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FLUORIDE AND HEALTH: DENTAL CARIES, OSTEOPOROSIS, AND CARDIOVASCULAR DISEASE

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INTRODUCTION

Water plays a major role in the distribution and availability of minerals that are essential for the development and maintenance of sound teeth. The high mineral content of dental tissues [enamel, 97%; dentine, 70%; cementum, 65% (approximately)] predisposes teeth to dissolution by acids produced by adherent bacterial biomasses (plaque) from dietary carbohydrate substrates. Since carious lesions develop through intermittent decalcification and recalcification, the mineral composition of teeth and the minerals in their environment are important determinants of the process.

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The quantitatively and structurally dominant components of teeth are calcium and phosphorous in the form of hydroxyapatite, but many other elements are also present in varying quantities (13). Of the lesser and trace mineral constituents of the dentition and the oral environment, fluoride (F) is especially pertinent to the theme of this chapter, partly because water is often its principal source (27) and partly because of its proven cariostatic properties. In contrast, the bulk of other minor or trace minerals is usually obtained from different sources, and despite plausible claims that certain elements increase resistance or susceptibility to caries, none has gained general acceptance leading to practical application for caries prevention at the public health level. Accordingly, this review deals with specific effects of F on human health.

FLUORIDE AND DENTAL CARIES

The identification of water-borne F as a cariostatic agent followed sporadic observations of brownish mottling of the teeth in parts of Italy, Mexico, North Africa, and the United States around the turn of this century. The epidemiological surveys that related enamel mottling to the presence of excessive amounts of F in the water and, in turn, to relatively low caries experience have been extensively reviewed (33, 34, 58). Subsequent studies (14) showed a remarkably close inverse association between the natural F concentration of domestic water and the caries experience of 12- to 14-year-old children in 21 US cities. Later surveys disclosed similar associations in England, Denmark, and Sweden, despite marked differences in mean caries experience (24).

The finding (14) that
maximum protection against caries in the practical absence of enamel mottling (fluorosis), considered in relation to climate (21), average water consumption (22), and water-borne F intake (20), opened the way for optimal fluoridation. This entails adjustment of the F concentration of water supplies to a level that enhances the appearance of teeth (15) and offers the greatest caries reduction at a negligible risk of fluorosis. An early recommendation set the optimal range at 0.7–1.2 ppm F, and more recently, 0.5–1.3 ppm has been suggested (41). Both ranges are based primarily on climatic conditions in the US; their safety is unquestionable (17), but in view of wide global variations of factors that affect caries experience and F intake and utilization, such as diet, nutritional state, cultural patterns, and geochemical background, higher or lower F concentrations may be appropriate for some populations.

Partial defluoridation or change of water supplies containing excessive amounts of F is warranted (26) and has been accomplished (26, 37), but the need for increasing the F concentration in deficient supplies for caries control arose much more frequently. Since the first fluoridation trials (35) confirmed that F was the protective agent and added F reduced caries experience as did naturally occurring F, community water fluoridation became the method of choice for caries prevention, benefiting some 200 million people on five continents.

Specific Aspects of Protection

Children exposed to fluoridated water continuously during and after tooth development benefit by an average reduction of caries experience of 50–60% and the proportion of caries-free children may increase sixfold (2). Deciduous (46) and permanent (5) teeth benefit equally and the consistency of protection often overrides ethnic, cultural, and geographic differences. Exceptions may be due to the operation of other influencing factors. The overall 70% caries reduction for 6- to 10-year-old children in Perth (38) and 81% reduction for 6 year olds in Canberra (11) after 10 years of fluoridation coincided with a 20–50% decline in caries experience in Brisbane, a non-fluoridated Australian city (36). The latter trend probably reflects improving oral health awareness and increasing utilization of preventive care, such as F tablet supplements (16) and topical F therapy (25). The reasons may be similar for the 80% and greater reductions reported in Eastern European countries (61). Teeth first exposed to fluoridated water some time after eruption benefit less (6), but increased protection with the length of exposure is apparent beyond the effect of other protective factors, including plaque F (52). Fluoride protects anterior teeth more than posteriors, because of the high caries susceptibility of the pits and fissures of the latter; the approximate order of caries reduction is 35–40% on surfaces with pits and fissures, 70–75% on approximal surfaces, and 85–90% on open smooth surfaces of permanent teeth in optimal F areas (5). Apart from reducing the incidence of caries, F retards the progress of lesions (54, 57) and thus reduces tooth mortality by 75–95% (6).

