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Oak Poisoning in Livestock

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Summary

Oak poisoning is a major problem in the production of livestock in areas where oak occurs. The blossoms, buds, young leaves and acorns are poisonous. Cattle, sheep, goats, swine, rabbits and guinea pigs are susceptible to oak poisoning.

A gallotannin isolated from oak has been demonstrated to be poisonous. Calcium hydroxide is an antidote for tannic acid. Calcium hydroxide, supplied in a supplementary feed, is an aid in preventing oak poisoning in cattle.

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Acknowledgements

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MORE THAN 40 SPECIES and numerous varieties of oak are found in Texas (5). Some occur in every section of the state. The buds and young leaves of many of the oaks are poisonous when they make up a major portion of the diet of livestock. The low-growing forms of oaks usually cause the most trouble, and the species *Quercus havardii* is considered to be the most serious offender.

Q. havardii grows in sandy soil from Crane and Ward counties north through the Plains and Panhandle area of Texas. Several counties have reported losing more than 1,000 cattle in a single year. The total annual loss in Texas from sand shin oak *(Q. havardii)* has been estimated to be more than $10,000,000. Total annual loss from all other oaks combined probably would equal this figure.

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In 1936, Boughton and Hardy (3) reported severe losses among cattle and sheep eating *Q. durandii* var *breviloba* in the Edwards Plateau region of Texas. *Q. gambellii* was reported by Marsh *et al.* (16) to be toxic. Losses have been observed by the authors in cattle that have eaten varieties of *Q. incana* (sandjack oak)[1] and *Q. stellata* (post oak) (10).

The authors have observed numerous cases of poisoning in cattle that resembled oak intoxication and were attributed to the ingestion of acorns.

Economic losses attributed to oak occur: When acute poisoning results in a high mortality rate; when chronically poisoned animals remain unthrifty for long periods; when other feed is substituted when animals are removed from oak infested areas; when use of the range is lost during this period; and when grass production is lowered because of the competition with oak for water and soil nutrients.

History of the Problem of Oak Poisoning

Probably the earliest recorded statement on the toxicity of oak as a forage was made by Mascal (17) in 1662, who wrote, "Again oak leaves, if sheep eat thereof green, it is evil for them; especially for young lambs, which will kill them; and likewise of other cattel". The next reference on oak toxicity did not appear until 1893, when Cornevin (6) reported a severe outbreak of oak leaf poisoning in France during the spring. The lesions found in this outbreak were primarily gastroenteritis and nephrocystitis. Hemoglobinuria was reported. Cornevin presented a theoretical discussion on tannin as the toxic principle of oak leaves, but his views were not supported by experimental evidence.

The reported work on oak from 1893 to 1915 was limited to case histories and discussion of forage values. In 1901, Harting (13)

[1]Field observations in 1960.

reported that goats could safely eat oak twigs, but deer and cattle were fatally poisoned. Mackie (15), in 1903, reported on the nutritional value of oak as a forage, but did not mention any poisonous properties of the plant material. The Breeder's Gazette, in 1909, reported a case history of oak poisoning. Glover and Robbins (12), in 1915 proposed that oak poisoning was due to the ingestion of larkspur growing in scrub oak thickets.

The scrub oaks, *Q. gambelii and Q. havardii,* were reported to be toxic by Marsh, *et al.* (16) in 1919. These investigators conducted feeding trials at the Salina, Utah Experiment Station during the summers from 1915 to 1918 and at Monahans, Texas, in the spring of 1917.

Q. breviloba was reported to be toxic to sheep and cattle in 1936 (3).

Towers (22) described signs and lesions of acorn poisoning in cattle which resembled those seen in animals poisoned by oak leaves and buds.

Clark and Cutchin (4) isolated a hydrolyzable tannin from *Q. penduculata* acorns. This tannin was toxic to a rabbit, a calf and to mice when injected intraperitoneally. The lethal dose to mice was of the same magnitude as commercial tannic acid.

For almost 300 years, the toxicity of oaks has been recognized, but the causative agent has been largely speculative. Oak tannins have been suspected since the time of Cornevin, but only limited experimental work has been reported to confirm or refute this hypothesis. Work done at the Texas Agricultural Experiment Station in the past 5 years has demonstrated the toxicity of oak tannin (19).

