FLUORIDE POISONING OF HORSES FROM ARTIFICIALLY FLUORIDATED DRINKING WATER

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SUMMARY: On a farm in Pagosa Springs, Colorado, Quarter horses consuming artificially fluoridated water (up to 1.3 ppm F) for extended periods of time developed classical symptoms of chronic fluoride intoxication including dental fluorosis, crooked legs, hyperostosis and enostosis, hoof deformities, and microscopic evidence of reduced bone resorption. These symptoms began to appear about two years after fluoridation started in 1985 and gradually became more severe. Representative postmortem F concentrations in dry matter bone were: 587 ppm in a 20-year-old gelding drinking F water for the last 10 years; 936 ppm in a 17-year-old gelding on F water for the last 11 years; and 757 ppm in a 21-year-old mare on F water all her life. Blood serum thyroxin in a 19-year old mare (T_4) was "very low", and interference with reproduction was noted after five years. Radiographs of the third metacarpal bone revealed osteomegaly with thick lamellae from both ends extending throughout the medullary space. The levels of F ingestion and the bone F concentrations of these horses are far below those claimed to cause F intoxication in cattle. After fluoridation was terminated on March 29, 2005, colic gradually ceased and other significant improvements have occurred.

Keywords: Artificially fluoridated water; Chronic fluorosis; Fluoride poisoning of horses.

INTRODUCTION

Although the literature on fluorosis in cattle is extensive, information about fluorosis in horses is almost nonexistent. A 1974 US National Academy of Sciences–National Research Council report on effects of fluoride in animals gave only one reference to horses in which dental and radiographic skeletal changes were described in horses grazing in areas where cattle and sheep had developed severe fluorosis.[1] The report stated: "No carefully controlled studies have been conducted to determine the effect of excessive fluoride ingestion on horses." Here we present evidence that artificial water fluoridation can cause serious fluorosis in horses.

MATERIALS AND METHODS

Background: Most of the horses in this study belong to the Justus Farm in Pagosa Springs, Colorado. Over the years this farm has included up to ten Quarter horses, numbering six in the year 2005. In 1985 artificial water fluoridation was introduced by the Pagosa Area Water and Sanitary District at a concentration ranging from 0.35 to 1.3 ppm F. This water was the only source of water for the horses on the farm. No other sources of fluoride were present; fluoride-containing phosphate fertilizer was not used on the pastures, nor were fluoride-containing mineral supplements ever fed to the horses.

Clinical observations: An early sign of intoxication was seen in a regular incidence of colic in all the horses, which promptly disappeared when the affected

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horses were removed from the farm. After about five years, hoof deformities reaching the extreme shown in Figure 1 were seen. Moreover, crooked legs were observed in some horses, as were exostoses in six horses, especially in the distal MCIII (third metacarpus) but also in the back and rib bones. Also after about five years, low conception rates were noted, and one mare delivered a fetal monster with partial aplasia of the skull. In a 19-year-old mare the serum thyroxin (T_4) was found to be "very low."

