FLUOROSIS IN HORSES DRINKING ARTIFICIALLY FLUORIDATED WATER

Pixi Macicek,[a] Lennart P Krook[b]

Hitchcock, Texas, and Ithaca, New York, USA

SUMMARY: Quarter Horses drinking water artificially fluoridated at 0.9 to 1.1 ppm over long periods of time developed dental fluorosis. Even when the horses had not been exposed to artificially fluoridated water (AFW) during formation of enamel, brown discoloration occurred and progressed. Pronounced loss of tooth-supporting alveolar bone with recession of bone and gingiva was also present as more severe signs of chronic fluorosis than enamel changes alone. The scene of these events was a lowland part of Texas just above sea level. AFW at these low concentrations can obviously induce chronic fluorosis in its own right without the support of high altitude.

With a change to low fluoride (0.1 ppm) well water, a remarkable improvement was observed in the general health of the horses in a very short time. Most notably, bothersome episodes of colic promptly ceased.

Keywords: Alveolar bone loss; Artificial water fluoridation; Chronic fluorosis; Colic and fluoride; Gingiva recession; Hitchcock, Texas; Hoof deformity; Quarter Horses.

BACKGROUND AND HISTORY

Recently, fluoride poisoning in horses drinking artificially fluoridated water (AFW) with up to 1.35 ppm F has been described.[1,2] These reports were of fundamental importance in bringing the current case from being undiagnosed by ignorance to, hopefully, a rewarding change. Our experience is closely similar to that recorded in these earlier reports but with some differences.

The farm: The farm, owned and operated by the senior author PM, is located in Hitchcock, Texas, about 15 miles north of Galveston, and is about 18 feet above sea level.

Artificially fluoridated water (AFW): In 1999 county authorities took over AFW treatment with hydrofluorosilicic acid. Before that, fluoridation was from "a treatment plant in Houston" with a fluoride concentration ranging from 0.9 to 1.1 ppm.

No other sources of fluoride were present. Fluoride-containing phosphate fertilizer was not used on the pastures, nor were fluoride-containing mineral supplements fed to the horses.

The horses: The horses are Quarter Horses and one Paint Horse. Currently (2008) there are 12 horses on the farm. When the horses were first moved to the farm in 1992, community fluoridated water was the only source of water.

Medical history: Colic stands out as the *piece de resistance*. Colic appeared here and there for no apparent reason. One horse in particular, bred and raised on the farm, suffered from colic once a week over an extended period of time. When he was 3 years old, his hooves became very deformed and abscessed constantly.

[a]Pixi Macicek, 10610 Jay Rd., Hitchcock, TX 77563 USA; E-mail: pmacicek@yahoo.com
[b]For correspondence: Lennart P Krook, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853 USA; E-mail: lpk3@cornell.edu.

There was a period of about three months when he suffered off and on from unmistakable urticaria over his head and body (Figure 1). He has suffered so much from joint and muscle soreness that, throughout his 11 years of life, he has been ridden a total of only about four months.

Figure 1. "Red Bugs Got Money," PM farm. Quarter Horse, 11-year-old stallion, on AFW from age 1 year. Note the raised, gray vascular reactions all over the body, typical of urticaria.

Most of the horses have been diagnosed with low thyroid function. Three horses have stopped sweating. Two horses have suffered from anemia, one chronic, the other acute and severe. All horses have developed hoof deformities, some to a severe degree. Two horses have suffered leg fractures; one horse had to be destroyed. Legs have been growing crooked. Ligaments have become hard, tight, and contracted.

Lameness was often the reason for veterinary consultation. The lameness was thought to be caused by barrel racing (PM is a rodeo competitor). The lameness did not respond to conventional treatment but got worse. Without sound horses, PM has not been able to compete for the past two years.

Examinations by veterinarians never included inspection of teeth.

Treatment was attempted in one case, "Jaguar Jones," a sorrel Quarter Horse (roping and barrel horse), born in 1990 and on AFW since age 7 years, had a blood test in 2004. Actual values of the thyroid hormone tests were not reported, but treatment with high iodine medication was nonetheless given. The horse became a "better horse within weeks." That horse had a low erythrocyte count of 2.97×10^6/μL, but treatment with iron injections was unsuccessful.

PRESENT INVESTIGATION

The teeth: Photographs of the incisor teeth of all 12 horses on the PM farm were taken and examined, as were those of four horses from a neighbor's farm. Horses of that farm, one mile away and on the same community fluoridated water, had exactly the same lameness problems. The horses were examined by veterinarians as often as every week. A diagnosis was never reached. As in our earlier reports,[1,2] the veterinarians did not examine the teeth.[3]

In the photographs, dental fluorosis was present in all cases to various degrees and in various expressions. Enamel hypoplasia presented itself as defects, especially at the gingival margin, i.e., the enamel last formed (Figures 2, 3, and 4). Note that these horses were on AFW during the time of amelogenesis of the permanent incisor teeth.

