RACEHORSE BREAKDOWNS AND ARTIFICIALLY FLUORIDATED WATER IN LOS ANGELES 2013

SUMMARY: The incidence of racehorse fatalities at two Southern California racetracks before and after providing fluorosilicic acid treated water supplies correlated with fluoridated water consumption and increased breakdowns. Possible contributing factors involved are discussed. Known toxicologic effects of fluoride that could contribute to breakdowns are presented. A working hypothesis is that fluoride ingestion under certain conditions can lead to increased breakdown incidence.

Key Words: Fluoridated water; Fluoride in bone; Hydrofluoric acid; Racehorse breakdowns; Silicic acid; Stomach ulcers

Although 99% of racing starts at horse racetracks are successful, fatalities have been steadily rising in the USA with 5,000 horses being killed from 2003–2008 with most being euthanized after bone breaks.[1-4] This prompted Congress to hold hearings, to recommend alterations in racing practices, and to find causes.[2-4] Because fluoride from synthetic sources, including sodium fluoride (NaF) and fluorosilicic acid (H_2SiF_6), is a known calcium chelator,[5,6] and horses turn over whole body water in a few days, it is prudent to examine whether materials used to artificially fluoridate water supplies could be involved.

Fluoride is not a normal component of the horse bloodstream and has no physiologic role in the horse. Ingested fluoride could possibly impair racehorse performance by decreasing calcium assimilation or by direct toxicity. Studies on research animals and man indicate that long-term consumption of fluoride from water with sufficient elevation of blood fluoride can cause anemia,[7] bone weakening following incorporation into bone,[6,8] and alterations in lung[9] and brain[8]. Fluoridated calcium-deficient water over many years caused pathology and lethality in horses.[10-12]

RESULTS

Results of an observational study of quarter horses at the Los Alamitos racetrack as a function of time are shown in Figure 1. This is the only track in the state that retained a dirt surface throughout the entire study period. The observations, on age-matched, 2–6-year-old, horses with well regulated diets, are prospective and the results are compared for the seven years before the commencement of the fluorosilicic acid treatment of water supplies in November 2007 and for the following five years. All horses at the track consumed municipal water. Water fluoride was increased five-fold from 0.2 ppm of natural fluoride to a total of 1.0 ppm, including artificial fluoride from fluorosilicic acid, and the breakdown incidence increased precipitously. The last data value in 2011 represents 7 fatalities per 1,000 racing starts. The peak breakdown incidence was the highest in the country at 10.8 fatalities per 1,000 starts. In a two-year period 186 horses perished at Los Alamitos. After the fluoride level was reduced in February 2011, following a recommendation of the US Health and Human Services, there was a

downward trend in the fatalities. In 12 years, for 170,000 racing starts, there were 708 fatalities.

