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FLUORIDES AND ROOT SURFACE DENTAL CARIES

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B.D.S. (Mysore)

A THESIS SUBMITTED IN PARTIAL REQUIREMENT FOR THE
DIPLOMA IN PUBLIC HEALTH DENTISTRY

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SUMMARY

This thesis is purely a literature review concerned with root dental caries and whether the use of fluorides has any effect on it. The increase in the number of older people and the increase in tooth retention has raised the possibility that root caries will become a significant problem over the next decades. Once developed, root caries can cause problems in aspects of restorative and prosthetic dentistry. Hence prevention of the lesion would be preferable. Of all the preventive programs available, water fluoridation is the most effective, practical and feasible method. Like coronal caries, root caries has been shown to be inhibited by fluoridated water and some topical fluorides.

The structure of the root cementum and dentine is not as hard as that of the enamel due to their higher organic content. Like enamel, the basic inorganic component of the cementum and dentine is apatite although the size of the crystal is much smaller. The structural entities of the cementum, dentine and enamel vary.

Studies describing the prevalence of root surface caries have been extremely diverse regarding age, sex, socio-economic status, sample selection as well as in methods of reporting. Despite this, there has been an observable pattern in the findings within the various groups. The prevalence in healthy adults is between 20-40 % while there has been a dramatic increase in the elderly who are chronically ill are institutionalized. The prevalence is also found
to increase with age and decreases with lifelong consumption of fluoridated water. Males tend to display slightly higher rates of root caries lesions. The most susceptible teeth to be affected are the mandibular molars followed by the mandibular premolars and the maxillary canines. In terms of surfaces, the buccal, proximal and lingual surfaces are the primary sites of attack. With the advent of the root caries index, there will be uniformity in reporting the levels of root caries and any future descriptive studies can be confidently interpreted.

High sucrose diets and microbial plaque have been implicated as the main etiological factors of root surface caries. Other factors that have also been related to root caries are periodontal diseases, saliva, morphology of the tooth root as well as oral hygiene.

The main methods by which fluorides, both systemic and topical act to inhibit root caries are by its action on the plaque, saliva as well as the cementum/dentine complex. It has been successfully shown that some form of daily self-application of topical fluorides is useful in preventing the onset of the root caries lesion and this is based mainly on observations in reduction of caries in the extremely high risk cases of xerostomia. No definite data was available regarding clinical studies reporting the effectiveness of various topical fluoride solutions or dentifrices although there were a number of studies conducted in vitro. More clinical trials will have to be carried out to determine which fluoride agent or vehicle is the most effective.
In the case of systemic fluorides, studies have shown that life-long residence in fluoridated areas have significantly reduced prevalence of root caries. However, more studies will have to be carried out to determine optimal concentrations of fluoride required to inhibit root caries formation (only comparison studies between two levels of fluoride have been carried out) as well as to clarify the relationship between fluoride concentration in the water supply and prevalence of root caries.
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1 INTRODUCTION

1.1 FLUORIDES AND DENTAL CARIES

Dental caries or tooth decay is a pathological process of localized destruction of tooth tissue by microorganisms. It is a multifactorial disease in which there is an interplay of three principal factors: the host (primarily the saliva and teeth), the microflora, and the substrate or diet. In addition, time is also an important factor to be considered. Much emphasis has been placed on the prevention and treatment of caries of the enamel and dentine and fluorides have been found to play a central role in this.

Research into the biology of fluorides has been extensive within the last fifty years especially after it was shown in the 1930’s that in small quantities, fluorides were beneficial to the dental system and in larger quantities caused defective enamel. Studies also showed that fluorides were beneficial not only to young children prior to and after eruption of the teeth, but also to adults who had continually consumed fluoridated water.

Fluorides can be administered in two ways: systemic and topical. The most common method of systemic administration of fluorides is by community water fluoridation. This has proved to be the most cost effective way of providing fluorides to large groups of people. The recommended fluoride concentration for community water fluoridation ranges from 0.8-1 ppm. Systemic fluorides have been found to be effective during the development and eruption of teeth. Incorporation of fluorides in the teeth during this phase results in the formation of hydroxyfluorapatite crystals which are more
resistant to the dissolution of acids than apatite, the principal inorganic material of enamel.

Systemic fluorides are also excreted in small amounts into the oral fluids through the salivary glands and crevicular fluid of teeth. Some researchers believe that the continuous low secretion of fluorides produces a protective topical effect on the teeth (Stallard 1982). Two more important actions of systemic fluorides are currently thought to be the ability of fluorides to assist in remineralization and to inhibit the glycolic activity of plaque. Other vehicles suggested for systemic fluorides have been fluoridated salt, milk and fruit juices as well as tablets and drops (Murray and Rugg-Gunn 1982).

Topical fluorides are thought to act mainly by increasing enamel resistance, causing remineralization of the enamel, altering tooth morphology and inhibiting plaque formation. Clinical preparations of topical fluorides include the application of concentrated fluoride solutions (around 1% F) to the teeth. The fluoride can be in water based solutions, gels, varnishes, or incorporated in prophylactic pastes. Mouthwashes and toothpastes are other types of vehicles, the fluoride concentration being 0.1% F or less (Murray and Rugg-Gunn 1982).

With the use of fluorides, the prevalence of dental caries in permanent dentition can be reduced by 60%. 
1.2 THE PROBLEM OF ROOT SURFACE DENTAL CARIES

Yet another aspect of dental caries which has been neglected in the past and has surfaced during the last ten years or so is the problem of root surface dental caries.

Current demographic data from the industrialized part of the world indicates that there will be an increasing percentage of individuals above 65 years during the next twenty five years. Also, improved dental techniques and greater utilization of dental services during youth and middle age together with an increased awareness of oral hygiene has resulted in a greater retention of the natural dentition in old age which previously would have been lost due to dental caries at an early age. Furthermore, the decreasing trend in caries prevalence in children will lead to an increased number of teeth present in older individuals.

There is a general agreement that the exposure of cementum to the oral environment is a prerequisite for the development of root surface caries. The root surface, due to its structure seems to be more vulnerable to chemical and mechanical destruction than enamel. All reports have also made references to periodontal disease either prior to or at the time of initiation of root surface lesions. A study by Schamschula et al. (1974) has confirmed that a significant relationship exists between root caries and periodontal disease. However not all types of periodontal disease may be conducive for the initiation of root caries. Sumney et al. (1975) have reported that root surfaces associated with periodontitis pockets frequently showed signs of root caries whereas root surfaces associated with periodontosis pockets of similar depth were often relatively free of
plaque and signs of caries.

Due to the fact that research on root caries has been neglected in the past, knowledge on its etiology is quite limited. However, bacterial plaque and diet have been implicated as the main etiological factors of root surface dental caries. Studies (Beck et al. 1976) have also suggested that root caries may have a complex etiology in which overall physical and oral health, behaviour and social factors are important contributors. These contributors should be interpreted as correlates of root caries and not as true risk factors.
1.3 AIM OF THESIS

Root surface dental caries is one of the most frustrating conditions that a dentist can encounter.

From the operative point of view, it is difficult to restore, especially when it extends below the gingiva. Also a restoration in this area may be sensitive to thermal changes due to the proximity of the pulpal tissues. Furthermore, recurrent caries is also a frequent problem. Severe recurrent cemental caries also seems to be a problem in geriatric patients using prosthodontic appliances. Hence the best approach to this would be the prevention and/or the arrest of the lesion. It has been proved that fluorides have a preventive effect on coronal caries. Hence it would be logical to assume that fluorides would have a similar effect on root caries.

The aim of this thesis is to study the relationship between fluorides and root surface dental caries by reviewing some of the available literature.
2 HARD STRUCTURES OF THE ROOT

2.1 CEMENTUM

Cementum is the mineralized dental tissue covering the anatomic roots of human teeth. The main function of the cementum is that it acts as a medium for the attachment of collagen fibres that bind the tooth to the alveolar bone.

2.1.1 Physical Characteristics

Cementum is lighter in colour and not as hard as dentine. It lacks the lustre of enamel and is of a darker yellowish hue (Armitage 1980). Two types of cementum have been described based on the presence or absence of cells in its matrix - acellular type and cellular type (Avery 1987). Acellular cementum develops when no trapped cementoblasts are embedded in the matrix. It generally covers the coronal half of the root, is thinner near the cemento-enamel junction and increases in thickness towards the apex. Acellular cementum thickness varies between 20-50 μm (Freeman 1985).

The cellular cementum develops when some cementoblasts get embedded in the matrix (Avery 1987). It has been observed in the apical third of the root, and was reported to be more uneven than the acellular type (Furseth 1967). The thickness varies between 150-250 μm (Freeman 1985). However, layers of acellular and cellular cementum may alternate at any site (Armitage 1980). [Figure 1]

Another type of cementum has also been described - intermediate cementum (Armitage 1980; Avery 1987). It appears as a layer between the cementum and dentine and does not exhibit either feature. It is
Figure 1: Diagrams of human teeth, illustrating what parts of the roots are covered with acellular cementum (darkly shaded) or cellular cementum (hatched). The middle of the root may be covered with acellular cementum, overlapped by a layer of cellular cementum.

Source: Salentijn and Klyver 1985, p260
Figure 2: Graph showing wet weight (left) and volume (right) composition of cementum.

Source: Furseth and Mjor 1979. p106
predominantly observed in the apical two-thirds of molars and premolars (Armitage 1980).

Freeman (1985) has also described another classification of cementum - intrinsic, extrinsic and mixed fibre type and this is based on the type of collagen fibres in the cementum matrix. Unlike bone, cementum is avascular (Avery 1987).

2.1.2 Chemical Composition

There is a general agreement that cementum consists of both inorganic and organic substances (Freeman 1985; Furseth and Mjor 1979; Armitage 1980). The cementum on a dry weight basis consists of 45-50% inorganic substances and 50-55% organic material and water (Armitage 1980). Furseth and Mjor (1979) state that on a wet weight basis, the inorganic content is 65% while the organic content is 23% and the remaining 12% is water. [Figure 2] In contrast, enamel comprises 96% inorganic matter and only 4% of organic substance and water while dentine has 70% inorganic matter and 30% organic matter and water.

The main bulk of the inorganic substances is calcium phosphate being in the form of hydroxyapatite. Other minerals in smaller quantities like magnesium, sodium, fluoride, zinc, iron, copper, silver and tin have been reported present (Hals and Selvig 1977). It is also of interest to note that cementum has the highest concentration of fluoride than any other calcified tissue in the body. The organic matrix is primarily made up of collagen (~90%) while the remaining component is made up of protein polysaccharides (Armitage 1980; Furseth and Mjor 1979).
Figure 3: Incremental lines in acellular cementum.

Source: Armitage 1980, p189
2.1.3 Structure

Several structural features are observed in the cementum.

a. Collagen Fibres

Two types of fibres are present.

(i) The fibres of periodontal origin which run at approximately right angles to the cemento-dentinal junction (Furseth 1967; Selvig 1965). They get embedded in the cementum through continuous cementum deposition and are known as Sharpey's fibres and they connect the alveolar bone to the tooth.

