prevention

Fluorides in Dentistry

Fluoride appears to provide its benefit when present in the oral cavity. Its effectiveness depends on how frequently it is administered in the mouth. Fluoride can be used to increase the resistance to dental caries while minimizing the risk of fluorosis via maximizing topical exposure of fluoride throughout life and minimizing systemic absorption during the developing period of dentition. It is commonly expressed in parts per million (ppm) which is equivalent to 1mg fluoride per kilogram or liter of water.

Fluoride in Environment:

In soil: Fluoride concentration of soil increases with depth. In high mountain areas the fluoride content of the soil is usually higher. In rock and soil, fluoride may occur in a wide variety of minerals, including fluorspar contains calcium fluoride, cryolite contains aluminum fluoride.

In atmosphere: fluoride originating from dust of fluoride-containing soils from gaseous industrial waste, the burning of coal fires in populated areas and from gases emitted in areas of volcanic activity in nature. The principal source of pollution are industries and mining of phosphate and fluorspar, where fluoride rich dust travel long distances by wind and enter food chain by depositing on plants. Pesticides containing fluoride can have a similar effect.

In waters:. All water contains fluorides in varying concentrations .As many of the minerals in the soil are soluble in water, fluoride is found in varying concentrations in the groundwater, Sea water contains significant quantities of fluoride at levels 0.8–1.4 mg/lt. In water from lakes, rivers, and artesian wells the fluoride content is usually below 0.5 mg /L .

Fluoride Metabolism

Fluoride intake: The major sources of fluoride are

1. Food► Most foods have fluoride as fish.
2. Liquid► drinking water and beverages, tea contains up to 7 ppm.
3. Fluoride-containing dental products.► tooth paste, fluoride gel.

Fluoride metabolism

1-Absorption of fluoride:

Approximately 75 -90 % of the fluoride ingested each day is absorbed from the alimentary tract. Fluoride may also be inhaled from air borne fluoride. Readily soluble fluoride compounds such as NaF tablets or aqueous solution of NaF are completely absorbed whereas compound with solubility such as CaF2, MgF and A1F3 , are less completely absorbed. So the presence of Ca may lead to formation of insoluble salts with fluoride and absorption reduced to 70% and in food rich with Ca to 60%. The ingestion of fluoride with food retards its absorption. Absorption from stomach occurs readily and is inversely related to the pH of the gastric content. The absorption process occurs by passive diffusion. The absorption of fluoride is unusual in that it can occur from the stomach to a considerable extent. The rate of gastric absorption is directly related to the acidity of the contents so that, for any given dose, the peak plasma level is higher and occurs sooner when the contents are more acidic. Most of the fluoride that escapes absorption from the stomach will be absorbed from the proximal small intestine.

2- **Distribution of Fluoride in the Body**

Fluoride is distributed as follow

1. **in Plasma:** Plasma is the biological fluid into which and from which fluoride must pass for its distribution elsewhere in the body and for its elimination from the body.

There are two general forms of fluoride in human plasma. The ionic form (also called as inorganic fluoride or free fluoride) and the non-ionic or bound fluoride.

Ionic form is of significance in dentistry and public health and is detected by ion-specific electrode. Together the ionic and non- ionic fraction is called "total" plasma fluoride. Ionic fluoride is not bound to proteins, to other components of plasma or to soft tissue. The concentration of ionic fluoride in soft and hard tissue is directly related to the amount of ionic fluoride intake. Since plasma fluoride levels are not homeostatically regulated, there is no normal physiologic concentration. Plasma fluoride levels increase with age. Fluoride balance in infants can be positive or negative during the early months of life, depending on whether intake is sufficient to maintain the plasma concentration that existed at the time of birth.

2. **in Soft Tissues**

The intracellular fluoride concentrations are from 10–50 % lower than those of plasma, but they change simultaneously and in proportion to those of plasma . The tissue-to-plasma ratios of radioactive fluoride are consistent with the hypothesis that hydrogen fluoride (HF) is the form in which fluoride migrates and establishes diffusion equilibrium across cell membranes. Since the pH gradient across the membranes of most cells can be decreased or increased by altering extracellular pH, it is possible to promote the net flux of fluoride into or out of cells. This is the basis for the suggestion that alkalization of the body fluids is a useful adjunct in the treatment of acute fluoride toxicity.

3. **in Calcified Tissues**

Approximately 99 percent of the body burden of fluoride is associated with calcified tissues. The fluoride concentration in bone is not uniform. In long bones, for example, the concentrations are highest in the periosteal region . They decline sharply within a few millimeters of the periosteal surface and increase slightly as the endosteal region is approached. Cancelous bone has higher fluoride concentrations than compact bone. Dentine and bone appear to have similar fluoride concentrations which increase with age, while that of enamel is markedly lower. Surface enamel fluoride concentrations tend to decrease with age in areas subjected to tooth wear but increase in areas that accumulate plaque . Dentine fluoride levels decline progressively from the pulpal surface to the dentino-enamel junction (DEJ). Enamel fluoride concentrations are highest at the surface and decline progressively toward the DEJ Bulk enamel (all the enamel from a tooth) fluoride

concentrations mainly reflect the level of fluoride exposure during tooth formation, while dentine and bone fluoride concentrations are generally proportional to the long-term level of intake.

3- Fluoride Excretion

1. In Urine

Fluoride is excreted primarily via urine. The percentage of the filtered fluoride reabsorbed from the renal tubules can range from about 10 to 90 percent. The degree of reabsorption depends largely on the pH of the tubular fluid, urinary flow and renal function. Urinary fluoride clearance increases with urine pH due to a decrease in the concentration of HF. Among the halogens, the renal clearance of fluoride is unusually high. Numerous factors (e.g. diet and drugs) can affect urine pH and thus affect fluoride clearance and retention. The renal clearance of fluoride in the adult typically ranges from 30 to 50 ml/min. The excretion of fluoride in urine is reduced in individuals with impaired renal function.

2. In Feces

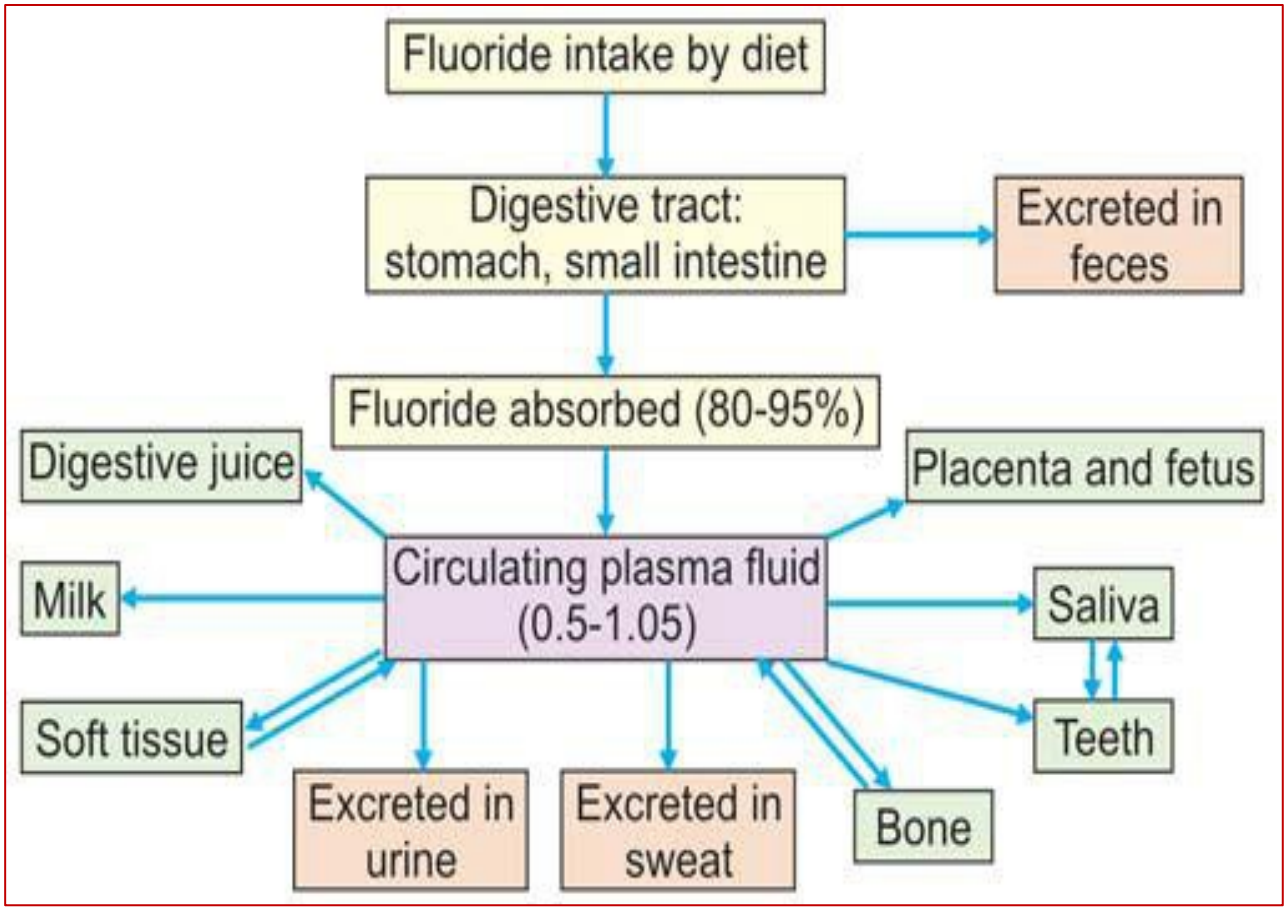
It is generally accepted that most of the fluoride in the feces is not absorbed. Fluoride present in feces results from two sources: the ingested fluoride that is not absorbed and the absorbed fluoride that is re-excreted into the gastrointestinal tract. Fecal fluoride usually accounts for less than 10 percent of the amount ingested each day.

3. In Sweat

Usually, only a few percent of the fluoride intake is excreted in the sweat. However, under excessive sweating as much as 50 percent of the total fluoride excreted may be lost via perspiration.

4. In Saliva

Less than 1 percent of absorbed fluoride is reported to appear in the saliva. The concentration of fluoride in saliva is about two-thirds of the plasma fluoride concentration and seems to be independent of flow rate, in contrast to the situation for most electrolytes. In fact, saliva does not represent true excretion, because most of the fluoride will be recycled in the body. However, the fluoride content of the saliva is of major importance for maintaining a fluoride level in the oral cavity.



Systemic fluoride

low levels of fluoride intake help to prevent dental caries. The dental effects of fluorosis develop much earlier than the skeletal effects in people exposed to large amounts of fluoride.

Ingestion of excess fluoride, most commonly in drinking-water, can cause fluorosis which is a developmental disturbance of dental enamel, caused by excessive exposures to high concentrations of fluoride during tooth development, leading to enamel with lower mineral content and increased porosity.

Dental fluorosis is generalized within the dentition and over the entire tooth surface which makes it easy to distinguish fluoride-induced enamel changes from other enamel defects (non-fluoride origin) which may be symmetrically distributed in the oral cavity. Infants and toddlers are especially at risk for dental fluorosis of the anterior teeth since it is during the first 3 years of life that the permanent front teeth are the most sensitive to the effects of fluoride (The central incisor takes approximately 3 years to go through complete enamel mineralization. Timing of chronic daily fluoride ingestion and the corresponding dental fluorosis pattern that can be expected.). Fluoride accumulates at the transition/ maturation stage of tooth development so that the entire tooth surface can be affected.

1. Child from birth -3year takes excess fluoride from tap water used for infant formula, Incisors, and first molars are most affected teeth.
2. Child from 3–6 years takes excess fluoride from early toothpaste use, premolars, canines and second molars are most affected teeth.

3. Child from 0-6years takes excess fluoride from Fluoride supplements and fluoridated water (drinking water >4 ppm fluoride), all teeth affected.

There is a direct relationship is present between dental fluorosis and level of F ingested and the severity of dental fluorosis depend on:

1. Stage of tooth development.
2. Duration of exposure to fluoride.
3. Concentration of fluoride in foods and drinks.

Dental fluorosis is characterized by staining and pitting of the teeth. It is difficult to differentiate between dental fluorosis and other enamel disturbances. Clinically dental fluorosis is characterized by a white opaque appearance of the enamel, caused by increased subsurface porosity. The earliest sign is a change in color, showing many thin white horizontal lines running across the surfaces of the teeth, with white opacities at the newly erupted incisal end. At higher levels of fluoride exposure, the white lines in the enamel become more and more defined and thicker. Some patchy cloudy areas and thick opaque bands also appear on the involved teeth. With increased dental fluorosis, the entire tooth can be chalky white and lose transparency.

With higher fluoride doses or prolonged exposure, deeper layers of enamel are affected; the enamel becomes less mineralized. Damage to the enamel surface occurs in patients with moderate- to- severe degrees of enamel fluorosis. Teeth can erupt with pits, with additional pitting occurring with post eruptive enamel fracture. In the individuals with moderate dental fluorosis, yellow to light brown staining is observed in the areas of enamel damage. In very severe cases, the enamel is porous, poorly mineralized, stains brown, and contains relatively less mineral and more proteins than sound enamel. Severely fluorosis enamel can easily chip post eruptively during normal mechanical use . Although teeth with mild dental fluorosis may be more resistant to dental decay because of the higher levels of fluoride contained in the enamel surface, severely fluorosis teeth are more susceptible to decay, most likely because of the uneven surface or loss of the outer protective layer.

Dean's Classification of Dental Fluorosis

Normal 0 Enamel (trance lucent, smooth, glossy and creamy white color).

Questionable (0.5) Enamel discolored(slight aberration from the traclucency of normal enamel, ranging from a few white flecks to occasional white spot

Very mild (1) Small, opaque, white area scattered irregularly over the tooth, but not involving as much as approximately 25% of tooth surface(no more than 1-2 mm of white opacity at the tip of cusps of bicuspids or second molar.

Mild (2) The white opaque areas in the enamel of teeth are more extensive, but not involve as much as 50% of tooth.

Moderate(3) All enamel surfaces of teeth are affected and subject to attrition wear, brown stains is a disfiguring feature.

Sever(4) All enamel surfaces of teeth are affected and hypoplasia is so marked that general form of the tooth may be affected, discrete pitting, brown stain wide spread teeth often present a corroded like appearance.

Pathogenesis of dental fluorosis:

It is related to physiological conditions, like body weight, rate of skeletal growth and remodeling, nutrition, and renal function. It is widely known that F- affects the kinetics of bio mineralization, , triggering the incomplete mineralization of enamel crystals and producing porous enamel-which is typical of dental fluorosis. Bone is a reservoir of fluoride, as fluoride is incorporated in the forming apatite crystals, and this ion can also be released from these crystals as bone remodels. Therefore, rapid bone growth, as occurs in the growing child, will remove fluoride from the blood stream, possibly reducing the risk of dental fluorosis by lowering serum fluoride levels. Also Nutrition is also important for controlling the serum level of fluoride, as ions such as calcium, magnesium and aluminum can reduce the bioavailability of fluoride. A deficiency in these ions in food can also affect (enhance) fluoride uptake.