Clinical Signs and Lesions

The clinical signs of oak poisoning (16) in ruminants usually become apparent 8 to 14 days after the animals start eating the plant material. The first indication is listlessness and loss of appetite. The hair coat becomes rough, and the animals appear gaunt and have a tucked-up appearance. A pronounced constipation occurs frequently followed in 1 to 8 days by a profuse diarrhea. The feces contains mucus and blood. Affected animals have a tendency to stay close to watering places and drink frequently, though only small quantities of water are consumed each time. The animals may become anemic and lose weight. The respiration remains normal during the course of the illness, but the pulse is weak and slower than in healthy animals. The pulse may become more rapid just before death.

Edema may occur with subcutaneous swellings containing a clear gelatinous material and no evidence of hemorrhage. Large quanties of clear fluid also may be present in the peritoneal cavity and in the pericardial sac. The abomasum, or true stomach, usually shows a severe hemorrhagic gastritis. The lesions in the first part of the small intestines usually are similar to those seen in the stomach. The kidneys frequently are inflamed and may contain petechial hemorrhages.

Smith (21) reported the presence of a red staining mass of solid material in some of the proximal convoluted and ascending tubules of sections stained by the hematoxylin and eosin method. He stated that this material replaces the epithelium and occupies the lumen of the tubules. He believed these findings to be an important aid in the diagnosis of oak poisoning.

Recent Studies on Oak Toxicity

A study to determine the cause and control of oak poisoning in cattle was initiated in Texas in 1959. This investigation had specific fundamental objectives: To determine the time of the year and the stage of growth in which the plant could best be used for chemical and feeding studies; to develop suitable methods of collection and storage of plant material for these studies; to determine if a laboratory animal could be used for chemical assay work; to isolate and identify the toxic agent in the plant; to develop an antidote or some method of neutralizing the toxic agent; and to attempt to supply supplemental feeds which would prevent animals from eating sufficient amounts of oak to produce injurious effects.

Practical experience had taught ranchers that animals could more safely be turned into oak pastures when the leaves had lost their tender green tint and had deepened in color. The first spring growth of oak was demonstrated to be more toxic by feeding trials. Rabbits were fed the fresh blossoms, buds and leaves of shin oak collected in the spring and mature leaves collected in November. Of 64 rabbits receiving the fresh spring growth, 32 showed definite signs of oak poisoning and 21 of these died. Those rabbits fed the November growth showed signs of emaciation and 2 pregnant rabbits aborted. No other signs were observed, and all animals gained weight rapidly when placed on other rations.

Buds, blossoms and stems of shin oak were collected by hand as they became available. The plant material was spread on wire racks for air drying or was sealed in plastic bags, then frozen within 2 hours from the time of collection. Drying the oak for 3-5 days did not alter its toxicity for calves or rabbits. Oak collected in the spring and stored frozen was palatable and toxic to rabbits. As the length of storage time increased, the oak became less palatable. After 10 months storage in a deep freezer, the oak was not palatable to rabbits.

Fresh oak was fed to sheep and goats that were nursing lambs and kids, but was withheld from the lambs

and kids. The toxic principle did not appear to be transmitted through the milk. The lambs and kids lost weight, but it is believed that this was due to the decreased milk yield.

Guinea pigs and rabbits also were found to be susceptible to oak poisoning. The clinical signs and pathologic lesions in rabbits were similar to those seen in cattle, except that less severe lesions occurred in the kidneys and more severe lesions were found in the liver. The ease of force feeding rabbits made this animal more suitable for the assay of chemical fractions derived from oak.

A gallotannin was isolated from shin oak in 1962 (19). "Tannin" is a generic name for a group of complex structures widely distributed in the higher plants. The "gallotannins" yield gallic acid when subjected to acid hydrolysis. Commercial "tannic acid" is an example of a gallotannin and is obtained from nutgalls, an insect excrescence on the young twigs of *Q. infectoria* and other allied species.

The oral toxicity of the isolated shin oak tannin was measured by determining the amount necessary to kill 50 percent (LD_{50}) of the assay animals. The multiple dose LD_{50} for the oak tannin was determined with adult rabbits, using four animals per group. Four different levels of oak tannin were administered to the rabbits daily for 5 days. The data were analyzed (2) and the oral multiple dose LD_{50} was calculated to be 6.9 gm./kg./day, for the tannin isolated from *Q. havardii* (19).