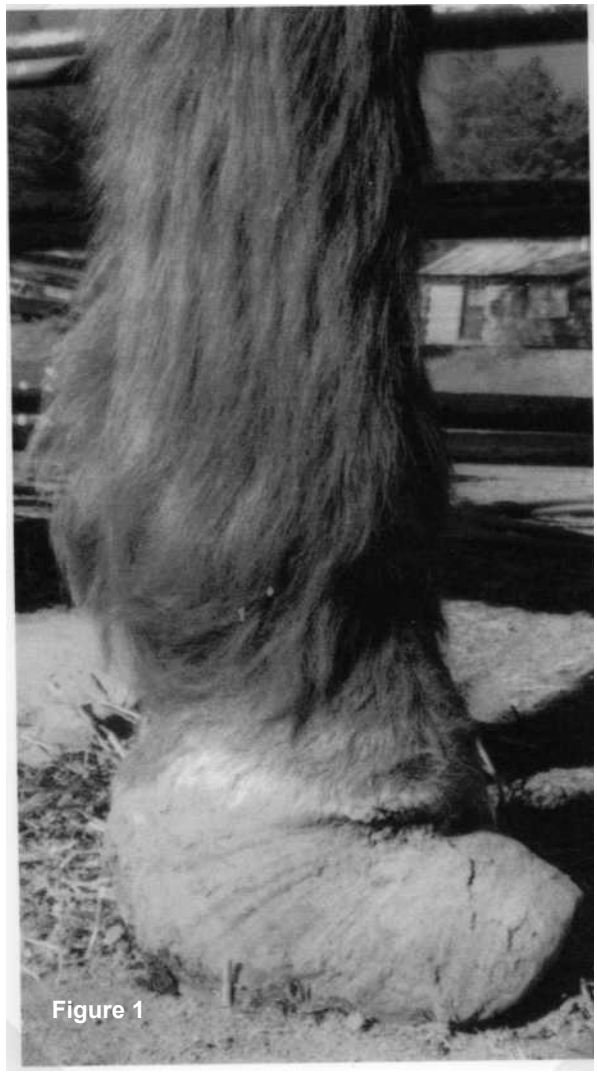

Figure 1. Severe hoof deformity in left thoracic limb of 22-year-7-month-old Quarter horse mare on fluoridated water for 21 years.

After many years, the farm owner, co-author CJ, suspected fluorosis, which, however, was not confirmed by local veterinarians. Remarkably, clinical examinations of the horses by the veterinarians apparently never included the teeth, since their records show nothing about dental conditions. Over the years, five horses had to be sacrificed because of ailments that, in retrospect, appear to have been induced by fluoride.

On March 29, 2005, water fluoridation of the Pagosa Springs area was terminated. Since then, colic among the horses has gradually entirely ceased, and other significant improvements have been noted.

Laboratory examinations: When the senior author LPK was engaged to investigate the problem, he requested photographs of the teeth of horses on the Justus farm as well as the most easily accessible MCIII bone specimens from any horse on the farm terminated for whatever reason. Dental photographs were supplied together with MCIII samples from three Quarter horses, one from the Justus farm and two from nearby farms using the same Pagosa Springs fluoridated water. One "control" MCIII sample was obtained from a "very old" thoroughbred horse at the necropsy laboratory of the Cornell University College of Veterinary Medicine. For the radiographs the MCIII

samples were split longitudinally in the dorso-palmar midline and radiographed on fine screen Kodak Lanex Fine TML film at 300 mA and 62 kV for 0.1 sec.

PRESENT FINDINGS

Dental disturbances: Figures 2–4 illustrate dental changes observed in the F-exposed horses with increasing age.

Figure 2 shows the incisor teeth of a Quarter horse foal 2 years and 10 months old. The foal came to the Justus farm at age 7 months. The photo shows the permanent central pair of incisors with extensive enamel defects in the maxillary teeth.

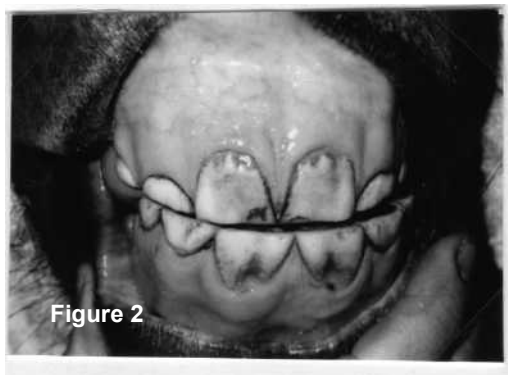

Figure 2. Incisor teeth of 2-year-10-month-old Quarter horse foal introduced to farm with fluoridated water at 7 months of age. Erupted permanent central incisors have extensive enamel defects distally of the maxillary teeth.

Figure 3 shows the incisor teeth of a 6-year-8-month old Quarter horse gelding that drank fluoridated water from birth. All teeth have severe brown discoloration, and the enamel of these areas is thinner and has receded from the surrounding less-disturbed enamel. The maxillary gingiva is recessed, and the exposed distal enamel exhibits extensive defects. The mandibular gingiva has receded and is bulging. The entire masticatory surface of the mandibular teeth has a severe brown coloration.

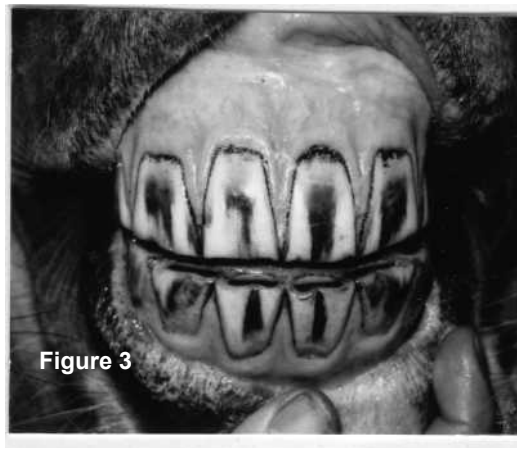

Figure 3. Incisor teeth of 6-year-8-month-old Quarter horse gelding on fluoridated water from birth. There is severe brown discoloration of the central enamel of all teeth, and this enamel is thinner and has receded from surrounding enamel. There is also recession of the maxillary gingival, and the exposed distal enamel shows extensive defects. The mandibular gingival has receded and is bulging, and the entire masticatory surface of the mandibular teeth exhibits severe brown discoloration.