(ii) The fibres of the cementum itself are produced by the cementoblasts (Salentijn and Klyvert 1985). They are usually parallel to the tooth surface although some have been reported to be irregularly arranged. These fibres do not take part in the attachment of the tooth (Selvig 1965).

b. Incremental Lines

The incremental or resting lines are formed as a result of the rhythmic pattern of cementum formation with alternating phases of activity and quiescence. These lines have a higher mineral content than adjacent cementum (Furseth and Mjor 1979; Avery 1987) as well as a higher content of ground substance and lower content of collagen (Furseth and Mjor 1979). [Figure 3]

c. Precementum

This zone covers the cementum. It is about 3-5 μm in acellular cementum (Selvig 1965) and slightly wider in cellular cementum (Furseth and Mjor 1979). Electron microscopic studies by Selvig (1965) demonstrates the presence of collagen bundles as well as
Figure 4: Cellular cementum from human premolar.

Source: Armitage 1980. p187
single fibres. Furseth (1969) observed that cells in this zone showed features similar to cementoblasts.

d. Cementoblasts
The cementoblasts are found on the surface of the cementum and are responsible for the formation of the organic matrix. These cells have numerous mitochondria, a well formed Golgi apparatus and large quantities of granular, endoplasmic reticulum (Furseth 1969; Armitage 1980).

e. Lacunae and Canaliculi
These are usually observed in the cellular cementum. The lacunae are roundish or ovoid in shape and approximately 8 μm in diameter (Furseth 1967). They are mineralized and usually contain the cementocyte. [Figure 4]

The canaliculi extend from the lacunae, are uncalcified and have regular, circular appearance in cross section. They contain the cellular processes of the cementocytes. The majority of the canaliculi point towards the periodontal ligament (Freeman 1985).

f. Cementocytes
The cementocytes are cementoblasts embedded in the cementum during matrix formation. They usually contain fewer processes than the cementoblasts (Furseth 1969).

Typical Features of Acellular Cementum
Sharpey's fibres constitute the major part of the organic matrix of the acellular cementum and in some cases these fibres may continue a short distance into the dentine (Furseth 1974; Kvam 1973). Some
fibres in different directions have been observed and has been interpreted as the intrinsic matrix (Furseth 1974; Selvig 1965) although Kvam (1973) doubted the presence of an intrinsic matrix component. In only very few instances could incremental lines be discerned (Furseth 1967). This is attributed to slow cemental deposition, resulting in the incremental lines being very close together (Furseth and Mjor 1979). However, where the cementum is thicker they can be observed more easily (Furseth 1967).

The mineral crystals in acellular cementum are almost homogeneously distributed except in the inner zone where they seem to be less in quantity and in some areas completely free of it. The crystals are normally observed with their axes parallel to the collagen fibres of the organic matrix (Selvig 1965).

Typical Features of Cellular Cementum

Sharpey's fibres have been observed at varying lengths along the surface as well as in deeper layers of cellular cementum. They appear as circular structures separated from each other by calcified tissue containing randomly arranged matrix fibres. They usually consist of an irregularly shaped unmineralized central core, surrounded by a highly mineralized periphery (Selvig 1965).

The incremental lines are situated further apart than in acellular cementum as cellular cementum is formed at a faster rate than acellular cementum (Furseth and Mjor 1979).

The hydroxyapatite crystals at the surface appear as thin, plate-like structures and reach their maximum mature crystal size of 80 A close to the calcification zone (Selvig 1965).
Figure 5: Intermediate layer of cementum.

Source: Armitage 1980. p193
Lacunae containing cementocytes and canaliculi containing their processes are typical features of cellular cementum (Furseth and Mjor 1979). In the deeper layers of the cementum, the cementocytes display few processes and exhibit stages of degeneration while the deepest layer may show empty lacunae (Avery 1987).

*Intermediate Cementum*

An intermediate layer of cementum has also been described (Armitage 1980; Avery 1987). It appears between the dentine and cementum and is predominantly observed in the apical two-thirds of molars and premolars (Armitage 1980). [Figure 5]

Avery (1987) describes it as being similar to aprismatic enamel and describes it as an amorphous layer of noncollageneous material containing no odontoblastic processes or cementocytes.

This layer appears to be formed by cells of the epithelial root sheath when they are trapped in rapidly deposited cementum or dentine (Armitage 1980). This layer then becomes more mineralized than either cementum or dentine. It probably serves to seal the sensitive surface of root dentine (Avery 1987).
Figure 6: Schematic drawing showing Hertwig's root epithelium and adjacent structures prior to onset of cementogenesis (a) and a more advanced stage of development with breakdown of Hertwig's root epithelium and onset of cementum formation (b).

Source: Furseth and Mjor 1979. p114

Note:  
O - odontoblast layer  
PD - predentine  
D - dentine  
E - enamel  
R - reduced dental epithelium  
H - Hertwig's root epithelium  
C - cementum  
PC - precementum  
CB - cementoblasts  
M - Malassez epithelial rests
2.1.4 Cementogenesis

Root development begins sometime after the crown is formed (Furseth and Mjor 1979; Armitage 1980) and when eruptive movement of the developing tooth has begun (Salentijn and Klyvert 1985). Cementum formation is preceded by the deposition of dentine along the inner epithelium of the Hertwig's root sheath. Following this, there is a breakdown of the Hertwig's epithelium resulting in contact between the newly formed dentine and dental follicle. Mesenchymal cells from the connective tissue of the dental follicle, which differentiate into cementoblasts are responsible for the formation of the cementum matrix. [Figure 6]

Ultrastructural changes occurring in the epithelial root sheath have been studied in animals (Furseth and Mjor 1979). These include degeneration of the basal lamina and presence of collagen fibrils between the epithelial cells. There is migration of some of the epithelial cells towards the dental sac and these become the epithelial rests of Malassez found in the periodontal ligament of fully formed teeth. Some epithelial cells also get incorporated into the cementum (Furseth and Mjor 1979; Armitage 1980).

The unmineralized layer of cementum is known as cementoid. Mineralization begins after some cementoid is deposited. Calcium and phosphate ions derived from tissue fluids are deposited as hydroxyapatite crystals on the surface of and between the collagen fibrils in an orderly manner.
**Figure 7:** Schematic drawing showing the cemento-enamel junction and the three relationships that can exist between cementum and enamel. I, cementum and enamel just meet. II, there is a layer of dentin devoid of cementum next to the enamel. III, cementum overlaps the enamel.

**Source:** Furseth and Mjor 1979. p119

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**Note:**

E - enamel  
D - dentine  
C - cementum
2.1.5 Cemento-Enamel Junction

Ramsey and Ripa (1969) after studies on premolar teeth, reported that at the cemento-enamel junction, the cementum just met the enamel in 61.25% of the cases, failed to meet the enamel in 31.25% of the cases and overlapped the enamel in 7.5% of the cases.

Other authors however (Armitage 1980; Furseth and Maj 1979) report that in about 30% of the cases they meet, forming a distinct cemento-enamel junction, in about 60% of the cases overlapping occurs while in 10% they do not meet. [Figure 7]

Overlapping occurs when some of the reduced dental epithelium is broken down, resulting in development of cementoblasts and formation of cementum on enamel. Where enamel and cementum do not meet occurs when the Hertwig's root epithelium does not disintegrate, thus preventing contact between the dentine and connective tissue.

2.1.6 Cemento-Dentinal Junction

Light microscopic studies revealed that acellular cementum stained heavier than dentine and the cemento-dentinal junction and the surface of the cementum formed straight smooth lines. Microradiographs showed a radiodense line about 10 microns wide separating the cementum from dentine (Furseth 1967).

However, cellular cementum did not stain as heavily as the acellular type so it was difficult to determine the exact location of the cemento-dentinal junction in light microscopic studies, although a radio-dense line was sometimes observed in microradiographs (Furseth 1967).
Kvam (1973) described the principal fibres branching out as finger-like projections rendering an irregular border between the dentine and cementum. There was intertwining of the cementum and dentine fibres, hence making it difficult to distinguish between fibres of cemental origin and those of dentinal origin.

2.1.7 Age and Functional Changes

Cementum is deposited throughout life and its width increases with age. With aging, the surface of the cementum becomes irregular with more deposition at the apical region. [Figure 8] Microscopically, only the surface cementocytes appear viable. The older root surface also has more fibre bundles than the younger ones (Avery 1987; Armitage 1980).

Cementicles, which are calcified bodies, are seen on or in the cementum and in the periodontal ligament. Cementicles are seen in older individuals and are a response to local trauma or hyperactivity (Avery 1987).

In addition to ordinary continuous cementum formation, additional cementum deposition can be observed for other reasons like repair of primary teeth, repair of resorption cavities in permanent teeth, cementum deposition in connection with root fracture, compensatory cementum deposition in attrition, as well as in reattachment of regenerated periodontal ligament fibres (Furseth and Mjor 1979).

When cementum deposition exceeds its physiologic limit, it results in a localized or general hypercementosis. Localised hypercementosis is associated with chronic periapical inflammation while general hypercementosis is associated with some systemic diseases.
Cementum is more resistant to resorption than bone. This is clinically significant as orthodontic tooth movement is made possible because of this.

Pathologic resorption can occur after trauma or excessive occlusal forces. Repair in these cases can occur either by the formation of cellular or acellular cementum, or both, resulting in the formation of a reversal line (Avery 1987).
2.2 DENTINE

Dentine is a vital tissue and comprises the bulk of the tooth structure. Next to enamel, it is the second most highly mineralized tissue. It serves as a protective covering for the connective tissue of the pulp organ.

2.2.1 Physical Characteristics

Crown dentine is covered by enamel up to the cervical margin while root dentine is covered by cementum. Dentine is light yellowish in colour and is very resilient. It is softer than enamel but harder than bone (Cox 1987; Avery 1980).

2.2.2 Chemical Composition

Dentine consists of about 30% organic material and water and about 70% inorganic substances. The inorganic substance is mainly hydroxyapatite crystals with small amounts of phosphates, carbonates and sulphates (Silverstone and Hicks 1985; Avery 1980). The organic substance comprises collagenous fibrils and a ground substance of mucopolysaccharides (Silverstone and Hicks 1985). The organic phase also contains small quantities of carbohydrates as proteoglycans and glycoprotein components as well as some liquid components. (Cox 1987).

2.2.3 Dentinogenesis

Dentinogenesis occurs in two phases - the formation of the uncalcified organic matrix, the predentine and its subsequent mineralization (Avery 1980; Cox 1987).
Figure 9: Relationship between odontoblast process and dentin tubule.

Source: Cox 1987, p153
2.2.3.1 Predentine Formation

The differentiation of the odontoblast forming root dentine is initiated by the Hertwig’s root sheath (Ten Cate 1985). The first sign of predentine formation is the appearance of bundles of collagen fibres between the differentiating odontoblasts. These fibres are the Von Kroff’s fibres and together with ground substance comprise the major portion of the first formed matrix or mantle predentine (Mjor 1979; Avery 1980). These fibres have a diameter of approximately 0.1–0.2 μm. The remainder of the mantle dentine comprises fibrils around 0.05 μm in diameter. These smaller fibrils predominate through the remaining predentine layers (Avery 1980). As collagen is being formed, the odontoblasts buds off a series of vesicles, the matrix vesicles which lie between the Von Kroff fibres.

As more of the matrix gets deposited, the odontoblasts begin to recede towards the pulp. Each odontoblast leaves a long cytoplasmic cell extension, the odontoblastic processes in the matrix which is enclosed in individual dentinal tubules. [Figure 9] The odontoblasts and their processes remain a vital part of the dentine throughout the life of the tooth (Ten Cate 1985; Salentijn and Klyver 1985).

