

Clinicopathologic Aspects of Chronic Fluoride Toxicosis in Cattle

James L. Shupe and A. E. Olsen

Veterinary Science

Utah State University

Logan, Utah 84322-5600

Summary

Cattle normally ingest small amounts of fluoride without evidencing adverse effects. Excessive fluorides may be from a variety of combination of sources. Responses to fluorides are influenced by a number of factors. Excessive levels can induce an acute, subacute or an insidious chronic toxicosis. Fluorides accumulate primarily in developing teeth and in bone, where major fluoride-induced lesions occur.

Characterization of clinical signs and lesions and classification criteria have been developed to help standardize the diagnosis and evaluation of fluorosis. Teeth are adversely affected during their development, but bones may be affected throughout an animal's lifetime. Supportive diagnostic criteria and aids are discussed and procedures and techniques that may partially protect cattle from chronic fluoride toxicosis are presented. Recommended tolerance standards for various ages and types of cattle are also given.

Introduction

Fluorides occur universally and may be either beneficial or harmful. Cattle normally ingest small amounts of fluorides without evidencing adverse affects. However, excessive fluoride intake can induce definite clinical signs and lesions.

Several sources may contribute to total fluoride intake. Potential sources include: 1. Forages contaminated by industrial effluents (2), 2. Water with high fluoride content, 3. Mineral supplements that contain excessive fluoride (1), 4. Vegetation contaminated by wind-blown or rain-splashed high fluoride content soil (3), and 5. Combinations of the sources.

Primary lesions of chronic fluoride toxicosis in cattle occur in developing teeth and in bone (9). No pathognomonic lesions attributable to excessive chronic fluoride ingestion have been found in any soft tissue or organs (6, 11, 13).

The biological responses of cattle to ingested fluorides depend on the: amount ingested, duration of ingestion, bioavailability of the compound, species of animal, age of animal, variations of intake level, sufficiency of diet, animal health, concurrent exposure to other toxic substances, stress factors, and individual biologic response (7-11, 14). Pathologic changes in the teeth and bones may reflect either continuous or intermittent fluoride intake (11).

Fluoride tolerance levels have been recommended for different ages of cattle (6-8, 11, 13). Management and feed additives may alleviate the effects of excessive fluoride ingestion (6, 11).

Materials and Methods

Data in this report are from carefully controlled experimental studies and long-term extensive clinical examinations of cattle under routine farm and ranch conditions (5, 12, 13, 15).

One hundred seventy six dairy and beef cattle 4, 6, 8, 12, or 24 months of age were involved in experiments lasting up to 7.5 years. The offspring of these cattle were also studied. Experimental procedures included individual or group feeding and close observations with frequent complete clinical examinations. Metabolism and digestion trials were conducted on some animals and periodic rib biopsies supplied specimens for pathologic evaluation and chemical analyses. Some physiologic tests of liver and kidney function and enzyme analyses were performed.

Fluoride intake was correlated with clinical signs, lesions and animal age. The toxicity of various fluorine compounds was compared. Compounds reported to alleviate fluoride toxicity were tested.

In addition to the experimental animals, 98,775 dairy, beef or crossbred cattle were clinically examined, many annually. Nine-hundred-eighty-eight of these animals were selected, necropsied and evaluated. Dietary samples, including mineral supplements and water, were collected to correlate symptoms and lesions with fluoride intake.

Field examinations were conducted in areas with high fluoride feed levels, and areas with high fluoride water sources.

Results and Discussion

Dental Fluorosis

Fluoride-induced lesions in permanent incisor teeth may include mottling, which appears as alternating white opaque horizontal areas or striations; enamel discoloration, which may vary markedly, hypoplasia which may appear as smaller than normal teeth, dysplasia, which may be indicated by abnormal development of soft chalky enamel or

horizontal zones of constriction; and erosion or pitting of enamel and abnormal abrasion. These lesions are usually bilateral.