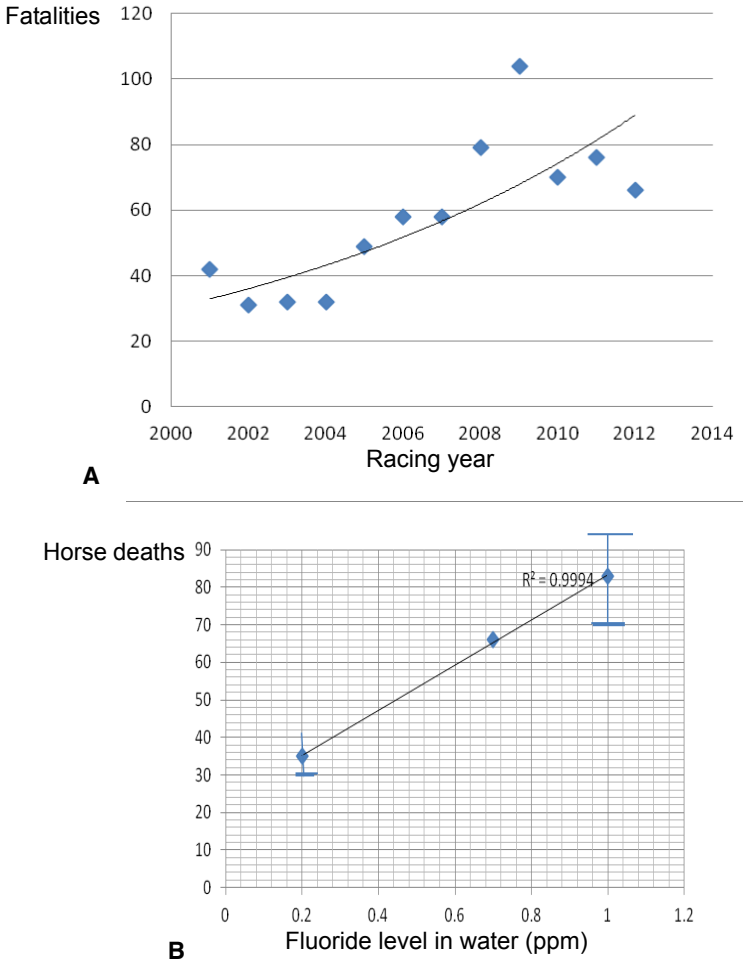

Figure 1. Los Alamitos quarterhorse fatalities per season (A) and as a function of water fluoride level (B). Data are from the California Horse Racing Board[1] for all 181 day racing seasons since 2000–2001. Water was infused with H_2SiF_6 at 1.0 ppm fluoride from November 2007 and then at 0.7 ppm from February 2011. Data from (A) were averaged for 2000–2004 before fluoridation (0.2 ppm natural fluoride), and from 2008–2011 (artificial fluoride 1.0 ppm, 0.8 ppm silicic acid, 93 ppm sodium) and the center value is from 2012 (0.7 ppm fluoride, 0.6 ppm silicic acid, 85 ppm sodium). All water contained 60 ppm calcium. Error bars are standard deviations.

Similar findings occurred at Hollywood Park in Inglewood, CA, (Figure 2). Again, an upward trend after 2007 was followed by a slight drop in 2011. The same year fluoridation began, the dirt track was replaced with a synthetic surface known to reduce fatalities when dry. Nevertheless, with typically 5,300 racing starts per season, the fatality incidence increased from 0.5 to 1%, comparable to Los Alamitos. All values represent fatalities per season but not exactly per racing

start because some breakdowns occurred during training. In 8 years, for 40,000 racing starts, there were 328 fatalities. For all horses examined to 2005 the breakdown incidence was 0.2%. After 2007 it was 0.7%.

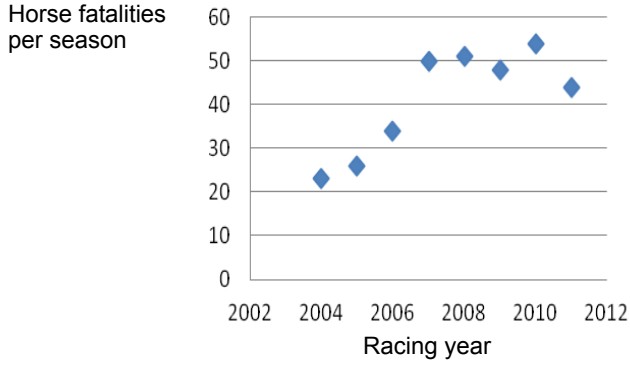

Figure 2. Hollywood Park thoroughbred fatalities per season. Entries for each year represent the season spanning into the next year (from mid-November to December 2006, April to July 2007, and early- to mid-November 2007).

DISCUSSION

Fluoride assimilation from drinking water is substantial in horses but depends on water hardness. Throughout India, where the maximum permissible water fluoride concentration is 1.2 ppm, the blood fluoride in thoroughbred horses ranged from 0.096 ppm to 0.3 ppm.[13] Horses, weighing 1,000 pounds, drinking 1 ppm treated water consume 0.26 mg of F per kg body weight daily (120 mg/455 kg). This is nine times higher than the intake able to cause acute gastrointestinal (GI) symptoms in humans of 0.028 mg/kg bw.[14] Racehorses consume 25–30 gallons of water daily with more in hot weather. Horse temperatures range from 99–102ºF and water loss through sweat is significant during racing. Most horses were stabled for long periods without access to pasture grazing, which would restrict fluoride assimilation from the GI tract, and may have had higher blood fluoride levels as a result. Almost all retained fluoride, 95%, resides in bone and this magnitude of uptake should not be assumed to simply exert no adverse biochemical effect.

The close agreement in the horse fatality trends for the two racetracks indicate a correlation between artificially fluoridated water and horse breakdowns but does not prove causation. This would require having control horses that consumed only hauled non-fluoridated water for comparison. However, the biphasic trend at both tracks that correlated with the biphasic fluoride levels would be unlikely to be due to causes other than the water supply. The correlations from the age-matched, 2–6-year-old, horses under strict dietary management and in prime health are consistent for both thoroughbreds and quarter horses.