2.2.3.2 Mineralization

Mineralization begins after about 4 μm of predentine has been laid down (Avery 1980). The apatite crystals first appear in the matrix vesicles as single crystals which grow, rupture and spread to form crystallites or calciospherites. These crystallites fuse and form the mineralized matrix of the mantle dentine. (Ten Cate 1985)
Figure 10: Schematic diagram of odontoblasts and odontoblast processes. Endoplasmic reticulum, Golgi apparatus, and mitochondria are limited largely to the cell body. The odontoblast processes contain mainly filaments and microtubules.

Source: Mjor 1985. p623
Following this initial calcification, crystals appear on the surface and within the collagen fibrils and are orientated parallel to the fibres. Mineralization takes place throughout the predentine until only the newly formed predentine along the pulp is uncalcified (Cox 1985). Hence there is always a layer of predentine between the odontoblasts and mineralized dentine (Ten Cate 1985).

Dentine formation continues until formation of the tooth is complete and at this stage is known as primary dentine. Following this it is deposited at a much slower rate and this is called secondary dentine.

2.2.4 Structure

The basic structural entities of the dentine are the odontoblasts and odontoblastic processes, the dentinal tubules, the periodontoblastic space, the peritubular and intertubular dentine.

The odontoblasts are found on the pulpal surface of the dentine and are responsible for the formation of the organic matrix of the dentine (Avery 1980). The cells are elongated and contain Golgi complex, rough endoplasmic reticulum, light to dense staining bodies, mitochondria and secretory granules (Cox 1987) with their nuclei situated in the basal end (Silverstone and Hicks 1985).

The odontoblastic processes are long cytoplasmic extensions from the odontoblasts and traverse through the dentine. These processes contain mainly filaments and microtubules (Mjor 1985). [Figure 10]

Each of the odontoblastic processes is enclosed in a dentinal tubule ranging from 1-4 µ in diameter. The course of the tubules in the
root is almost straight. In the dentine the tubules have lateral branches called canaliculi (Cox 1987). Branching is also present in the terminal part of the tubules and is more profuse in the root dentine than coronal dentine. There are less tubules per unit area in the root than in the crown (Avery 1980).

The dentinal tubules are surrounded by a highly mineralized collar of peritubular dentine (Avery 1980; Cox 1987) ranging from 0.5 to 2 μm in width. The collagen fibres in the peritubular dentine are delicate with a diameter of 25-50 nm and are smaller and lesser in quantity than elsewhere in the tissue (Silverstone and Hicks 1987). This dentine has a 9% higher mineral content than the remaining dentine (Cox 1987).

The remaining bulk of dentine between the tubules is the intertubular dentine which has large amounts of coarse, collagen fibres and is less mineralized than the peritubular dentine (Salentijn and Klyver 1985).

Another structural entity, the periodontoblastic space has been described as being between the tubule wall and odontoblastic process containing tissue fluids and few organic components like collagen fibres. This space together with the odontoblastic processes comprise the dentine soft tissue and any tissue changes in the dentine occur here (Mjor 1979).

The interglobular dentine describes areas of hypomineralized dentine and is normally observed in the coronal portion of the mature circumpulpal dentine (Cox 1987) and represents failure of fusion of crystallites at the dentine-predentine interphase (Mjor 1985).
The Tomes granular layer is another hypomineralized area seen only in the root and appears as a narrow layer adjacent to the cementum (Cox 1987) and represents an interface with mineralization of the entire root surface dentine layer prior to cementum formation (Avery 1980).

A hyaline layer between the cementum and dentine has also been described (Ten Cate 1985). It is believed to be a product of the root sheath cells and functions to seal the cementum to the dentine.

The mineral crystals of the dentine are arranged parallel to the long axis of the collagen fibrils. They are plate-like and are 15-30 nm in width (Silverstone and Hicks 1985). Generally, mineral distribution is fairly homogeneous except for the mantel dentine and dentine close to the pulp in newly erupted teeth, which have less mineral content, interglobular dentine and Tomes granular process which are unmineralized (Mjor 1979).

Incremental lines in the dentine are of two types - incremental lines of Von Ebner and contour lines of Owen. The incremental lines of Von Ebner indicate the growth pattern of the dentine with the distance between the lines corresponding to the daily apposition of the dentine (Avery 1980). Dentine deposition in the root is slower, hence these lines are observed more frequently and separated by a distance of about 3-5 µm (Ten Cate 1985).

The contour lines of Owen are observed in the dentine and are the result of disturbances in the mineralization process and represent hypocalcified bands (Avery 1980).
2.2.5 Innervation

Ultrastructural studies on dentine of human premolars have revealed the presence of intratubular nerve fibres which are probably derived from the inferior alveolar nerve, a branch of the trigeminal nerve (Lilja 1979) and also from branches of the cervical sympathetic ganglion (Cox 1987). The fibres are mainly confined to the predentine and most pulpal part of the dentine (Lilja 1979; Dahl and Mjor 1973) and are significantly lower in number in the apical region. These nerves follow the course of the odontoblastic processes and are located in the periodontoblastic space (Dahl and Mjor 1973; Avery 1980) and they contain neurofilaments, neurotubules, mitochondria and small vesicular structures (Cox 1987; Avery 1980). Some fibres are close to the cell body of the odontoblast in the odontoblastic layer and are characterized by lack of neurofilaments (Dahl and Mjor 1973).

2.2.6 Age and Functional Changes

Dentine is considered as being vital as the odontoblasts and its process are an integral part of it. Various age and functional changes have been observed in the dentine and these are:

2.2.6.1 Secondary Dentine

Dentine formed before the completion of the apical foramen is called primary dentine. Once this occurs, dentine formation occurs at a slower rate and is called secondary dentine (Salentijn and Klyvert 1985) and is deposited on the entire pulpal surface of the dentine (Avery 1980). However, the rate and formation is not even in all the areas especially in the molar teeth where there is a greater
deposition of dentine on the root and floor of the pulp chamber (Ten Cate 1985). The dentinal tubules follow a more irregular course and are lesser in number. An increase in the width of secondary dentine is observed in the older age group (Mjor 1979). The interface dentine – the junction of the primary and secondary dentine is often atubular and may contain cell inclusions (Mjor 1985).

2.2.6.2 Reparative Dentine

Also referred to as tertiary dentine, it is produced as a response to external stimuli, caries or restorative dental procedures (Ten Cate 1985). This dentine is formed only in localized areas affected by the stimulus (Mjor 1985). Reparative dentine is deposited rapidly and morphologically displays a sparse irregular tubular pattern with some cellular inclusions (Ten Cate 1985).

2.2.6.3 Sclerotic Dentine

Sclerotic dentine is formed as a protective mechanism in the existing dentine to loss of dentine or enamel by attrition or environmental trauma (Avery 1980). In this case, the tubules are occluded by a mineral complex rendering them less permeable and thus prolonging pulp vitality (Cox 1987; Salentijn and Klyver 1985). When ground sections of sclerotic dentine are viewed through transmitted light, it assumes a glossy appearance and hence is also known as transparent dentine (Salentijn and Klyver 1985).

Sclerotic dentine is also observed as a natural occurrence in the aged and is most commonly observed in the apical third of the root and dentine so affected is referred to as physiologic sclerotic dentine (Ten Cate 1985).
Figure 11: Diagram of root caries, dead tracts, sclerosed tubules, and reparative dentine.

Source: Cox 1985. p159
2.2.6.4 Dead Tracts

Areas of dentine characterized by degenerated odontoblastic processes are called dead tracts (Avery 1980). This degeneration may occur as a result of some severe irritation of the dentinal tubules. When observed in ground sections, the air-filled tubule appears as black dead tracts (Ten Cate 1987). The pulpal end of the tract may get occluded by sclerotic dentine which may be covered with reparative dentine (Cox 1987).

[Figure 11] illustrates dead tracts, sclerosed and reparative dentine.
3 ROOt SURFACE DENTAL CARIES

3.1 PREVALENCE

Epidemiological investigations on caries have been confined almost exclusively to enamel and dentinal caries of the crown, this being in spite of the fact that caries of the root surface has plagued man since ancient times. Ancient skulls (Moore and Corbett 1971-1973; Lunt 1974) and primitive communities of the present day (Mehta and Shroff 1965; Schanschula et al. 1974) typically show a predominance of root surface caries occurring at the cemento-enamel junction in adult life, coronal caries being relatively uncommon.

The state of development regarding epidemiological studies of root surface caries has been described as being inadequate, rudimentary and even non-existent (Banting and Courtright 1975; Hazen et al. 1973; Jordan and Sumney 1973). Despite the lack of current epidemiological data on root caries, the need for it will definitely arise in the next twenty-five years due to the increase in the aging population as well as the increase in tooth retention. Only in 1987 did the World Health Organization make provisions to record root surface caries by including age groups of 65-74 years as they felt that the data for this group was necessary for planning appropriate care for the elderly and for monitoring the overall effects of oral health care services in a population.

Different methods have been used in reporting the frequency of root caries (Katz 1980). One of the most popular methods was to report the percent of study populations exhibiting one or more lesions (Table 1). This method was only useful for comparative descriptive
Table 1: Prevalence* of root caries and/or fillings (RDF), in selected adult population groups aged 18+.

*Proportion of all persons examined.

<table>
<thead>
<tr>
<th>Group</th>
<th>Investigator</th>
<th>N</th>
<th>%RDF</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Community</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) non-fluoridated</td>
<td>Hazen et al. 1972</td>
<td>500</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>Stamm &amp; Banting 1980</td>
<td>465</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>Vehkalahti et al. 1983</td>
<td>5028</td>
<td>17</td>
</tr>
<tr>
<td>(b) fluoridated</td>
<td>Stamm &amp; Banting 1980</td>
<td>502</td>
<td>21</td>
</tr>
<tr>
<td>Military Personnel</td>
<td>Sumney et al. 1973</td>
<td>172</td>
<td>44</td>
</tr>
<tr>
<td></td>
<td>Lohse et al. 1977</td>
<td>281</td>
<td>15</td>
</tr>
<tr>
<td>Periodontal Patients</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) treated</td>
<td>Hix &amp; O'Leary 1976</td>
<td>120</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>Hix &amp; O'Leary 1976</td>
<td>124</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>Ravald &amp; Hamp 1981</td>
<td>35</td>
<td>87</td>
</tr>
<tr>
<td>(b) untreated</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronically Ill,</td>
<td>Sumney et al. 1973</td>
<td>135</td>
<td>59</td>
</tr>
<tr>
<td>institutionalized</td>
<td>Banting, et al. 1980</td>
<td>59</td>
<td>83</td>
</tr>
<tr>
<td>Primitive Tribesmen</td>
<td>Schamschula et al. 1972</td>
<td>22</td>
<td>72</td>
</tr>
<tr>
<td>Elderly,</td>
<td>Beck et al. 1985</td>
<td>520</td>
<td>63</td>
</tr>
<tr>
<td>non-institutionalized</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
studies and had less potential for analytical studies for risk factors or etiologies.

The second most common method to report root caries was to count the root caries lesions per person. This was an improvement from the first method as it introduced a measure of severity and permitted more analysis of the risk factors as well as etiologies.