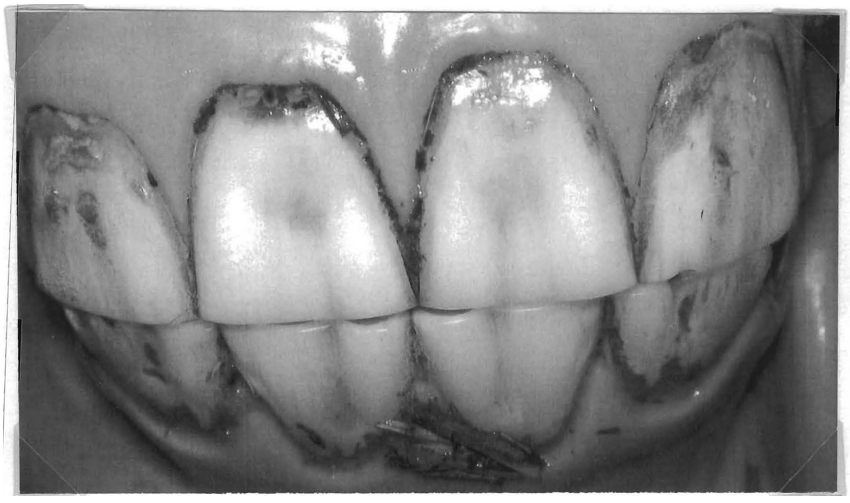

Figure 2. "Wallig," neighbor farm. Incisor teeth of a 6-year-old thoroughbred gelding, on AFW since age 5 months. Extensive areas of enamel hypoplasia are seen at the gingival end of the maxillary teeth. There is slight brown discoloration of the enamel, notably of the second incisor teeth. Slight recession of the alveolar bone with exposure of the root and slight bulging of the gingiva is visible at left I_2.

The teeth in Figure 4 are those of a 15-year-old horse on AFW all his life. The toxic effects by F on the odontoblasts resulted in smaller teeth than normal. The maxillary incisor teeth were fan-shaped rather than rectangular (normal) with the same width at the bottom as on the top. The mandibular incisor teeth were smaller with resulting diastemata between the teeth.

Brown discoloration of the enamel ranged from minimal (Figure 3) to very severe (Figure 5). That horse, it should be noted, had not been on AFW during amelogenesis. The dentin exposed on the masticatory surfaces by abrasion showed brown discoloration (Figure 4).

Recession of the alveolar bone was observed in all cases and ranged from mild (Figure 2) to profound (Figure 3). There was corresponding exposure of the proximal part of the roots and bulging of the gingiva.

.

Figure 3. “Buck,” neighbor farm. Incisor teeth of a 14-year-old Quarter Horse, on AFW for 9–10 years. Note the very severe bulging of the gingiva and pronounced recession of the alveolar bone of all four maxillary teeth with corresponding exposure of the roots.