Treatment of Dental Fluorosis:

Mild dental fluorosis is treated with bleaching, to make the color of the tooth surface uniform ,while moderate type Composite restorations combined with micro abrasion or application of aesthetic veneers is the treatment of choice. Sever fluorosis requires prosthetic crowns.

It is important to differentiate visually between incipient caries and developmental white spot hypo calcifications (fluorosis) of enamel. Dental fluorosis is unaffected by drying and wetting. So, a white spot that is an incipient lesion will disappear upon wetting and a hypo calcification will remain whether dry or moist, also they can be differentiated by position, white spot carious lesions usually occur around margins of gingival (the favorable site for plaque deposition)

Dental and skeletal fluorosis:

Skeletal or bone fluorosis affects children as well as adults. It does not easily manifest until the disease attains an advanced stage. Symptoms of bone fluorosis:

1. Early symptom include sporadic pain, back stiffness, burning like sensation, pricking and tingling in the limbs, muscle weakness, chronic fatigue, abnormal calcium deposits in bones and ligaments.
2. The advanced stage is osteoporosis in long bones and bony outgrowths may occur. Vertebrae may fuse together and eventually the victim may be crippled. Generalized dental fluorosis of all the permanent teeth indicates that the bone is potentially a major source of the excess fluoride that causes dental fluorosis in children. People ingesting fluoridated water for many years have higher levels of fluoride in their entire skeletal systems.

Community Water Fluoridation

It's the upward adjustment of the natural fluoride level in a community's water supply to a level optimal for dental health. By consuming the water directly or indirectly through incorporation in foods and beverages, consumers accrue preventive benefits regardless of age or socioeconomic status. Dean's results showed that both a reduction of dental caries and an acceptable level of enamel fluorosis could be attained with water containing fluoride levels at approximately 1 ppm of fluoride. Michigan city in 1945 , was the first city in the world to fluoridate its drinking water as a measure to promote dental health and prevent disease.

Fluoridation is the controlled adjustment of a fluoride compound to a public water supply in order to bring the fluoride concentration up to a level which effectively prevents caries.Water fluoridation requires a level of dental caries in the community that is high or moderate, or a firm indication that the caries level is increasing.

Water fluoridation has advantages of low cost, no motivation or behavioral changes necessary., had pre and post eruptive benefit., and caries reduction 50-60% in permanent teeth, and 40-50% in primary teeth. The disadvantage is the possibility of mild to moderate fluorosis, political and/or emotional objections to water additives, possibility of mild to moderate fluorosis if other sources of fluoride are ingested, and Alleged toxicity.

Fluoride compound used in water fluoridation

1. Fluorspar: It is a mineral containing calcium fluoride [CaF2].
2. Sodium fluoride.
3. Silicofluorides.
4. Sodium silicofluorides: Most commonly used due to its low cost. Solutions of this compound are corrosive hence materials for piping, etc. should be chosen accordingly.
5. Hydrofluosilicic acid.
6. Ammonium silicofluoride [(NH)2SiF6].

Artificial water fluoridation level

World Health Organization (1984)guidelines suggested that the level of artificial water fluoride according to climate as:

1. In areas with a warm climate, the optimal fluoride concentration in drinking water should remain below 1 mg/ liter (1 ppm or part per million).
2. While in cooler climates it could go up to 1.2 mg/liter. (A range of 0.7-1.2 ppm). The differentiation derives from the fact that perspiration is more in hot weather and consequently intake is more.

Then the National Advisory Committee on Oral Health suggested a range 0.6-1.1 mg/L with variation within that range according to the mean maximum daily temperature.

According to World Health Organization's monograph 'fluoride and human health' there is evidence that ingestion of fluoride at recommended levels presents no danger to humans.

Health benefits and risk of fluoridation has been the subject of searching reviews by expert committees throughout the world including the WHO. None has found evidence that drinking water with a concentration of around 1 ppm is harmful to health. In fact other than dental fluorosis only endemic skeletal fluorosis is known to result from long-term ingestion of water containing high levels of fluoride. In recent years opponents of fluoridation have attempted to link fluoridation with a wide range of diseases, e.g. cancer, Alzheimer diseases or that it interferes with the immune function, but there is overwhelming agreement between the scientific, medical and dental community worldwide that fluoridation of water is a safe and effective public health measure.

School Water Fluoridation

It is an alternative method to community water fluoridation in rural schools, where fluoridation of community water is not feasible. Reduction in dental caries was found to be about 40 percent.

It has disadvantages that the children do not receive the benefits until they begin school(belated) , also children consume the fluoridated water only when the school is in session(abbreviated)

In order to compensate for this belated and abbreviated exposure, the school water is usually fluoridated at 4.5 times the optimum concentration recommended for that place

There are two major concerns about school water fluoridation:

1. By age 6 all teeth expect 3rd molars are in an advanced stage of mineralization, thus reducing the pre-eruptive benefits of fluoride.
2. There are no data to indicate expected caries incidence following graduation.

Systemic effect of fluoride:

1. **Pre-eruptive Systemic Effects:** During tooth development, fluoride is incorporated into the developing tooth's mineralized structure. Although this is no longer believed to be the most important reason for the effect of fluoride in dental caries, the presence of fluoride in the dental enamel probably increases resistance to demineralization when the tooth surface is exposed to organic acids.Systemic fluoride may enhance the resistance of the tooth by way of an alteration in tooth morphology, and a conversion of the hydroxyapatite mineral to a fluoridated state with an attendant reduction in

solubility and an enhancement of the remineralization phase of the caries process.

2. **Post-eruptive Systemic Effects:**

After tooth eruption, fluoride is no longer involved systemically in tooth formation. However, consumed fluoride is excreted through the saliva and can aid in tooth protection throughout the lifetime. At the time of tooth eruption the enamel is not completely calcified and undergoes a post-eruptive period of approximately 2 years during which enamel calcification continues. Throughout this period of enamel maturation, there is continuous accumulation of fluoride as well as other elements in the superficial part of enamel.

Fluoride concentration (mg/L)	Effects
<1.0	**Safe limit**
1.0–3.0	**Dental Fluorosis**
3.0–4.0	**Brittle and stiff bones and joints**
4.0–10	**Dental fluorosis, skeletal fluorosis (pain in neck bones and back)**

Fluoride Supplements

Fluoride supplements were originally designed to provide the systemic fluoride that a child would not consume living in a non-fluoridated area. However fluoride supplements can be prescribed for children ages 6 months to 16 years who are at high risk for tooth decay and whose primary drinking water has a low fluoride concentration. Fluoride tablets became the method of choice for fluoride supplementation. Fluoride supplements should only be prescribed by dentists where there is clear evidence for high risk of caries and non- compliance with using other fluoridated products. Supplements contain a measured amount of fluoride typically 0.25mg,0.5mg, and 1mg usually as sodium fluoride and it should only be prescribed by dentists where there is clear evidence for high risk of caries and non-compliance with using other fluoridated products; and the parents must be cooperative. 2.2 mg sodium fluoride tablet gives 1mg ion fluoride, and 1.1 mg sodium fluoride tablet gives 0.5mg ion fluoride.

Instruction to use fluoride supplement (tablet or lozenges or drop):

If fluoride level is unknown, drinking water should be tested for fluoride content before supplements are prescribed. It is indicated to

children living in area with none or low level of fluoride in water. Especially children with high risk to dental caries, children with chronic systemic disease and handicapped children. Fluoride supplement is daily used from 6months to 16 years to give their maximum effect. Fluoride tablets and lozenges are intended to be chewed or sucked for 1–2 minutes before being swallowed To maximize their topical effect. Before considering supplementing fluoride, it is relevant to take into account the natural sources of fluoride in food and drinking water. It has also been shown that when exposure to fluoride is discontinued, its caries-reducing effect gradually wanes. This is entirely logical, because fluoride is affecting the dynamics of lesion formation. Fluoride supplement Should not be given with milk. Fluoride supplement during pregnancy until dental formation is completed through pharmaceutical products, i.e. tablets or drops, according to variable doses (0.25 and 1 mg). During pregnancy and breast feeding, mothers should take 1 mg a day. In fact, theoretically, during intrauterine life, the fluoride taken by the mother may work in the pre- eruptive phase, during the Amelogenesis of deciduous teeth with a consequent beneficial effect on the newborn's deciduous teeth. Fluoride passes through the placenta freely, until it reaches excessively high levels in the mother's blood, and thus triggers this passage (barrier effect) to protect the fetus from excessive doses. The threshold concentration that pushes the placenta to trigger this function is 0.4 ppm of fluoride in maternal blood . Some Authors consider the systemic administration of fluoride as a further supplement during pregnancy, as it is identified as the first step to caries prevention. The children until they are old enough to swallow use fluoride supplement as Fluoride Drops; they are available as 0.125mg, 0.25mg,0.50mg drops . 10 drops equal to 1mg, if 10 drops placed in a liter of water the result concentration of 1ppm of fluoride.

Fluoridated salt

Where water fluoridation could not be initiated, some countries have introduced salt fluoridation. Salt is usually fluoridated at 250 ppm (which is 250 mg F/kg salt, or 0.25 mg/gm. salt). Table salt in the kitchen can contribute 1 to 4 g of the daily salt intake. Thus, a person could potentially ingest 1 mg of fluoride a day at a salt intake of 4 grams a day.

It has the advantages of wide coverage ,need little action by the individual, low cost, freedom for the consumers as both fluoridated and non-fluoridated salt is available, also it is safe with minimum possibilities of fluorosis, but has many disadvantages that it need community education and promotion and international efforts to reduce sodium intake to help control hypertension, also its c consumption is lowered during early life when the need for fluoride is the maximum.

Fluoridated milk

Milk fluoridation is the addition of a measured quantity of fluoride to bottled or packaged milk to be drunk by children .Both bovine and human milk contain low level of fluoride about 0.03ppmF. Milk fluoridation is suggested instead of water fluoridation. Fluoridated milk promotes remineralization of lesions in enamel *in vitro* and *in vivo*, and inhibits demineralization in enamel and dentin. Milk itself has a protective effect in intra-oral caries models as well as *in vitro* . it has the disadvantages of its consumption varies between different socioeconomic groups , also its uptake decrease with age so long term benefit is less than water fluoridation and require high level of technical expertise. A high concentration of fluoride is needed because the children did not drink the beverage throughout the day and calcium in the milk complexes with fluoride, which would reduce its availability for topical benefits.

To be continued

Topical Fluorides

It refers to the use of systems containing relatively large concentrations of fluoride that are applied locally or topically, to the erupted tooth surface to prevent the formation of dental caries. Application of topical fluorides immediately after eruption hastens fluoride uptake and makes enamel more resistant to dental caries. Its effect depends on: The concentration of fluoride used, and the frequency with which it is applied and the duration of application , and the specific fluoride compound used.

Its advantages are

1. Does not cause fluorosis.
2. Cariostatic for people of all ages.
3. Available only to people who desire it.
4. Easy to use.

Its disadvantages are

1. Person must remember to use.
2. High cost compared to water fluoridation.
3. More concentrated professional use products can cause short-term side effects like nausea immediately after use.

Mechanisms of Fluoride Action:

Scientists have proposed that fluoride has several functions

1. Increased enamel resistance or reducing enamel solubility [acid dissolution].
2. Interferes in the formation and functioning of dental plaque microorganisms.
3. Increases the rate of post-eruptive maturation.
4. Remineralization of incipient lesions.
5. Improves tooth morphology.

The presence of elevated concentration of fluoride in enamel surface makes tooth surface more resistant to development of dental caries.

Fluoride ions when substituted into the hydroxyapatite crystals fit more perfectly than do hydroxyl ions. Also the greater bonding potential of fluoride makes the apatite crystals more compact and more stable, thereby more resistant to the acid dissolution. When concentrated topical fluoride agent reacts with enamel there is formation of calcium fluoride: $Ca_{10}[PO_4]_6[OH]_2 + 20F^- \rightarrow 10\ CaF_2 + 6[PO_4]^{3-} + 2[OH]^-$ Hydroxyapatite ↔ 10 Calcium fluoride

Most topical fluoride agents have a fluoride ion concentration of between 10,000–20,000 ppm which leads to the formation of calcium fluoride and eventually**Fluorhydroxyapatite**.

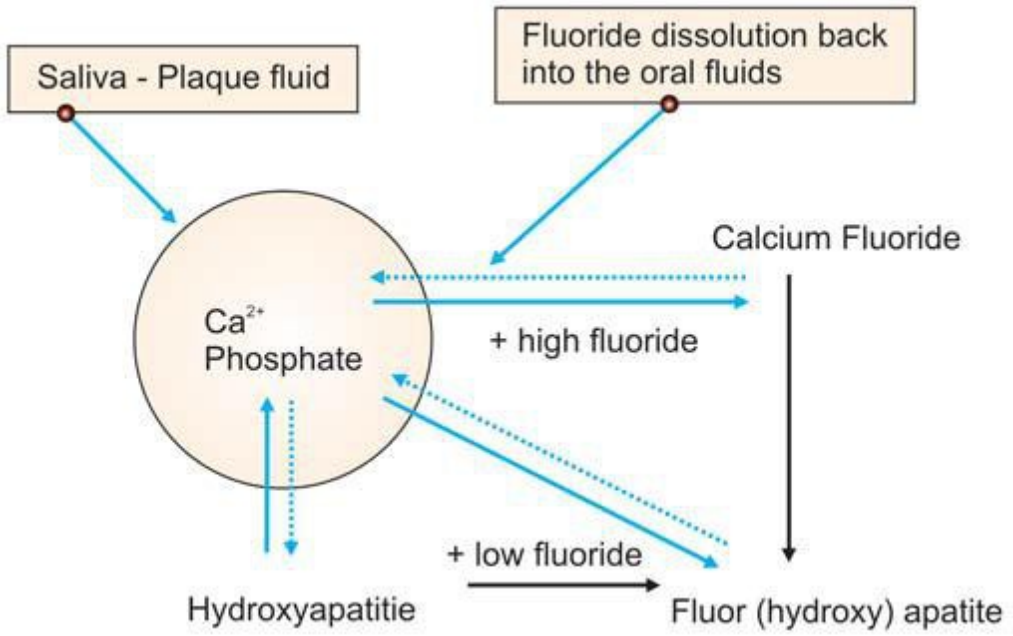


Fig (1) Mechanism of action of fluoride on enamel

Commonly used topical fluoride agents include ***Sodium fluoride, Sodium monofluorophosphate. Stannous fluoride and Amine fluoride***.

Fluoride's effect on tooth mineral:

Fluoride, however, substituting for the hydroxyl group fits extremely well and stabilizes the hydroxyapatite (HA) molecule forming fluoridated apatite fluorapatite (FA). If all of the hydroxyl ions are substituted fluorapatite (FA) forms. The fluoride ion is extremely electronegative and forms very strong hydrogen bonds with hydroxyl and acid phosphate groups in the HA crystal rendering the enamel surface more difficult to protonate. Essentially this makes the enamel more difficult to demineralize, and it also favors the remineralization process. This is the primary chemical mechanism of fluoride's action to protect the tooth against acids produced by plaque metabolism. Fluoridated apatite and/or fluorapatite are generally found in the surface layers of enamel that contains high fluoride concentrations of fluoride. This can arise both during development and from topical exposure.

