As if a cold wet spring and summer flooding were not adequate adversity for Missouri cattle, a heavy acorn crop, possibly associated with high tannin concentrations, resulted in numerous instances of oak intoxication throughout the state during October, 1993. Pathologists and the toxicologist at the Veterinary Medical Diagnostic Laboratory at the University of Missouri report 25 cases of renal disease associated with known or probable acorn ingestion during October, compared to 3 cases during the previous 7 years. This brings the total reported cases for this year to 26, compared to 13 previously.

Ingestion of acorns or oak buds by cattle is often fatal. The toxic principle is believed to be the gallotannin, which is hydrolyzed by rumen microflora to the toxins gallic acid and pyrogallol. Cattle, sheep, and less commonly goats, swine and horses are victims of acorn ingestion. Deer appear to be resistant.

Attack rates in affected herds during the present month have ranged from 1 to 30% (median 6%). The majority of affected animals (20/31) were less than 1 year of age.

Clinical signs reflect GI and renal dysfunction. Death can occur in as little as 24 hours, or up to 2 weeks following the onset of clinical signs. Cattle have usually been eating acorns for 1-2 weeks before intoxication is initially noted. Non-specific signs of lethargy, anorexia, respiratory distress, hypersalivation and rumen atony are often the first clinical signs to be observed.

This year clinical disease developed in epidemic proportions in early October, and cases have been spread evenly throughout the month. Although losses may decline 1 to 2 weeks after the first hard freeze, our experience in previous years demonstrates that cases can occur during late fall and early winter, especially in southern Missouri.

Tannin intoxication during spring months is usually due to ingestion of immature shoots and leaves, which also have a high tannin content.
In previous years cases tended to be located in southern Missouri, the problem is more widespread during 1993. Cases have thus far been reported from 21 counties.

It is generally stated that cattle do not prefer oak as a feed source, but eat acorns under conditions where poor pasture or harsh weather conditions restrict access to other forage. This year, we have received several reports of cattle becoming intoxicated while on relatively good pasture, without feed restriction. Acorn intoxication is more likely to occur when there is a heavy crop. In addition, immature acorns contain higher concentrations of toxin than mature ones. Wet weather, such as occurred in Missouri throughout much of the summer, delays ripening and means that green acorns may be available for a longer period of time. Environmental factors, such as heavy mid-September rains may have dislodged green acorns from the trees, making them available to cattle. Studies have shown that red oak acorns have a higher concentration of tannins and are more likely to be toxic than those from white oaks. Red oaks appear to be producing more acorns this year and this may further explain the toxicity observed.

After renal damage occurs, the effects of oak intoxication are likely to be permanent, and usually result in death. Although mildly affected animals will make growth recoveries after the acute illness is over, calves with extensive renal damage will remain small and stunted. Thus, prevention is the most efficient means of limiting losses. Cattle appear to acquire a taste for the acorns and may continue to ingest them. When access cannot be controlled, animals can be supplemented ration containing 10-15% calcium hydroxide (hydrated lime). It is recommended that mature cattle be given 4 pounds of this supplement daily and calves be fed 2 pounds. The lime effectively binds tannins and prevents their absorption. When signs of intoxication do occur, supportive treatment and laxatives usually have little effect.

Contributed by Gayle Johnson, Lenny Pace, Stan Casteel, Margaret Miller, John Kreeger, Hames Turk, Lucky Pittman, Sue Turnquist, Keith Bailey and Havey Gosser, UMC VMDL. Reprinted from Under the 'Scope' - Special Edition, November 1993, from the University of Missouri, Veterinary Medical Diagnostic Laboratory, with permission by Dr. Stan W. Casteel.