Dear Drs Reichardt, Braddock, Medford, Newberry, Stolzberg, and Taylor,

March 19, 2010

RE: Fairbanks Fluoridation Task Force
Letter #10: Fluoride and Effects on the Endocrine System

Fluoride and the Endocrine System with Emphasis on the thyroid, parathyroid, and pineal glands. NRC 2006 Chapter 8.

Because some children ingest too much fluoride from toothpaste and food, intentionally adding any more fluoride to water which further increases their excess fluoride exposure, simply makes no sense. Dispensing a drug through the public water systems, which provide only a part of total fluoride exposure, is simply an impossible method to control total dosage for each patient.

I must strenuously object to any suggestion that the NRC 2006 report has no application to water fluoridation at 1 ppm. Not only do many of the studies directly apply, but fluoridation increases total exposure and fluoridation cannot be evaluated as an isolated or precise dosage. Concentration of fluoride in water is not the same as the milligrams of fluoride ingested per kilogram of body weight. Water is a contributing factor for total fluoride exposure.

This task force must be protective of all residents, not just average, mean, or to the 90th percentile.

Summary:
“..."optimal" intake of fluoride has been widely accepted for decades as between 0.05 and 0.07 mg fluoride per kilogram of body weight (mg F/kg bw) but is based on limited scientific evidence... CONCLUSIONS: Given the overlap among caries/fluorosis groups in mean fluoride intake and extreme variability in individual fluoride intakes, firmly recommending an "optimal" fluoride intake is problematic."[1][1]

In other words, 2.5 to 3.5 mg for a 110 pound person and 5 to 7 mg for a 220 person. Keep in mind 0.05 to 0.07 mg F/kg bw and the numbers 2.5 to 7 mg total daily fluoride intake as the "recommended" or "optimal" amount of fluoride suggested by fluoridation proponents. Sometimes these numbers have been simplified to 3 mg for women and 4 mg for men. And not only is the "optimal" intake the same amount causing dental fluorosis, but there is an extreme variability in individual fluoride intake.

The NRC 2006 reported several lines of information indicate an effect of fluoride exposure on thyroid function. Until the 1970's, European doctors used fluoride to suppress the thyroid, to treat hyperthyroidism,[2][2] with doses of between 2 to 10 mg/day, the same dosage range recommended in the USA for everyone, and even today fluoride is a component of some attention deficit disorder drugs. In the USA, hypothyroidism is a major problem with Synthroid, to treat hypothyroidism, one of the top four or five prescribed drugs. Hypothyroidism results in symptoms such as fatigue,
depression, weight gain, hair loss, muscle pains, increased LDL cholesterol and heart disease.

Kathy Thiessen, one of the 12 NRC 2006 members, has a “must see” power point at http://www.fluoridealert.org/health/thyroid/thiessen.pdf

I. The NRC 2006 Report: p 189, Thyroid Follicular Cells.

Fluoride & the Thyroid - US National Research Council (2006):

“In summary, evidence of several types indicates that fluoride affects normal endocrine function or response; the effects of the fluoride-induced changes vary in degree and kind in different individuals. Fluoride is therefore an endocrine disruptor in the broad sense of altering normal endocrine function or response, although probably not in the sense of mimicking a normal hormone. The mechanisms of action remain to be worked out and appear to include both direct and indirect mechanisms, for example, direct stimulation or inhibition of hormone secretion by interference with second messenger function, indirect stimulation or inhibition of hormone secretion by effects on things such as calcium balance, and inhibition of peripheral enzymes that are necessary for activation of the normal hormone.” National Research Council. (2006). Fluoride in Drinking Water: A Scientific Review of EPA's Standards. National Academies Press, Washington D.C. p 223. (NRC 2006)

“The effects of fluoride on various aspects of endocrine function should be examined further, particularly with respect to a possible role in the development of several diseases or mental states in the United States.” NRC 2006, p 224.

“several lines of information indicate an effect of fluoride exposure on thyroid function.” NRC 2006, p 197

“It is difficult to predict exactly what effects on thyroid function are likely at what concentration of fluoride exposure and under what circumstances.” NRC 2006, p 197.

“Fluoride exposure in humans is associated with elevated TSH concentrations, increased goiter prevalence, and altered T4 and T3 concentrations; similar effects on T4 and T3 are reported in experimental animals.” NRC 2006, p 218

“In humans, effects on thyroid function were associated with fluoride exposures of 0.05-0.13 mg/kg/day when iodine intake was adequate and 0.01-0.03 mg/kg/day when iodine intake was inadequate.” NRC 2006, p 218.
"The recent decline in iodine intake in the United States could contribute to increased toxicity of fluoride for some individuals." NRC 2006, p 218

"Intake of nutrients such as calcium and iodine often is not reported in studies of fluoride effects. The effects of fluoride on thyroid function, for instance, might depend on whether iodine intake is low, adequate, or high, or whether dietary selenium is adequate." NRC 2006, p 222

"Between 4% and 5% of the U.S. population may be affected by deranged thyroid function (Goodman 2003), making it among the most prevalent of endocrine diseases (Larsen et al. 2002). The prevalence of subclinical thyroid dysfunction in various populations is 1.3-17.5% for subclinical hypothyroidism and 0.6-16% for subclinical hyperthyroidism; the reported rates depend on age, sex, iodine intake, sensitivity of measurements, and definition used (Biondi et al. 2002)." P 189

"Several authors have reported an association between endemic goiter and fluoride exposure or enamel fluorosis in human in India (Wilson 1941; Siddiqui 1960; Desai et al. 1993), Nepal (Day and Powell-Jackson 1972), England (Wilson 1941; Murray et al. 1948), South Africa (Steyn 1948; Steyn et al. 1955; Jooste et al. 1999), and Kenya (Obel 1982)." P. 192.