1-Decreases the solubility of the crystals

The presence of elevated concentration of fluoride in enamel surface makes tooth surface more resistant to development of dental caries. Fluoride ions when substituted into the hydroxyapatite crystals fit more perfectly than do hydroxyl ions. Also the greater bonding potential of fluoride makes the apatite crystals more compact and more stable, thereby more resistant to the acid dissolution.

2- Remineralization

As the saliva flows over the plaque and its components neutralize the acid, raising the pH, demineralization is stopped and reversed. The saliva is

supersaturated with calcium and phosphate, which can drive mineral back into the tooth.
Fluoride enhances remineralization by adsorbing to the crystal surface and attracting calcium ions, followed by phosphate ions, leading to new mineral formation. The partially demineralized crystal surfaces within the lesion act as ―nucleators, and new surfaces grow on the crystals. These processes constitute remineralization—the replacement of mineral in the partially demineralized regions of the carious lesion of enamel or dentine
(including the tooth root). Fluoride enhances remineralization by adsorbing to the crystal surface and attracting calcium ions, followed by phosphate ions, leading to new mineral formation. FAP contains approximately 30,000 ppm F and has a very low solubility in acid.

Increased Enamel Resistant:

The topical effect of fluoride in reducing enamel solubility occurs during the repeated cycles of demineralization and remineralization in the early stages of carious lesion. When tooth is exposed to a pH of about 5.5 or lower enamel dissolution starts. This occurs beneath bacterial plaque. The concentration of calcium, phosphate and other ions increases in the solution. When the pH returns back to normal the dissolved minerals get precipitated. Hence, carious dissolution of enamel is a cyclic phenomenon consisting of phases of demineralization when precipitation occurs. The presence of low levels of fluoride in the plaque leads to a gradual establishment of well-crystallized and more acid resistant apatite in enamel surface. The other mechanisms which have been postulated in rendering decreased enamel solubility are:
• That under the influence of fluoride, larger crystals are formed with fewer imperfection thus stabilizing the lattice and presenting a smaller surface area/unit volume for dissolution.
• Enamel which mineralizes under the influence of fluoride has a lower carbonate content thus giving a reduced solubility.
(FAP = $Ca_{10}(PO_4)_6F_2$) in which the OH^- ion in pure hydroxyapatite is completely replaced by an F^- ion. The resulting mineral FAP is highly
resistant to dissolution by acid.

Inhibition of Bacterial Enzyme System:

Fluoride interferes with oral bacteria in two ways. In high concentrations it acts as bactericidal and in low concentration it decreases acid production. Fluoride inhibits **glycolysis**—the process by which fermentable carbohydrates are metabolized by cariogenic bacteria to
produce acids. Plaque fluoride can inhibit the production of extracellular polysaccharides by cariogenic bacteria—this is necessary for plaque adherence to smooth enamel surfaces. Fluoride can interfere with enolase, an enzyme which is used by bacteria in the fermentation of carbohydrates.

Topical Fluoride admistration could be applied through.
1. Those applied by professional.
a. Topical solutions and gels.

b. Fluoride containing varnishes. c.
Fluoride prophylaxis paste.
d. Restorative materials containing fluoride.
e. Fluoride containing devices (Slow Release).
2 Self- applied fluoride agents. a.
Fluoride dentifrices.
b. Fluoride rinses. c.
Fluoride gels.

Classification of Topical Fluoride:
Fluorides Applied by Dentist/Professionally Applied
A. Aqueous solutions
• Sodium fluoride - 2 %
• Stannous fluoride - 8% B.
Fluoride Gels
• Acidulated phosphate fluoride - 1.23 % C.
Fluoride varnishes
• Duraphat
• Fluorprotector
D. Fluoride prophylactic paste
E. Restorative materials containing fluoride (composite resin) F.
Fluoride containing devices (slow release)
Self -Applied
• Fluoride dentifrices
• Fluoride mouth rinses.
• Fluoride gels

Fluoride Compounds
1- Inorganic compounds: (e.g. Sodium fluoride (NaF))
readily soluble salts that provide free fluoride; the most commonly used fluoride compound (both selfapplication and professional use); when in solution, NaF salt readily releases fluoride into saliva, dental plaque, pellicle and enamel crystallites.
Used in dentifrices , mouth rinses, chewing- gums,Solutions , gels ,varnishes ,prophylaxis pastes,slow- release devices
➢ Ammonium fluoride (NH4F) although investigated intensively some decades ago, it is currently unused – mainly due to its unpleasant taste and lack of superiority in clinical performance over NaF Formulations used in solutions
➢ Titanium tetrafluoride (TiF4) able to significantly reduce enamel solubility (as solution),due to the formation of a glaze on enamel and dentine;
currently being tested in solutions/varnishes as preventative for caries and erosion
used: solutions, varnishes
2 -Monofluorophosphate-containing compounds: fluoride is covalently bound to $PO3^{-}2$ions and requires hydrolysis to release fluoride ions;
e.g. dentifrices (neutral pH)

gels (neutral and acidic pH)
(Na2FPO3)one of the main advantages is its compatibility with abrasives.
3- organic fluorides: fluoride bound to organic compounds.
e.g. Amine fluoride which used in dentifrices, gels, mouth rinses , prophylaxis pastes, associated with a reduction in plaque adhesiveness due to the greater affinity of hydrophilic counter- ions to the enamel.

Goals of Fluoride (F) Administration

1. Do not harm the patient.
2. Prevent decay on intact dental surfaces.
3. Arrest active decay.
4. Remineralize decalcified tooth surfaces.

Self-Applied Fluoride

Self-applied fluoride products are usually brought and dispended by the individual patient but at the recommendation of a dental professional. These fluoride products are of low concentration ranging from 200-1000 ppm or 0.2-1 mg/ml. These self – applied fluorides are:

1. Fluoride dentifrices.
2. Fluoride mouth rinse.
3. Fluoride gel.

Requisites for self-applied fluoride agents: **[community and individuals]:**

Should be completely safe.
Should be effective for preventing caries
Method should be suitable for use by large groups and at a reasonably low cost.
Should be acceptable to participants.
Should be easy to use to ensure compliance.
Should require few professional personnel.
Should be able to be supervised by non-dental personnel after short periods of in-service training.

1. Fluoride Dentifrices

Fluoride adding fluoride to tooth-paste has been carried out since 1945 and covers a wide range of active ingredients in various abrasive formulations. Compounds that have been tested for caries-inhibitory properties include sodium fluoride, acidulated phosphate fluoride, stannous fluoride, sodium monofluorophosphate and amine fluoride. Most toothpaste nowadays contain sodium fluoride or sodium monofluorophosphate as active ingredient, usually in concentration of 1000–1100 mg F/g.

Fluoridated Toothpaste for Children

Concern about the fluorosis risk from children swallowing toothpaste has led to trial of lower-strength dentifrices. Findings from studies of 500–550 mgF/g products suggest efficacy equivalent to 1000 mgF/g toothpaste.

Since children can swallow between 0.12 and 0.38 mg of toothpaste per brushing, lower fluoride toothpaste may reduce the risk of fluorosis while substantially retaining the caries preventive benefits.

The production of candy like flavors and toothpaste containing fluoride at 1500 ppm or more should not be encouraged for use by children, as it may lead to an excessive ingestion of fluoride.

Toothpaste Formulations

Most 1000 ppm fluoride containing toothpaste achieve this concentration, i.e. [0.1%F= 1 mgF/g paste] by adding one of the following fluoride salts.

- Sodium fluoride [0.2% NaF]

  Sodium monofluorophosphate [0.76% Na_2PO_3F]
- Stannous fluoride [0.4% SnF_2]

Stannous Fluoride toothpaste has one major disadvantage that they lead to unsightly black/ brown extrinsic staining of tooth surface, especially around margins of tooth colored restorations. The discoloration is probably due to precipitation on the acquired pellicle of oxides and sulphides of tin.

Both sodium fluoride and sodium monofluorophosphate dentifrices can be recommended freely as available evidence fails to support the superiority of one fluoride over the other.

A unique characteristic of sodium monofluorophosphate is its compatibility with a wide variety of dentifrice abrasive system.

In contrast to other fluoride compounds such as stannous fluoride, which are almost completely dissociated in aqueous solution to yield fluoride ions that readily react with available cations, the fluoride in sodium monofluorophosphate remains largely complexed as PO_3F^- in solution. This fluoride complex is compatible with a wide variety of abrasive system. By far the greatest number of dentifrices on sale in the world today has sodium monofluorophosphate (MFP) as their active ingredient.

Mechanism of Action of sodium monofluorophosphate (MFP)

Two possible mechanisms have been suggested. One suggests that MFP ions is incorporated into the hydroxyapatite crystal lattice with a subsequent slower release of fluoride ion which then replaces hydroxyl groups to form fluorapatite.

On the other hand it is suggested that it is MFP ions itself which is incorporated into the apatite crystals by means of a substitution reaction with one or more of the phosphate groups. Fluoride in toothpaste is taken up directly by demineralized enamel and it also increases the fluoride concentration in dental plaque, thus leaving a store of fluoride available for remineralization when pH drops.

Manner of Use of Fluoridated Toothpaste

- Fluoridated toothpaste should be used daily for tooth cleaning by person of all ages to control development and progression of dental caries.
- In children under the age of 6 years, brushing should be supervised in order to prevent excessive ingestion.
- In children only a very small amount (less than 5 mm) which approximates the "pea size" should be placed on the brush**.**

2. Fluoride Mouth rinses

Frequent use of low concentration of fluoride is more cariostatic than less frequent use of higher concentration of fluoride for topical application. In areas where water fluoridation is not possible or has not been implemented, the fluoride mouth rinses have been found to be an effective tool in prevention of dental caries. Over the past few decades fluoride mouth rinsing has become one of the most widely used caries-preventive public health measure.

Sodium fluoride mouth rinse is now widely used in school based programs as well as by individuals at home. Other less extensively tested fluoride mouth rinses include those containing APF, stannous fluoride, ammonium fluoride and amine fluoride. Caries reduction by 30 %.

For reasons of lowest expense, convenience in handling as well avoidance of unpleasant taste, NaF became the most widely used of these tested products in public health programs.

Recommendations

- Mouth rinses designed to be rinsed and spit out, either prescribed by the dentist.
- The American Dental Association recommends the use of fluoride Mouth rinses, but not for children under six years of age because they may swallow the rinse.
- In communities with fluoridated water supplies or with natural occurring optimum fluoride level in drinking water, mouth rinsing programs would give a super added benefit.
- Over-the-counter daily fluoride mouth rinses generally contain 0.05 % NaF (200–220 ppm F). A 10 mL volume should be swished around the mouth vigorously once each day for one minute (ideally just before bedtime) and then expectorated. Patients should not rinse afterwards for 30 minutes. Pharmacy-only "weekly fortnightly" fluoride mouth rinses typically contain 0.2 percent NaF (900 ppm F). They are designed to be used under adult supervision, once each week for

one minute.

- Fluoride mouth rinse should be used at a time of day when toothpaste is not used, and it should not be a substitute for brushing with fluoridated toothpaste. After rinsing, mouth rinse should be spat out, not swallowed.

Indications

1. Patient with low salivation and high caries level because of systemic disease, use medication, surgery, radiotherapy.
2. Patient wearing orthodontic appliance which act as traps for plaque accumulation.
3. Patient unable to achieve good oral hygiene.
4. Patient with gingival gum recession and susceptible to root caries.
5. Patient with rampant caries.

3. Fluoride Gel .

Many fluoride gels have been become available recently. Fluoride gel contain component formulated in a non-aqueous gel base that does not contain an abrasive system. It contain:

1. Stannous fluoride 0.4% equal to 1000 ppm.
2. Sodium fluoride 1.0% equal to 5000ppm.

Recommendations

Use as tooth brushing with gel as the use of dentifrices(allowing the gel to remaine in the oral cavity for 1 minute and then expectorate).

Use as alternative to the use of fluoride rinses and adjunct to the use of professional topical fluoride application and dentifrices to achieve caries control in patient who are especially prone to caries formation as patient with rampant caries.

Restricted use only to the period required to achieve caries control like fluoride rinses.

Fluoride gel is not substitute to dentifrices, but it use in combination with dentifrices to achieve caries control in active caries patient

Fluoride exposure from multiple sources

Fluorides are found naturally throughout the world. They are present to some extent in all foods and waters so that all humans ingest some fluoride. This can be extremely beneficial in terms of the prevention of dental decay.

Multiple fluoride therapy →fluoride combination programs included application of fluoride in dental clinic in form of topical applied fluoride and fluoridated prophylactic past use and the home use fluoride dentifrices in addition to systemic ingestion of fluoride (communal water fluoridation).

This combination programs between systemic and topical fluoridation may give about 75% reduction in dental caries.

Tooth erosion

Tooth erosion is the term used to describe tooth wear caused by acid (extrinsic and intrinsic acid) that is not of bacterial origin, leading to painless and irreversible loss of hard tooth tissue. This phenomenon should be early diagnosed in children and adult to stop its progress. Fluoride use to formation of a protective layer on dental hard tissue , which compose calcium fluoride (CaF2) in case of amine fluoride and sodium fluoride or of metal rich surface precipitate in case of stannous fluoride, appear to be most effective on enamel. There is convincing evidence that fluoride in general can strength teeth against erosive acid damage. The uses of high concentration fluoride agent and/or frequent application are considered potentially effective in prevention of dental erosion.

Professionally applied fluorides

Fluoride agents have been applied by dental personal since 1940 in concentration of between 10,000–20,000 ppm which leads to the formation of calcium fluoride and eventually Fluor hydroxyapatite.

Topical fluoride applications are indicated for:

1. Patients with active smooth surface caries and those patients in high caries risk groups.
2. Patients who are at high risk for caries on root surfaces
3. To reduce tooth sensitivity
4. White spots lesion.
5. Active decay
6. Special patient groups, such as:

– Orthodontic patients
– Patients undergoing head and neck irradiation
– Patients with decreased salivary flow

7. Children whose permanent molars should, but cannot be sealed.
8. Additional protection if necessary for children in areas without fluoridated drinking water.

Type of Fluorides Applied by Dentist/ professionally applied include:

A. Aqueous Solutions

1. **Sodium Fluoride [Knutson's Technique]:** Available in both powder and liquid form. Neutral pH, 9,200 ppm of available fluoride, 29% effective in caries reduction. The recommended and approved concentration is 2 %.

Method of preparation:

Dissolving 0.2 gm of powder in 10 ml [20 gm in 1 liter] of distilled water → 2% NaF solution.

Advantages:

1. Taste is acceptable.
2. It is stable if stored in plastic bottle.
3. Non-irritating to gingiva .
4. Does not cause discoloration of tooth structures.

Disadvantage:

1. Patient had to make four visits in relatively short period of time.
2. If stored in glass bottle, the fluoride ion of prepared solution can react with silica of glass forming SiF2 [silicon fluoride], thus reducing the availability of free active fluoride. Hence reducing its anti caries action.