As the isolation procedure became more refined, the toxicity of the more purified tannin was increased. The purified tannin produced death in all rabbits in 1-2 days when administered orally at doses of 2 gm./kg./day and death in 75 percent of the animals in 5 days when fed doses of 1 gm./kg./day.[2]

The isolated tannin, purified tannin, fresh shin oak leaves and frozen shin oak leaves were fed to rabbits, and the clinical signs and gross post mortem lesions were compared. The signs and lesions were similar in all cases.

Sick rabbits, which had received 1 gm./kg./day of shin oak tannin[3], were sacrificed at 4-7 day intervals for post mortem examination. The characteristic lesions of oak poisoning were observed.

Purified shin oak tannin and commercial tannic acid were fed to rabbits in parallel studies. The serium tannin levels (18) (expressed as "tannic acid" equivalent) were determined periodically. The times of death of rabbits receiving lethal doses of these materials corresponded to the amounts of tannin consumed and the level of tannin measured in the blood serum.

[2]Dollahite, J. W. and B. J. Camp, unpublished research notes, 1962.

[3]Dollahite, J. W. and B. J. Camp, unpublished research notes, 1963.

Samples of shin oak were collected at several stages of growth from a 1-acre plot near Andrews, Texas. Chemical analyses indicated the following variations in tannin content on a dry weight basis (expressed as percent): April, 15.1; May, 8.7; August, 7.7; and October, 4.2.

From the similar clinical signs and post mortem lesions produced by oak tannin and *Q. havardii* leaves, the oral toxicity of the isolated tannin in rabbits, and the high tannin content of the plant concomitant with the most toxic period of the plant, it was concluded that the tannin of *Q. havardii* was the toxic principle.

During the fall and early winter, from 1959 to 1965, the authors observed numerous cases of poisoning in cattle which apparently resulted from the ingestion of acorns. These cases occurred in the eastern part of Texas and were more numerous in 1965. Typical clinical signs and post-mortem lesions of oak poisoning were observed. The blood-urea-nitrogen (BUN) was elevated, indicating extensive damage to the kidneys.

Evaluations of Possible Antidotes

Commercial tannic acid representing the same class of tannins as isolated from shin oak was used in the preliminary attempts to find an antidote, or neutralizing agent, for oak poisoning. Numerous chemical compounds were administered to rabbits along with tannic acid in an effort to find one with beneficial effects. Ferrous citrate, calcium chloride, calcium lactate, calcium carbonate and calcium acetate failed to provide any protection against tannic acid poisoning (8). Bone meal, defluorinated phosphate, calcium gluconate, and dicalcium phosphate provided a slightly beneficial effect when administered in equal quantities with tannic acid. Calcium hydroxide prevented tannic acid poisoning when administered in the ratio of 1 part calcium hydroxide to 6 parts tannic acid. This chemical reduced the losses when administered in the ratio of 1 to 8. This finding suggested that calcium hydroxide be evaluated as a possible antidote for shin oak tannin.

Numerous feed formulas containing varying amounts of calcium hydroxide were tried to find a combination that was effective, palatable, economical and practical to manufacture. Two feeds, containing 9 percent and 15 percent calcium hydroxide were used in the first evaluation against shin oak poisoning in cattle (9). These feeds were prepared as follows:

	9% Feed	15% Feed	Control Feed
Ground alfalfa	36%	30%	45
41 percent protein cotton seed meal	20	20	20
Rice oil	5	5	5
Calcium hydroxide	9	15	0
Molasses	30	30	30

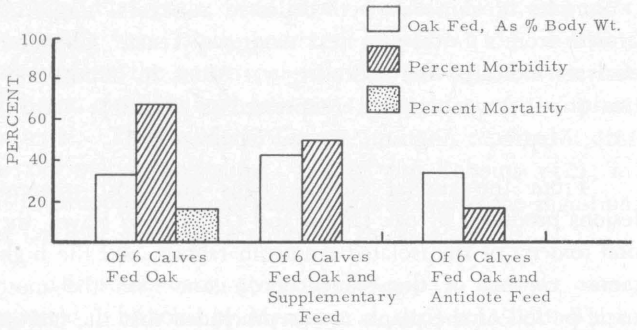

Figure 1. Comparison of average oak consumption, percent morbidity and percent mortality of calves fed *Quercus harvardii* (sand shin oak) and supplements. Antidotal feeds contained 9 percent and 15 percent by weight of calcium hydroxide.