Incisor teeth are evaluated and classified according to severity of fluoride-induced effects. These classification categories range from 0-5: 0. Normal: 1. Questionable effect: 2. Slight effect: 3. Moderate effect: 4. Marked effect: 5. Severe effect: Erupting permanent incisor teeth are usually difficult to evaluate.

Dental fluorosis in cattle is usually diagnosed by examining the incisor teeth. Permanent incisor teeth lesions may indicate whether excessive fluoride ingestion has been constant or intermittent. There is a correlation between fluorotic lesions in incisor and cheek teeth that form and mineralize at the same time. Abraded cheek teeth can cause improper mastication and utilization of roughage. Abrasion feeds increase the rate of dental abrasion. Cheek teeth should be examined when diagnosing and evaluating chronic fluoride toxicosis in cattle.

Osteofluorosis

Fluorides have a marked affinity for bone. They normally accumulate and increase gradually throughout an animal's lifetime without any demonstrable changes in bone structure or function.

Excessive ingested fluorides may affect the bones at any time during an animal's life, although bones in young animals are more responsive to excessive fluoride.

The type and severity of bone changes observed in osteofluorosis vary with the 11 factors listed earlier that influence the expression of fluoride toxicosis. One or more of the following conditions may occur: osteosclerosis, osteoporosis, hyperostosis, osteophytosis or osteomalacia. Characteristic changes are associated with osteofluorosis.

The most consistent gross changes are abnormal bone formation on the periosteal surface with thickening of the cortex. Bone changes reflect whether elevated fluoride intake has been continuous or intermittent. Grossly, bones that are severely affected by fluoride appear chalky white with a roughened irregular periosteal surface. In cattle, the first clinically discernible fluoride-induced bone lesions usually occur on the ribs and mandible, and on the medial surface of the proximal third of the metatarsal and metacarpal bones.

Histological signs may include abnormal bone remodeling and mineralization; coarse, haphazardly-arranged collagenous fibers; and excessive osteoid tissue. Osteones may have irregular shapes and sizes with irregular and abnormal distribution of osteocytes (4).

Lameness, Stiffness and Joint Involvement

Non-specific intermittent stiffness and lameness are sometimes observed in severely affected animals. However, they alone are inconclusive measures of fluoride toxicosis. Most animals are often associated with periods of physiologic stress such as late gestation and heavy lactation.

The stiffness and lameness appear to be associated with periosteal overgrowths and ossification of ligaments, tendon sheaths and tendons. These lesions can deter the affected animals from grazing or standing and may subsequently impair performance.

The primary bone lesions associated with fluoride toxicosis are not intra-articular. In contrast, lesions associated with degenerative joint disease (osteoarthritis) or infectious arthritis are primarily intra-articular.

Radiology

Characteristic bone changes have been observed in radiographs of animals that have osteofluorosis. Increased density (sclerosis), decreased density (porosis), periosteal and endosteal hyperostosis, and osteophytosis, or a combination of these bone changes, may occur.

Urine

When supportive clinical evidence is lacking, urinalysis can add to roughly estimate current fluoride intake. The urine of normal cattle ingesting a normal diet generally contains less than 6 ppm F. By itself urinalysis is insufficient to make a definitive diagnosis and evaluation of fluoride toxicosis (12).

Fluoride Retention in Tissues

Elevated fluoride level in bone is one of the most definitive indicators that animals have ingested increased amounts of fluoride. The rate of increase in skeletal uptake usually decreases with age.

Fluoride-induced bone lesions contain more fluoride than normal bone. Analytical results should include a description of the sampling site and whether results are reported on ash weight or dry fat-free weight basis.

Kidneys, which eliminate fluorides from the body, routinely contain higher levels of fluoride than other organs.

Fluoride ingestion levels do not directly affect reproduction although low conception rates and small calf size may occur as a secondary effect in unthrifty animals. Fluorides have not been shown to have teratogenic or carcinogenic effects.

There is little transfer of fluoride from dam to offspring across the placental barrier.

Fluoride levels in milk were low and safe for human and animal consumption. Lactogenesis is not directly affected by fluoride ingestion (15). Milk production decreased only when clinical symptoms and lesions were evident.