Recommended ages: It is recommended that a series of 4- weekly applications of 2 % NaF be given at ages 3,7,11 and 13, coinciding with the eruption of different groups of primary and permanent teeth.

Method of application

1. Cleaning and polishing of teeth is done.
2. Teeth are isolated with cotton rolls and dried with compressed air.
3. Teeth can be selected quadrant wise.
4. 2 % aqueous NaF solution is applied with cotton applicator for 4 minutes.
5. Procedure is repeated for remaining quadrants until all of the teeth are treated.
6. Second, third and fourth applications are recommended at intervals of approximately 1 week and they are preceded by cleaning and polishing.
7. Patient is advised to avoid rinsing, drinking and eating for next half hour.

Mechanism of Action of Sodium Fluoride

When sodium fluoride solution is applied on the tooth surface it reacts with hydroxyl apatite crystals rapidly to form calcium fluoride (CaF2). The calcium fluoride forms a layer on the tooth surface blocking further entry of fluoride ions. This sudden stop of the entry of fluoride is termed as "Chocking off effect". Fluoride then slowly leaches from the calcium fluoride. Thus calcium fluoride acts as a reservoir for fluoride release and that is the reason why sodium fluoride is kept untouched on the tooth for 4 minutes.

The calcium fluoride reacts with hydroxyl apatite to form fluoridated hydroxy apatite. This increases the concentration of surface fluoride, making the tooth structure more stable, and surface more resistant to caries attack. It also helps in remineralization of the initial decalcified areas.

The chemical reaction involved is:

$Ca10\ [PO4]6[OH]2 + 20\ F– \leftrightarrow$ **10CaF2** $+ 6PO4 – + 2OH–$

$CaF2 + 2Ca5\ [PO4]3\ OH \rightarrow$ **2Ca5 [PO4]3F** $+ Ca\ (OH)2$

2.Stannous Fluoride- [Muhler's Technique]:

Available in powder form either in bulk containers or pre-weighed capsules., *19,500 ppm of available fluoride and 32% effective in caries reduction. The recommended and approved concentration is 8 %.*

Method of Preparation

The solution has to be **freshly prepared** as they are not stable. It can be prepared by dissolving 0.8 gm of powder in 10 ml of distilled water. The solution is acidic, with a pH of about 2.4– 2.8.

Method of Application

1. Cleaning and polishing of teeth is done.
2. Teeth are isolated with cotton rolls and dried with compressed air.
3. Freshly prepared SnF2 solution is applied using cotton applicator. Care should be taken that all teeth surfaces are treated.
4. Repeated loading of cotton applicator should be done and swabbing is continuously done so as to keep tooth surface moist for 4 minutes.
5. Patient is allowed to expectorate after cotton rolls are removed.

Recommended Schedule

A six monthly interval treatment schedule is advised.

Mechanism of Action

Stannous fluoride reacts with hydroxy apatite and in addition to fluoride the Tin of solution also reacts with enamel and forms Stannous tri-fluorophosphate, which is more resistant to carious attack.

Chemical reaction *at low concentration* is:

$Ca_5[PO_4]_3OH + 2SnF_2 \rightarrow \underline{2CaF_2} + Sn_2[OH]PO_4 + Ca_3[PO_4]_2$

At High concentration:

$Ca_5[PO_4]_3OH + 16SnF_2 \rightarrow CaF_2 + \underline{2Sn_3F_3PO_4} + Sn_2[OH]PO_4$

[Tin Trifluorophosphate] [Tin hydroxyl phosphate]

$+ 4CaF_2[SnF_3]_2$

[Calcium trifluorostannate]

Tin hydroxy phosphate gets dissolved in oral fluids and is responsible for the metallic taste.

Tin trifluorophosphate which is the main end product is responsible for making the tooth structure more stable and less susceptible to decay.

Advantage

Recommended frequency is 6–12 months interval much less than as in case of sodium fluoride**.**

Disadvantages

1. Solution has to be freshly prepared each time before use.
2. A bitter metallic taste.
3. Can cause gingival irritation.
4. It causes brown pigmentation of teeth particularly in hypocalcified areas and around margins of restorations.
5. It is highly acidic in nature (pH 2.1-2.3).

B. Fluoride Gels

Fluoride gels and foams contain a high concentration of fluoride, typically up to 12.3 mg fluoride.

1. **Acidulated Phosphate Fluoride *(Brudevolds Solution)***: This is available as either as a solution or gel. *Both are stable(1.23 percent). 12,300 ppm of available fluoride, 3.0 pH, 28% effective in caries reduction.*

Method of Preparation

1. Solution*:* It is prepared by dissolving 20 gms of sodium fluoride in 1 liter of 0.1 M phosphoric acid. To this is added 50 % hydrofluoric acid to maintain a pH of 3.0 and fluoride ion concentration at 1.23 %.

2. Gel*:* for preparation of gel [APF], a gelling agent methylcellulose or hydroxyethyl cellulose is added to the solution and the pH is adjusted 4-5.

3. **Another form of APF Thixotropic gels** is available.

Thixotropic denotes a solution that sets in a gel like state but is not a true gel. Upon the application if pressure, thixotropic gels behave like solutions.

Recommended Frequency

Recommended frequency of APF application is twice a year topically.

Procedure for the Application of Fluoride Solution

a. Oral prophylaxis is done.

b. Teeth are isolated with cotton rolls and dried with compressed air.

c. Fluoride solution is then applied continuously with cotton applicator so as to keep teeth moist with fluoride solution for 4 minutes.

d. After all the teeth are treated patient is asked to expectorate and instructed not to rinse, drink or eat for next half hour.

Procedure for the Application of Fluoride Gel

a. Mouth trays should be tried in the patient's mouth. It may be necessary to adapt or trim trays.

b. Patient should be seated upright and suction should be used during the procedure.

c. Teeth should be air-dried before gel application. For caries prevention, cleaning or prophylaxis is prior to APF.

d. Enough gel, or foam, should be used to completely cover the teeth, but should be no more than 2–2.5 grams per tray or 40 % of the tray's volume.

e. Upper and lower trays should be inserted separately.

f. Fluoride should be applied for 4 minutes, not 1 minute.

g. Patient should expectorate for 1–2 minutes after tray removal.

h. Patient should not rinse, eat, or drink for at least 30 minutes after the procedure.

Clinical application

The frequency of gel application varies based on the caries risk level of the patient, and is usually provided at least every 6 months.

Mechanism of Action

APF when applied on teeth initially leads to dehydration and shrinkage in the volume of hydroxyapatite crystals. There is further hydrolysis and formation of di calcium phosphate dehydrate [DCPD], which is highly reactive. The fluoride ions start penetrating into the deeper crystalline structure of enamel and forms fluoro apatite which is stronger to acid dissolution.

Advantages

1. It is stable when stored in a plastic container.
2. No staining of teeth.
3. Gels can self -applied.

Disadvantages

1. Cannot be stored in glass container because it may remove minerals from the glass [etch].
2. Repeated exposure of porcelain or composite restorations to APF can lead to loss of material leading to surface roughening and cosmetic changes hence not advisable to use acidic topical fluoride agent in patients with these type of restorations.
3. It has an acidic taste.
4. Repeated application necessitates the use of suction, limiting its use in field programs.

Guidelines for the Application of Topical Gels

These are designed to minimize the amount of fluoride that may be swallowed.

1. Limit the amount of gel placed in each commercially available disposable mouth tray to no more than 2 ml or 40 % of the tray capacity.
2. Sit the patient in an upright position with the head inclined forward.
3. Use suction throughout the gel application procedure.
4. Instruct the patient to expectorate, or use a saliva ejector for 30 seconds after the gel application.

C. Fluoride Varnishes:

A fluoride varnish is a professionally applied adherent material. They permit the application of high fluoride concentrations in small amounts of material. These products are much more concentrated than gels, with typical concentrations of 22,600 ppm fluoride (in NaF varnishes) .

Advantages:

1. The use of fluoride varnish increases the fluoride concentration in saliva, which remains significantly higher 2 hours after its application than after the use of other fluoride agents.
2. Simple application and requires minimal training.
3. Prolonged contact time between fluoride and the tooth surfaces (increases fluoride uptake by dental hard tissues, as well as the formation of CaF2 reservoirs), and the possibility of using very small amounts of the product (a thin layer), which minimizes the risk of excessive fluoride ingestion.

Duraphat [NaF]: It was first fluoride varnish to be tested. It contains 2.26 % NaF or 22.6 mgF/ml. It is a viscous, resinous which should be applied to dry, clean tooth. Duraphat hardens into a yellowish brown coating in the presence of saliva. The efficacy of Duraphat between 30 to 45 %.

Mechanism of Action

When varnish is painted on the tooth surface, it acts as a fluoride depot from which fluoride ions are continuously released. These ions react with hydroxyapatite over a longer period of time as varnish is not quickly washed away by saliva. This leads to deeper penetration and significant anti caries effect.

Method of Varnish Application

1. Oral prophylaxis is done.
2. Teeth are dried and but not isolated with cotton rolls as varnish sticks to cotton.
3. First lower arch is taken up for application and then upper arch as saliva collects rapidly on the lower arch.
4. Dispense a small amount of varnish (0.3 ml to 0.5 ml, or 2 drops, for the entire primary dentition) to the applicator dish or pad.
5. Application is done with single tufted brush starting with proximal surfaces (Dental floss can be used to ensure that the varnish reaches interproximal areas).
6. Since varnish sets rapidly when they come in contact with saliva, no drying is necessary.
7. After application, patient is made to sit with mouth open for 4 minutes.

Recommended Dosage

Fluoride varnish has a high fluoride concentration, but its safety is acceptable. Varnish is fast setting, fluoride is slowly released, and a small amount is needed for the complete dentition. Measurements of fluoride after topical treatments with varnish. show levels far below those considered toxic Consequently, varnishes may be a better alternative to fluoride gels, especially for young children.

The only disadvantage of sodium fluoride varnishes is that they cause a temporary change in tooth color, which dental professionals need to inform their patients of.

Indications:

Fluoride varnishes are used for:

- Disabled children
- Incipient caries lesion
- After restorative treatment is complete under general anesthesia
- Very young children who cannot expectorate the gel. Fluoride varnishes are safe because the amount of varnish usually used is 0.3-0.5 ml which delivers only 3-6 mg of fluoride.

Note: Patient is advised not to eat or brush for at least 4 hrs. after varnish application.

D.Fluoride Prophylactic Paste

The major functions of prophylactic paste are:

1. To clean the tooth surface through the removal of all exogenous deposits.
2. Polish the dental hard tissues, including restorations.

Prophylactic paste contains abrasive particles which abrade the deposits and debris from tooth surface.

E. Restorative Materials Containing Fluoride

- The purpose of adding fluoride to restorative material is to capture its anti -cariogenic property. A major reason for the failure of restorations is recurrent or secondary caries, incorporation of fluoride into restorations may be beneficial because of the observed cariostatic action of fluoride.
- The fluoride ions are slowly released from the materials. Fluoride has also been added to amalgam in an attempt to reduce the risk of recurrent caries at restoration margins.

- Fluoride containing restorative materials includes glass ionomer cements, resin modified glass ionomer cements, poly acid modified resin composites (compomers), resin composites, fissure sealants and dental amalgam.
- Fluoride releasing components have included fluoro alumino silicate glasses (FAG), stannous fluoride (SnF2), organic amine fluorides (CAFH) and ytterbium fluoride (YbF).
-

F. Fluoride Containing Devices (Slow Release):

As the current scientific consensus regards a constant supply of low levels of fluoride, especially at the biofilm/ saliva/dental interface, as being of the most benefit in preventing dental caries, it is reasonable to expect a positive effect on caries prevalence of a treatment able to raise intraoral F concentrations at constant rates, without relying on patient compliance. Considering that intraoral levels of F play a key role in the dynamics of dental caries, it has been suggested that the use of controlled and sustained delivery systems can be considered as a mean of controlling dental caries incidence in high-risk individuals.

There are three types of slow-release F devices: the copolymer membrane type, developed in the United States, and the glass bead, developed in the United Kingdom. More recently, a third type, which consists in a mixture of sodium fluoride (NaF) and hydroxyapatite.

Fluoride Toxicity

Inadequate ingestion of fluoride is associated with dental caries while ingestion of fluoride in excessive quantities can be toxic. The fluoride toxicity can be *chronic* or *acute*.

Chronic fluoride toxicity refers to long term ingestion of fluoride in amounts that exceed the approved therapeutic level.

Acute fluoride toxicity: Acute means rapid intake of an excess dose over a short period time.

Factors affecting fluoride toxicity:

The differences in toxic potential of different fluoride compounds are related to:

1. Solubility of the compound.
2. Content of the compound, e.g. stannous fluoride is slightly more toxic than sodium fluoride because high doses of tin ion.
3. route of administration
4. Age.
5. Rate of absorption.
6. Acid-base status.

1. Acute toxicity.

Acute fluoride poisoning is rarely seen. Symptoms of acute fluoride poisoning.

1. Salivation

2. Nausea
3. Vomiting
4. Abdominal pain
5. Diarrhea
6. Cramps
7. Cardiac arrhythmia
8. Coma

Management

Management based on the amount of fluoride ions ingested if < 5.0 mg/ kg, it requires Initial Emergency Response in the Oral Care Setting:

1. Induce vomiting by administering an emetic, to reduce the fluoride absorption (this should occur only if the client has a gag reflex, is conscious, and is not convulsing)

2. This is followed by the oral administration of 1% calcium chloride or calcium gluconate; if these are not available milk should be ingested.

3. Increasing fluoride excretion by increasing the alkalinity of the urine and fluid replacement.

If fluoride ingested level >5.0 mg/kg need induce vomiting ingested Milk, and 5% calcium gluconate, hospitalization.

Lethal and Safe doses of Fluoride

Certainly Lethal Dose (CLD)

A lethal dose is the amount of drug likely to cause death if timely interception by antidote is not initiated.

In Adult*:* CLD is 5–10 gm of sodium fluoride taken at 1 time. The fluoride ion equivalent is 32–64 mg Fluoride (F) per kg body weight.

In Children*:* CLD is approximately 0.5–1.0 gm. It varies with size and weight of the child. children under 6 years of age, however 500 mg is lethal.

probably toxic dose (PTD). The minimum dose that could cause toxic signs and symptoms, including death, and that should trigger immediate therapeutic intervention and hospitalization for fluoride intoxication has been set at 5 mg/kg body weight this is called probably toxic dose (PTD).

Recommendations for parents about use of fluoride agent by children:

• Parental supervision
• Child-proof containers (for fluoride tablets)
• Keep products out of reach of young children
• Supervise children when brushing / rinsing , Small amount of tooth paste to be used
• Products with low fluoride level to be used
• Teaching children not to swallow paste or rinse
• Strict adherence to professional advice

2. Chronic Toxicity

The various forms of fluorosis arising due to excessive intake of fluoride over a prolonged period of time. It can cause dental and skeletal changes referred to as dental and skeletal fluorosis respectively.

Dental fluorosis

It is a hypoplasia or hypo mineralization of tooth enamel or dentine produced by chronic ingestion of excessive amount of F during the period when teeth are developing. In relation to the stage of tooth development and exposure to fluoride. The central incisor takes approximately 3 years to go through complete enamel mineralization .