The 9 percent calcium hydroxide feed was made into 3/8-inch cubes and the 15 percent feed made into 3/4-inch cubes for purposes of identification.

Eighteen calves were fed as much fresh shin oak *(Q. havardii)* as they would consume for 19 days. Six calves were fed fresh shin oak exclusively. Four of these animals developed severe signs of intoxication and one died (Figure 1). Six calves were fed shin oak and 3 pounds of control feed daily without calcium hydroxide. Three became ill. Six calves were fed shin oak and 3 pounds of supplemental feeds daily containing calcium hydroxide (hydrated lime). Three animals received the 9 percent lime formula and three were started on the 15 percent formula. Difficulty was encountered in getting the calves to continue eating the 15 percent lime feed as prepared in this formulation. Only one animal ate this feed for the entire feeding period. The other two calves were changed to the 9 percent formula after 10 days, but one calf refused this formula also. By the thirteenth day, signs of oak poisoning developed in this calf. The other calves remained free of oak intoxication.

A similar feeding trial was performed in 1964 to evaluate a 10 percent calcium hydroxide supplementary feed (10). Post oak *(Q. stellata)* was fed during this trial. The antidotal and supplementary feeds were prepared as follows:

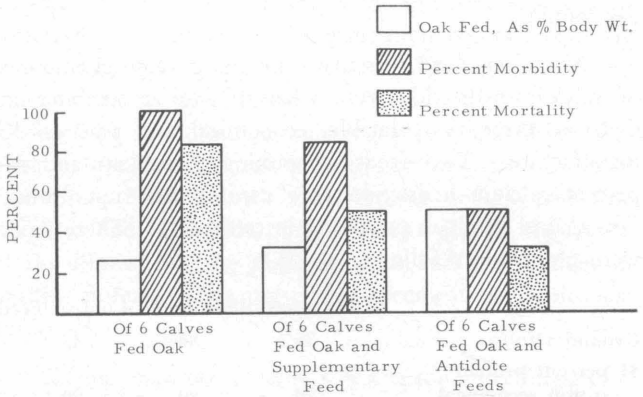

Figure 2. Comparison of average oak consumption, percent morbidity and percent mortality of calves fed *Quercus stellata* (post oak) and supplements. Antidotal feed contained 10 percent by weight calcium hydroxide.

	Supplementary feed, pounds	Antidotal feed, pounds
Cottonseed meal	1,040	1,040
Dehydrated alfalfa leaf meal	600	600
Vegetable oil	160	160
Calcium hydroxide (hydrated lime)	0	200

Eighteen calves of mixed breeding from 8 to 12 months of age and weighing from 160 to 393 pounds were fed as much post oak *(Q. stellata)* as they would consume for 12 days. Six calves were fed an average of 26.5 percent of their body weight on post oak blossoms, buds and leaves. All developed severe signs of poisoning and five died (Figure 2). Six other calves were fed oak in quantities averaging 32.1 percent of their body weight and 1.8 pounds daily of supplementary feed. Five of these developed signs of oak poisoning, and three died. The surviving calves in this group lost weight. A third group of six calves was fed a total amount of oak which averaged 50.2 percent of their body weight and 2 pounds daily of the antidotal feed. Three developed signs of oak poisoning and two died. The surviving calves made substantial weight gains while they were eating oak and after returning to a normal diet.

The calves in this feeding trial were permitted free choice of post oak in all three conditions of feeding. This allowed unusually large quantities of oak to be consumed with a fixed amount of the antidotal feed. The two fatal cases of oak poisoning in the group fed this feed were the animals that consumed the greatest quantity of oak. They ate oak in total amounts which averaged 53.6 and 60.6 percent of their body weight. At these high rates of oak intake the protective ratio of calcium hydroxide to oak tanning probably was exceeded. To better define this ratio it became necessary to compare the feed at two levels of oak consumption.