For adults, the benefit is less well defined, because loss of teeth through periodontal disease complicates the assessment; moreover, the protection against root caries is yet to be determined. However, 60% caries reduction was reported at age 20–44 years in Colorado at 2.5 ppm naturally occurring F, where only 7 of the 385 persons examined had severe fluorosis (47). In terms of decayed tooth sites, the difference was 44% at 45 years and over, in favor of Hartlepool (1.5–2.0 ppm F) compared to York ($\leq$0.25 ppm F) residents (40). Where the caries risk is extreme, the benefit may be more profound: 20-year-old Australian Aborigines of comparable lifestyles had an average of 1.8 decayed, missing, and filled teeth (DMFT) in an area where the water contained 1.3–1.8 ppm F (8) and 17.1 DMFT in a F-deficient area (55).

The effects of F warrant changes in dental care delivery in fluoridated areas. Chemical treatment could replace interceptive and small restorations (12), especially in permanent molars and deciduous teeth. More tooth structure can be retained in cavity preparation, and by routinely establishing the average progress of lesions by using suitable indices (53), treatment frequency could be reduced to match actual needs. Simpler treatment and conservation of dental manpower further enhance the economy of water fluoridation (30), one of the most cost-effective public health measures at an average annual per capita cost of US $0.15 (6). Individual preventive measures, such as restriction of sweet intake, plaque removal, and topical F therapy, can result in freedom from caries for those who have access to intensive preventive care and are enlightened and motivated to utilize it. Water fluoridation benefits all, but particularly the poor, the ignorant, and the negligent, who could or would not use individual measures for caries prevention.

Protective Mechanisms of Fluoride

The cariostatic mechanisms of F have been extensively studied but are not yet fully understood (10). Several mechanisms may act in combination, resulting in alteration of the morphology of teeth so that less plaque is retained, reduction of the solubility of enamel and promotion of remineralization, and interference with the adherence and the metabolism of plaque bacteria. As most studies have been carried out in the laboratory, epidemiological data are limited.

Permanent molar teeth formed in fluoridated areas tend to be smaller and have shallower fissures than in control groups of children (31). An inverse trend was also found between the F content of soil and molar tooth size (51). However, the differences are usually small, not always significant, and the etiological relevance appears tenuous.

We are not aware of epidemiological studies comparing enamel solubility in communities exposed to F-deficient and optimal or high F-containing water under conditions that simulate the caries process, but exposure to optimal or low F concentration in the water during tooth formation is reflected in the F content of deciduous (59) and permanent (1) tooth enamel. Moreover, the F concentration of subsurface and deep enamel was inversely related to the caries experience of 14- to 16-year-old children (50). The relationship was obscured in the surface layer, where F incorporated as a result of caries challenge (62) may retard further dissolution (27) and may aid recalcification of early lesions (29).

The higher mean plaque F content in fluoridated than in F-deficient areas

(27) indicates that water is one of the sources of the F concentrated by plaque (48). The protective role of plaque F is supported by inverse associations found between individual caries experience and the total F content of plaque in Australian (3) and primitive New Guinean (49) communities. Determinations of bound and ionized F in resting and fermenting plaques of 75 children showed that low caries experience was associated with high bound F concentration and the latter was associated with reduced acid production (4); these results lend epidemiological support to the view that plaque F protects against caries by interfering with bacterial metabolism (28). Inverse associations found between the mass and F content of plaque in vivo (3, 48) are in accordance with the hypothesis that F interferes with bacterial adherence to teeth.