Skeletal Fluorosis

Skeletal fluorosis affects the bones/skeleton of the body. Skeletal fluorosis affects children as well as adults. It does not easily manifest until the disease attains an advanced stage. Fluoride mainly gets deposited in the joints of neck, knee, pelvic and shoulder bones and makes it difficult to move or walk. The symptoms of skeletal fluorosis are similar to spondylitis or arthritis. Patients who consume large quantities of water or who have renal problems should avoid fluoridated water altogether . Physicians should at least consider that some joint pain complaints may simply be the result of exposure to too much fluoride and develop a strategy to reduce the fluoride intake.

The concentration in PPM of fluoride that prevents dental caries as followings

Water supplies → 0.7–1.2ppm
Fluoridated salt →200–250 ppm
0.05% NaF Mouth rinse,→ Once daily 230 ppm
0.2% NaF Mouth rinse,→ Once weekly 920 ppm
Dentifrices, children→ 250–500 ppm
Dentifrices, adult Twice daily →1,000–1,500 ppm
1.1% NaF gels Once daily →5,000 ppm
Professionally applied solutions →(2 % NaF) 9,200 ppm
Professionally applied solutions, →12,300 ppm gels, foams →(1.23% APF) ppm

Professionally applied solutions (8% SnF2) 19,500 ppm
Professionally applied varnishes (5% NaF) 22,600 ppm

Pit and fissure sealant

Pit and fissure sealant it's an additional preventive tool to halt caries progression. Caries potential is directly related to the shape and depth of the pits and fissures. The success of fluoride in caries prevention on smooth tooth surfaces has made caries primarily a disease of pits and fissures of the tooth. The cariostatic properties of sealants are attributed to the physical obstruction of the pits and grooves. This prevents the penetration of fermentable carbohydrates, and so the remaining bacteria cannot produce acid in cariogenic concentration. Sealants are effective caries prevention agent to the extent they remain bounded to teeth. Sealants are material that are chemico-mechanically retained within the pit or fissure, and thus prevent the penetration bacteria and there is no marginal leakage, such vulnerable sites remain free of caries.

Pit& fissure sealant can be defined as " a cement or resin material which is introduced into unprepared occlusal pits and fissures of caries susceptible teeth forming a mechanical and physical protective layer against the action of acid producing bacteria and their substrate".

The epidemiological studies shown that the molar pit and fissure are the most likely to become carious. Occlusal caries has been shown to occount for 83% of the total caries in children between 5 and 17 years of age (in the UK).

In 1955 Buonocore wrote about the technique of acid etching as being a simple method of increasing the resin materials to dental enamel. He used 85% phosphoric acid to etch enamel for 30 seconds. This produces a roughened surface at a microscopic level, allowing mechanical bounding of low viscosity resin materials. By 1965 Bowen and others developed the Bis-GMA resin, which is the chemical reaction product of bisphenol A and glycidyl methacrylate. Urethane dimethacrylate and other dimethacrylates are alternative resins used in sealant materials. For the chemically curing sealant, a tertiary amine (activator) in one component was mixed with another component containing benzoyl peroxide, and their reaction produces free radicals, thus initiation polymerization of the sealant material. The other sealant materials are activated by an external energy source. The early light-activated sealants were polymerized by the action of the ultra violate rays

(which are no longer used) on a benzoin methyl ether or higher alkyl benzion ethers to activate the perioxide-curing system. The visible light-curing sealants have diketones and aromatic ketones, which are sensitive to visible light in the wave length region of 470 nm (blue-light). Some sealants contain filler, usually silicon dioxide micro fill or even quartz.

Types of pit and fissure sealants according to:

1- **Generations.**
   a- First generation sealants which activated by ultra-violet light, no more used, as u-v light is harmful to the body.
   b- Second generation sealants which is chemical curing resins, based on catalyst-accelerator system e.g. Concise (3M)
   c- Third generation sealants which activated by visible light e.g. Fissurit (Voco) Delton (Johnson & Johnson).
   d- Fluoride containing sealants which have double protection.
2- **Fillers**.
   a- Free of filler which is flow better.
   b- Semifilled which is more resistant to wear.
3- **Colour** of the sealants.
   a- Clear which is more esthetic but difficult to detect at recall examination.
   b- Tinted: - can be easily identified.
   c- Opaque: - can be easily identified.

Recently, glass-ionomer cements (GICs) and resin modified GICs have also been introduced as caries preventive fissure sealants. These materials should not be regarded as semi permanent fissure sealants, but as efficient, slow-release fluoride agents, in whom the depleted fluoride reservoir can be replenished from fluoride sources as varnishes, gels, etc. placement of GICs would be more appropriate during eruption, when the fissures are most caries susceptible, even though most of the GICs material is lost shortly after full eruption, by which time the risk of caries in these fissures is negligible.

Newer materials as fissure sealant (1966- present day)

1- Cyanoacrylate resine.
2- Urethane resins.
3- Bis-GMA resins

4- Glass ionomer cements.
5- Resin-modified glass ionomer cements.

Procedure of pit and fissure sealant application:-

1- Polish the tooth surface:
   Remove plaque and debris from the enamel and the pits and fissures of the tooth. Any debris that is not removed will interfere with the proper etching process and the sealant penetration into the fissures and pits. Polishing can be carried out by using prophylaxis cup and pumice.
2- Isolate and dry the tooth surface:-
   Rubber dam provide the best isolation of the tooth from the salivary contamination.
3- Etch the tooth surface:-
   The tooth should be etched with a 37% of orthophosphoric acid for 15-30 seconds. The etchant should be applied to all the pits and fissures with at least a few millimeters beyond the final margin of the sealant. Do not allow the etchant to contact with soft tissue. If this occurs, rinse the soft tissue thoroughly.
4- Rinse the tooth:-
   The tooth should be rinsed for approximately 15 seconds.
5- Isolation and dry the tooth:-
   The tooth is dried until it has a chalky appearance if not we should re-etched for 15 second. The moisture contamination at this stage of the process is the most common cause of sealant failure.
6- Apply bonding agent:-
   Apply the intermediate bonding agent and cure it.
7- Material application:-
   The sealant material is then applied to the tooth according to the manufacturer's directions. Be careful not to incorporate air bubbles in the material. After the sealant has set, the operator should wipe the sealant surface with a wet cotton pellet. This allow for the removal of the air-inhibited layer of the non-polymerized resin. Failure to perform this step will leave an objectionable taste in the patient's mouth.
8- Evaluation the sealant:-

The sealant should be evaluated visually and tactically. If there are any deficiencies in the material, more sealant material should be applied.

9- Check occlusion:-

Check for occlusal high points and if present correct them.

10- Retention and periodic maintenance:-

It is necessary to re-evaluate the sealant at recall visits. If there has been any sealant loss, new sealant can be applied over the old material.

*Note:- polyacrylic acid is used to prepare fissures for sealing with glass-ionomer cements. This procedure should be regarded as a chemical conditioning of the surface and not an etching treatment. The reaction between GIC and the dental tissues is chemical, and the bond strength is considerably weaker than that achieved with the acid-etching technique.

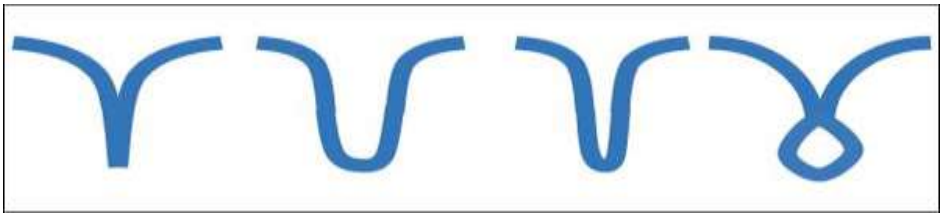
Pit and fissure sealants require:-

1- Good moisture control when being placed.
2- Clean surfaces.
3- Appropriate etching and drying time.
4- Appropriate coverage of the surface.
5- Checking occlusion for interferences.
6- Regular monitoring and maintenance after placement.

Indication of the sealant:-

Originally routine application of sealant was recommended for all posterior deep occlusal fissure, fossa or lingual pit present in teeth.

1- Sound teeth, with many occlusal lesions or with few proximal lesions.
2- If pits and fissures are separated by transverse ridge, a sound pits or fissure may be sealed.
3- Deep narrow pits and fissures.



4- Recently erupted teeth (within three years).

Thus, teeth in caries free patient and caries free occlusal surfaces, which have been fully erupted for more than 3 years, do not need application of sealant. While where caries has affected one or more permanent molar teeth (and those who have experience of caries in primary teeth) the remaining sound fully erupted pits and fissure should be sealed. Preventive program for the occlusal surfaces of the molars, selected at the beginning of the molar eruption. The aims are maintain caries-free occlusal surfaces and, at the most, use of a fissure sealant or so-called" fissure blocking".

The sealant of primary teeth may be advised where a child or a young person is compromised in some way for the development of caries and / or its treatment, while in adult; the sealant should be placed if there is evidence of existing or impeding caries susceptibility as would occur following excessive intake of sugar or as a result of drug or radiation induced xerostomia. In all cases it’s the disease susceptibility of the tooth that should be addressed not the age of the individual.

The decision to place sealant on sound teeth based on:-

1- Oral hygiene of the patient.
2- Individual history of dental caries.
3- Dietary habits.
4- Patient cooperation and reliability in keeping recall appointments.
5- Tooth type and tooth morphology.

Contra indication

1- Caries pits and fissures.
2- Broad, well-coalesced pit and fissures.
3- Teeth caries free for 4 years or longer.
4- Many proximal lesions.
5- Patient's behavior does not permit use of adequate dry field technique throughout the procedure.

Factors affecting sealant retention in the mouth:-

1- Type of sealants.
2- Position of teeth in the mouth.

3- Clinical skill of the operator.
4- Age of the child.
5- Eruption status of the teeth.

Plastic sealant is retained better on recently erupted teeth than in teeth with more mature surface, while retention better on mandibular teeth than on the maxillary teeth, this is possibly due to the fact the lower teeth are:-

1- More accessible.
2- Direct slight is possible.
3- Isolation of the teeth is easier.
4- Gravity aids the flow of sealant in to the fissure.

Teeth that have been sealed and then have lost the sealant have had fewer lesions than control teeth. This possibly due to the tags that are retained in the enamel after the bulk of the sealant has been sheared from the tooth.

Fluoride containing sealants

The addition of fluoride to sealants was considered since 1976 and efforts to combine the two continue today. Basically, two methods of fluoride incorporation are used. In one, a soluble fluoride salt is added to unpolymerized resin. After sealant is applied to a tooth, the salt dissolves and fluoride ions are released. The other method involves an organic fluoride compound which is chemically bound to the resin. Since there is no lasting effort on salivary fluoride concentration, any additional benefit from the use of the fluoride-releasing sealant would have to be derived from fluoride absorbed into the enamel underlying the sealant.

The sealant-composite combination

In this case a small carious lesion has penetrated to the dentin. A clinical series showing the sequence for this conservative preparation and restoration that by doing ordinary cavities for carious lesion then filled with a light-curing composite or resin-modified glass ionomer. A light-curing sealant is placed over the remaining susceptible areas and brushed into the pit and grooves.

Diet and dental caries

One of the many factors that contribute to the development of dental caries is diet that plays an important role. In conjunction with oral hygiene and other measures such as widespread use of fluoride, dietary control makes an important contribution to the multifaceted strategy for caries control.

The role diet and nutrition in the etiology and pathogenesis of caries may be viewed as systemic and local effect. In general, the term diet refers to food and drink consumption (*diaita* 'way of life') while nutrition refers to a process by which living organisms physiologically absorb and metabolize food to ensure growth, energy production, repair of tissue and ultimately reproduction of the species (*nutritio* 'to nourish')

Role of carbohydrates in caries development

It has been firmly established that dietary carbohydrate are caries conducive and they exert their cariogenic effect locally on tooth surface by influence the quality and quantity of dental plaque. Dental caries occurs as plaque bacteria metabolize fermentable carbohydrate, producing organic acids. These acids diffuse through the plaque into the enamel and dissolve mineral. If mineral diffuses out of the tooth and into the oral environment, then demineralization occurs. If the process is reversed the damaged crystal are rebuilt (remineralization).

Caries occurs when demineralization exceeds remineralization

Fermentable carbohydrate: any carbohydrate that can be hydrolyzed by salivary amylase and subsequently fermented by bacteria

Carbohydrate is an essential nutrient and can be classified in to **three** main categories:

1- Free-form monosaccharide (simple sugars) include ***glucose*** and ***fructose*** which are found naturally in fruit, vegetables and honey while ***galactose*** occur only as a result of the breakdown during digestion of lactose.

2-Disaccharides (two simple sugar molecules linked together): the most common:

- **Sucrose** (refined from sugar cane or sugar beets, it's a major part of dietary sugar) formed when one molecules of glucose combine with one molecules of fructose.
- **Lactose** is formed when a molecule of glucose combine in a molecules of galactose (milk sugar).
- **Maltose** is formed when two molecules of glucose combine; it is mainly derived from hydrolysis of the starch.

3-Polysaccharides unlike the mono and disaccharide the polysaccharides not sugar. All polysaccharides are made up of many individual sugar molecules, usually glucose, joined together. The digestible form**s** include ***starch***, which is found in rice, potatoes, peas; about half of dietary carbohydrates are composed of starch.

The fact that dental caries occur beneath dental plaque is now quite clear and this distinguish it from dental erosion that is dissolution of enamel caused by acids of non-bacterial origin.

Types of study providing evidence for the relationship between diet and caries development*:* The evidence come from a number of type of studies, these include:

- ***Observational (epidemiological) studies***: Numerous world-wide epidemiological studies show that caries prevalence is low in population adhering to a primitive way of living and a diet of local products with little sugar.
- ***Interventional studies*** : in which diets of groups of people are purposefully altered and the effect of this intervention observed. Those that have been reported are now decades old and were conducted on highly selected groups of people, before the strong link between sugars and caries was established. Such studies would not be possible to repeat today because of ***ethical constraints*** they include.

The Hopewood House was an orphanage in Australia. From its beginning sugar and other refined carbohydrate were excluded from the children diet Dental surveys of these children from the ages of 5-11 years revealed a greatly reduced caries incidence compared the state school population in that age group When the children became old enough to earn wages in the outside economy, they deviated from the original diet. A steep increase of decayed missing and filled teeth (DMFT) after the age of 11 years indicates that the teeth did not acquire any permanent resistance to caries

The Vipeholm study was conducted shortly after the Second World War in an adult mental institution in Sweden between 1945-1953. The study investigated the effect of consuming sugary foods of varying stickiness (i.e different oral retention times) and at different time through out the day on the development of caries by measuring caries increment in subjects

Main conclusions of the Vipeholm study:

- Sugar intake, even when consumed in large amounts , had little effect on caries increment if it was ingested up to a maximum of four times a day at mealtimes only
- Consumption of sugar in between meals was associated with a marked increase in dental caries
- The increase in dental caries activity disappear on withdrawal of sugar- rich foods
- Dental caries experience showed wide individual variation

- ***Animal studies***: various animal experiments were conducted to evaluate the effect of sugar intake
- ***Enamel slab experiments***: These observe the effects of diet on dmineralization in slabs of enamel (cut from extracted teeth) which are held in the mouth of human volunteers in a removable plate constructed like an orthodontic appliance.
- ***Plaque pH studies***: This type of experiment investigates the effect of food, meals, or component of foods on the pH of dental plaque. These studies are relatively easy to do, but they measure acidogenicity of diet rather than cariogenicity (only an indication of the possible effect of diet on the development of dental caries).
- ***Incubation studies***: these are the simplest but weakest method. Test foods are incubated with plaque or saliva (which contains plaque organisms) and the rate of acid production is recorded. In some experiments, whole enamel, powdered enamel, or calcium phosphate are added to the saliva/substrate mixture and the rate of dissolution of mineral is taken as a measure of cariogenic potential.