Figure 4 shows the teeth of a 23-year-8-month old Quarter horse mare that has been on fluoridated water for 21 years. Brown discoloration of the enamel and extensive defects of the distal enamel of the maxillary teeth are present. A severe loss and recession of apical alveolar bone resulted in exposure of the distal clinical crown and the upper part of the roots of the maxillary teeth along with recession and bulging of the gingiva of the mandibular teeth.

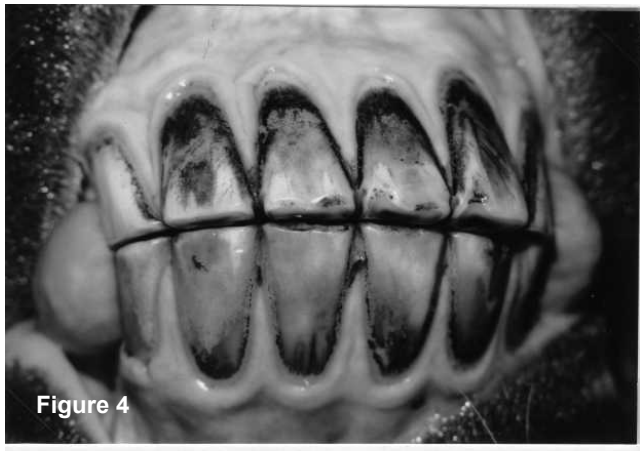

Figure 4. Incisor teeth of 23-year-8-month-old Quarter horse mare on fluoridated water for 21 years. There is brown discoloration of the enamel with extensive defects of the distal enamel of the maxillary teeth. Severe loss and recession of apical bone have resulted in exposure to the distal clinical crown and the upper part of the roots of the maxillary teeth, together with recession and bulging of the gingiva of the mandibular teeth.

Bone fluoride and radiographs: Three MCIII (thoracic leg third metacarpus = "cannon bone") samples were submitted for fluoride analysis by CJ from terminated Quarter horses (QH) at the Justus farm. A control MCIII sample was obtained by LPK from a "very old" thoroughbred gelding at routine necropsy at the Cornell University College of Veterinary Medicine. Fluoride in dried bone tissue was determined by the fluoride ion electrode method for measurement of F ion activity in standard and sample solutions. The analytical results for these samples are tabulated below.

Table. MCIII bone fluoride analyses of four deceased horses performed in 2005

Horse	Age (years)	Fluoridated water	Dry bone F (ppm)
QH mare	21	All her life	757.1
QH gelding	20	Last 10 years	587.1
QH gelding	17	Last 11 years	936.1
Thoroughbred	"Very old"	Never	162.2

The radiographs of MCIII bone samples from the thoroughbred "control" horse and the 17-year-old Quarter horse gelding in the table above are presented in Figure 5. In the control horse the subchondral bone plate is well differentiated from the lamellar epiphyseal bone. The metaphyseal lamellae become gradually thinner and disappear well below the midshaft. Especially noteworthy is the sharp demarcation of the cortex from the medullary cortex. By contrast, the radiograph of the MCIII sample from the fluoride-exposed 17-year-old QH gelding shows that the subchondral bone is blended diffusely with the epiphyseal bone. The metaphyseal trabeculae remain thick and extend through the entire medullary cavity. The cortical surface of the medulla is less sharply defined than in the control, most strikingly at the upper palmar cortex.