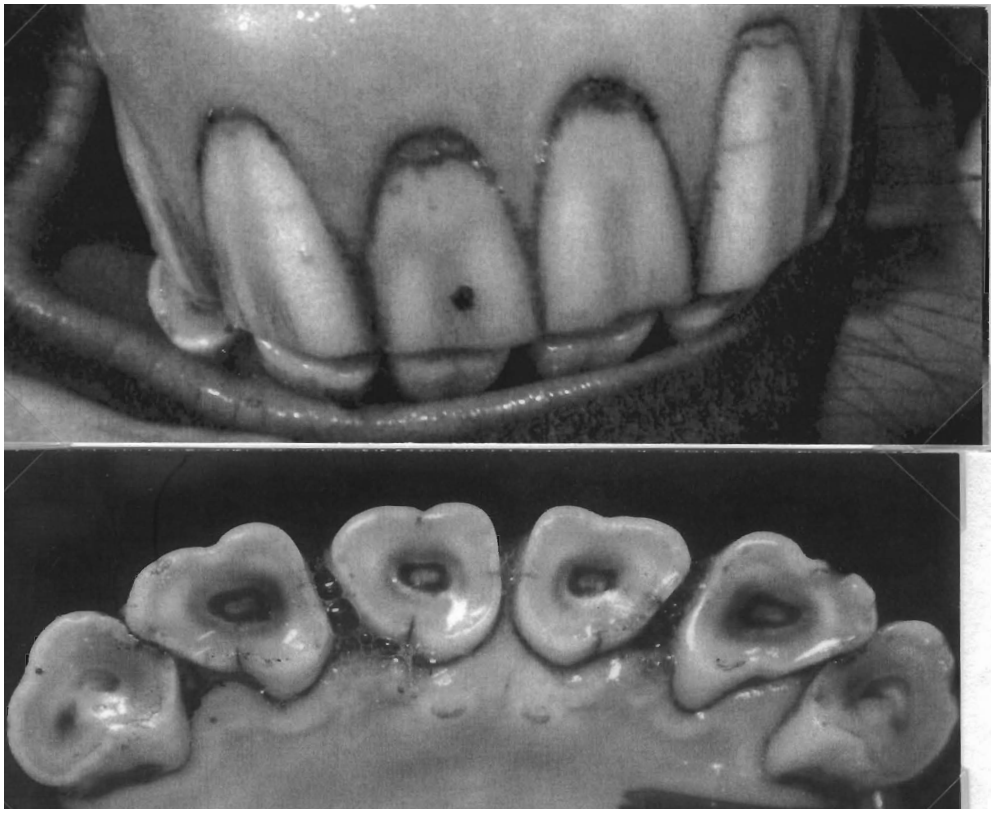

Figure 4. “Pert,” neighbor farm. A 15-year-old gelding Quarter Horse, on AFW all his life. The maxillary incisor teeth are fan-shaped. There is pronounced hypoplasia of the distal end of the enamel with exposure of the roots. The gingiva is slightly bulging at left I_1. The mandibular incisor teeth (lower part) are hypoplastic with diastemata between the teeth. There is also brown discoloration of the dentin next to the pulp cavity.

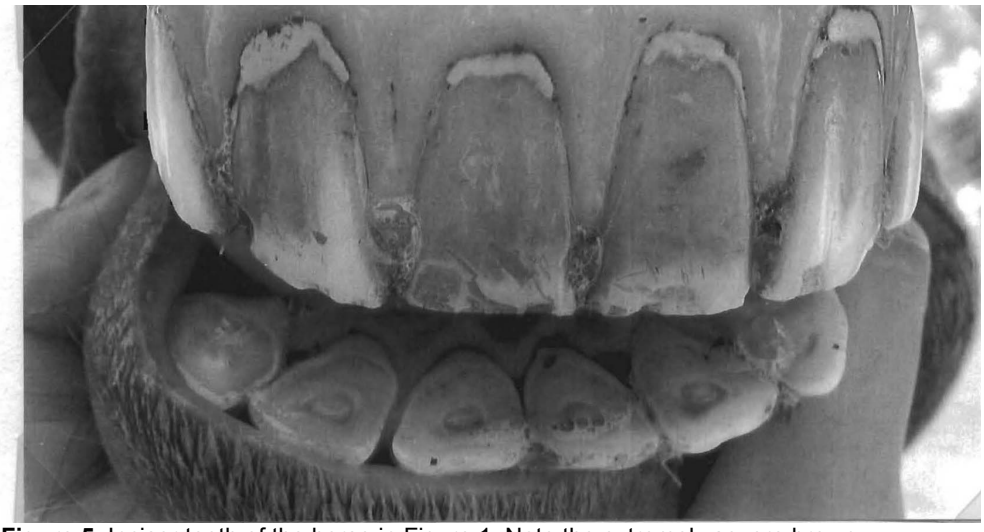

Figure 5. Incisor teeth of the horse in Figure 1. Note the extremely severe brown discoloration of the enamel of the maxillary teeth. The exposed parts of the roots are covered by a grayish amorphous mass.

Change in water: Recently, a well was drilled on the PM farm, and the horses began consuming water from this well on February 22, 2008. As analyzed by Accutest Lablink Gulf Coast in Houston, Texas, the water contained 0.10–0.11 ppm F. All horses started to drink more water than ever before. There were no further cases of colic. A huge decrease in muscle and joint stiffness was noted. Hooves began to grow normally, ligaments appeared to be softening, and the overall appearance of the horses was significantly improved.

The stallion shown in Figure 1 was ridden after 10 weeks following being off AFW and was traveling well. He was schooled on the barrels and was able to rate at the barrel without his back legs buckling at the fetlock!

DISCUSSION

At hand are:

• A source of fluoride – artificially fluoridated water (AFW);

• A similar history of damage to horses on two farms, which is identical to those established in fluorosis in horses on AFW;

• A documentation of dental fluorosis in the current cases – the pathognomonic signs of fluorosis, i.e., chronic fluoride poisoning.