The water district officials had no awareness that they were treating the horses, the horses were unaware of the treatment, the owners were not informed that treatment was occurring, and the author/observer had no role in either the treatment or handling of the horses. The most logical objective explanation of

these data is that the breakdown incidence parallels the level of fluoride in the fluoridated water supply. It is useful to consider possible mechanisms.

A suggested possibility is that silicosis-induced bone deformations may be involved in racehorse breakdowns in the state.[15] Silicic acid, H_4SiO_4, levels in the treated water exhibited a biphasic trend in conjunction with the added fluoride. Horses that consume treated water rather than well water ingest daily 8 mg of silicic acid and 9.3 g of sodium (93 ppm = 93 mg/L × 100 L/day). Silicate has been reported to strengthen bone in chickens. Some argue it may be a mineral nutrient. It is not regulated by the EPA. Pure sodium silicate ingested in acute doses is tolerated and has no published LD_{50}[16] but liver silicosis has occurred in alligators from residing in fluorosilicic acid treated water.[10] Silicic acid and fluoride might act synergistically. Sodium levels in water before (85 ppm) and after (93 ppm) the infusions are not consistent being the cause of the 2–3 fold increases in breakdowns.

The FDA ruled fluoride does not strengthen bone from studies commissioned in 1989.[17] Bone fluoride at 3,000 mg/kg from consumption of 1 ppm fluoride water for decades weakens human bone making it more subject to fracture.[6] Fluoride is an EPA regulated water contaminant with a secondary maximum contaminant level (SMCL) of 2 ppm assessed for natural calcium fluoride (CaF_2 LD_{50} 3,000–5,000 mg/kg).[16] There is no maximum contaminant level (MCL) for the more toxic fluoride compounds made by industrial reaction used to treat water that lack calcium (LD_{50}~ 60 mg/kg).[16] It would be unethical to administer sodium fluoride versus fluorosilicic acid treated waters to horses to clarify which substances may be involved since these compounds are calcium chelators. Such controlled human studies have also not been done as required for FDA approved drugs.

Systemic fluoride abnormally alters bone hydroxyapatite[6] by ion exchange. Normal bone is a repository for blood calcium during dietary insufficiency[18] but parathyroid hormone is not designed to mobilize fluoride which has a 20 year half-life in bone.[6,19,20] Horse blood ionized calcium ranges from 2.6–3.5 mM (10–14 mg/dL)[21] and is crucial for normal heart function. Systemic low level fluoride can cause hormonal mobilization of normal bone regions to prevent hypocalcemia, but this can eventually cause bone mineral depletion affecting performance.

Thoracic leg cannon bone fluoride levels in horses consuming soft water infused with 0.35–1.3 ppm fluoride from diluted H_2SiF_6 over a 19 year period were 587–936 mg/kg. This is 4–6 times higher than that in control horses without fluoridated water. Quarter horses exhibited effects on bone-resorbing and bone-forming cells with osteomegaly and osteopenia. Bone bulged deep into the internal marrow and the bones exhibited extra, low-density growths along their shafts. Noticeable structural abnormalities occurred with fluoride levels as low as 500 mg/kg.[10] Since fluoride during continuous ingestion typically accumulates in bone,[6] the more years a horse consumes soluble fluoride the more bone must be synthesized to maintain blood calcium homeostasis. Chemically altered bone will break with smaller forces. Bone breaks in California were accompanied with a pathologic bone structure of unknown cause which was observed on X-rays before the breaks

occurred.[15] Fatal injuries usually started as mild ones that went undetected.[22] A lateral manœuver during a race can place most of the force on one of two lateral positioned bones. At full gallop, all four hooves are airborne. Stopping abruptly from distraction or pain can be disastrous if bone is fluoride-enriched as $Ca_{10}[PO_4]_6F_2$. It is possible that silicate plus fluoride act synergistically.

Fluoride is converted to hydrofluoric acid, HF, in the acidic stomach. Most fluoride in the horse stomach pyloric region, where the pH of is 2.6,[18] is protonated to HF^6, the fully assimilated form.[23] Stomach ulcers have been diagnosed by video endoscopy in 80–90% of racehorses.[18] Poor racing performance is the chief resulting complaint but veterinarians often certify racing soundness, believing the condition to be mild in most cases.[24] Ulcers are absent during natural pasture foraging but are enhanced by stress.[21] Sodium fluoride at 1 ppm in plain drinking water causes GI discomfort from HF in 7% of humans in the absence of ulcers.[25-27] At low concentrations, HF can aggravate ulcerated tissue and in horses could conceivably cause heartburn during racing and possible missteps.