The third method, a modification of the second, was a count of the number of root caries lesions per person. Only teeth present are used as a unit of risk. When reported as a count of root caries lesions per teeth present in an individual, it allows for analysis of risk factors associated with an individual.

Although each method of reporting acquired a better standard and was more refined, there was a need for developing a universal method in reporting root surface caries. Katz (1980) put forward the root caries index (RCI) which is:

\[
\text{Number of root caries lesions} \times 100 = \text{RCI Score}
\]

\[
\text{Number of teeth with gingival recession per person}
\]

which in epidemiological terms is an attack rate for the disease of root caries. In future, the RCI should prove to be a feasible method for reporting root caries data in descriptive and analytical epidemiologic studies as well as in clinical trials for assessing preventive and treatment agents.

The prevalence of root surface caries has been reported on specific groups of populations [Table 1]. The prevalence of root caries ranges between 15-87%. For healthy ambulant adults, it is between 20-40%. A dramatic increase is observed in specific groups like
Figure 12: Prevalence (percent of all persons examined) of root caries and/or fillings by age and population group.

Source: Banting 1986. p6
Figure 13: Prevalence (percent of all persons examined) of root caries and/or fillings by age and sex.

Source: Banting 1986. p8
primitive tribesmen, the institutionalized, chronically ill patients, the elderly and those patients with destructive periodontal disease.

An early comparative study between drug addicts and non-drug addicts showed a high prevalence of root caries in drug addicts. General malnutrition, the addict's craving for sweets resulting in increased sugar intake and poor oral hygiene were considered as contributing factors (Hecht and Friedman 1949).

One of the earliest studies to demonstrate that the prevalence of root caries increases with age was the Vipsholm study where more than half of all new lesions in subjects above 50 years of age developed in the root surface (Gustafsson et al. 1954). Subsequently, other studies have also shown that root caries increases with age [Figure 12]. Furthermore, Katz et al. (1985) having observed that RCI values are much the same in each decade of life from 20-80 years suggested that the increased prevalence of root caries lesions observed in older patient age groups may be directly related to the occurrence of recession rather than age.

Studies by Stamm and Banting (1980) and Burt et al. (1986) have also shown that life-long consumption of fluoridated water is capable of significantly reducing the prevalence of root caries.

Differences in prevalence rates have also been shown for males and females. [Figure 13] It has been observed from studies that males tend to display slightly higher rates of root caries lesions than females (Vehkalahti et al. 1983; Katz et al. 1985). Vehkalahti et al. (1983) suggest that this difference may be due to many factors,
Figure 14: Prevalence (percent of all persons examined) of root caries and/or fillings by age and country.

Source: Banting 1986. p8
Figure 15: RCI rates and percent of surfaces at risk for each tooth type for the total population.

Source: Katz et al. 1982. p269
Figure 16: Distribution of root caries and/or fillings by surface.

Source: Banting 1986. p8
Table 2: Relative likelihood of specific surfaces to show recession and relative ratios for the RCI rates on specific surfaces for each tooth type.

Source: Katz et al. 1982. p268

<table>
<thead>
<tr>
<th></th>
<th>Recession B:L:I</th>
<th>RCI B:L:I</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maxillary</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Molars</td>
<td>7:2:1</td>
<td>1:5</td>
</tr>
<tr>
<td>Premolars</td>
<td>13:1:2</td>
<td>1:2</td>
</tr>
<tr>
<td>Canines</td>
<td>12:1:2</td>
<td>1:1</td>
</tr>
<tr>
<td>Incisors</td>
<td>6:1:5</td>
<td>2:1</td>
</tr>
<tr>
<td><strong>Mandibular</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Molars</td>
<td>7:1:1</td>
<td>2:1</td>
</tr>
<tr>
<td>Premolars</td>
<td>13:1:3</td>
<td>8:1</td>
</tr>
<tr>
<td>Canines</td>
<td>5:1:2</td>
<td>8:1</td>
</tr>
<tr>
<td>Incisors</td>
<td>3:1:1</td>
<td>3:1</td>
</tr>
</tbody>
</table>

B = Buccal; L = lingual; I = interproximal.
for example the subject's oral hygiene, health habits and frequency of dental visits. Also, previous reports on the Finnish adult population have shown that women have better gingival conditions (Markkanen 1982), less retained roots than men (Vehkalahti et al. 1982) and the mean number of teeth is consistently 1-2 teeth lower for women than men (Vehkalahti et al. 1981).

Only two sets of data are available to make a direct comparison of the prevalence rate of root caries between countries [Figure 14]. The occurrence of the disease is much higher in North America (Katz et al. 1982) than in Finland (Vehkalahti et al. 1983). This may be due to differences in cultural background.

Assessment of teeth and teeth surfaces most susceptible to root surface caries has been difficult to document since in the older, susceptible individuals, tooth loss may already be considerable. However, Katz et al. (1982) have observed that the most frequently attacked tooth types were the mandibular molars (RCI=40%), mandibular premolars (RCI=25%) followed by the maxillary canines (RCI=23%). The least frequently attacked tooth type were the mandibular incisors (RCI=2%). [Figure 15]

In terms of surfaces attacked, studies (Sumney et al. 1973; Banting et al. 1980) have shown that the buccal proximal and lingual surfaces are the primary sites of attack. [Figure 16]. Katz et al. (1982) have further compared the likelihood of different surfaces becoming carious by tooth types and recession rates. [Table 2] For example, although the buccal surface of maxillary molars are seven times more likely to exhibit gingival recession
than the interproximal surface, the RCI rate for the interproximal surface is four times that of the buccal surface. This suggests that specific intraoral environmental factors may play a significant role in determining which surfaces with recession will succumb to the root caries attack.
3.2 CLINICAL FEATURES

Four destructive lesions that may affect the root surface of a tooth exposed to the oral environment have been described (Hazen et al. 1973). These are abrasion, erosion, idiopathic resorption and root caries.

Katz et al. (1982) described the root caries as soft, progressive destructive lesions either totally confined to the root surface or involving undermining of the enamel at the cemento-enamel junction but clinically indicating that the lesion initiated at the root surface. Similar descriptions were adopted by Vehkalahti et al. (1983), Sumney et al. (1973), Hazen et al. (1973).

Hix et al. (1976) described root surface caries as a cavitation or softened area in the root surface - generally well established, discoloured and characterized by penetration and destruction of the root surface and underlying dentine. The point of an explorer can easily be inserted into the carious area with moderate finger pressure.

The lesions have been described as being shallow, about 0.5-1 mm in depth, but could range from a slight surface etch to a 3 mm cavity. The lesions have well defined margins which could be classified as elliptical, round or band like, with the latter being described as the most invasive and involving more than one surface. Discolouration is a prominent feature. The lesions begin as one or more circular areas that spread concentrically, coalesce or do both, but rarely penetrate the pulp. It has also been suggested that the direction and extent of the spread of the lesion depend on the tooth
Table 3: Root surface caries severity index.

*Source:* Billings 1986, p44

Grade I (Incipient)
i. Surface texture: soft, can be penetrated with a Dental Explorer
ii. No surface defect
iii. Pigmentation: variable, light tan to brown

Grade II (Shallow)

i. Surface texture: soft, irregular, rough, can be penetrated with a dental explorer
ii. Surface defect (less than 0.50 mm in depth)
iii. Pigmentation: variable, tan to dark brown

Grade III (Cavitation)

i. Surface texture: soft, can be penetrated with a dental explorer
ii. Penetrating lesion, cavitation present (greater than 0.50 mm in depth) no pulpal involvement
iii. Pigmentation: variable, light brown to dark brown

Grade IV (Pulpal)

i. Deeply penetrating lesion with pulpal or root canal involvement
ii. Pigmentation: variable, brown to dark brown
surface involved and that the outline of the lesion is indicative of its stage of development (Banting and Courtright 1975).

Other studies (Nyad and Fejerskov 1982; Banting and Ellen 1976) have described two types of lesions - active lesions as being yellowish or light brown in colour and softened without obvious cavitation and passive or remissive lesions as being darkly stained, often almost black with a leathery consistency. These lesions may be as hard or even harder than the non-diseased root surface.

Katz (1986) describes a slightly modified set of visual-tactile criteria for the identification of root caries and diagnosed root caries as being present when either of the following criteria are satisfied.

a. Active Lesion
Any root surface area with or without frank cavitation which exhibits:

(i) a darkened discoloured appearance and

(ii) a tacky or leathery feel upon probing with moderate pressure.

b. Inactive Lesion exhibiting gross cavitation
Any root surface area which exhibits a frank cavitation with a darkened, discoloured appearance but without a tacky or leathery feeling upon probing with moderate pressure.

Billings (1986) has developed a root surface caries severity index [Table 3] for the purpose of segregating lesions by type and also to serve as a basis for treatment planning.

Not much literature is available on the radiographic diagnosis of root caries. Buchholz (1965) has discussed the difficulties in
diagnosing dental caries. However, textbooks (Frommer 1981; Bhaskar 1979; Wuehrmann and Manson-Hing 1977) have described them as saucer-shaped radiolucencies with poorly defined borders and associated bone loss. These lesions radiographically can be mistaken for cervical burnouts and cervical abrasions.
3.3 HISTOPATHOLOGICAL FEATURES

Bacterial enzymes and organic acids play an important role in the development of root surface dental caries. The acids primarily cause demineralization while the enzymes denature the collagen matrix of the cementum and dentine. However, from studies of lesions in extracted teeth, it has been difficult to establish the sequential change of root caries formation, i.e., whether demineralization of the inorganic substance precedes degradation of the organic matrix by bacterial enzymes (Phankosol et al. 1985).

The mineral phase of the cementum has been shown to undergo several changes in the carious process. These include demineralization and remineralization (Furseth and Johansen 1968). Caries of the cementum has been reported to start as a partial decalcification which soon reaches the peripheral dentine and may extend to a depth of a third of a millimetre without any loss of surface contour at the light microscopic level of observation (Hals and Selvig 1977).

According to Nyad and Fejerskov (1982), the early carious lesion shows an outer radiopaque zone about 10-30 μm wide and appears to be hypermineralized relative to the adjacent cementum. The underlying cementum appears as radiolucent zones except at the region of the cemento-dentinal junction which appears radiopaque. This is in agreement with descriptions provided by Furseth and Johansen (1968), Selvig (1969) and Hals and Selvig (1977). This hypermineralized layer is also a consistent finding on exposed root surfaces although it is thinner than that found on exposed carious cementum (Furseth and Johansen 1968). It is not found on non-exposed tissue.
Selvig (1969) suggested that this hypermineralized layer might be due to mineral exchange at the cementum-saliva interface. Furseth (1970) suggested that it might be due to exposure to fluorides while Mellberg (1986) says that it might be due to redeposition of mineral from the advancing front of the lesion.

In the initial stage, the surface zone of the carious cementum shows a variety of ultrastructural changes. This highly mineralized surface layer of carious cementum often reveals plaque as well as masses of bacteria attached to the surface (Furseth and Johansen 1970). The surface also appears uneven. Micrororganisms were also reported within lacuna-like spaces in the surface cementum indicating the apparent loss of crystals. There were changes in the shape of the hydroxyapatite crystals which were of varying sizes. The collagen fibres close to the bacterial plaque appear split (Furseth and Johansen 1970; Furseth 1971; Nyad and Fejerskov 1982). Demineralization was reported to begin in the subsurface cementum (Furseth and Johansen 1968). Hals and Selvig (1977) attribute this to the very high fluoride content of the surface cementum which makes it less soluble than the subsurface cementum.

