

TOXICOLOGICAL EFFECTS OF FEEDING CONTAMINATED HAY TO LIVESTOCK

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In California, the most prominent plants involved are fiddleneck and common groundsel. Pyrrolizidine alkaloid (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 \geq roots. The plants contain PA's at all stages of growth. The PA's are resistant to heat, and also maintain their toxicity in dried plant material and hay.

The PA's toxic action is principally exerted on the liver, and depending on the animal species, the kidney and lungs may also be affected but to a lesser extent than the liver. Pyrrolizidine alkaloids are activated by liver metabolism to toxic pyrrole metabolites which have been shown to be carcinogenic, mutagenic, and embryotoxic. Most of the toxic effects of the PA's can be traced to their destructive effect on liver cells, and thus the clinical signs and symptoms of PA intoxication are those of progressive liver failure.

Pyrrolizidine alkaloids produce very characteristic pathological changes in the structure of the liver. The characteristic hepatic lesion of the PA toxicity is hemorrhagic centrilobular necrosis without inflammation, occlusion of the central veins, sinusoid distension, and megalocytosis (enlarged cells). The severity of the changes reflect the degree of damage. Extensive fibrosis (cirrhosis) may be seen in the liver of patients that recover from acute PA poisoning and patients that have had chronic exposure to PA's.

Pyrrolizidine Alkaloid Poisoning of Animals

Cattle and horses are the most sensitive species to the toxic effects of PA's, pigs and chickens are less sensitive, and sheep, goats and turkeys are the least sensitive. The young of all species are more sensitive than are adults. Numerous feeding trials have been conducted using cattle, sheep, goats and horses, and the results of these trials, although somewhat confusing, indicate that the PA's produce a classical time-dose-response toxicity picture. Thus high doses of PA's fed over a period of a few days will produce toxicity in a short period of time whereas low doses fed over a longer period will produce a very similar but much delayed toxicity. There is presumably a no effect level for PA plants and it is probably less than 5% of the total ration. One of the more resistant species, the goat, has to consume a quantity of tansy ragwort at least equal to its body weight in order to be poisoned.

The clinical signs of PA poisoning in animals vary from species to species. In cattle, common signs are: dull haircoat, dry muzzle, lack of conditioning, photosensitization, diarrhea with tenesmus (straining), rectal prolapse, depression and death. In horses the neurological signs predominate, and they gradually lose condition, become anorexic, depressed, and wander aimlessly (Walk-about Disease). In sheep, acute deaths are rare but the liver damage produced by the PA's often predispose sheep to copper toxicity. In goats the clinical picture is similar to that seen in cattle. The amount of plant material that must be ingested in order to produce serious toxicity is dependent on the species, the period of consumption and the alkaloid content of the plants. Estimates range from 5-10% of body weight for cattle and horses to more than 100% for sheep and goats. Smaller amounts may depress production without producing observable signs.

The degree of liver damage produced by the PA's can be assessed by measuring serum enzyme activities. Certain enzymes are released when the liver cells are damaged and increased enzymatic activity in blood serum is indicative of cellular damage. A study at the Veterinary Medical Teaching Hospital at the University of California at Davis looked at the effects of PA's on plasma amino acid patterns in the horse. Changes in the amino acid pattern have been used in human medicine as markers of hepatic damage and it has been proposed that these changes in amino acid ratios in the serum may be responsible for some of the toxic effects seen in hepatic failure. Because serum enzyme levels and amino acid levels are non-specific markers of hepatic damage, definitive diagnosis of PA intoxication requires microscopic examination of biopsy or necropsy material. At necropsy, the liver is small, pale, firm and may have a nutmeg appearance. The gall bladder is almost always full

and very distended. Edema of the gastrointestinal tract (especially the abomasum) may also be seen. Jaundice, ascites and petechial (paintbrush) hemorrhages are more variable findings. The discovery of the characteristic histopathological changes in liver structure produced by the PA's can be used to make a definitive diagnosis. Treatment of PA induced liver damage is supportive because there are currently no specific antidotes and in general, the damage is irreversible. In many cases diagnosis of PA intoxication occurs post-mortem, but if the diagnosis is made in time and feeding of PA's is discontinued, the animals may recover due to the liver's reserve capacity and ability to regenerate.

Summary

Prevention is the best "cure" for PA intoxication. Preventing exposure to PA containing plants often means preventing the growth of the plants in forage. Because fiddleneck and groundsel are principally spring seasonal plants, most problems are associated with first cut hay. Most animals find PA plants unpalatable and will not eat them if better forage is available, but they will ingest toxic amounts of PA plants if they have nothing better. Animals can't select PA plants out of cubes or pellets and in 1980, hay containing PA plants was made into a pelleted feed which was responsible for losses in a feed lot in southern California. In Oregon, ranchers have used sheep to clear the tansy ragwort from their pastures. Apparently there were no problems in these sheep, however in feeding trials, liver damage in sheep was often not seen until after 2 seasons of exposure.

Because the chemical analysis of PA's in plant material is difficult and expensive, the procedure is not readily available for checking samples. Occasional low level exposure would not present the hazard to health that continuous usage would. Ingestion of these alkaloids should certainly be kept at a minimum in humans and livestock. If PA's are fed to lactating animals, low levels of PA's do get into milk, however the health implications of milk contamination by PA's is unknown.