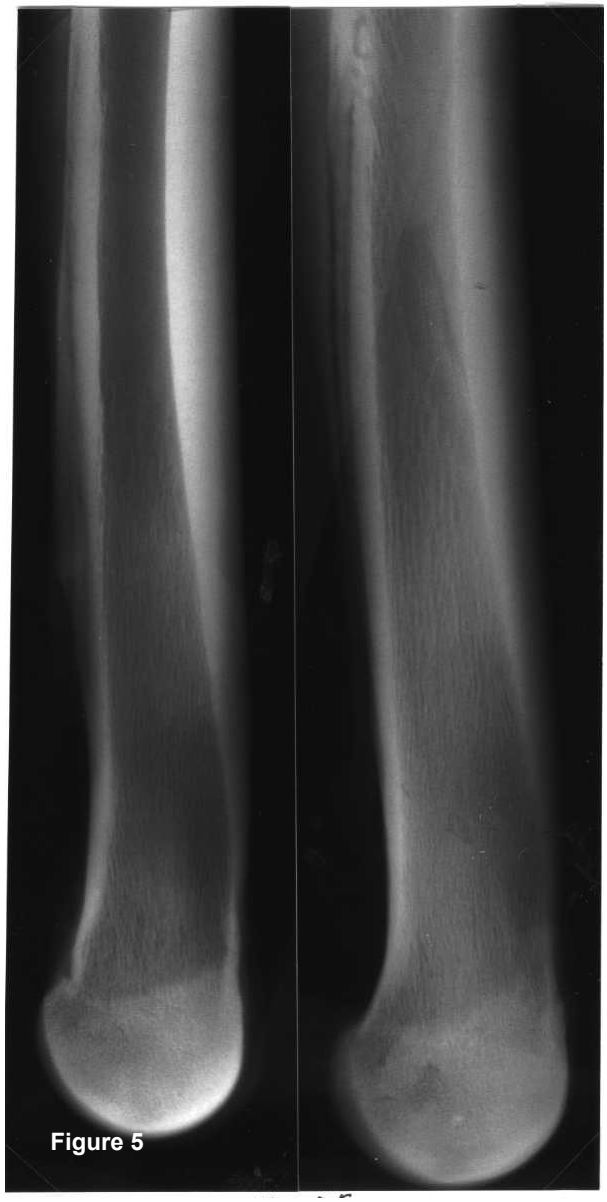

Figure 5. Radiographs of lower two-thirds of left thoracic third metacarpus (MCIII) cut longitudinally at the dorso-palmar midline (palmar is to the left). The left radiograph is from an old thoroughbred horse (routine necropsy at Cornell College of Veterinary Medicine); the right radiograph is from a 17-year-old Quarter horse gelding on fluoridated water for the last 11 years.
Left: The subchondral bone plate is well defined from the lamellar epiphyseal bone. The metaphyseal lamellae become gradually thinner and disappear at the lower half of the picture. The cortex is sharply demarcated from the medullary cavity.
Right: The subchondral bone plate blends diffusely with the epiphyseal bone. The metaphyseal trabeculae remain thick and extend throughout the entire medullary cavity. The cortical surface facing the medulla is less sharply defined, most eloquently so at the upper palmar cortex.

Figure 6. Photo of the left MCIII of 21-year-old Quarter horse mare on fluoridated water all her life. The bone is cut lengthwise in the dorso-palmar midline with the lower end, not including the joint cartilage, at the bottom. The dorsal contour is to the left. The dorsal cortex of the wall bulges severely into the marrow space, beginning just proximal to the epiphysis, creating endosteal hyperostosis “enostosis”. The added bone is less dense than the original cortex; the contour of the original cortex is well defined.

As seen in Figure 6 for the 21-year-old Quarter horse mare in the Table, the poor definition of the inner surface of the MCIII cortex reaches a more extreme degree than in the 17-year-old Quarter horse gelding depicted in Figure 5. In the 21-year-old mare, the inner surface of the dorsal wall of the left MCIII bulges severely in the narrow space, beginning just proximally to the metaphysis. The bone of this endosteal hyperostosis is less dense than the original cortex, the contour of which is obvious.

Examination of microscopic sections of MCIII samples of lamellar as well as compact bone of the fluoride-exposed horses shows retarded activity of resorbing osteocytes, and the normal basophilia of the adjacent matrix has receded into sharply defined bands giving the field a mosaic appearance. Failure of normal resorption also caused osteosclerosis as seen radiographically in Figure 6. Likewise, the endosteal hyperostosis was evidently the result of failure of normal maturation of the lamellar bone which, instead, is piled up in multiple layers.

DISCUSSION

The foregoing clinical and morphological observations, together with the bone fluoride analyses, establish the diagnosis of chronic fluoride intoxication of horses in this study caused by consumption of artificially fluoridated drinking water.