The 1965 feeding trial evaluated a lime feed containing 15 percent by weight of calcium hydroxide in an attempt to get the animals to eat more of the antidote. The content of vegetable oil was reduced from 8 to 6 percent, since the cubes manufactured in 1964 became soft during storage. This resulted in a much harder cube and reduced the total cost. The following formulations were used in the experimental feedings:

	Supplementary feed, pounds	Antidotal feed, pounds
Cottonseed meal	980	980
Dehydrated alfalfa leaf meal	600	600
Vegetable oil	120	120
Hydrated lime ($Ca(OH)_2$)	0	300

Six calves were used as controls. They were fed free choice post oak buds, blossoms and leaves, and the quantity

of oak they consumed was determined. This same quantity of oak was fed to six other calves together with 2 pounds of the new formulation. This condition of feeding represented one level of oak consumption. The second level of oak intake was established by allowing another group of six calves free choice of oak together with 2 pounds of the antidotal feed daily. A final group of six calves was fed post oak with 1.7 pounds of supplementary feed daily. As is seen in the tabulation, the supplementary feed was the same formulation as the antidotal feed, but it contained no calcium hydroxide. The 1.7 pounds of supplementary feed contained the same amount of nutrients as 2 pounds of the antidotal feed.

The control calves ate an average of 37.9 percent of their body weight of post oak (Figure 3). One calf was unaffected; five developed severe signs of poisoning, and four of these died. The six calves fed supplementary feed daily consumed an average of 41.7 percent of their body weight of oak. Two remained free of signs of post oak poisoning, two became ill but recovered and two died. The calves receiving the antidotal feed and post oak free choice consumed the plant material in an amount which averaged 46.9 percent of their body weight. Five calves were unaffected, but one refused to eat the feed after 1 week, developed signs of poisoning and died. The group of animals receiving antidotal feed together with post oak in quantities approximating that eaten by the control group remained free of signs of oak poisoning.

At both levels of oak intake, the feed containing hydrated lime was adequate in preventing illness and death as long as the calves continued to eat. One calf ate feed and oak in an amount which averaged 60.4 percent of body weight, but no signs of poisoning developed. This amount of oak had been fatal in the 1964 evaluation of a feed containing 10 percent by weight of calcium hydroxide.

With the exception of the calf which refused to eat the medicated feed, all animals fed the antidote and post oak were safely maintained on a diet containing approximately 1 part calcium hydroxide to 1 part oak tannin. This ratio was calculated from the chemical assay of plant material for tannin content and the known amounts of hydrated lime and plant material fed.

The results of these controlled feeding trials indicate that a good supplementary feed will reduce livestock losses from oak poisoning but will not always prevent intoxication. The beneficial effects of a supplementary feed were reflected in a lowered incidence of intoxication and lowered death rate. A good supplementary feed containing hydrated lime had more beneficial effects, as reflected by still lower morbidity and mortality rates (Figures 1, 2 and 3).

The determination of blood-urea-nitrogen proved useful in following the renal impairment subsequent to oak

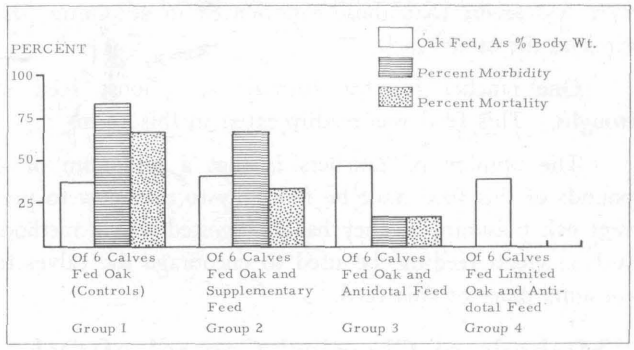

Figure 3. Comparison of average oak consumption, percent morbidity and percent mortality of calves fed *Quercus stellata* (post oak) and supplements. Antidotal feed contained 15 percent by weight of calcium hydroxide.

ingestion. The extent of change in this serum constituent as determined for calves on the different oak and supplemental diets may be seen in Figure 4.