Studies have also shown that destruction of the apatite crystals takes place prior to bacterial penetration of root cementum. However, bacterial invasion seems to occur much earlier than in coronal caries (Furseth 1971; Nyad and Fejerskov 1982). As the carious process advances deeper into the cementum, the minerals that are released from the advancing front of the lesion may be reprecipitated loosely along the previously demineralized collagen matrix, resulting in remineralization of an area with decreased
mineral content (Phankosol et al. 1985). Electron microscopic studies have shown thinning of crystals as well as small and large areas devoid of crystals (Furseth 1971).

The caries in the cementum has been observed to penetrate primarily along Sharpey's fibres (Awazawa 1961; Kostlan 1963). Furseth and Johansen (1968) also reported the occurrence of two patterns of demineralization which originated below the highly mineralized surface layer and extended to the cemento-dentinal junction and attributed the course of the Sharpey's fibres to the patterns of demineralization. The brush-like pattern observed could be due to a fairly straight course of Sharpey's fibres while the more uniform pattern of demineralization could be due to a more irregular orientation of Sharpey's fibres.

In addition to this, electron microscopic studies have shown that partial or complete loss of the crossbanding of the collagen fibres occurs in the subsurface regions of carious cementum (Awazawa 1961; Johansen 1965).

Furseth and Johansen (1968) have reported that the relatively high caries resistance of the radiopaque layer near the cemento-dentinal junction probably represents secondary mineral deposition, most likely rich in fluoride.

Advanced carious lesions sometimes shows fracture lines and loss of the surface layer which were usually parallel to the tooth surface and followed the incremental lines (Furseth and Johansen 1968). As the cariogenic attack progresses into the dentine, it
demineralizes the more highly mineralized peritubular dentine more rapidly than the intertubular dentine (Selvig 1968). However, Phankosol et al. (1985) suggested that the peritubular dentine is more resistant to acid destruction than intertubular dentine. This is observed as finger-like projections at the advancing fronts of the dentinal lesions and represents the dissolution of the intertubular dentine around the dentinal tubules. Wagg (1984) has suggested that the progress of the root caries dentine would be identical to coronal dentinal caries. However, the apparent slow progress of root caries as compared to coronal caries maybe due to the lesser number of dentinal tubules per unit area in the root than the crown (Avery 1980).

Furseth and Johansen (1968) reported that the carious dentine had a lower mineral content than carious cementum and spread of the lesion often occurred along the granular layer of Tomes.

Not all root surfaces are covered with cementum. Ramsey and Ripa (1969) have reported that in about 30% of teeth, the enamel and cementum do not meet. Caries at the enamel-root junction may then start at the dentine. Majer (1967) has also observed a hypermineralized zone over these surfaces of exposed dentine.

Electron microscopic studies of carious dentine have revealed that a collagenous matrix with typical crossbanding is present together with fibres with no crossbanding as well as areas with absence of fibres (Johansen and Parks 1961).

Sarnat and Massler (1965) described the 'basic unit' of dentine as consisting of the intertubular contents, the peritubular structures
Table 4: Active carious lesion in dentin.

**Source:** Sarnat and Massler 1965. p1390

<table>
<thead>
<tr>
<th></th>
<th><strong>Intratubular</strong></th>
<th><strong>Peritubular</strong></th>
<th><strong>Intertubular</strong></th>
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<tbody>
<tr>
<td><strong>Necrotic</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Upper zone</td>
<td>Lumen enlarged</td>
<td>Absent</td>
<td>Absent but entirely absent</td>
</tr>
<tr>
<td>Decalcified layer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep zone</td>
<td>Bacteria-free</td>
<td>Absent in parts</td>
<td>Absent but not entirely</td>
</tr>
<tr>
<td>Sclerotic zone</td>
<td>Calcified</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Normal Dentin</td>
<td>Calcified material present</td>
<td>As in normal</td>
<td>As in normal</td>
</tr>
</tbody>
</table>
and the intertubular area. They also distinguished four layers in
the active lesion - a necrotic surface layer, a decalcified layer
subdivided into upper and deep zone, a sclerotic zone and underlying
normal dentin. The changes occurring in the layers are illustrated
in Table 4.
3.4 ARRESTED CARIES

The formation of arrested caries is mainly a defensive reaction of the dentine to prevent the further spread of the carious process.

No literature was available regarding the prevalence of arrested root surface caries.

Clinically, arrested caries appears as darkly stained, often almost black with a leathery consistency. These lesions may be as hard or even harder than the sound root surface (Banting and Ellen 1976; Nyad and Fejerskov 1982).

Katz (1986) described the arrested lesion as exhibiting gross cavitation with a darkened discoloured appearance but without a tacky or leathery feel on probing.

These lesions may remain inactive for a long period of time but in the presence of altered bacteriology of root surface caries, it can become active again (Mount 1986).

Histologically, obvious distinction between arrested and active dentinal lesions is difficult to detect, as active caries, arrested caries and remineralization may all be found in the same carious lesion (Massler 1967). However, Daculsi et al. (1979) have described two stages of arrested caries at the crystal level. Firstly, the material dissolved at the beginning of the carious process precipitates and secondly, there is remineralization of decalcified dentine by growth of the residual crystals and maturation of whitlockite to apatite. In sound dentine, the mean thickness of apatite crystals is about 32 A whereas in arrested
Table 5: Arrested carious lesion in dentin.

*Source:* Sarnat and Massler 1965, p1391

<table>
<thead>
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<th>INTRATUBULAR</th>
<th>PERITUBULAR</th>
<th>INTERTUBULAR</th>
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<tr>
<td><strong>SURFACE LAYER</strong></td>
<td></td>
<td></td>
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<tr>
<td>Upper zone</td>
<td>Some bacteria</td>
<td>Absent</td>
<td>Heavily mineralized</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Calcified</td>
<td>Mineralized</td>
</tr>
<tr>
<td>Pigmented layer</td>
<td>Bacteria and coalesced bacterial bodies inside enlarged lumen</td>
<td>Very thin</td>
<td>Present</td>
</tr>
<tr>
<td>Deep zone</td>
<td>Bacteria-free</td>
<td>Wider</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Transitional zone</strong></td>
<td>No bacteria</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>Some lumen calcified</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>SCLEROTIC DENTIN</strong></td>
<td>Lumina filled with calcified material</td>
<td>Normal</td>
<td>Normal to hypermineralized</td>
</tr>
</tbody>
</table>
caries, it is about 86 A.

Mendis and Darling (1979) reported that most of the tubules in the translucent or sclerotic zone were occluded. The occluded material was found to consist of rod-like plugs of crystals and were continuous for 50 μm or more in length and varied between 2-5 μm in width. Frank et al. (1964) showed that in the early stages of dentinal caries the occluding material was apatite together with larger crystals of whitlockite. The thickness of this hypermineralized zone varies and ranges from 100-2000 μm (Mellberg 1986).

Sarnat and Massler (1965) also agreed that a higher degree of mineralization was a feature of arrested caries and was carried out by three mechanisms - remineralization of the surface layer, source of minerals being the saliva, reprecipitation of the dissolved apatite of the active lesion to form large angular crystals in the area below and in the deeper layers sclerosis, and obliteration of the tubules with the source of minerals being the pulpal blood supply (Sognnaes and Shaw 1952). Four layers were identified (Sarnat and Massler 1965) - a leathery surface layer, a pigmented layer with an upper and deep zone, a transitional zone and a sclerotic zone; the characteristics of each layer is illustrated in Table 5. They also attributed the brown pigmentation of the arrested carious lesion to the degenerated bacteria and their products.
3.5 MICROBIOLOGICAL FEATURES

Numerous studies have implicated streptococcus and lactobacillus species as playing a strong role in the etiology of enamel caries. However, only few microbiological data are available regarding root surface caries in man.

Jordan and Hammond (1972) examined carious lesions in the root surface of periodontally involved extracted human teeth, the subjects being residents of a state institution for the mentally retarded and they sampled these teeth for the presence of filamentous bacteria. Microscopic examination revealed the filamentous nature of the plaque. Samples of dentine from deeper layers of the lesions were also examined. Strains of A.Viscous, A.Naeslundii, A.Odontolyticus and R.Dentocariosa as well as other unidentified Actinomyces-like organisms were isolated. S.Mutans strains were also identified. Gnotobiotic rats later developed periodontal infections and root surface caries when infected with A.Viscous and A.Naeslundii isolates obtained from these teeth.

Later, Sumnerry and Jordan (1974) also isolated bacterial forms from root surface carious lesions. Microscopic examination of the plaque layer covering the cementum revealed a dense layer of gram-positive filamentous bacterial forms. The deeper layers of the dentine revealed S.Mutans as the predominant type though S.Sanguis strains, S.Mitis strains and other streptococci with characteristics of enterococci were identified. In addition, the presence of A.Viscous, A.Odontolyticus and A.Naeslundii was also confirmed. Of interest was the fact that aerobic gram-positive diptheroids with characteristics of the genus Arthrobacter were isolated from the
advancing front of the lesion using a roentgenological grid technique (Sumney and Jordan 1973). This technique when used to sample deep dentinal lesions on the coronal surface showed organisms with characteristics similar to lactobacilli. In both the above studies, surface plaque was not cultured.

Syed et al. (1985) sampled plaque flora associated with human root surface caries lesions in situ in periodontally treated patients. A.Viscous was the dominant bacterial species found with lesser numbers of A.Naeslundii. S.Mutans was also found in high numbers in certain plaque samples and the inverse relationship between S.Mutans and S.Sanguis noted in coronal caries (Doesche et al. 1973) was also observed in these samples. Veillonella strains were also isolated from both plaque groups.

Emilson et al. (1988) examined the presence of selected microorganisms associated with root surface caries in periodontally treated patients. Both saliva and dental plaque were examined. They reported a low prevalence of root surface caries and a low level of salivary mutans streptococci and lactobacilli. There was also no significant difference in proportions of the S.Mutans in plaque from carious root surfaces and carious free surfaces. A significant inverse relationship between noncarious and carious root surfaces was noted for S.Sanguis. No significant difference was observed for population of A.Viscous and A.Naeslundii, however elevated numbers were observed in subjects with five or more new root surface lesions.
Ravald and Hamp (1981) in a study on periodontally treated patients, reported that patients whose salivary lactobacilli count remained high after periodontal and restorative treatment subsequently developed more root surface lesions.

Studies by Brown et al. (1986) and Hill et al. (1977) on root lesion plaque samples find streptococci to be the predominant microorganisms while a large proportion of the remaining microflora are actinomycetes.

Ellen et al. (1985) found a high correlation between S. Mutans and lactobacilli in that both these organisms were isolated more frequently from caries-free surfaces in caries-active subjects than caries-inactive subjects. They also reported that the caries risk was five times higher for surfaces with both mutans streptococci and lactobacilli compared with surfaces free of these bacteria. The risk was more than three times higher for surfaces with only S. Mutans than those with lactobacilli.

From all the above studies, although a variety of microorganisms have been identified, no specific micro-organism has been shown to be responsible for the development of root surface caries.
4 FACTORS RELATING TO ROOT SURFACE CARIES

4.1 SALIVA
Saliva is a complex fluid produced by the salivary glands which discharge the secretion into the oral cavity via the salivary ducts (Dale 1985). The total volume of saliva secreted daily is about 750 ml of which about 90% is produced by the submandibular and parotid glands (Hand 1980).