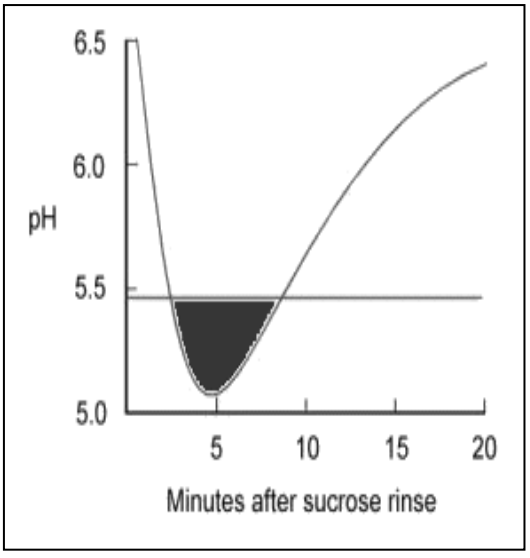
The basic Stephan curve

The resting PH of dental plaque was mostly between pH 6.5 and 7, the term ***resting plaque*** refers to plaque 2-2.5 hours after the last intake of carbohydrate. But when the plaque exposed to sucrose or glucose the pH of plaque falls rapidly below the critical value within 2-5 minutes.

(the value of pH 5.5 has become accepted as critical pH below which dental enamel will begin to dissolve because the environment is no longer saturated with enamel mineral), this rapid fall was then followed by slow recovery over the next 30-60minutes.The plot of plaque pH against time has become known as the **Stephan curve**.

The rapidity with which the pH falls is a reflection of speed on which sucrose come diffuse in to plaque and the activity of the concentration of enzymes produced by the great number of bacteria in the plaque. The slow rate of recovery to the resting pH, critical factor in caries production depend mainly on

- **Rapid production of high concentration of acids within the plaque , temporarily overcomes local buffering**
- **Escape of acids in to saliva, delayed by the diffusion-limiting properties of plaque and its thickness**
- **Diffusion of salivary buffers in to plaque hampered by the diffusion-limiting properties of plaque and its thickness**
- **Continued sugar production from bacterial intracellular polysaccharides**



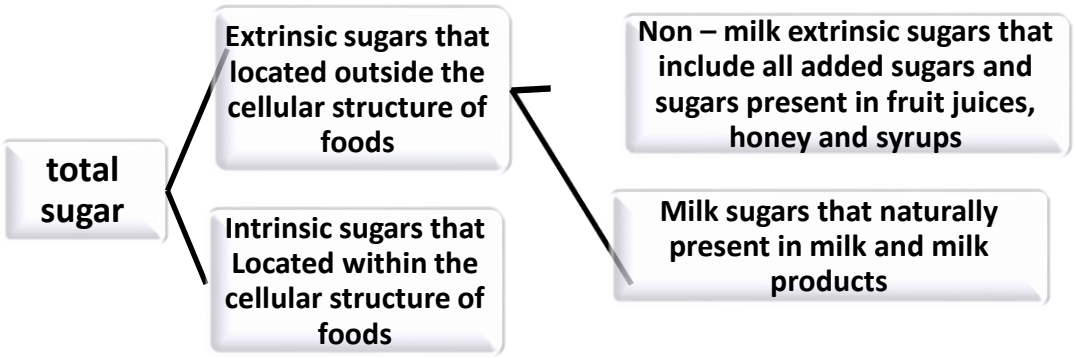
➡ Lack of saliva means long and deep Stephan curve, lengthening the time of demineralization and reducing the time when remineralization can occur.

➡ Caries free subjects tend to have a slightly higher resting plaque pH, a higher minimum pH following consumption of fermentable carbohydrate and a faster return to resting levels, when compared with caries susceptible subjects (as shown in figure above).

Factors affecting food cariogenicity:

1-Types of carbohydrates

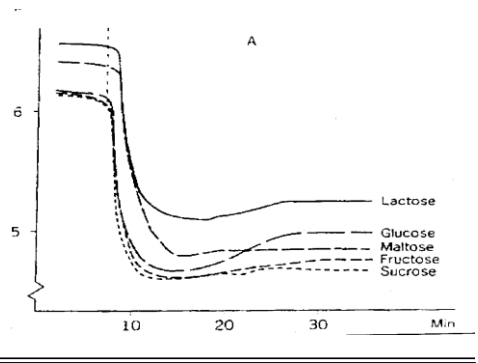
For dental health purpose , there is no evidence from epidemiological studies that sugar located within the cellular structure of a food are harmful to teeth, and therefore it is important to distinguish between theses and sugars in free form, therefore they classified sugars for dental health purpose into intrinsic and extrinsic sugars, extrinsic sugar were subdivided into milk sugars (as lactose naturally present in milk is not thought to be harmful to the teeth) and non milk extrinsic sugar which include all added sugars and sugars present in fruit juices, hony and syrups. In term of dental caries, it's the intake of non milk extrinsic sugar (or free sugars) that need to be reduced.



Sucrose is the most abundant sugar. It is used by food manufacturers all over the world as a food ingredient.

For several reasons sucrose has been called the *arch-criminal* in dental caries. The epidemiological evidence for sucrose as the cause of dental caries:

- **Low caries prevalence in population with low sucrose intake**
- **The decline in caries prevalence during wartime sucrose shortages**
- **The rise of caries prevalence with increasing availability of sucrose**
- **Archaeological evidence of low caries prevalence in eras before sucrose became freely available**
- **Low caries prevalence in disorders of sucrose metabolism hereditary fructose intolerance**



There seems to be little difference in the cariogenicity of glucose, fructose, and maltose, but the same source of evidence show that lactose is less cariogenic

Sucrose is unique because it is a substrate for extracellular dextrin synthesis by *S. mutans*. The cariogenicity of plaque depend on its ability

- ✓ to adhere to the teeth,
- ✓ to resist dissolution by saliva and
- ✓ its protection of bacterial acids from salivary buffering.

These properties depend on the formation of insoluble polysaccharides produced particularly by cariogenic strains of S. mutans. In addition, colonization by S. mutans (cariogenic bacteria) is highly dependent on the sucrose content of the diet

In the absence of sucrose, S. mutans cannot colonize the mouth, therefore its plaque counts appear to depend on the sucrose content of the diet. Sever reduction in the dietary sucrose cause S. mutans to decline in number or disappear from plaque.

Nevertheless, no sugar has been shown to be more cariogenic than sucrose and, it is the most widely available dietary sugar, it is has been subject of the greatest criticism.

Starch constitutes a heterogeneous food group, it may be highly refined or consumed in its natural state, it may be consumed raw or in cooked form- all these factors should be considered when assessing the cariogenicity of starches. They argue that all carbohydrate including starch cause dental caries, because although the starch (polysaccharide) molecules are, too large to diffuse into the plaque, however they are broken down by salivary amylase releasing ***maltose, maltotriose, and glucose*** that may be metabolized by oral bacteria to produce acids that cause dental caries.

Plaque pH drop very little following consumption of raw starch but soluble starch and starch containing food such as bread cause a pH fall which is somewhat smaller than with sugar

The starch granules of plants are only slowly attacked by salivary amylase, because the starch is in an insoluble form and protected by cellulose membrane, therefore the cariogenicity of uncooked starch is very low. Heating at temperature used in cooking cause a partial degradation to a soluble form, which can be further broken down by salivary and bacterial amylases to ***maltose, maltotriose, and glucose***.

Mixture of starch and sucrose cause a more dental caries than starch alone and the amount of dental caries was positively related to the amount of sugar in the mixture probably due to prolonged retention.

2-Physical form of food and clearance time:

In addition to the chemical composition of food, physical and organoleptic properties (particle size, solubility, adhesiveness, texture and test) are important for cariogenicity, since they influence eating pattern and oral retention of the foods. Diets that results in the greater retention of refined carbohydrate over the longest period are the most cariogenic.

- The carbohydrate in various drink are eliminated within 5 minutes while sweet such as sugar containing chewing gum, toffees, lozenges generally give high oral sucrose concentration and clearance time from 40 minutes for chewing gum to 15-20 minutes for other sweets.
- The texture of the diet is also important, for both salivary secretion and elimination of fermentable carbohydrate from the oral cavity. A diet that require thorough chewing will result in the secretion of large amount of saliva with a high pH and strong buffering capacity, in contrast to a finely textured diet that require little mastication tends to be retained in the oral cavity and eliminated slowly

The caries producing potential could possibly be reduced by modifying their physical properties (roughage, adhesiveness, solubility).

➡ Practical way to speed up carbohydrate clearance are

- ✓ tooth brushing immediately after meal, *or*
- ✓ induction of rapid salivary flow by mechanical or gustatory stimuli through eating tough or highly flavored foods at the end of meals

Chewing sugar free chewing gum or peanuts immediately after eating sugar also speed up sugar clearance and neutralization of plaque acid through saliva stimulation

Mouth rinsing with water has a very limited effect, partly because it is generally done too late: two minutes after a sucrose challenge, the sugar concentration in saliva is usually lower than that in plaque so rinsing with water at that time would not be expected to reduce the diffusion of sugar into plaque, unless the sugar clearance were excessively slow, as in xerostomic subjects.
The advantages of mouth rinsing after meal is that it also help to remove sugars in solution and food debris.

Chewing sugar-cane yield a less pronounced pH drop and a quicker pH recovery in dental plaque than is seen following a mouth rinse with sucrose. The difference probably result from stimulation of salivary flow associated with the chewing

It's often advised to consume sugar- rich foods at meal times rather than alone, or in between meals; this is because, when consumed with other foods the effect on pH is minimized probably due to
(1) The dilution effect
(2) The increased salivary flow rate due to mastication of other foods.

Frequency of intake sugar and dental caries

The relative importance of frequency versus the total amount of fermentable carbohydrate consumption is difficult to evaluate. Most studies point to the frequency of eating as being of greater etiological importance for caries than the total consumption of sugar. Frequent intake sugars will induce a prolonged and intense acid attack on the tooth surface; moreover, the time available for remineralization is thus decreased.

The pH of dental plaque falls rapidly when sugar are eaten, the more occasion sugar is taken the greater the number of times plaque pH will fall below a level where demineralization can occur (critical pH), the less time there is for remineralization to occur. With a more frequent intake of refined carbohydrate, damage time is increased and tooth repair time (remineralization) decreased proportionately.

Nutrition and oral health

Nutrition is an essential for the growth, development, and maintenance of oral structures and tissues. During periods of rapid cellular growth, nutrient deficiencies can have an irreversible effect on the developing oral tissues. Early malnutrition increases a child's susceptibility to dental diseases in the deciduous teeth, throughout life, nutritional deficiencies or toxicities can affect host resistance, healing, oral function, and oral-tissue integrity.

The oral cavity is a mirror of the nutritional status of the body. Nutrition is considered to be one of the most important factors that plays an important role in tooth formation as well as in bone development and metabolism, it may involved in disease process affecting the tooth and its supporting structure, deficiencies in many nutrients are thought to be linked to the development and progression of oral diseases.

Nutrition dental caries

During the pre-eruptive period of development of teeth, food exerts a systemic (nutritional) effect on the formation of the dental matrix and its mineralization. The first sign of tooth development of dental tissue occur around 28 days of intrauterine life and mineralization of dentin and enamel of primary teeth occur about 4-6 months in uteri so formation of teeth take long time and pass through a number of stages these are:

1-Secretary phase when the organic matrix is formed

2-Mineralization phase *which consist of crystal formation and crystal growth*

3-Maturation phase *during which water and organic matter withdrawn and the mineral content increase*

These stages are considered critical periods (critical period of human development) that define as: time interval when specific nutrient are needed by a particular tissue programmed to develop at prescribed time and rate, inappropriate supply of nutrient at such time can result in sever irreversible changes that affect the growth of the ***organ these changes in turn can result in permanent defects in function and decreased resistance to disease.***

Aberrations that occur during period of formation may have potentially irreversible effect on the developing organism. Malnutrition during these critical periods of growth can result in a dentition that is more susceptible to dental caries.

Systemic effect: In the first half of previous century there was strong believes that good nutrition while tooth were forming was the principal way to prevent dental caries and caries was considered to be a deficiency disease.

Nutritional factors may have an effect on the following:

• Morphology of the teeth

• The quality of dental hard tissues

• The quality of saliva .

Morphology of the teeth

It is well known that teeth with deep narrow fissures and marked pits and grooves are more susceptible to caries than those with fewer plaque retentive areas. The morphology of the tooth is largely determined by genetic factors but in many studies nutritional imbalance of protein, fat and carbohydrate affect the morphology of the teeth.

Enamel defect

The tooth developments include the formation of an organic protein matrix, followed by mineralization and maturation. The process, which follows a well-defined chronological pattern, involves several critical stages, Nutritional insult to protein synthesis or mineralization may disturb the tooth structure as well as the form of the teeth.

- **If matrix formation is affected enamel, hypoplasia will ensue**
- **If maturation is lacking or in complete hypocalcification of enamel will result in which deficiency in the mineral content of the enamel is found.**

"Poor nutrition is one of many causes of dental defect, clear relationship between specific dietary nutrients deficiency during critical periods of developments and poorly calcified tooth had been demonstrated.

Based on many studies awareness has arisen that nutritional disturbances, such as deficiencies of calcium, phosphate, vitamin A, D and C and **protein-energy**, affect tooth tissue formation according to their biological roles.

Protein energy relation

When dietary energy intake is adequate, the amino acids derived from dietary protein are immediately used for whatever protein synthesis is required such as for growth and maintenance of the body tissue. While when dietary energy intake falls below a certain critical level (insufficient fat and carbohydrate are available to meet immediate energy needs), amino acids are used as a source of energy.

Vitamin D, calcium, phosphorus

It well known that 96% of dental enamel is apatite mineral principally calcium and phosphorus. Vitamin D is intimately involved with calcium metabolism and its intestinal absorption and therefore, it has a role in tooth formation.