The response to removal of AFW provides a most eloquent confirmation fluoride as the cause of observed disorders. Moreover, the low thyroid hormone levels and the anemia documented in the current horse material equate these symptoms of fluorosis to those recorded in cattle.[4]

Examination of the teeth showed that the brown discoloration of the enamel and rapid progression thereof can occur in teeth not exposed to F during amelogenesis. The rapid increase in brown discoloration has also been found in cattle.[5] These

findings clearly discredit the widely-held assertion by a US Federal agency: "Teeth that have erupted, however, are not influenced adversely by subsequent fluoride ingestion."[6]

The increase in brown discoloration of the enamel is explained by the rapid incorporation of ions as described in experiments with monkeys.[7] Intravenously injected isotopes (^{135}I, ^{32}P, and others) were traced to the enamel within five minutes, and the concentration of the isotopes was greater in the *external* than in the internal enamel layer.

Recession of alveolar bone and the gingiva is a very important expression of chronic fluorosis. In the rat, alveolar bone has the greatest turnover rate of any bones in the skull.[8] Alveolar bone cells are, accordingly, very sensitive to F exposure. Atrophy of osteoblasts and even necrosis of osteocytes can, alone or together, cause osteopenia. Furthermore, although enamel formation occurs only before eruption of teeth, bone turnover is life-long. Thus, alveolar bone and gingiva recession can occur with or without changes in the enamel.

Finally, with respect to fluorosis and altitude, there is consensus that altitude is a significant risk factor.[9,10] In other words, even with less F, there is increased risk of dental fluorosis at higher altitudes.

F poisoning in horses drinking water fluoridated up to 1.35 ppm has been described.[1,2] The home of the horses, Pagosa Springs, Colorado, USA, is at 7200 feet above sea level,[11] and this has been proposed as a factor to explain how 'safe' F could cause severe fluoride poisoning. In the present study, the altitude was slightly above sea level, and the horses were drinking AFW at the same, or lower, concentrations than those in Pagosa Springs. We do not know if the horses drank more fluoridated water because of higher temperatures or high humidity, but it can certainly be stated that artificial fluoridation at the 'safe' level of 1 ppm did not need high altitude to induce chronic fluoride poisoning in horses.

The report, *Effects of fluoride in animals*,[6] an official publication by a US Federal agency, states that horses can tolerate 60 ppm F in the total feed. The absurdity of this figure has been analyzed and refuted.[1-3]

ADDENDUM

When the AFW was replaced by low-F well water, not only did the horses recover but owner PM did, too. Up to the end of January 2008, she had recurring migraines every six weeks like clockwork for four years. These caused her to be immobilized for 8 hr with headache and vomiting. She had not been drinking the AFW, but every day she had used it to prepare and consume two pots of coffee. When the horses were diagnosed with fluorosis, she then switched to the low-F well water and has not had any migraines since.

REFERENCES

1 Krook LP, Justus C. Fluoride poisoning of horses from artificially fluoridated water. Fluoride 2006;39:3-10. Correction: Fluoride 2006;39(2):156.

2 Justus C, Krook LP. Allergy in horses from artificially fluoridated water. Fluoride 2006;39:89-94.

3 Burgstahler AW. Editorial: Failure to diagnose fluoride poisoning in horses caused by water fluoridation. Fluoride 2006;39:1-2.
4 Hillman DD, Bolenbaugh L, Convey EM. Hypothyroidism and anemia related to fluorosis in dairy cattle. J Dairy Sci 1979;63:416-23.
5 Krook L, Maylin GA, Lillie JH, Wallace RS. Dental fluorosis in cattle. Cornell Vet 1983;73:340-62.
6 National Academy of Sciences – National Research Council Committee on Animal Nutrition, Subcommittee on Fluorosis. Effects of fluoride in animals. Washington, DC: US National Academy of Sciences; 1974.
7 Sognnaes RF, Shaw JH, Bogoroch R. Radiotracer studies on bone, cementum, dentin and enamel of Rhesus monkeys. Amer J Physiol 1955; 80:408-20.
8 Baumhammer A, Stallard RE, Zander, HA. Remodeling of alveolar bone. J Periodont 1965;36:439-42.
9 Yoder KM, Mabelya L, Robison VA, Dunipace AJ, Brizendine EJ, Stookey GJ. Severe dental fluorosis in Tanzanian population consuming water with negligible fluoride concentration. Community Dent Oral Epidemiol; 1998;26:382-93.
10 Rwenyonyi C, Bjorvatn K, Birkeland J, Haugejorden O. Altitude as a risk indicator of dental fluorosis in children residing in areas with 0.25 and 2.5 mg fluoride per litre in drinking water. Caries Res 1999; 33:267-74.
11 Justus C. Personal communication. 2008.