Field trials were made in Andrews County, Texas in 1963, 1964 and 1965 to evaluate 10 percent calcium hydroxide antidotal feeds under range conditions. The formulation used in 1965 contained vegetable oil at a concentration of 6 percent. Approximately 100 tons of the feed were used during these evaluations. On most of the ranches, the feed was supplied to one or more pastures while other types of supplementary feed, such as 41 percent cottonseed cake, 20 percent range cubes and protein blocks were fed in the remaining pastures. In all cases, ranchers reported that cows fed antidotal feed were in better condition at the end of the feeding trial than cows on any other supplementary feed. The cows ate the cubs satisfactorily, but young calves would not always eat their share. Losses in cows eating the feed were practically nil at a time when there were losses due to oak poisoning in adjoining pastures. The losses in calves due to oak poisoning usually

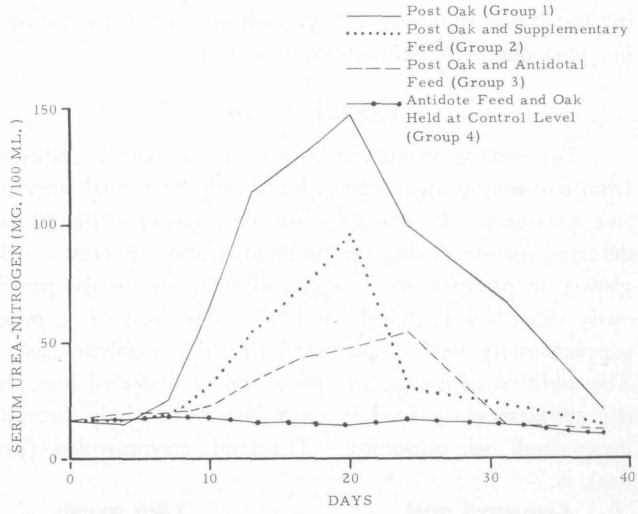

Figure 4. Average daily urea-nitrogen (B.U.N.) values for four groups of calves fed *Quercus stellata* (post oak) and supplements.

were less severe than those experienced in adjoining pastures on the same ranch.

One rancher fed the formula as a loose feed in troughs. This feed was readily eaten in this form.

The opinion of ranchers is that a minimum of 4 pounds of this feed must be fed daily to each cow to prevent oak poisoning. They have suggested that a method, such as creep feeding, be used to encourage the calves to consume more of this feed.

Methods of Chemical Control of Oak

The success of chemical control of oak depends on the consecutive annual re-application of the herbicides, as described below. The growth of grass after chemical spraying is excellent, if moisture conditions are favorable, but regrowth of oak is frequent and complete eradication is rare.

Aerial spray applications of herbicides have been used as an aid in the control of sand shin oak. One-half pound of low-volatile esters of 2,4,5-T in 4 gallons of a diesel oil-water emulsion (1 gallon diesel oil and water sufficient to make 4 gallons) per acre has been an effective aerial spray. Maximum control is obtained when applications are made for 3 consecutive years.

Shin oak on limestone soil does not respond readily to aerial applications of herbicides. Effective control is obtained after two consecutive treatments of 1 pound of 2,4,5-T in 4 gallons of a diesel oil-water emulsion.

Shin oak in tree form, post oak and blackjack oak may be controlled by trunk base application of 16 pounds of 2,4,5-T of low-volatile ester in 100 gallons of diesel oil (20).

Post and blackjack oaks may also be controlled by the aerial spray application of 2 pounds of 2,4,5-T esters in 4 gallons of diesel oil-water emulsion per acre. Repeating the application with 1½ pounds of 2,4,5-T the following year obtains maximum control (7).

Conclusions

The severe economic losses to the cattle industry from oak poisoning can be reduced only by annual preventive measures. If available, oak-free pastures should be deferred for use during the problem season. Excessive oak growth in pastures may be controlled by one of the previously described chemical methods. The use of a good supplementary feed is needed during the problem season. The addition of a calcium hydroxide, or hydrated lime, to the supplementary feed is recommended to help prevent losses from oak poisoning. The feed recommended (per ton) is:

Cottonseed meal	**1,080 pounds**
Dehydrated alfalfa leaf meal	**600 pounds**
Vegetable oil	**120 pounds**
Calcium hydroxide	**200 pounds**

This feed may be used in the form of cubes or in loose form in troughs. A minimum of 4 pounds per cow should be put out daily. Calves should be fed a minimum of 2 pounds per day in a creep feeder.

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