins circulating in the bloodstream, and acts as a photodynamic agent to cause photosensitization. Tetradymia spp. (horsebrush) and Senecio spp. are two common forage plants that can cause liver damage, resulting in secondary photosensitization.

Feeding Trials

Numerous ranchers and extension personnel suspected that tansymustard (Descurainia pinnata) was causing the photosensitization in Montana. The plant was growing in most pastures where cattle were being affected, generally in grain stubble or fallow fields or in disturbed sites within the pastures.

We conducted 2 feeding trials during 1987 and 1988 to determine if tansymustard would cause photosensitization under controlled feeding conditions. The 1987 study was conducted in mid-May near Miles City, Mont., using white-faced range cattle familiar with the mustard. Three cow-calf pairs were fed hay only, and 8 other cows (4 dry and 4 lactating) were grazed in a 2-acre pasture where 45% of the vegetation was tansymustard. The penned cows receiving the mustard were fed several times each day. In order to increase intake, yellow sweet clover was mixed (about 10% of the feed) with the mustard. Bite counts were used to determine the diets of the grazing animals during the study.
Various clinical tests were used to determine if primary or secondary photosensitization occurred. At the beginning and end of the trial, blood samples were taken to monitor liver enzyme levels. A dye clearance test (BSP) was used to evaluate how rapidly the liver could break down a dye injected into the bloodstream. Liver samples were also taken by biopsy for microscopic examination of liver structure.

Penned cows consumed about 5.5 lb. (dry weight) of mustard each day during this trial. Blood values were generally within normal limits, and clearance of the dye was normal. The cows showed no evidence of photosensitization during the trial or during a subsequent 10-day period. The grazing cows ate from 0 to 40% of their diets as tansymustard. During the first 3 days, some animals ate from 15 to 30% of their diets as tansymustard, although they could have selected other forage, indicating that tansy was of moderate palatability. As forage was depleted, the animals were forced to consume more mustard. No photosensitization occurred in these grazing animals. About 20 cases of photosensitization were reported in this general area during spring, 1987.

The mustard was in the early to late flower stage during the 1987 trial, and the results may have been influenced by plant maturity; many poisonous plants become less toxic with maturation. Therefore, a similar trial was undertaken in 1988 while the mustard was still in the rosette stage.

The 1988 trial was conducted near Roundup, Mont. Twelve Hereford cow-calf pairs were purchased in western Montana; the animals had no known experience with tansymustard. The cow-calf pairs were assigned to 3 different groups during the 12-day trial: (1) 4 pairs fed only alfalfa hay, (2) 4 pairs fed only tansymustard, and (3) 4 pairs fed flixweed, with some supplemental hay provided because of the low palatability of this plant. A severe drought influenced forage growth in 1988. About 10 days before we began this trial, 2 inches of rain induced forage growth, including the mustards. The mustards were immature at the start of the trial, but matured rapidly, and many plants were beginning to flower at the end. The mustards were hand-harvested 3 to 4 times each day, and fed fresh to the cattle.

Blood samples, the BSP test, and liver biopsy samples were collected several weeks before the feeding trial was initiated, and repeated at the end of the trial to evaluate any changes in liver function.

The cows ate large amounts of the tansymustard, and smaller amounts of flixweed (Table 1). As the animals adapted to the feeding trial, daily consumption of tansymustard increased from 18 lb. per head to 62 lb. per head (wet weight) on day 12. Protein and phosphorus content of the tansymustard (rosette to the bud stage) was 22.4 and 0.38%, respectively. None of the cows showed any evidence of photosensitization. Blood chemistry values were within normal limits, as were BSP clearance rates. Liver biopsy samples indicated normal liver structure at the end of the trial. The cows were pastured
Table 1. Daily intake of tansymustard and flxweed by lactating cows during a 12-day feeding trial in Montana 1988.

<table>
<thead>
<tr>
<th>Item</th>
<th>Tansymustard</th>
<th>Flxweed</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mustard intake</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>wet weight (lb/head)</td>
<td>39.6</td>
<td>24.0</td>
<td>0</td>
</tr>
<tr>
<td>dry weight (lb/head)</td>
<td>4.1</td>
<td>6.0</td>
<td>0</td>
</tr>
<tr>
<td>Mustard intake (% of body weight)</td>
<td>4.4</td>
<td>2.4</td>
<td>0</td>
</tr>
<tr>
<td>wet weight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dry weight</td>
<td>1.0</td>
<td>0.6</td>
<td>0</td>
</tr>
</tbody>
</table>

for 2 weeks near Bozeman on lush forage after the trial to eliminate the possibility that photosensitization symptoms would appear later. No cases of photosensitization were reported in this general area during spring, 1988.

Weather Patterns

Precipitation is the dominant factor influencing plant growth in Montana, with temperatures playing a secondary role. Annual plants such as tansymustard germinate and initiate growth during wet, cool fall months. If subsequent springs are dry, mustards have a competitive advantage over perennial forage plants, and represent a much larger proportion of the herbage available for grazing animals. We suspect that climatic variability is responsible for much of the yearly variation in severity of photosensitization problems from area to area. Changes in weather influence amounts of a plant present in relation to other forage species, the rate of plant growth, and perhaps the concentration of toxic compounds.

Extension service records were used to determine the location of reported cases of photosensitization in Montana from 1983 to 1987. Climatic data, provided by the National Climatic Data Center, Asheville, N.C., were analyzed to determine seasonal trends in rainfall and temperature during that period.

Monthly temperature and precipitation patterns (deviations from 30-year normal) for 2 locations that had photosensitization problems during April and May, 1985 to 1987, are shown in Figure 1. Photosensitization problems generally occurred during warm springs with above-normal temperatures and below-normal precipitation. However, cool weather, with above-average precipitation characterized the preceding July to September period. Winter temperatures were usually above normal from January to March.

Conclusions

The difficulty we experienced in reproducing photosensitization under controlled conditions is not unique to this study. Other plants such as horsebrush (Tetradymia spp.), kleingrass (Panicum coloratum), smartweeds (Polygonum spp.), and rain lily (Cooperia pendulcuta) have been suspected causes of photosensitization, but have failed to cause the condition in research trials. The classic case may be Tetradymia, which was fed in trials for many years with little progress. The problem was solved when it was observed that photosensitized sheep had been grazing on black sage (Artemisia nova). It was then verified that previous consumption of black sage predisposes sheep to photosensitization by horsebrush.

Tansymustard is readily grazed by cattle in the Southwest, but photosensitization has not been reported. However, the plant causes a condition known locally as "wooden tongue," in which cattle become partially blind and lose the ability to swallow. We saw no evidence of wooden tongue in our studies.

Note added in proof: Grazed tansymustard plants were collected from a Montana pasture with numerous photosensitized cows during 1989. This material, and tansymustard grown in a greenhouse, was fed to hamsters for 21 days. We found elevated liver enzyme levels in the grazed tansy group, but not the greenhouse group, thus providing the first direct evidence that tansymustard contains a liver toxin.

It appears possible that some unknown factor is operating in conjunction with consumption of tansymustard to cause the photosensitization problems in Montana. There is a possibility that mycotoxins from a fungus or mold growing on some plant causes liver damage. We plan to continue to investigate the problem by visiting affected herds, and by making detailed observations on pastures and animals.