FLUORIDE AND OSTEOPOROSIS

Primary osteoporosis, a disease characterized by fractures resulting from idiopathic loss of bone mass and strength, has been treated with F for 20 years. The use of F was indicated by its osteosclerotic effect and by the low prevalence of osteoporosis in high F areas (9). A review of 1961–1970 reports on F therapy (45) attributed conflicting results to variations in F dosage, duration of treatment, and supporting Ca administration. More recent studies were based on improved, yet incomplete, understanding of the metabolic effects of long-term ingestion of therapeutic F doses (usually 20–60 mg/day): stimulation of osteoblastic and, to a lesser extent, osteoclastic activity and consequent increase in trabecular density (43). The net result is increased skeletal mass and decreased negative calcium balance (44). Impaired calcification of the new bone matrix can be offset by Ca administration (1.0–1.5 g/day), often supported with vitamin D therapy (44).

Clinical Tolerance

Prolonged use of therapeutic F doses could induce skeletal fluorosis; early clinical signs, observed after 6 years of treatment, such as bone spurs, osteophytes, and ligamentous calcification (44), indicate finite duration (5–8 years) of therapy. Adverse rheumatic and gastrointestinal reactions to high doses of F tend to occur in 30–50% of patients and led to the discontinuation of therapy for 22% of patients in one study (44), but only to temporary suspension in another (43). Reduction of dosage would alleviate or eliminate side effects. Administration of Ca and F as soluble complex ions (19) may permit reduction of the high dosages of $CaCO_3$ and NaF (needed because CaF_2 is sparingly soluble) without reducing the available amount of Ca and F.

Benefits

The benefits are usually expressed as the reduction in vertebral fracture rate. Control groups were rarely used, but most studies showed a reduction in fracture rate with increasing length of treatment; by far the greatest number of new fractures occurred during the first year of treatment (43, 44). In a study that did include controls (39), 35% of patients treated with NaF, Ca, and vitamin D had new fractures after 1 year, compared to 69% in calcitonin treated controls, but the number of patients was small (26 and 16 per group) and they were not grouped randomly or treated concurrently. The main advantage of F treatment in osteoporosis is that it can reverse osteoporotic changes, whereas other forms of therapy only retard or arrest their progress. However, the risks involved must be weighed against the benefits individually. Moderately increased bone density found in a 1 ppm F area (23) warrants epidemiological studies comparing the prevalence of osteoporosis in optimally fluoridated and F-deficient areas.

FLUORIDE AND CARDIOVASCULAR DISEASE

In a study of rural communities exposed to contrasting levels of F in the water (4.0–5.8 ppm vs $\leq$0.3 ppm) in North Dakota (9), the number of persons with calcified aortas was significantly lower in the high F area. However, in the high F area, water was obtained from artesian wells and presumably contained high concentrations of other minerals, which may have played a role; moreover, the F concentration was in excess of that recommended for consumption. Similar comments apply to a study in Finland (32), where the prevalence of cardiovascular diseases was highest in the low F (0.05 ppm) and lowest in the high F (2.57 ppm) area, where the F and Mg contents of the water were correlated [$r = +0.98$ (our calculation)]. No such objections can be raised in the case of a US study (60), which showed a 2.5% greater decrease in mortality attributed to heart disease in 20 fluoridated towns than in 15 control towns between 1950 and 1970. In contrast, F was not implicated in a study of cardiovascular death rates in relation to 35 constituents of municipal water in 94 US cities (56), nor in other extensive investigations in the US and in England (42).

Further studies are needed in communities where the effect of other elements in water is negligible or can be isolated from that of F. Although further confirmation is needed, the beneficial effect of F in heart disease remains an exciting possibility.

SUMMARY

Water fluoridation is the preferred method of caries prevention. It should be promoted wherever technically feasible, whereas fluoridation of other vehicles, such as salt, milk, and sugar, should be considered (18) where no

reticulated water supplies exist, as in many developing communities where caries prevalence is increasing sharply (7).

Fluoride treatment of osteoporosis results in tangible improvement, but its pharmacological basis is incompletely understood and therapeutic doses are not tolerated indefinitely or by all patients. Epidemiological studies are needed to examine further the possible benefits of optimal fluoridation in relation to the prevalence of osteoporosis and heart disease.

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