Lady May Mellanby in the first half of previous century showed that vitamin D deficiency led to hypoplastic teeth (she recognized that children with vitamin D deficiency had delayed development of teeth, had very deficient poorly calcified enamel). She noticed improved appearance of teeth by provision of cheap milk or cod liver oil to pregnant and lactating mother, infants, and young children also addition of vitamin D to margarine and calcium carbonate to bread

@ In addition to the study by Mellanby, the importance of the role of vitamin D and plasma calcium level in the etiology and prevention of enamel hypoplasia was illustrated by many studies showed that enamel hypoplasia is related to disorder in calcium homoeostatic, which in turn is controlled in a complex way involving vitamin D, parathyroid hormone and calcitonin. They also suggested that hypocalcaemia was the mechanism by which chronic diarrhea caused dental hypoplasia.

Indeed the health education authority in developed countries stopped ging" concerning calcium and vitamin D and strongly healthy teeth many years ago, order to concentrate the dietary aspects of dental health education on me uncontroversial messages reduce sugar consumption and using fluoride. It should be emphasized that these remarks concerning the unimportance of nutrition (other than fluoride), while teeth are forming apply at present time to developed countries where standards of nutrition are generally adequate, the same may not apply in countries when sever malnutrition is prevalent

The quality of the hard tissues:

The quality of the hard tissues of the tooth can influenced by nutrition, the changes may be importance for development and progression of dental caries.

Evidences that nutritionally derived variation in structure increase caries susceptibility have been presented.

In many studies deficiency of protein energy, vitamin A, zinc and iron during preeruptive period are reported to cause increased caries development that claimed to be related to impaired tooth tissue

Acid solubility of enamel is increased in protein energy deficiency during preeruptive period

It has been shown that feeding a diet high in sugar during pregnancy and lactation will result in changes in the offspring dental tissues namely higher level of carbonate, mucopolysaccharides in the enamel that later in life made them more susceptible to caries. Feeding diet high in protein during pregnancy and lactation resulted in offspring with lower level of carbonate; mucopolysaccharides in the enamel were found to be more resistance to caries.

Quality of saliva

Nutrition may effect the quality of saliva it has been shown that individual on lactovegetarian, high protein or high fat diet produce saliva with a high buffering capacity.

where as individual on a high carbohydrate diet produce saliva lower buffering ability.

Salivary lactoferrin, lysozyme, sialin and statherin are protein molecules that are part of the defense arsenal secreted by salivary glands. All of these can be diminished in volume or altered in structure during sever periods of malnutrition.

However marginal protein energy deficiency during pre-eruptive period; impair the condition for the development of the salivary glands, which causes a decreased secretion rate and amount of protein secreted per minutes.

Evidences of the effect of some nutrients on dental caries

- Vitamin A deficiency is known to impair enamel and dentin formation, impair immune function, reduces synthesis of specific glycoprotein such as salivary bacteria agglutinating glycoprotein (BAGP) and in cases of sever deficiency to reduce saliva secretions rate. It was reported that dental caries was increased when they fed vitamin A deficient diet

-The possible influences of the hardness and calcium content of water on prevalence of dental caries has been suggested with inverse relation. Concerning people living in primitive village who were still not exposed to European civilization, demonstrating strong inverse association between the prevalence of dental caries and concentration of calcium in soil, also found an inverse association with phosphorus composition of local food in that village.

-Iron was classified as caries inert element, deficiency of iron during pre-eruptive period of tooth development in animal caused increase caries development.

-Caries susceptibility is increased among children with chronic malnutrition (stunted means deficient height for age) and acute malnutrition (wasting means deficient weight for height), as a result of systemic effect of protein energy malnutrition during pre-eruptive period. It has also been shown that protein-energy deficiency induced after completed tooth formation but during the early

post-eruptive period, increases caries susceptibility to standardized cariogenic challenge. During the latter condition, impairment in saliva secretion rate and composition was found along with increased caries development.

Nutrition and eruption of teeth

Among children with protein energy malnutrition, protein deficiency may be the reason for delayed eruption of their deciduous teeth, on the other hand, early eruption of teeth were noted among children whose height and weight were greater than average.

Several studies have demonstrated that the rate of dental development and dental eruption affected by pre-term birth, children with the lowest birth weight and shortest gestational age (prenatal malnutrition) have lowest rate of dental development

The weaning period, which is a critical time, during which the infants diet predominantly liquid and soft food, begins to include more solid and usually going from a law protein to a high protein diet. If solid protein rich food intake (meat with 20% protein compared to with milk 5% protein) is not adequate, jaw and tooth malformation occur. As protein nutrition is a basic consideration in the growth and development of the oral cavity, if the diet include too little **essential amino acids** during critical period of active growth, permanent structural damage can occur, synthesis of protein in the cell disrupted, resulting in a disturbed tissue growth and development, this will affect the maxillary and mandibular bone, resulting in adverse effect on tooth alignment and alveolar bone integrity.

All proteins are synthesized from amino acids molecules; these can be classified into two groups: **essential amino acids** that cannot be synthesized by the body at a rate sufficient to meet the needs for growth and maintenance. It is essential that these amino acids be provided in the diet. **Nonessential amino acids** that the body can make adequate amounts from other compounds if necessary.

Disease associated with unduly delayed eruption of teeth includes rickets that is caused by vitamin D deficiency, which is essential for calcification and

growth of the jaw, and regulates the level of calcium in serum. So in many of this condition, where the level of calcium is lower than normal, found delayed tooth eruption.

From epidemiological view:

Under nutrition result in delays shedding of the primary teeth and delayed eruption of the permanent teeth, this may influence the caries prevalence at given age.

New approach in restorative dentistry

Traditional dental restoration was based on the principle created by G.V. Black in 1908, removing a lesion by operation and then restoring the damaged part. Modern material science proved that dental restoration material could not match the healthy dental tissue in terms of physical, mechanical, and biological properties. Removing healthy dental tissue and restoring the cavities with traditional filling material certainly could not meet the functional requirements.

In the twenty-first century, modern dentistry suggested a more reasonable theory, which was minimally invasive treatment; other terms were used such as minimal intervention dentistry, minimally invasive dentistry, and micro dentistry. Minimal intervention dentistry focuses on the knowledge of how caries develop, including early diagnosis, prevention, and treatment, and placed emphasis on the treatment switch from dental operation to biological method, to prevent the development of dental caries and preserve as much healthy dental tissue as possible.

They focus on the preservation of healthy dental tissue when removing caries lesions, instead of the "extend to prevent" principle of G.V. Black

In terms of the biological study of dental tissue, in addition to the etiology of caries, especially the process of remineralization, the revolution of diagnosis measurement, and the novel view of prevention, the development of dental material has laid the foundation for minimally invasive dentistry as dental caries treatment has shifted from dental surgery to biological treatment.

Four basic principles for was proposed for minimal invasive dentistry:

- lesion control
- remineralization of early caries
- minimal surgical trauma
- restoration.

Modern caries treatment pays more attention to the

- biological response of the pulp–dentin complex
- to the relationship between the restored tooth and periodontal health
- between occlusion and periodontal health
- proximal contact between the prosthetic and the adjacent teeth

Minimally Invasive Treatment Technique

Minimally Invasive Cavity Preparation

The cavity can be roughly divided into two layers from outside to inside:

1. Infected layer: this layer of the tooth structure has been completely denatured and bacteria settled.

2. Demineralized layer: this layer has a certain level of demineralization, but the collagen scaffold still exists and can be re-mineralized. In the past it was thought that the demineralized layer should be removed, but now they suggested that the demineralized layer is a precarious status instead of caries-active status, this layer can be remineralized. Therefore, the modern view is that the removal of diseased tooth structure should be limited to the infected layer(minimal surgical intervention) by new technologies including ***sandblasting caries removal, LASER, chemical–mechanical caries removal,*** and other, all these have overcome the excessive loss of the healthy tooth structure caused by traditional dental drilling.

Non-machinery Preparation

Air Abrasion The principle of air abrasion is to apply highly pressurized, nontoxic particles, such as aluminum oxide ions, to accurately remove the enamel, dentin, carious tissue, and old fillings. Air abrasion can partially replace the high-velocity gas turbine cavity preparation. It is quieter, more time- and energy-efficient, and requires no anesthesia as it does not produce vibration and heat. It is well received by patients and maximizes the conservation of the tooth structure. The interior of the prepared cavity is smooth, making it easier to fi ll. It reduces the likelihood of micro-fracturing.

The disadvantage of this method is that because it is easier to remove dentin than enamel, it causes the overhang of enamel, which requires trimming of the enamel with the drill.

Contraindications to air abrasion include patients with:

1. A severe allergy to dust, asthma, and chronic obstructive pulmonary disease.
2. Open wound or recent tooth extraction.
3. Active periodontal disease.
4. Recent placement of an orthodontic appliance
5. Subgingival caries

LASER

The ideal laser should be able to manage both dental hard and soft tissues. Clinically used lasers that can cut through dental hard tissues, all types have selective abrasive properties whilst conserving healthy tooth tissue. Laser cavity preparation is precise, non -vibrating, has no smell, and does not require anesthetics. As lasers can seal dentinal tubules, they can also prevent hypersensitivity postoperatively. On the downside, the machinery is bulky and expensive, thus limiting its role in clinical practice.

Chemo mechanical Caries Removal

Chemo mechanical caries removal (CMCR) uses chemical agents to soften the dental tissues before eliminating infected tissue. This solution causes the partial disintegration of the collagen in the cavity, accelerating the removal of dental caries,a hand tool can be used to remove the softened carious tissue. This method can selectively dissolve carious tissue quickly (around30s), whilst not affecting any healthy dentin.

CMCR can effectively remove the smear layer of the cavity, reinforce the bond between the filling and the tooth, there is no noise, vibration or anesthetics, and patient acceptance is high. However, when compared with the high-velocity turbine, the operating time is longer and requires alternative methods to gain access to and repair some undermining caries.

The CMCR method should be first considered for the following patient group: root/cervical caries, coronal caries (especially deep coronal caries), caries located on the edge of the crown or bridge abutment, completion of canal preparation, those in whom anesthetic is contraindicated, especially needle-phobic patients, those with a dental phobia, and pediatric patients.

Preventive Resin Restorations treat suspicious fissure caries and provides a new approach to the treatment of fissure caries. Preventive resin restorations only remove the infected enamel or dentin at the lesions, according to the size of the caries, using etching technology and the resin material filling up the early fissure caries, and the occlusal surface coated with sealant. It is a preventive measures combined between pits and fissure sealing and fissure caries filling.

Because it does not use the traditional extension for prevention, only amount of carious tissue is removed and restored with composite resin or glass ionomer, then the pit and fissure caries without caries is protected by the sealant, thus preserving more healthy dental tissue, and is an effective method for preventing the further development of caries.

The advantage of preventive resin restorations is using glass ionomer composite resin as filling and binding with enamel mechanically or chemically, and then bonding with sealant by chemical bonding reduces the possibility of generating micro-leakage.

Remineralization Treatment

For early enamel caries that have been demineralized, the appropriate drug treatment to remineralize, is called remineralization treatment.

Early enamel caries on the smooth surfaces (buccal, labial, lingual, palatal or proximal), such as white spots, and people susceptible to caries are suitable for remineralization therapy. There are many types of mineralized fluid, which divided into single component and complex components. The single component is mainly fluorine-containing, the complex component mainly containing different ratios of calcium, phosphate, and fluoride salts, while calcium or fluoride salt is the main ingredient

In recent years, a new remineralization agent, casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), has been used clinically. CPP-ACP has a wide range of applications in biology, including the promotion of remineralization of the tooth surface and bone calcification, promoting the absorption of minerals, and has an effect on cariogenic bacteria.

Currently, CPP-ACP is used in the treatment of early caries remineralization, dentin hypersensitivity, dental erosion treatment, and as prevention in caries-susceptible patients. The remineralizing agent with CPP-ACP as the main ingredient shows broad application prospects in caries prevention.

Sealing over caries lesions

Clinicians are often concerned regarding the inadvertent sealing of surfaces that are already carious because of the possibility that sealed lesions will remain active. However they indicated that sealants can be considered a viable modality for *arrest* of pit-and-fissure caries, many studies have shown that carious lesions that are effectively sealed do not progress for as long as 3-5 years. When sealing incipient lesions, care should be taken to monitor their retention at subsequent recall/annual dental examinations. Sealed lesions generally become arrested because the microorganisms do not remain vital within the lesion and what bacteria remain are not capable of maintaining progression of the lesion in addition, the acid-etching process alone can reduce the bacterial load

Sealants for proximal enamel surfaces

Adhesive resins can be applied for sealing early carious enamel lesions on proximal surfaces in order to arrest their progression .The proximal sealant technique is likely to be suitable for the primary dentition as the progression of enamel caries into dentine occurs relatively quickly, and the timely placement of sealants can halt progression of the lesions. However, this technique may be hampered by the requirement for separation of the teeth prior to placement of the sealants that usually involves two appointments, so it is not practical for young children. An alternative method would be to seal the teeth while it is possible to gain access to the proximal smooth surfaces before the contacts with adjacent teeth are established, another disadvantage is the need for repeated radiographs at initial and periodic examinations to check for progression of the lesions

Cariogenic potential of bacteria

Virulence of microorganism

The low level of nutrients in the oral cavity gives rise to complex pattern of competition for the available nutrients among the different microbial populations of the oral microflora. Most oral bacteria are only able to grow if the pH is within a narrow range, but some oral bacteria are aciduric and will grow at significantly low pH values. These organisms are favored when the environment on the teeth becomes acidic. Sugar is the main energy source for the micro-flora, some of micro-flora on the teeth may utilize carboxylic acids, amino acids rather than sugars to drive cellular activity. However, many organisms are able to utilize a wide variety of sugars by the induction of specific enzymes that are synthesized only in the presence of the specific sugars.

Oral bacteria protect themselves by:

- ✓ Regulating the rate of glycolysis
- ✓ The efficient conversion of pyruvate to metabolic end-products
- ✓ Synthesis of intracellular polysaccharites
- ✓ Inhibition of sugar transport by phosphotransferase system

Microorganisms have a variety of systems for the conversion of cellular end products which vary according to:

- Type of organism
- Amount and type of sugar available
- Presence of oxygen and carbon dioxide

Bacteria must possess certain caries-promoting characteristics to play a role in caries include:

- ✓ The ability rapidly to transport fermentable sugars to acid.
- ✓ The ability to maintain sugar metabolism under extreme environmental conditions, such as at a low pH. Few oral bacteria are able to tolerate acidic conditions for prolonged periods. *Mutans streptococci* and *lactobacilli* not only remain viable at a low pH, but preferentially grow and metabolize, they are both acidogenic and aciduric.
- ✓ The production of extracellular (EPS) and intracellular polysaccharides (IPS). EPS include glucans and fructans, both of which contribute to the biofilm matrix. Fructans are labile and can be metabolized by biofilm bacteria under carbohydrate- restricted conditions. IPS is glycogen-like storage compounds that can be used for energy production and converted to acid when free sugars are not available in the oral cavity. Thus, the metabolism of IPS can prolong periods over which biofilms can generate acids (formic and acetic acids). Many factors may influence the types and amount of acids formed by such a complex microflora as dental plaque.

Major dental caries-associated bacteria

Oral streptococci

The current classification of the human oral streptococci puts them into four species groups, the *mitis, mutant, salivarious,* and *sanguinosus* groups. *Streptococcus sanguis* was found to play an important role in the initiation of plaque formation while *S. mitis* was reported to a role in gingivitis and periodontitis as well as it was isolated from carious dentine and infected root canal. *Mutant Streptococci* (MS) are gram-positive facultative coccus commonly found in the oral cavity. Species of streptococci may vary in their cariogenic determinant,

streptococcus mutans were found to be the predominant bacteria in caries process.