4.1.1 Composition
Saliva is composed of 99% water and the remaining 1% comprises inorganic ions like Na⁺, K⁺, Cl⁻, HCO₃⁻, with smaller quantities of Ca²⁺, Mg²⁺, HPO₄⁻², I⁻, SCN⁻, and F⁻ as well as secretory glycoproteins, digestive enzymes and immunoglobulins (Hand 1980). The most common cellular elements are desquamated epithelial cells, lymphocytes, and polymorphonuclear leucocytes. In addition, salivary smears yield clumps of mucin to which bacteria are attached. The pH of whole saliva varies from 6.7 to about 7.4 (Avery 1987).

4.1.2 Functions
Mandel (1984) has described a variety of functions of saliva. These are: the lubrication and protection of the mucous membrane, mechanical cleansing to remove food, cellular and bacterial remnants, as a buffer system to regulate the pH level of the oral cavity, maintaining the integrity of the tooth as well as a role in antibacterial activity.
Not much data is available on the role of saliva in root surface caries. Mandel (1984) is of the opinion that there is no diminution of the salivary gland function in healthy, aging individuals. However, salivary flow is decreased with alterations in composition in the elderly on medications or therapeutic interventions, undergoing head and neck irradiation or cancer chemotherapy. This in turn reduces the protective mechanisms of saliva and leads to the formation of caries and other oral conditions.

Studies by Baum (1981) and Makila (1977) have further supported this by noting that aging individuals showed an increased degree of salivary diminution than aging healthy individuals.

However, Waterhouse et al. (1973) and Scott (1977) describe a loss of 25-40% of acinar tissue in the submandibular gland over the life span, suggesting that aging per se was also a factor in diminished salivary gland functions.
4.2 DIET

Production of acid from dietary carbohydrates by plaque bacteria has been implicated as one of the factors responsible for root caries (Jordan and Sumney 1973).

Early animal studies showed a strong relationship between dietary carbohydrates and root surface caries. Keyes (1946) noted that periodontal disturbances and carious lesions of root surfaces were observed in 26 old hamsters which were put on a high carbohydrate diet. However, in his discussion he mentioned that root caries could occur in humans with periodontal diseases and physiological root exposure but that it was an uncommon finding.

King (1950) reported that golden hamsters developed root caries whether fed on a predominantly cereal diet or a high (65%) sucrose diet. Caries if present were due to bacteria. He also noted that none of the animals developed primary coronal caries.

Gibbons and Banghart (1968) observed that alveolar bone loss and root surface caries could be induced in gnotobiotic rats with lesion producing streptococci and a high sucrose diet.

Much more recently, De Palma et al. (1983) by experiments on the rice rat have demonstrated a direct relationship between the amount of sugar consumed and root surface caries.

Root caries in the human population also appears to be enhanced by dietary sugars. The Vipeholm Dental Caries Study (Gustafsson et al. 1954) showed that an increased daily intake of sugar especially
between meals, resulted in an increased frequency in both cemental and enamel caries.

Hix and O'Leary (1976) reported that in patients with periodontal disease, both treated and untreated, those that had the most root caries had a significantly higher number of carbohydrate exposures per week.

A higher prevalence of root caries was also observed in drug addicts by Hecht and Friedman (1949). They attributed this increase of root surface caries to the addict’s craving for sweets resulting in an increased intake of sugar-containing food.

Banting and Ellen (1976) were of the opinion that once gingival recession occurs, there may be accumulation of plaque on the root surfaces. A high sugar diet will increase the risk of the cementum to acid attack by organisms in the plaque resulting in root caries.

In short, carbohydrates in the diet plays an important role in the etiology of root caries.
4.3 PLAQUE AND MICROFLORA

Literature reviews have strongly implicated that the only difference between plaque on the enamel and root surface plaque was in the microflora inhabiting it. As mentioned in a previous section, streptococcus and lactobacillus species seem to play an important role in enamel caries whereas in the case of root plaque, although a variety of microrganisms have been identified, no specific microorganism has been shown to be responsible for the development of root surface caries.

4.4 OTHER FACTORS

Banting and Ellen (1976) have also described the morphology of the tooth root and oral hygiene as factors relating to root caries.

The surface of the root being not as smooth as enamel might predispose the cementum to greater plaque accumulation and bacterial retention and hence root caries.

With advancing age, manual dexterity and interest diminishes, resulting in less efficient cleaning, which in turn causes retention of the plaque and food debris which subsequently leads to root caries formation.
5.1 EFFECT ON PLAQUE

Fluorides in dental plaque have been shown to inhibit dental caries in a number of ways.

5.1.1 Chemistry of Plaque Fluorides

Fluoride concentration in plaque has been reported to range from 5-10 ppm with higher concentration in high fluoride areas (Harper and Loesche 1986) and existing either as free ions (2-5% of total plaque fluoride) or as tightly or loosely bound ions of which the loosely bound or ionizable fluorides are of metabolic significance (Jenkins and Edgar 1977) as they can be released under acidic conditions (which often prevail after eating or drinking), thereby increasing the level of free fluoride ions in plaque (Ferguson and Jenkins 1965) which would result inhibition of further acid production which would contribute in retarding the carious process (Harper and Loesche 1986). Although only a small proportion of plaque fluoride is present normally in ionic form, its proximity to the caries prone site would compensate for this. As a reservoir, plaque usually contains higher quantities of fluoride, calcium and phosphates than saliva and for this reason, plaque may play an important role in the remineralization of the early carious lesion (Duckworth et al. 1987). Some researchers are of the view that the bound fraction of fluoride in plaque is associated with or held in the bacteria (Jenkins 1969; Edgar and Jenkins 1972) while others (Birkeland and Rolla 1972; Birkeland 1973) suggested that the greater proportion of fluoride is attached to extra cellular mineral components.
Figure 17: Diagrammatic representation of the pathways involved in glucose metabolism in intact cells of S. salivarius. Numbers indicate possible sites of fluoride inhibition.

Source: Hamilton 1977. p263

Note: Pathways: ----- glycogen synthesis
----- glycogen degeneration
----- exogenous glucose metabolism
In fluoridated areas, fluoride in the plaque is probably derived from the water. It has been shown that the concentration of fluorides in plaque in fluoridated areas is about twice as high as that in non fluoridated areas (Dawes et al. 1965) and this is probably due to the frequent contact between the water and plaque (Jenkins and Edgar 1977). In non fluoridated areas, the fluoride in plaque is derived from saliva and gingival fluid (Murray and Rugg-Gunn 1986).

5.1.2 Plaque Fluoride and Acid Production

It is a well accepted fact that the degradation of carbohydrate by plaque bacteria into acid which in turn causes demineralization of tooth substance is one of the most important etiological factors in the carious process. There is sufficient evidence that fluorides derived from topical fluorides (Geddes and McNee 1982) or water fluoridation (Jenkins et al. 1969; Agus et al. 1980) may decrease the acidogenic potential of plaque though the exact mechanism of action was not known.

However, Hamilton (1977) suggested that fluoride could inhibit acid production by inhibiting any or all of the following stages of carbohydrate metabolism (glycolysis) by the bacteria, i.e., the enzyme enolase in bacteria and consequently the transport of glucose into cells; the translocation of sugar in cell membranes; cation transport and accumulation in cells and cellular phosphates which dephosphorylate sugar phosphates resulting from transport. [Figure 17] With decrease in pH (increase in hydrogen ion concentration) there is increased permeability of the bacterial cell wall to fluorides which results in a decrease in carbohydrate utilization.
5.1.3 Antibacterial Actions of Plaque Fluorides

Fluorides have been found to affect microorganisms in several ways. Jenkins (1978) reported that at low concentrations it reduces acid formation, at higher concentrations it produces a larger reduction in acids and at very high concentrations, it completely inhibits bacterial growth.

Inhibition in growth of S. Mutans was observed by Loesche et al. (1973) using topical fluorides. Edgar (1981) suggested that high fluoride levels might disinfect the tooth and prevent recolonization of the organism on the tooth.

Gibbons and Van Houte (1973) also suggested that fluorides prevent the synthesis of extracellular glucans and fructans by S. Mutans and S. Sanguis during sucrose degradation which is of significance in the colonization of these bacteria to the tooth surface. However, other researchers (Sharma et al. 1974; Carlsson 1970) have reported otherwise.

Certain strains of oral streptococci have also been reported to undergo lysis in the presence of fluorides (Marquis et al. 1976). Based on all the above, although plaque cannot be considered totally non-pathogenic, the fluoride concentration in it might contribute significantly in retarding the carious process by inhibiting acid production as well as by inhibiting bacterial growth, causing lysis of bacteria and perhaps preventing the colonization of bacteria to the tooth surface.
5.2 EFFECT ON SALIVA

Fluoride is also present as two forms in the saliva - as free F ions and bound to bacteria, epithelial cells or inorganic constituents at neutral PH but in forms which are readily ionizable in an acidic medium (Jenkins and Edgar 1977). The basal fluoride concentrations found in saliva is reported to range from 0.5-2.6 mg (Yao and Gron 1970; Bruun et al. 1982) and is believed to increase with systemic ingestion or after various applications of topical fluorides (Bruun et al. 1982). Whole saliva samples were found to contain higher fluoride levels than simultaneously collected submandibular and parotid samples (Edgar and Jenkins 1977). In the case of ingestion of fluoridated drinking water, the source of salivary fluoride is likely to be the hard tissues with transfer of fluoride occurring via the blood (Ekstrand et al. 1986). In the case of topical fluorides, the fluoride reservoir of the saliva may be by sources in the mouth like the plaque (Duckworth et al. 1987).

Various studies (Svanberg and Westergen 1983; Svanberg and Rolla 1982; Bruun et al. 1982) have shown a significant reduction in the number of viable microrganisms in the presence of fluorides in whole saliva. Although no literature was available on the mode of action of fluorides on salivary microrganisms, one could assume that it might be similar to that in dental plaque.

Featherstone et al. (1985) have also suggested that the continuous presence and redistribution of F by saliva may be important in caries prevention as it would be available during each acid attack of the teeth from acids produced by fermenting carbohydrates. Also, the release of free fluoride, calcium and phosphate ions at an
acidic medium would make saliva an important reservoir of these ions by replenishing fluoride which is lost from plaque to the tooth structure.

Hence, salivary fluorides like plaque fluorides may be a contributing factor towards the prevention of dental caries.
5.3 EFFECT ON CEMENTUM-DENTINE COMPLEX

Root surfaces appear to react with topical fluorides in basically the same way as enamel does, with some fluorides being more effective than others (Shannon et al. 1976). However, fluoride uptake of cementum and dentine has been shown to be greater than enamel (Ericsson 1977). Saxegaard et al. (1987) attribute this to the fact that larger surface areas are available on these tissues and thus more Ca\textsuperscript{++} may be available for reaction with F. Interestingly, F concentration was found to be higher in acellular than cellular cementum (Nakagaki et al. 1987) with the concentrations being higher in the outer surfaces (Hals and Selvig 1977).

In the case of dentine, much of the fluorides penetrates along the dentinal tubules, thus resulting in the peritubular zone being more resistant to subsequent acid attacks than intertubular dentine (Selvig 1968).

