

Dental Fluorosis

'Mottled enamel' was first described by Eager in 1901¹ as a strange condition in the teeth of people living in a small village near Naples, Italy. In 1931,²⁻⁴ three different groups of scientists announced their discovery of the primary cause of the widely occurring endemic tooth defect known as dental mottling and first associated it with fluorides in the drinking water of children. Thereafter, this type of mottled teeth was designated dental fluorosis or enamel fluorosis. The permanent teeth are particularly affected, though it occasionally affects primary teeth.⁵⁻⁷

Dental fluorosis is easily recognizable. It is a clinical manifestation of chronic exposure to high intakes of fluoride through drinking water. The signs of dental fluorosis range from a few white flecks to confluent pits in the enamel surface and unsightly dark brown stains.⁸⁻¹³ Although fluoride from any source can cause dental fluorosis, the high concentration of naturally occurring fluorides in drinking water is the most important cause in India.^{14,15} The minimum level of fluoride at which perceptible changes appear in the developing enamel of permanent teeth is well below 1 ppm.^{15,16} People living in a tropical climate have to consume more water and hence more fluoride is ingested.

Several classifications have been proposed to assess the severity of dental fluorosis. Dean¹⁶ based his classification on the clinical appearance of the enamel, and it varied from normal to severe:

Normal: Translucent, smooth enamel with a glossy appearance.

Questionable: Seen in endemic areas, borderline between normal and very mild.

Very mild: Small opaque, paper-white areas scattered irregularly over the labial and buccal surface of teeth.

Moderate: Entire tooth surface involved, minute pitting often present on labial and buccal surfaces, brown surface, brown stains, frequently disfiguring.

Moderately severe: Entire tooth surface involved, marked pitting with intense brown stain.

Severe: Widespread, deep brown or black areas, corrosion type of mottled enamel.

To determine the severity of dental fluorosis as a public health problem, Dean devised a method of calculating the prevalence and degree of severity in a community, which he termed the community fluorosis index (CFI). When the CFI was <0.4, Dean¹⁶ considered it of little or no public health concern, while indices between 0.4 and 0.6 were borderline, and the removal of excess fluoride was indicated when the CFI was >0.6. Hence, a CFI of 0.6 sets the upper limit of fluoride concentration for aesthetic reasons. Dean's index is based upon the clinical appearance only and no attempt has been made to correlate the diagnosis with histological changes.

Teotia and Teotia^{14,17} proposed a simplified classification to assess the severity of dental fluorosis.

Grade 0: Normal, translucent, smooth and glossy teeth.

Grade 1: White opacities, faint yellow line.

Grade 2: Brown stain.

Grade 3: Pitting and chipped off edges.

Grade 4: Brown plaques, corrosion and falling of teeth.

This classification is easy to use, less time-consuming, has only five categories and is therefore more practical for community and epidemiological surveys.

Gopalakrishnan *et al.* in their study published in this issue of the journal (pp. 99-103) have reported the prevalence of dental fluorosis and associated risk factors in Alappuzha district of Kerala. In their community-based, cross-sectional survey of 1142 schoolchildren in the age range of 10-17 years, 35.6% had evidence of dental fluorosis, with a CFI of 0.69. The incidence of dental fluorosis was greater in urban (55.3%) compared to rural areas (16.8%) and in girls (39.2%) as compared to boys (31.3%).

They also report a higher incidence of dental fluorosis in children who consumed

pipe water (44.8%) as compared to those who consumed well water (12.7%) and the increasing prevalence of dental fluorosis with increase in the fluoride content of drinking water. However, as they mention, there is no information on the fluoride content of drinking water consumed by individual children in rural and urban areas, nor any information on the fluoride content of drinking water collected from different sources except those in various *panchayats*.

The high incidence of dental fluorosis in urban areas needs explanation, since endemic fluorosis is almost exclusively confined to villages where water used is mostly from superficial sources such as wells and hand pumps.^{17,18} A statement that dental stains caused by excessive consumption of fluoride were differentiated from other causes should also have appeared in the paper.

The paper, although pointing to interesting observations, omits several important factors that should have been investigated to link the 'exposure to fluoride' with the 'prevalence and severity' of dental fluorosis. Some major concerns are:

1. Clinical details of the children studied, the duration of stay in the urban or rural areas, had they been living in the respective areas since their birth or had migrated, fluctuations in the length of their stay periods and in the continuity of exposure to fluoride.
2. The concentration of fluoride in the drinking water, total daily fluoride intake, duration of exposure to high intake of fluoride and nutritional status of the children, particularly that of calcium and vitamin D.

Dental fluorosis occurs in children who are exposed to high intake of fluoride before completion of dental mineralization (12–14 years of age). We conducted an epidemiological study^{19,20} between 1963 and 1998 on 0.45 million children residing in non-endemic (fluoride content <1 ppm) and endemic (fluoride content >1 ppm) villages of India. This study revealed that in non-endemic areas, among children with adequate calcium nutrition (calcium intake >800 mg/day), 7% showed dental fluorosis and 2% had dental caries, while among children with inadequate calcium nutrition (calcium intake <300 mg/day), 14.2% showed dental fluorosis and 31.4% had dental caries. In endemic areas, of the children with adequate calcium intake, 59% had dental fluorosis and 10% dental caries, while in the calcium inadequate group, 100% had dental fluorosis and 74% dental caries.

Our findings indicated that dental fluorosis and caries were caused by high fluoride and low dietary calcium intakes, separately and jointly. Dental fluorosis and caries were more severe and complex in calcium-deficient children exposed to high intake of endemic fluoride in drinking water. Thus, adequate calcium nutrition is the strongest antagonist which effectively counteracts the toxic effects of fluoride on teeth.^{19,20}

The consumption of brick-tea and fish is not of much clinical or practical importance. Drinking water remains the only and major source of high intake of fluoride in India, causing dental and skeletal fluorosis.^{17,18}

The only practical and effective public health measure for the prevention and control of dental fluorosis and caries is limitation of the fluoride content of drinking water to <0.5 ppm, using deep bore drinking water supplies and adequate calcium intake (dietary calcium >1 g/day).²¹ The World Health Organization policy and recommendations on fluorides are not universally acceptable, especially in our environment of nutritional deficiencies, endemic fluorosis, and different trends of caries prevalence. In the light of the available data, the World Health Organization's recommendations need to be modified to achieve dental health for all by the year 2000.

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