Consumption of 1 ppm artificially fluoridated water in man for one year disrupts iron-assimilating intestinal cells causing anemia and reduced hemoglobin levels that return to normal after the withdrawal of fluoridated water.[7] Any possible anemia in racehorses could be dangerous during bouts of exercise with full cardiac output. Heart rates increase 10-fold, from 24 to 240 beats per minute (bpm) during racing.

Many race officials and jockeys fault, for causing horse breakdowns, the combination of painkilling drugs, steroids, and the nonsteroidal flunixin [2-methyl-3-(trifluoromethyl)phenyl]amino]pyridine-3-carboxylic acid].[28,29] In 2005, flunixin was approved by the FDA as a generic horse drug.[30] The incidence of breakdown could be complicated by any use of such drugs. Ulcers are common side-effects of flunixin[31] which was banned by the International Federation for Equestrian Sports in Switzerland.[32] The California Horse Racing Board (CHRB) raised its allowable blood level to 50 ppb in 2007 but then lowered it again in 2010.[1]

Two of the Southern CA horses had trace levels of an unidentified rodent poison.[33] Any warfarin-based anticoagulant could act at lower levels in fluoridated blood. Several deaths were from hemorrhage.

California leads the states in racehorse fatalities at 3 per 1,000 racing starts compared to a USA average of 1 per 1,000. Fatalities in Washington State, where the water is soft, increased recently to 2.64 per 1,000 starts. Twelve deaths in CA (2010–2011) were from sudden heart failure from aortic rupture or myocarditis.[22] Nineteen such deaths occurred in 2011–2012 and 17 in 2012–2013 with three months remaining in the fiscal season, 11 of these being heart failure. The CAHRB and the Animal Health and Food Safety Laboratory System have not found the cause after necropsy and toxicologic and histologic study.[33] Systemic fluoride

accumulates in aorta in man and animals[34] and in atherosclerotic plaque in human coronary arteries.[35]

At Churchill Downs, Louisville, KY, municipal fluoridated water is used but only temporarily by horses during racing visits. No horses are housed there during the winter season. This is significant since the severity of the effects on ulcerated tissue caused by HF would depend on length of exposure. Fluoride toxicity is dependent on conditions[36] such as water hardness and food availability during drinking.

Generally toxicologic tests for ingested substances exhibit wide biologic variability even within one animal strain. Administration of LD_{50} acute oral doses cause 50% of animals to die but the remaining 50% are either sickened and recover or are often seemingly unaffected. Variation in genetic factors, hormonal and other traits also apply to low level chronic exposure and explain why artificially fluoridated water could contribute to breakdowns in a small fraction of the population while most horses performed normally. The lifetime accumulation of fluoride and silicate would affect all horses to a varying degree but the effects would be difficult to assess since speed and stamina decline naturally with age.

The toxic effects of low level chronic exposure to chemicals in racehorses can be minimized with superior care such as providing sufficient dietary calcium, offering feed with drinking water, and pasturing horses frequently. Quality reverse osmosis defluoridated water equipment could be employed on site by owners at racing venues, but halting chemical infusions into water supplies is the best practice since adequate controlled trials for long-term safety in racehorses under conditions of quartering do not exist. Racehorse health and performance affect the attendance at race tracks. The 50-year-old Hollywood Park race track is now closing due to insufficient attendance, influenced in part by increased fatalities.

In the interest of animal welfare, it is prudent to follow Federal water laws designed to protect the natural chemistry of USA waterways. The FDA ruled that fluoride added into water is an uncontrolled use of an unapproved drug and that it is not a mineral nutrient.

Water districts infuse public supplies with materials to treat human caries, not horses. Topical fluoride is not used on horse teeth. The soft cementum layer covering teeth is synthesized by cells under the gum line forming a smooth shape during circular chewing.[18,37,38] Halting fluoride infusions would not eliminate racehorse fatalities but could prevent many.

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DEDICATION

This work is dedicated to Dr. Albert Burgstahler, Editor-in-Chief of *Fluoride*, who asked that an editorial on fluoride exposure in racehorses be written. He is sorely missed. His rigorous work on the toxicity of fluoridated water remains superior.

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