Saxegaard et al. (1987), after application of 2% NaF on dentine and cementum reported that the major component on the surface of the dentine and cementum was CaF (alkali soluble) although they did not rule out the possibility of hydroxyfluoroapatite formation (alkali insoluble). Recent data (Ogaard et al. 1983, 1984) has indicated that CaF is more stable in the oral environment than previously believed (Brudevold et al. 1967). The cariostatic effect of CaF seems to be that it acts as a significant reservoir of F ions which could be directly deposited in areas of demineralization and thus reducing the loss of mineral by promoting reprecipitation of stable apatite structures (Ogaard et al. 1984). Ellingsen et al. (1987)
were also of the similar opinion that exposure of root surfaces to topical fluorides would have a caries inhibiting effect due to formation of calcium fluoride. Furthermore, Ten Cate and Duijsters (1983) have also suggested that the calcium fluoride layer may also prevent the dissolution of the underlying dental tissues due to the acid resistance of calcium fluoride. Ogaard et al. (1984) are hence of the opinion that the main value of topical fluoride application may thus be to enhance the remineralization of the early carious lesions rather than to increase the content of stable fluoroapatite of the tooth tissue.

Cementum readily takes up fluoride from fluoridated water (Nakata et al. 1972; Stepnick et al. 1975) as does dentine (Yoon et al. 1960). Hoppenbrouwers et al. (1987), after studying the demineralization of human dental roots in the presence of fluoride summarized that the reduction of the rate of demineralization by fluorides was due to the specific adsorption of fluorides to the crystal and it is derived from the interstitial fluid of the root hard tissue. Fejerskov et al. (1981) concluded that the major cariostatic effect of water fluoridation was probably due to the regular increases in fluoride ion activity in the oral fluids where the fluoride ion might be exchanged with an OH group in the hydroxyapatite crystal to form fluoroapatite, a more stable structure, i.e.,

$$\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2 + 2F^- \rightarrow \text{Ca}_{10}(\text{PO}_4)_6F_2 + 2OH^-$$

($\text{hydroxyapatite}$) $\rightarrow$ ($\text{fluoroapatite}$)

Apatite crystals are also surrounded by other ions like magnesium, carbonate and fluoride. Unlike fluoride, the magnesium and
carbonate exhibit poor crystallinity. Fluoride also has the ability to replace the magnesium and carbonate in the apatite crystal thus enhancing the stability of the crystal structure (Murray and Rugg-Gunn 1982).

Fluorides may thus act on the cementum-dentine complex by the formation of calcium fluoride which acts as a reservoir of fluoride. The fluoride may also react with the hydroxyapatite crystal of the tooth structure to form the more stable fluorapatite crystals.
6 EFFECT OF FLUORIDES ON ROOT SURFACE CARIES

Previously, studies to determine the effects of fluorides on dental caries were mostly on enamel caries. However, in recent times, with the growing importance of the problem of root surface dental caries in the adult population, a few fluoride studies have been done on them and these studies have shown a positive relationship on the effect of fluorides on root surface dental caries.

6.1 SYSTEMIC FLUORIDES

Various studies have shown that long term consumption of fluoridated water has an inhibitory effect on root surface dental caries. However, further research is still necessary to clarify whether the mode of action is systemic and/or topical (Burt et al. 1986; Brustman 1986).

Murray (1971) conducted a study on adult dental health in fluoride (1.5-2.0 ppm F) and low-fluoride areas (0.2 ppm F). Although this study was not specifically on root surface dental caries, he reported that the DMFT of the life-long residents of the fluoride area of all age groups including the 60-65 years age group (DMFT 15.4) was less than that of those in the low-fluoride area (DMFT 17.8), thus demonstrating that continuous residence in a high fluoride area has lifelong beneficial effects.

Banting and Stamm (1978) studied the relationship between fluoridated water and cemental caries. They compared their findings with those of other researchers (Hazen et al. 1972; Sumney et al. 1973; Hix and O'Leary 1976) and concluded that lifelong consumption of fluoridated water would have an inhibitory effect on the
Table 6: Mean numbers of root caries lesions by age for all persons, and for those with root caries, Lordsburg and Deming, NM.

* Source: Burt et al. 1986. p1155

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<thead>
<tr>
<th>Age</th>
<th>Lordsburg</th>
<th></th>
<th>Deming</th>
<th></th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Root lesions</td>
<td>n</td>
<td>Root lesions</td>
<td></td>
</tr>
<tr>
<td></td>
<td>315</td>
<td>0.04</td>
<td>87</td>
<td>0.15</td>
<td>0.05</td>
</tr>
<tr>
<td>27-40</td>
<td>81</td>
<td>0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>41-50</td>
<td>37</td>
<td>0.17</td>
<td>46</td>
<td>1.35</td>
<td>0.004</td>
</tr>
<tr>
<td>51-65</td>
<td>46</td>
<td>0.08</td>
<td>18</td>
<td>1.61</td>
<td>0.005</td>
</tr>
<tr>
<td>Total</td>
<td>164</td>
<td></td>
<td>151</td>
<td>0.69</td>
<td>0.0000</td>
</tr>
<tr>
<td>All</td>
<td>persons</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(n = 315)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Persons with root caries (n = 48)

<table>
<thead>
<tr>
<th>Age</th>
<th>Lordsburg</th>
<th></th>
<th>Deming</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Root lesions</td>
<td>n</td>
<td>Root lesions</td>
<td></td>
</tr>
<tr>
<td>27-40</td>
<td>3</td>
<td>1.00</td>
<td>10</td>
<td>1.30</td>
<td>(n too small)</td>
</tr>
<tr>
<td>41-50</td>
<td>2</td>
<td>1.00</td>
<td>16</td>
<td>3.88</td>
<td>(n too small)</td>
</tr>
<tr>
<td>51-65</td>
<td>7</td>
<td>1.14</td>
<td>10</td>
<td>2.90</td>
<td>0.14</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>1.08</td>
<td>36</td>
<td>2.89</td>
<td>0.04</td>
</tr>
</tbody>
</table>

* Two-sample t-test.
occurrence of root surface caries. Subsequently, Stamm and Banting (1980) did a study to compare the prevalence of root caries in adults with life-long residence in fluoridated (1.6 ppm) and non-fluoridated areas. They reported that adults in the naturally fluoridated area had a mean of 0.4 root surface caries decayed and 0.16 filled against 0.99 and 0.37 respectively in the adults of the non-fluoridated area suggesting that life-long consumption of fluoridated water may significantly reduce the prevalence of root surface caries.

Studies by Banting and Stamm (1979, 1982) have been successful in demonstrating that the uptake of fluorides has a systemic benefit on teeth even after their development and eruption and this benefit increases with length of exposure and age although the former seemed to be the more influential variable of the two. Their in vitro studies on the unexposed middle third of the unexposed root surface indicated that fluoride from the surrounding tissue is incorporated into the cementum which is being deposited throughout life. This fluoride strengthens the cementum making it more resistant to carious attacks. Fluoride concentrations of cementum in the fluoridated areas (1.6 ppm) was significantly higher than in the non-fluoride area.

Burt et al. (1986) did a comparison between root surface caries in an optimally fluoridated area (0.7 mg F/L) and a highly fluoridated area (3.5 mg F/L). The mean number of lesions per person examined with root surface caries was 1.08 in the highly fluoridated community (Lordsburg) and 2.89 in the optimally fluoridated community (Deming). [Table 6] Their findings also indicated that
Table 7: Mean number of teeth with recession\(^1\) and root caries index values\(^2\) Lordsburg and Deming, NM.

Source: Burt et al. 1986. p1156

<table>
<thead>
<tr>
<th></th>
<th>Lordsburg</th>
<th>Deming</th>
<th>(p^3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. Persons Examined</td>
<td>164</td>
<td>151</td>
<td></td>
</tr>
<tr>
<td>Mean No. Teeth with</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recession</td>
<td>8.14</td>
<td>6.04</td>
<td>0.01</td>
</tr>
<tr>
<td>Root Caries Index</td>
<td>1.22%</td>
<td>6.68%</td>
<td>0.0002</td>
</tr>
</tbody>
</table>

\(^1\)At least 1 mm between the gingival crest and the cemento-enamel junction.

\(^2\)Katz (1980)

\(^3\)Two-sample \(t\)-test.
Table 8: Distribution of root surfaces by fluoride exposure.

*Source*: Brustman 1986. p206

<table>
<thead>
<tr>
<th>Root surfaces</th>
<th>Number</th>
<th>Full (Percent)</th>
<th>Fluoride exposure</th>
<th>None (Percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sound</td>
<td>665</td>
<td>(26.9)</td>
<td>869</td>
<td>(22.4)</td>
</tr>
<tr>
<td>Carious and/or</td>
<td>12</td>
<td>(0.5)</td>
<td>68</td>
<td>(1.7)</td>
</tr>
<tr>
<td>Filled</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>780</td>
<td>(31.6)</td>
<td>1760</td>
<td>(45.3)</td>
</tr>
<tr>
<td>Unexposed</td>
<td>1015</td>
<td>(41.1)</td>
<td>1191</td>
<td>(30.6)</td>
</tr>
<tr>
<td></td>
<td>2472</td>
<td>(100)</td>
<td>3888</td>
<td>(100)</td>
</tr>
</tbody>
</table>
although there was more gingival recession in Lordsburg, the RCI was five times greater in Deming [Table 7] suggesting that the action of fluoride might be greater in preventing otherwise susceptible patients from developing root caries rather than in reducing the number of root caries already present. They also concluded that a high concentration of fluoride in the water is associated with a lower prevalence of root caries. Brustman (1986) also conducted a study between residents in fluoride adequate communities (minimum length of exposure - 8 years; fluoride concentration 1.0-1.2 ppm) and fluoride deficient communities. She reported that the non-fluoridated communities had the higher percentages of carious and filled root surfaces [Table 8]. Based on this, Brustman discussed the possibility that the appositional uptake of fluoride by the root cementum might reach significant levels for root caries prevention within as little as eight years or that there may be some effect due to the uptake by the already exposed surfaces.
<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Teeth</th>
<th>No. of Carious Teeth</th>
<th>Root Caries No. of Surfaces</th>
<th>Coronal Caries No. of Surfaces</th>
<th>No. of Surfaces</th>
<th>% of Surfaces</th>
<th>No. of Surfaces</th>
<th>% of Surfaces</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radiation Therapy</td>
<td>1002</td>
<td>448</td>
<td>490</td>
<td>277</td>
<td>354</td>
<td>72*</td>
<td>144</td>
<td>52</td>
</tr>
<tr>
<td>Xerostomia</td>
<td>182</td>
<td>87</td>
<td>139</td>
<td>18</td>
<td>125</td>
<td>90</td>
<td>13</td>
<td>72</td>
</tr>
<tr>
<td>Sjögren's Syndrome</td>
<td>210</td>
<td>116</td>
<td>166</td>
<td>50</td>
<td>144</td>
<td>87</td>
<td>26</td>
<td>52</td>
</tr>
<tr>
<td>High Caries Susceptibility</td>
<td>308</td>
<td>114</td>
<td>149</td>
<td>23</td>
<td>102</td>
<td>68</td>
<td>5</td>
<td>22</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td><strong>1702</strong></td>
<td><strong>765</strong></td>
<td><strong>944</strong></td>
<td><strong>368</strong></td>
<td><strong>725</strong></td>
<td><strong>77</strong></td>
<td><strong>188</strong></td>
<td><strong>51</strong></td>
</tr>
</tbody>
</table>

* In 23 patients of the Radiation Therapy group, 100% of the 243 carious surfaces were remineralized.
### Table 10: Private practice study: remineralization of active carious lesions.