The curled hoof of one horse seen in Figure 1 mimics the hoof lesions found in 88 (57.9%) of 152 cows fed fluoride-containing phosphate mineral supplements.[2] The same report also provided conclusive proof that F interferes with thyroid function in cows, since lowered serum T_3 and T_4 hormones correlated significantly with urinary F concentrations. The "very low" serum T_4 found in one of the horses in the present study further supports the diagnosis of chronic fluorosis.

Also beyond question are the pathognomonic indications of dental fluorosis lesions seen in Figures 2–4.

As noted earlier, the F concentration in the fluoridated water ranged from 0.35 to 1.3 ppm. The later 1978 NAS report on "Nutrient Requirements of Horses"[3] states that the need for water intake in horses is 2 to 4 times that of dry matter intake, and the F tolerance levels are all based on dry matter intake. The equivalent ppm F in the dry matter would then be 4 x 1.3 = 5.2 ppm. The same report[3] continues: "Work may increase the water need about 20–30 percent, and lactation may increase it 50–100 percent above maintenance." If we consider the most extreme situation with the Pagosa horses, their intake of F in ppm of dry matter would be only a small fraction of the so-called "tolerance level" of **SIXTY** (60) ppm F of dry matter, cited by NAS.[1] This 60-ppm figure first appears in a 1974 article by Shupe and Olson titled: "Clinical Aspects of Fluorosis in Horses"[4] and concerns "breeding and lactation animals." The same figure is then repeated in the 1974 NAS-NRC publication *Effects of Fluorides in Animals*[1] this time as the "tolerance level" for "performance" defined as "Levels that, on the basis of published data for that species, could be fed without interference with normal performance." That publication further informs: "Analyses of pastures in these [fluorosis] areas (Shupe, 1972a, b) suggests that 60 ppm F is the tolerance levels for this species [horses]." The "Shupe, 1972a" is reference 5 of the current paper, but it contains

no mention whatsoever to support the above NAS-NRC statement.[1] Shupe's reference 1972b is reference 6 of the current paper. It too presents the same vacuum of its claimed support for the NAS-NRC 60-ppm figure.

Thus there is no justification for NAS to state that the "tolerance level" for F in horses is 60 ppm on a dry matter basis. On page 52 of its NRC Committee on Animal Nutrition report,[1] we read: "No carefully controlled studies have been conducted to determine the effects of excess fluoride ingestion in horses." Yet, three pages later, Table 4 in the report lists a "tolerance level" of 60 ppm F in horses as based on published data for that species. The second statement contradicts the first, and, as seen here, is in obvious conflict with the truth.

As is well known, when sufficient circulating F is present it promptly changes the mineral phase of hard tissue by replacing hydroxyl ions in calcium hydroxyapatite to form calcium fluoroapatite. The F so trapped is toxic to the respective cells of the hard tissues. In acting on the enamel ameloblasts F causes the dental fluorosis lesions shown in Figures 2 and 3. In acting on bone tissue F first affects the bone resorbing cells, resulting in osteomegaly. Over time and/or with greater F exposure, bone-forming cells are also affected, resulting in osteopenia.[7] Since the metabolic rate is higher in alveolar bone than in skeletal bone tissue at other sites,[8] the alveolar bone first shows evidence of a very important sign of chronic F poisoning, seen in recession of the alveolar bone and gingival so eloquently depicted in Figure 4.

Although enamel changes in dental fluorosis are acknowledged by NAS-NRC, its report[1] advocating a 60-ppm F tolerance in horses overlooks and fails to recognize that "The tooth should not, during clinical and morphological examinations, be considered as only a tooth, but as part of a functional unit, a unit which, in addition to the tooth, includes the supporting tissues such as the Sharpey's fibers, the transseptal fibers, the gingiva and, above all, the alveolar bone."[9] Thus, loss of alveolar bone with recession of gingiva tissue as observed here is a sign of far more advanced fluoride intoxication than enamel changes only.

In closing, it must be asked: How could the Subcommittee on Fluorosis, appointed by a US Federal authority, convey such blatant misinformation on F tolerance in horses and fail to note a critically important aspect of dental fluorosis? The subcommittee consisted of four biochemists and one veterinarian, at least four of whom had financial support from F polluting industries, and it was on the basis of "research" sponsored by these industries that the tolerance levels of F for cattle and horses were proposed by the committee. By sending out the skeletal material for description by a pathologist,[10] the subcommittee acknowledged its inability to examine the most important expression of F intoxication in animals.

It seems that the only beneficiaries of NAS-NRC (mis)information on fluorosis in animals are the F polluting industries by which and for which it was evidently written.

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