Results of experiments with dairy heifers and cows show bone alkaline phosphatase increased in response to increased dietary fluoride and was higher in the induced-abnormal bone (5).

Long-term ingestion of high levels of fluorides may cause dry, lusterless hair and thick non-pliable skin in cattle.

Decreasing Fluoride Toxicity

No known substance will completely counteract excessive

TABLE 1. Tolerance Levels of Cattle for Fluorine.

Animal Type	Pathology*	Water*** mg/1	Performance** mg/kg in dry matter
	Feed mg/kg dry matter		
Dairy or Beef Heifers	30	2.5-4	40
Dairy Cows	40	3-6	50
Beef Cows	50	4-8	50
Fattening Slaughter Cattle	100	12-15	100

*At these levels, characteristic lesions do occur, but the economic effects are minimal. Pathology precedes changes in performance.

**These levels could be fed without clinical interferences with normal performance.

***There is a wide range given for water because of differences in ambient air temperatures and level of physical activity that will affect water consumption.

fluoride ingestion (6, 7, 11). Feeding some aluminum and calcium compounds may ameliorate the effects of fluoride (11). Roughages may be blended to safely maintain fluoride levels below the recommended tolerance levels. All dietary components, including water, should be analyzed and evaluated to ensure that total intake does not exceed recommended tolerance levels (Table 1).

Animals already clinically affected by fluoride toxicosis and with severely affected teeth will not recover completely, but effects may be lessened with proper management.

This paper is published with the approval of the Utah Agriculture Experiment Station as journal paper No. 3234.

References

1. AAFCO. 1984. Official Publication Assoc. Amer. Feed Control Officials, Inc., Charleston, W. Va.
2. Bruns, K.N. & R. Allcroft. 1964. Ministry of Agr., Fisheries and Food Animal Disease Surveys Report No. 2, Part I. Her Majesty's Stationery Office, London, 51 pp.
3. Hobbs, C.S. & G.M. Merriman. 1962. Tenn. Agr. Exp. Sta. Bull. No. 351. Univ. of Tenn., Knoxville, TN, 183 pp.
4. Johnson, L.C. 1965. Fluorine Chemistry, Vol. 4, Acad. Press, New York, 424-441 pp.
5. Miller, G.W. & J.L. Shupe. 1962. Amer. J. Vet. Res. 23(92):24-31.
6. National Academy of Sciences, National Research Council 1974. Washington, D.C. 1-70.
7. Newell, G.W. & H.J. Schmidt. 1958. Amer. J. Vet. Res. 29:363-375.
8. Phillips, P.H., J.W. Suttie & E.J. Zebrowski. 1963. J. Dairy Sci. 46:513-516.
9. Roholm, K. 1937. H.D. Lewis and Co., Ltd. London, p. 364.
10. Shupe, J.L. 1967. IVth Intern. Meeting of the World Assoc. for Buiatrics. Publ. No. 4. Zurich, Switzerland, pp. 15-30.
11. Shupe, J.L. 1983. Proceedings of an International Symposium on Fluorides. Utah State University, Logan, Utah, 1-370.
12. Shupe, J.L., L.E. Harris, D.A. Greenwood, J.E. Butcher & H.M. Nielsen. 1963a. Amer. J. Vet. Res. 24:300-306.
13. Shupe, J.L., M.L. Miner, D.A. Greenwood, L.E. Harris & G.E. Stoddard. 1963b. Amer. J. Vet. Res., 24:964-984.
14. Shupe, J.L. & E.W. Alther. 1966. Handbook of Experimental Pharmacology, Vol. 20, Pt. I. Springer-Verlag, New York, pp. 307-354.
15. Stoddard, G.E., G.Q. Bateman, L.E. Harris, J.L. Shupe & D.A. Greenwood. 1963. J. Dairy Sci. 46:720-726.

Paper presented at the 14th World Congress on Cattle Diseases, Dublin, Ireland, August, 1986.