S. mutans commonly arranged in chains, subspecies are: S. Rattus, S. Sobrinus, S. Cricetus, S. Ferus.

Certain physiological characteristics of the *Streptococcus mutans* :

- The ability to adhere to tooth surfaces by either of two mechanisms:

(1) attachment to the acquired pellicle through extracellular proteins (adhesins) located on the fimbriae (fuzzy coat) of these organisms; and (2) sucrose-dependent mechanisms, in which bacteria require the presence of sucrose to produce sticky extracellular polysaccharides (glucans), that allows attachment and accumulation of additional waves of bacterial colonization.

- Rapid production of lactic acid from a number of sugar substrates,

- The production of intracellular polysaccharide (energy) stores.

As a general rule, the cariogenic bacteria metabolize sugars to produce the energy required for their growth and reproduction. The by-products of this metabolism are acids, which are released into the plaque fluid. The damage caused by oral streptococci is mainly caused by lactic acid, although other acids, such as butyric and propionic, are present within the plaque. A positive correlation is found between the counts of these bacteria in saliva and plaque.

Lactobacilli

Lactobacilli (LB) comprise a diverse collection of gram-positive bacilli. They are aero tolerant or anaerobic bacteria. It was thought previously that LB play

a major role in the carious process. Later, it was found that LB are more a consequence than a cause of caries initiation During the initial phases of the developing carious lesions, large numbers of MS are involved, only to decrease later in number as the LB population increases. *Lactobacillus* is acid tolerant (aciduric) and can carry out glycolysis at pH values as low as 3. However, lactobacilli are poor colonizer of smooth tooth surface. Lactobacilli are generally believed to exacerbate the initial enamel lesion to deep dentine lesion. A positive correlation is found between the counts of these bacteria in saliva, plaque and caries activity. Also, these bacteria have ability to produce both extracellular and intracellular polysaccharides.

Actinomyces

Actinomyces are a genus of gram-positive anaerobic pleomorphic rod-shaped bacteria. They have been frequently isolated from both root caries lesions and sound root surface, suggesting their association with root caries. Results of several studies documented that MS and LB were found in root caries lesions. However, the knowledge about the involvement of individual *Actinomyces* species in caries initiation and progression is not well-understood.

Veillonella

They are gram-negative anaerobic cocci appears as plaque oxygen levels fall, are unable to metabolize dietary carbohydrates but they are able to use lactate that is produced by other microorganisms and convert it to a less cariogenic and weaker acid. This may consider a beneficial effect of these bacteria in relation to dental caries.

Plaque hypothesis

The oral cavity is inhabited by hundreds of bacterial species, forming complex ecology system. The “specific pathogen hypothesis” has led to the identification of a single or very small numbers of species were actively involved in disease. In contrast, the “nonspecific plaque hypothesis” supports the concept that caries is the consequence of the overall acid production activity of the total plaque microflora rather than a few specific bacteria but also species that produce alkali or consume lactate need to be considered. An alternative hypothesis has been proposed the ecological plaque hypothesis suggested that the organisms with the disease may also present at sound sites but at a low level.

The converse situation is also not uncommon, where mutans streptococci are found in high numbers in plaque but in the apparent absence of any demineralization of the underlying enamel. This may be due to:

- The structure of the biofilm and the localization of mutans streptococci in plaque

- The presence of lactate-consuming species (e.g. Veillonella)

- The production of alkali to raise the local pH (by ammonia production from urea or arginine by S. salivarius and S. sanguinis, respectively).

Species interactions and caries

The viability of oral microflora is dependent not only on the host genetic and environmental factors but also on interactions between microbial residents. The residents interact extensively forming biofilms structures, carrying out physiological functions and inducing microbial pathogenesis.

Microbial interactions include:

- ✓ Competition between bacteria for nutrients
- ✓ Synergistic interactions for growth and survival

- ✓ Antagonistic interactions by secondary metabolites production
- ✓ Neutralization of a virulence factor that produced by another resident
- ✓ Interference in the growth-dependent signaling mechanisms of each other.

A dynamic balance of both synergistic and antagonistic interactions with the residents plays an essential role in determining whether these pathogenic factors cause damage or not. Hence, dental caries is the net result of the interaction of multiple acidogenic/aciduric bacteria with others within dental plaque.

Bacterial colonization can be controlled by:

- Antimicrobial approaches (fluoride, chlorhexidine and others).

- Mechanical approaches (teeth brushing and others).

- Dietary assessment (use of non-cariogenic sweeteners as xylitol, avoid frequent consumption of carbohydrates between meals).

- Probiotics are live microorganisms which, when applied in adequate amounts, will benefit the health of the host.

- Salivary antimicrobial substances.

Nutrition, diet & periodontal disease

Like caries, periodontal disease is an infectious disease, multifactorial in etiology, and occurs when virulence of the bacterial challenge is greater than the host defense and repair capability. Unlike the direct causative relationship between carbohydrates and caries, nutritional factors seem to play a much more subtle role in periodontal status.. Even when the periodontium is healthy, there is continual need for nutrients to maintain the tissues. Once inflammation is established, the need for nutrients increases.

It is generally acknowledged that gingivitis and periodontitis are the result of accumulation of supra and sub gingival plaque, calculus, or both. However the extent and the intensity of the gingival inflammatory process are

Directly affected by both the virulence of dental plaque bacteria around the supra and sub gingival margin of the teeth and **Indirectly** affected systemically by the relative innate of the periodontal tissue to infection.

The periodontal tissue is composed of epithelium, collagen fiber, blood vessels, cementum and bone so nutritional deficiency will affect adversely these tissues Nutritional deprivation affect rate and degree of periodontal disease rather than its initiation as nutritional deficiency apparently **do not initiate periodontal disease but may modify the severity and extend of the lesion** Over all nutritional deficiency affect the severity and extend of periodontal disease by modulating the responses and repair properties of the tissue

Food and nutrition can affect periodontal disease by

- contributing to the microbial in the gingival crevice
- affecting the immunological to bacterial antigens
- assessing the repair of the connective tissue at the local site after injury from plaque , calculus and so forth

The mechanisms by which nutrition may affect periodontal disease include the following:

Antimicrobial action: many nutrients have antimicrobial activity these may alter the quantity and/ or quality of dental plaque and thus be associated with a reduction in gingival inflammation.

Anti- inflammatory effect: nutrient that decrease the host response to injury may result in a reduction in the severity of gingivitis and /or development and progression of periodontitis. These work by affecting the enzymes involved in the production of the anti-inflammatory compounds or by altering which compounds are actually produced.

Immune system modification: (affecting the immunological response to bacterial antigens). Some nutrients are thought to act as immune system modifiers in that they optimize the host immune response so that the protective immune reaction outweigh the self destructive ones, this could also be accomplished by alteration of the permeability of the gingival epithelium thus changing host resistance to bacterial product.

Antioxidant effect: Antioxidants: are substances that protect other chemicals of the body from damaging by reacting with oxidizing agents within the body.

The oxidizing agents either are produced within the body as a part of its normal metabolic process or enter the body from atmosphere. For example, free radicals (highly reactive molecules carrying unpaired electron):

@ are producing during the normal oxidation of the energy yielding nutrients in the cell.

@ are produced by the presence in the body the various environmental pollutants (such as cigarette smoke).

When free radicals attack e.g. the lipids of the cell membranes, they can initiate a highly damaging chain reaction leading to widespread damage to the structure.

The intake and serum level of antioxidant nutrients have been associated with reduced risk of many diseased state. Although there is no evidence to suggest that increased intake of these nutrient are associated with a decreased risk of periodontal disease. It is known that several antioxidant nutrient and enzymes are present in the crevicular fluid and in the oral epithelium and secretion

considering the responses elicited by the host against pathogenic oral bacteria. Some of the nutrient that influences an individual oxidative status includes vitamin C, vitamin E, zinc, copper, manganese, and selenium. These antioxidant compounds are essential for helping to maintain cell integrity.

Effect of food texture on periodontal health:

It has been assumed that firm fibrous food may be beneficial to periodontal health and that can and that eating soft, sticky food might tend to have an adverse effect. These basic tents still seems to have some validity but perhaps for different reasons from those originally suggested

The following conclusions can be drowning from currently available evidence about the local effect of physical consistency of food on periodontal health:

• Fibrous food does not remove plaque at the gingival level of the tooth. Chewing on fibrous or firm food stimulate salivary flow and can therefore aid the oral clearance of food debris

• Chewing fibrous or firm food dose not increase gingival keratinization but it dose produce a type of local exercise that can stimulate and strengthen the periodontal ligaments and perhaps may also increase the density of alveolar bone adjacent to the root

• Another important positive effect of including fibrous food in the diet is that this food can replace empty calorie, sugar rich sweet that are retained in the mouth and may provide a substrate for increased formation of supragingival plaque bacteria

Nutrition and oral mucosal disease

Nutritional deficiencies can impair oral mucosal health and oral immune defense, and component of some diet may be harmful to the mucosa. Conversely, oral disease can interfere with feeding and nutrition as a consequence of compromised mastication and swallowing, pain, or discomfort. Protein-energy deficiency may in children vary in its effect from mild growth retardation to marasmus and kwashiorkor (sever protein malnutrition). Protein malnutrition decrease collagen synthesis in oral mucosa and oral lesion has been described in kwashiorkor; these include edema of the tongue and

papillary atrophy, angular stomatitis, hypo-pigmentation circumorally and Xerostomia. Interestingly, tolerance of dentures appears to be increased if the dietary protein intake is improved in edentulous patients.

Common oral mucosal manifestations of nutritional defeciciences

Candidiasis **Oral ulceration**

Glossitis **Angular stomatitis**

Burning Mouth syndrome **Gingival bleeding**

Post extraction haemorrhage

Nutrition and oral cancer

Oral cancer is largely a preventable disease, dietary factor seems to be important in the prevention of oral cancer. this has been shown in hundred of recent studies. Significant trend of increase risk with more frequent intake of meat and processed meat while significant inverse trend in risk were observed with more frequent intake of fruit and vegetables.

Prevention of oral cancer exerted as:

• **Primary prevention** focused on elimination of risk factor and inhibition of tumor initiation and activation.

• **Secondary prevention,** focused on inhibition of tumor promotion and progression.

Tumor initiation or activation commences when e.g. the DNA of a cell or a population of cells is damaged by exposure to carcinogenic elements, whether endogenous exogenous if this damaged remain unrepaired then mutation may occur, the sensitivity of the mutated cell to their microenvironment changes and more rapid growth takes place than non- affected cells.

Carcinogenic agents may be of two types:

1-Exogenous agents

Physical: ultraviolet rays gamma rays

Biological: viruses

Chemical: e.g. nitrosamines, which a class of carcinogenic amine that are form from nitrate and nitrites in food, either during drying and cooking or when the food is in the gastrointestinal tract, also found in cigarette smoke. This nitrosamine is known carcinogens that may be responsible for some cancer. Nitrites are also used as food additives to preserve the color of meat, inhibit oxidation, and discourage the growth of microorganism in meat.

2-Endogenous (normal products of oxidative metabolism that can cause damage to DNA and covert normal cell to cancer cell).

Oxygen is essential for sustaining life, but it could be harmful. Oxygen itself is not the problem, but once it transform into a **free radicals,** it assume a destructive powers. Free radicals are **unstable form of oxygen** they have lost an electron from their molecular structure, (normally these electron exist in pairs). To replace the lost electron free radicals actively seek out electron from other substances in the body. When these materials give up an electron to the free radicals their structure, become damaged.

Among favorite targets of free radicals are cell proteins, enzymes the fatty acids in cell membrane and the genetic material DNA. Damage to these structures can trigger the development of cancer. **A force that acts on oxygen to create free radicals are called oxidative stresses,** some of these stresses arise as a normal part of cell reaction. Fortunately, the bodies are armed with (for example) antioxidants, these substances can neutralize free radicals, and include:

• vitamins: Vitamin C, E, carotenoids, beta-carotene (provitamin A)

• Minerals: selenium, manganese, and zinc.

Antioxidants tackle free radicals by using a variety of tactics:

• Giving the free radicals one of its own electron, the antioxidants spares the cell material from damage, antioxidants that work this way are called **free radicals scavengers.** Vitamin C, beta carotene, and vitamin E work as a scavengers

these antioxidants reducing agents is able to convert these oxidizing agents to harmless substances that can be excreted so scavenge many type of oxidizing free radicals

The various antioxidants cooperate with one another to achieve their goals of protection against free radicals damage. They require this team effort because antioxidants exist in different places in the cell and attack different free radicals. Example vitamin C regenerates the reduced form of vitamin E by giving it another electron, once vitamin E losses its electron to a free radicals so converting this vitamin back in to the form in which it act as antioxidants.

• Other way antioxidants may protect against cancer by preventing chemicals from being transformed into cancer-causing substances or carcinogens in the first place. For example, vitamin C can stop the transformation of nitrates -into powerful carcinogens called nitrosamine but nitrates discourage the growth of microorganism in meats and therefore perform an important function so instead of eliminating nitrates they added vitamin C to these food to prevent their transformation into carcinogenic nitrosamine.

It has been found from epidemiological studies that protection against cancer was found among individuals who ate relatively large amount of fresh fruit and vegetables rich in vitamin C. It appears best increase intake of vitamins from food source rather than from large doses of vitamin tablets because benefit can be derived from the cellulose, and from other vitamins, minerals naturally present in the food.

Inhibition of tumor promotion and progression

Certain antioxidants such as selenium , ascorbic acids and some poly-phenolic compounds found in green tea , fruit and vegetables have been shown to be effective in inhibition of tumor promotion.

Vitamin A : large number of retinoid are inhibitors of substances specific to tumor promotion in the early stage of vitamin A deficiency, change that resemble the early stages of cancer occur in cells.)

The retinoid are <u>powerful antioxidants,</u> protecting cellular lipids from oxidation. In addition, vitamin <u>A direct cells to produce new cells that are</u>

identical reproduction of the original, restore normal cell and differentiation (tumor form when new cell that are different from the original cell, begin to reproduce) so they have been used in treatment of cancer, with some success.

Vitamin A also boosting immune function, enhances cell mediated immunity and induce a mononuclear infiltrate in the tumor suggesting that immune modulation may be a protective mechanism a against the tumor

Retinoid are chemoprevention agent for oral pre-malignant lesion, they have significant effect but wide spread use is limited by significant clinical toxicity.

Soybeans

It has been suggested that premalignant tumor tissue have elevated level of proteolytic activities that can be used as biomarker for human cancer prevention studies. The Bowman- Birk inhibitor is a soybean derived serine protease inhibitor and a potential chemo-preventive agent for human (potent anti-carcinogenic agent). Interest in use of soy beans products as a cancer preventive agent emanated from epidemiological studies demonstrating low incidence rate of several cancer in population with high soy intake. In Japan, which has a high dietary intake of soy product, the incidence rate of several of cancer is very low.