**Source:** Johansen et al. 1987, p.48

<table>
<thead>
<tr>
<th>Group*</th>
<th>No.</th>
<th>Age</th>
<th>DMFT</th>
<th>No of teeth present</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>12</td>
<td>50</td>
<td>25</td>
<td>22</td>
</tr>
<tr>
<td>B</td>
<td>18</td>
<td>58</td>
<td>27</td>
<td>21</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pre-treatment No. of active lesions</th>
<th>Post-treatment remineralized lesions year</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>77</td>
<td>100</td>
</tr>
<tr>
<td>129</td>
<td>100</td>
</tr>
<tr>
<td>Totals</td>
<td>206</td>
</tr>
</tbody>
</table>

* A - Patients without diagnosed medical problems.

B - Patients with medical problems.
6.2 TOPICAL FLUORIDES

Various studies have been carried out to determine the remineralization and solubility effects of topical fluorides on root surface carious lesions.

Johansen et al. (1987) did two studies on elderly patients to determine the remineralization of carious lesions using topical fluoride application (self administration of fluoride gel with custom made trays, usually two five-minute applications daily for two weeks followed by single daily applications). In addition to this, other procedures like proper oral hygiene, using of remineralizing mouthwash and salivary stimulation was also done. In both studies, incidences of new carious lesions decreased significantly and a high percentage of active lesions were remineralized and remained arrested. [Tables 9 and 10]

Stabholz et al. (1987) studied the in vitro demineralization and remineralization of exposed and unexposed root surfaces pretreated with 2% sodium fluoride (NaF). They reported that fluoride treatment significantly decreased the solubility of the root surfaces and suggested that application of the NaF may have resulted in the deposition of calcium fluoride and fluoridated hydroxyapatite which strengthens the root surface and decreased its solubility.

The effect of monofluorophosphate (MFP) dentrifice (50% slurries of MFP) on root dentine softened by artificial caries was studied by Sanchez and Mellberg (1988). They reported that there was significant mineral deposition on the surface as well as the deepest part of the lesion suggesting fluoride uptake by the dentine in both
the MPF and placebo dentrifrices although that of the MPF dentrifice was significantly better. They suggested that remineralization of the root surface carious lesion is a natural process and is further accelerated with the use of fluorides.

Brewer et al. (1987) studied the cementum fluoride uptake using various topical fluoride agents. The significant amounts of fluoride acquired by the cementum using these agents suggested that the application of topical fluorides to exposed root surfaces may inhibit the onset of the root caries lesion.

Al-Joburi and Koulourides (1984) studied the effect of three fluoride concentrations (0.7%, 1.23%, and 2.3%) on in vitro root surface lesions. They reported that root surfaces exposed to the oral environment were more resistant than unexposed surfaces. All three fluoride treatments increased the resistance of the root surfaces to acids by the formation of a thick, highly dense mineral layer over the surface of the lesion.

Uptake of fluoride from titanium tetra fluoride (TiF₄), acidulated phosphate fluoride (APF) and sodium fluoride (NaF) on scaled root surfaces was tested by Hals et al. (1981). Both TiF₄ and APF caused greater deposition of fluorides as compared to NaF.

Shannon et al. (1976) studied four major fluoride compounds - 1.23% each of sodium fluoride (NaF), sodium monofluorophosphate (Na₂PO₃F), acidulated phosphate fluoride (APF) and stannous fluoride (SnF₂) for their ability to reduce the solubility of intact human root surface in vitro. They found that all the compounds were effective in reducing the solubility of the root surface. However NaF and
Figure 18: Root surface solubility reduction by various fluorides (each at 1.23% F and native pH)

Source: Shannon et al. 1976. p202

[Graph showing root surface solubility reduction with mean (S.D.) values for NaF, Na₂PO₃F, APF, and SnF₂]
Na$_2$PO$_3$F reduced root surface solubility by approximately 30% while APF and SnF$_2$ showed a solubility reduction of more than 80%.

[Figure 18]
7 DISCUSSION

The predominant form of dental caries afflicting elderly patients are root surface and recurrent carious lesions. Current demographic data indicates that there will be an increasing percentage of individuals above 65 years within the next twenty-five years. With this, root caries will become a clinical problem of great significance and there will be an increased need to prevent or restore them.

A prerequisite of root caries seems to be periodontal disease with associated gingival recession resulting in the exposure of the root surface to the oral environment. Primary prevention can therefore be aimed at preventing periodontal disease with regular oral prophylaxis as well as motivating the patient to remove microbial plaque and maintain good oral hygiene habits. However, with age, lack of manual dexterity, interest as well as the time, effort and perseverance to continually maintain good oral hygiene may well exceed the ability of the average person (Loe 1970).

Microbial plaque has been strongly implicated as one of the etiological factors of root surface caries although it has not been possible to single out any specific organism as the causative factor. Suitable antimicrobial agents can be considered as a means of preventing the development of the lesion and it is surprising that not much effort has been made to study this. Since most studies have also indicated that root caries increased with increase of sugar in the diet, limiting dietary sucrose, both in amount and frequency of ingestion may be successful in inhibiting the
development of the lesion to a certain extent.

Probably the cornerstone to any patients with high risk of root caries would be some mode of fluoride therapy, either systemic or topical. As a public health preventive program for adults, water fluoridation would be ideal in terms of population covered as well as in cost-effectiveness. Studies have gone on to show that life-long or maybe even short term residence in fluoridated areas result in a highly significant reduction of root caries. These studies clearly disprove the theory of antifluoridationists that water fluoridation does not benefit adults. However, with the knowledge that water fluoridation is effective in inhibiting root caries, more research will have to be carried out to determine whether the action is purely systemic or a combination of both systemic and topical. These studies on water fluoridation compare only two levels of fluoride and do not include the optimal concentration of fluoride required to effectively inhibit root caries formation.

Two main categories of suggested mechanisms can be advocated for fluorides - those which affect the environment of the tooth, the saliva and plaque, and those which affect the tooth itself.

Fluoride in plaque may act in several ways - by affecting bacterial growth, glycolysis, glycogen synthesis and acid production. All these effects may occur with topical fluoride treatment. However, with oral hygiene procedures like scaling, toothbrushing and flossing, plaque might be removed and the fluoride thus incorporated into the plaque will be removed as well. In addition, as the amount of fluoride deposited on the tooth is limited, fluoride supplied to
the plaque by this method will be limited too. Hence effect of topical fluorides of plaque are likely to be of a shorter duration and to be effective might require frequent applications.

The continuous presence of effective but low concentrations of fluoride in the saliva as a result of topical or systemic fluorides may have a more lasting effect than plaque fluorides as they act as a reservoir for the fluoride ions and may enhance the remineralization of an early lesion sufficiently to prevent a clinical lesion from developing.

Fluorides act directly on the tooth by enhancing remineralization and decreasing the solubility of the tooth structure as well as by preventing the colonization of bacteria to the tooth surface.

The direct action of fluorides on the tooth structure, as well as the continuous presence of fluoride in the saliva would have the advantage of greater permanence than plaque effects.

One of the high risk groups where daily applications of topical fluorides have been used with encouraging results is in the prevention of root caries in patients suffering from radiation infused xerostomia. Two forms of treatment have been advocated for this group - one method is by the self-application of 0.4% SnF₂ gel daily for one minute which provides 1000 ppm F (Wescott et al. 1975; Keene et al. 1984). The other method is by the self-application of 1% NaF gel providing 5000 ppm F using a custom fitted soft plastic tray for 5-10 minutes daily (Daly et al. 1972; Dreizen et al. 1977). However, these daily applications of high concentrations of fluoride are not feasible for the normal population and may bring about the
problem of fluoride intoxication.

Johansen and Olsen (1979) in a three-year study on high risk root caries patients claimed that those patients treated with high concentrations of either 1.23% acidulated phosphate fluoride (12,300 ppm F) or 1% sodium fluoride (10,000 ppm F) in gel form together with daily rinsing using a Ca, P and F mouthrinse could discontinue the fluoride gel treatment after four weeks providing they followed a regime consisting of good oral hygiene, a fluoride toothpaste, daily use of supersaturated remineralizing mouthwash and using a special chewing gum to stimulate salivary secretion.

Although the topical efficiency of topical fluoride therapy for the prevention of root caries in healthy adults may not be as spectacular as for radiation caries (Billings et al. 1985), the available data on in vitro and in vivo studies definitely suggest that topical fluorides would be an effective weapon against root surface caries.

From these studies, it is probable that acidulated phosphate fluoride is the most effective topical fluoride agent on root caries, followed by stannous fluoride and titanium tetrafluoride with sodium fluoride and sodium monofluorophosphate being the least effective. However, since none of the studies have been replicated, it is difficult to determine the effectiveness of the various agents, dose-response relationships as well as the duration and mode of application. These may all be avenues for future research.
8 CONCLUSIONS

The review of the literature has shown without doubt that fluorides, both systemic and topical are effective in preventing and controlling root caries.

The main mechanisms by which systemic and topical fluorides act to inhibit root caries is by its action on the plaque, saliva and the cementum-dentine complex. In the case of plaque, the fluoride acts mainly by affecting bacterial growth, glycolysis, glycogen synthesis and acid production. The plaque fluorides may also act as a reservoir of fluoride ions which may be available for the remineralization of the early carious lesion. It can be assumed that the mode of action of fluorides in saliva may be similar to that of dental plaque. Fluoride acts on the cementum-dentine complex by the formation of calcium fluoride which may act as a significant reservoir of fluoride ions as well as by reacting with the hydroxyapatite crystal to form fluorapatite, a more stable crystal structure. However, since most plaque is removed by oral hygiene procedures, the direct action of fluorides on the tooth surface as well as the continuous presence of fluoride in the saliva would have the advantage of greater permanence than plaque effects.

It has been successfully shown that some form of daily self-application of topical fluoride is useful in preventing the onset of root caries lesions and this is based mainly on observations in reduction of root caries in high risk cases of xerostomia as well as in vitro and in vivo studies on fluorides and root caries. However, more studies will have to be carried out to determine the
effectiveness of the various agents, dose-response relationships as well as duration, mode of application and cost effectiveness.

In the case of systemic fluorides, studies have shown that life-long residence in fluoridated areas have significantly reduced the prevalence of root caries. More research will be necessary to determine the optimal concentration of fluoride required to inhibit root caries formation.

Although research in various avenues of fluoride and root caries is still needed, it is reasonable however to recommend topical fluorides for any adults who are caries prone and particularly for those with known risk factors such as xerostomia.

Probably, some form of acidulated phosphate fluoride, stannous fluoride or titanium tetrafluoride will be effective in preventing or inhibiting root caries. Monofluorophosphate dentrifices have also been found to cause significant mineral deposition and can be recommended for adults. Adults would also benefit from systemic fluorides from the systemic effects during tooth formation eventually as many areas have been fluoridated since the 1940’s. There could also be some reinforcing beneficial effect from the low concentration topical effect of the fluoridated water.
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