II. **Thyroid Parafollicular Cells (C cells, produce calcitonin)**

"... its (Calcitonin's) primary importance seems to be to protect against excessive bone resorption (Bringhurst et al. 2002; Goodman 2003)" p 198

"Calcitonin concentrations do not seem to have been routinely measured in cases of skeletal fluorosis, but elevated calcitonin does seem to be present when looked for. The effect has been noted at fluoride intakes as low as 3.8 mg/day in humans (approximately 0.06 mg/kg/day) and was found routinely at intakes of at least 9 mg/day (approximately 0.15 mg/kg/day)." P 200

III. **Parathyroid Glands (4 small glands normally posterior to the thyroid producing PTH)**

"The primary effect of PTH is to increase the calcium concentration and decrease the phosphate concentration in blood (Bringhurst et al. 2002; Goodman 2003). . . Circadian patterns of PTH concentrations differ in men and women (Calvo et al. 1991) and between healthy and osteoporotic postmenopausal women (Eastell et al. 1992; Fraser et al. 1998)." P 200

See the NRC report for in vitro and animal studies. See table of studies on pages 420-426.
In a review of skeletal fluorosis, Krishnamachari (1986) indicated that the nature (osteosclerotic, osteomalacic, osteoporotic) and severity of the fluorosis depend on factors such as age, sex, dietary calcium intake, dose and duration of fluoride intake, and renal efficiency in fluoride handling. In some cases, secondary hyperparathyroidism is observed with associated characteristic bone changes. He also noted the preponderance of males among fluorosis patients and discussed a possible protective effect of estrogens. In his review, Krishnamachari (1986) described a twofold model for the body’s handling of fluoride.

1. In the presence of adequate calcium, absorbed fluoride is deposited in the bone as calcium fluorapatite. Bone density increases, urinary fluoride increases, but urinary calcium and phosphorus are not altered. Osteosclerosis and calcification of many tendons and ligaments occur. Serum alkaline phosphatase activity is elevated, but no specific changes occur in other constituents of serum. There are minimal hormonal changes and only mild secondary hyperparathyroidism. If the situation progresses, there will be osteophytosis (bony outgrowths), neurological complications, and late crippling, producing an osteosclerotic form of fluorosis that primarily affects adults.

2. In the presence of inadequate calcium, fluoride directly or indirectly stimulates the parathyroid glands, causing secondary hyperparathyroidism leading to bone loss. Bone density is variably increased, with areas of sclerosis or porosis; there is evidence (radiological and densitometrical) of bone loss. There is renal conservation of calcium in spite of hyperparathyroidism, with no significant changes in serum biochemistry; urinary hydroxyproline excretion is significantly increased. In these conditions, an osteoporotic type of skeletal fluorosis occurs at a younger age, and growing children develop deformities due to bone softening.

“Thyroid Parafollicular Cell Function

Only one study has reported calcitonin concentrations in fluoride-exposed individuals. This study found elevated calcitonin in all patients with fluoride exposures above about 0.15 mg/kg/day and in one patient with a current intake of approximately 0.06 mg/kg/day (Table 8-2); these exposures corresponded to plasma fluoride concentrations of 0.11-0.26 mg/L. Results attributed to altered calcitonin activity have also been found in experimental animals at a fluoride exposure of 2 mg/kg/day (Table 8-1). It is not clear whether elevated calcitonin is a direct or indirect result of fluoride exposure, nor is it clear what the clinical significance of elevated calcitonin concentrations might be in individuals.” P 221

Parathyroid Function

In humans, depending on the calcium intake, elevated concentrations of PTH are routinely found at fluoride exposures of 0.4-0.6 mg/kg/day and at exposures as low as 0.15 g/kg/day in some individuals (Table 8-2). Similar effects and exposures have been found in a vriety of human studies; these studies indicate that elevated PTH and secondary hyperparathyroidism occur at fluoride intakes higher than those associated with other endocrine effects. In the single study that measured both calcitonin and PTH, all individuals with elevated PTH also had
elevated calcitonin, and several individuals had elevated calcitonin without elevated PTH (Teotia et al. 1978). P 221

Pineal Function
“The single animal study of pineal function indicates that fluoride exposure results in altered melatonin production and altered timing of sexual maturity (Table 8-1). Whether fluoride affects pineal function in humans remains to be demonstrated. The two studies of menarcheal age in humans show the possibility of earlier menarche in some individuals exposed to fluoride, but no definitive statement can be made. Recent information on the role of the pineal organ in humans suggests that any agent that affects pineal function could affect human health in a variety of ways, including effects on sexual maturation, calcium metabolism, parathyroid function, postmenopausal osteoporosis, cancer, and psychiatric disease. P 222

Glucose Metabolism
“Impaired glucose tolerance in humans has been reported in separate studies at fluoride intakes of 0.07-0.4 mg/kg/day, corresponding to serum fluoride concentrations above about 0.1 mg/L. The primary mechanism appears to involve inhibition of insulin production." P 222

Summary
“In summary, evidence of several types indicates that fluoride affects normal endocrine function or response; the effects of the fluoride-induced changes vary in degree and kind in different individuals. Fluoride is therefore an endocrine disruptor in the broad sense of altering normal endocrine function or response, although probably not in the sense of mimicking a normal hormone. The mechanisms of action remain to be worked out and appear to include both direct and indirect mechanisms, for example, direct stimulation or inhibition of hormone secretion by interference with second messenger function, indirect stimulation or inhibition of hormone secretion by effects on things such as calcium balance, and inhibition of peripheral enzymes that are necessary for activation of the normal hormone.” p 223

Fluoride & the Thyroid - Studies Available Online:


This is a very brief review of a complex section.

Sincerely,

Bill Osmunson DDS, MPH