

IS DENTAL FLUOROSIS CAUSED BY THYROID HORMONE DISTURBANCES?

SUMMARY: Although it has been known since 1917 that mottled dental enamel (later recognized as dental fluorosis—DF) is identical with that observed in thyroid dysfunction, disturbances of thyroid hormone metabolism during crucial periods of tooth development as the primary cause of DF have received very little consideration by dental researchers. New findings indicate that thyroid hormone metabolism is disturbed in peripheral tissue of children with DF, thereby helping to account for timing of events observed in DF and the delayed eruption of teeth in fluoridated areas and further suggesting the use of DF as a marker and diagnostic aid for iodine deficiency disorders.

Keywords: Dental fluorosis; Thyroid hormone metabolism.

Shortly after Professor G Vardiman Black and Frederick S McKay published their impressively illustrated reports in *Dental Cosmos* in 1916 on mottled dental enamel (later identified as dental fluorosis) they had observed in Colorado,¹ McKay in 1917 reported that Professor John E Grevers of Utrecht, Holland, to whom he had sent specimens of mottled teeth, found the *identical* condition in the teeth of people with goiter in Utrecht.² Grevers also obtained laboratory evidence from his drinking water studies on rats that there was a clear association of his clinical cases of goiter with mottled enamel, and, like McKay, postulated a connection with an unidentified component in the drinking water—found years later, in 1931, to be fluoride.

At about the same time, and independently of McKay, Leon Goldemberg, a medical practitioner in Buenos Aires, was conducting extensive research into areas of Argentina having “Kropfwässer”—the German word for “goiterous waters”. After studying the available literature of the time, Goldemberg became convinced that the goiters observed in many such areas were not actually caused by any deficiency of iodine—as was commonly held—but were, in fact, caused by excessive intake of fluoride from *all* sources: water, food, and air.³

Goldemberg’s further study led him to believe that—if fluoride was indeed able to cause iodine deficiency and symptoms such as goiter and cretinism—then it should also be able to reduce the excess levels of blood iodine in his patients who presented themselves with an enlarged thyroid gland, bulging eyes, and heart palpitations, a group of symptoms which had been given the name Basedow Disease, or Graves’ Disease in the UK. Iodine had earlier been identified as the “mysterious toxin raging through the body” of all such patients. After first conducting animal experiments to test his hypothesis and producing a condition he called “fluoride cretinism”, Goldemberg began treating his Basedow patients successfully with fluoride therapy.⁴ Later, Wilhelm May and others adopted Goldemberg’s treatment method to include various organic fluoride compounds, and, for many decades, “fluoride therapy” became the first line of treatment for hyperthyroidism in Germany and Austria.^{5,6}

McKay and his dental colleagues, however, paid no further attention to the clear association between dental disorders and thyroid dysfunction. Instead, their

focus shifted to the observation that the “mottled enamel”, identified to be a *developmental disorder*, was apparently more resistant to caries. Dental researchers, not endocrinologists, then became the experts on all matters related to fluoride.

Since McKay’s time, dental fluorosis (DF) has been studied extensively, but even to this date dental researchers state that the mechanisms underlying the pathogenesis of DF are not yet understood.⁷ Fluorosed (mottled) teeth are labeled only a “cosmetic defect”, as one can read repeatedly in public health reviews on the topic and in pro-fluoridation literature.⁸ The obvious question beckons: How can one declare a “developmental disorder” to be only a “cosmetic defect”, if the mechanisms are not understood?

In fact, DF is a developmental disorder—originating from aberrant thyroid hormone metabolism.

Perhaps the most obvious indication that DF is a condition caused by disordered thyroid hormone signaling during the time of enamel development is the long-standing observation of delayed eruption of teeth in fluoridated areas.⁹ DF is invariably associated with dental age and eruption of teeth, a process closely controlled by thyroid hormone (TH). TH deficiency leads to delayed tooth eruption, while TH excess leads to the acceleration of tooth eruption. The more fluoride ingested, the longer it takes for the tooth to erupt. The later in life maturation of enamel is completed, the greater is the severity of dental fluorosis.¹⁰

At the same time, other risk factors known to influence DF are identical to those observed in thyroid dysfunction. Thus, while DF gets more severe at higher altitudes, the same is generally true for iodine deficiency.¹¹ Furthermore, while the frequency of DF is significantly greater among Blacks, it is now known that they also have a more sensitive thyroid status.¹²

It is well established that DF can only occur as a result of excessive fluoride exposure during crucial times of development—*in utero to approximately 30 months for deciduous teeth and permanent incisors*— and is marked by events related to *timing*. Thus, it is associated with *delayed* tooth eruption,^{9,10} *delayed* removal of enamel matrix proteins,¹³ *delayed* enamel maturation,^{10,13} etc., clearly indicating that a tissue-specific differentiation program is being disturbed. Endocrinology has firmly established TH to be the crucial regulator of all tissue-specific differentiation programs during development. Appropriate TH levels at the precise time are critically important for the coordination of developmental processes. This is most clearly demonstrated in amphibia, in which metamorphosis does not occur in thyroidectomized larvae unless TH is present. The metamorphic transitions of individual organs in amphibia are all controlled by TH, with each event occurring at distinct developmental stages, requiring correct spatial and temporal manner.¹⁴ (Since the 1930s fluoride has been known to inhibit and delay metamorphosis in amphibia.⁶ Just a coincidence?)

In the physiology of human development, the importance of TH is especially evident in the central nervous system in which TH deficiency during fetal and

neonatal periods can lead to morphological and functional abnormalities, the most severe manifestation of which is cretinism.

The findings by AK Susheela and co-workers, as published in this issue of *Fluoride*,¹⁵ present not only the first reports on TSH and free TH levels in children and adolescents with DF, but, in addition, show that even in children without DF—but with elevated fluoride serum levels—abnormal TH metabolism is present, as previously observed in workers exposed to fluoride,¹⁶ as well as in children and adults with various amounts of fluoride in the water supply.^{6,17} This new evidence indicates that iodine metabolism is being disturbed in peripheral tissue through manipulation of the deiodinases, the three enzymes which delicately regulate TH metabolism through external TSH/G-protein activation. The disturbances in TH levels observed are identical with those observed in iodine deficiency disorders (IDD).

TH deficiency during the secretory stage of amelogenesis results in poorly calcified enamel,¹⁸ a hallmark of dental fluorosis. Since it is during this time that any alterations in TH may also influence the neurological development of the child, enamel defects in deciduous teeth and permanent incisors, such as dental fluorosis, should be used as a marker and aid in the diagnosis of neurological and iodine deficiency disorders, as has been suggested by others.¹⁹⁻²²

Understanding thyroid hormone metabolism is essential in understanding fluoride toxicity. Further research, be it on dental or skeletal fluorosis, effects on IQ, oxidative stress, etc., should focus on this matter with utmost urgency, since it is here that all observed adverse effects can be explained, thereby leading to a new toxicological assessment of “fluorosis”, and, most importantly, proper treatment and prevention.

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