Fluoride toxicity Lec -6-

By Assist. Prof. Azhar Alkamal

The fluoridation of public water supplies represents one of the most successful public health measures ever undertaken and the widespread use of F is reasonable for part of the remarkable decline in prevalence of dental caries. Fluoride is also a toxic substance when ingested in overdoses. The toxic effect of F overdose can be classified as:

1- **Acute toxicity**: - it is rapid excessive ingestion of a relatively high dose of F at one time.

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

I- Acute toxicity of fluoride: -

It is rapid excessive ingestion of a relatively high dose of F at one time that may be followed by signs & symptoms which may result in death. Because there are several variables that can affect the outcome at acute F poisoning, it is not surprising that the fatal dose is uncertain, for ex: - the action of compound may exert a toxic effect; thus, SnF_2 is slightly more toxic than NaF, because high doses of tin ion adversely affect kidney & other organs.

Certainly lethal dose (CLD): -

It is the dose that is going to cause death.

CLD = (5-10) g f/kg body weight (adult 70 kg).

Probably toxic dose (PTD): -

It is the threshold dose that should trigger immediate emergency treatment. **PTD = 5 mg f/kg** body weight (child).

Factors affecting the severity of acute toxicity of fluoride: -

1- The amount of F ingested.

2- Fluoride bioavailability (absorb ability), therefore there is differences in the toxic potential of different compound which is related to the solubility of these compounds.
3- Route of administration.
4- Age & weight of the individual (acute F toxicity occurs more rapid in younger & thinner individuals).
5- Acid-base status (pH of the administrated solution or the rate of gastric acid secretion).

Signs & symptoms of acute toxicity of fluoride: -

1- **GIT:** - in nearly all cases of F poising, the victim experience nausea, vomiting & abdominal pain within minutes after ingestion. There may or may not be a variety of nonspecific symptoms such as excessive salivation, tearing, mucous discharge from nose & mouth and diarrhea. In sever case spasm of the extremities & tetany often developed.
2- **CVS:** - weak pulse, hypotension, cardiac arrhythmias may develop in association with the hypocalcemia & hyperkalemia. Different cardiac irregularities that may end with heart failure.
3- **Neurological: -** paresthesia, tetany, CNS depression & coma. Fluoride binds to calcium, which is needed for nerve action, so drop in blood level of calcium leading to tetany.
4- **Blood chemistry:** - acidosis, hypocalcemia & hypomagnesemia & hyperkalemia will develop.

The series symptoms develop within (1-2) hours after ingestion. Death occurs in the first (2-4) hours after ingestion. If death has not occurred after 24 h, the prognosis for recovery is good.

Emergency treatment: - depending on the dose ingested.

1- **If less than 5 mg f/kg body weight**.
   a- Gives Ca^{+} orally (milk) to relieve GI symptoms & observe for few hours.
   b- Induce vomiting, if necessary, by stimulating the soft palate by mirror or spatula.
2- **More than 5 mg f/kg body weight**.

a- Empty stomach by inducing vomiting or by endotracheal intubation.
b- Given orally soluble calcium in any form (milk, 5% calcium gluconate or calcium lactate solution).
c- Admit to hospital.

3- **More than 15 mg f/kg body weight**.
a- Admit to hospital immediately.
b- Gastric lavage.
c- Cardiac monitoring & be prepared for cardiac arrhythmia.
d- IV administration of 10 ml of 10% calcium gluconate solution, because calcium reacts with F ion reducing the bioavailability of F (amount absorbed & distributed in the body).
e- Monitoring electrolytes.
f- Adequate urine output using diuretics if necessary.
g- General supportive measures for shock.

To avoid fluoride toxicity: -

1- The patient should be seated in an upright position or with the head inclined slightly for word with use of saliva ejector.
2- Using small amounts of professionally applied F not exceeding 4 ml &it is preferable to use a tray with an absorbent foam lining.
3- The buccal vestibule & sublingual space should be evacuated by suction during & after the application procedure.
4- The patient requires expectorating multiple times after the procedure.
5- For self-applied topical F parents' supervision is required.
6- ADA reported that for each tablet, the total amount should not exceed 264 mg f.

II-Chronic toxicity of fluoride

It is long term ingesting of a relatively small amount of F. it may produce dental fluorosis, when somewhat larger amounts are ingested over a period of year, changes in the quality & quantity of skeleton may occur. This, in fact, is the bas is of the use of F for the treatment of osteoporosis. The skeletal changed may become sever enough to be classified as crippling skeletal fluorosis.

The effect of chronic fluoride toxicity: -

1- **Dental fluorosis:** - it is the first clinical sign of a toxic effect of F characterized by opaque, white patches in the enamel which may become molted, stained, & or pitted.
2- **Skeletal fluorosis:** - it is the general name describing any changes in the bone due to excessive F intake. Skeletal fluorosis affects children as well as adults. There will be variety of skeletal changes as: periosteal apposition, increase density of trabecular bone, crippling exostoses, osteomalacia, and increased amounts of woven bone altered remodeling &calcification of ligaments & tendons. i.e., skeletal fluorosis affects the bones/skeleton of the body. 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 like spondylitis or arthritis. The change produce appears to differ depending on factors such as dose of F, duration of exposure, age of the patients & supply of calcium and vit D (malnutrition). Fluoride increases the prevalence of rickets for children from high F area. In some cases, these will be outward bending of legs, hands in which these parts lose their shape & contours & such condition called **Knock-Knee** syndrome.

The left panel indicates the stages of tooth permanent tooth development in the human dentition. The right panel shows the pattern of fluorosis that one can expect with the exposure periods indicated on the right (white areas = fluorosis). Note that fluorosis can still occur after the age of 5 years, but only the posterior teeth, which develop later than the incisors, are affected.

3- **Kidney changes:** - over 2 mg F/L in drinking water causes renal damage. Fluoride conc in urine & serum increase gradually with increased F level in drinking water, so individual with kidney diseases have decreased ability to excrete F in urine & are at risk of developing fluorosis even at normal recommended limit at (0.7-1.2) mg/l
4- **Thyroid gland changes:** - fluoride affects the calcium homeostasis. Effects on calcium regulating mechanisms have been

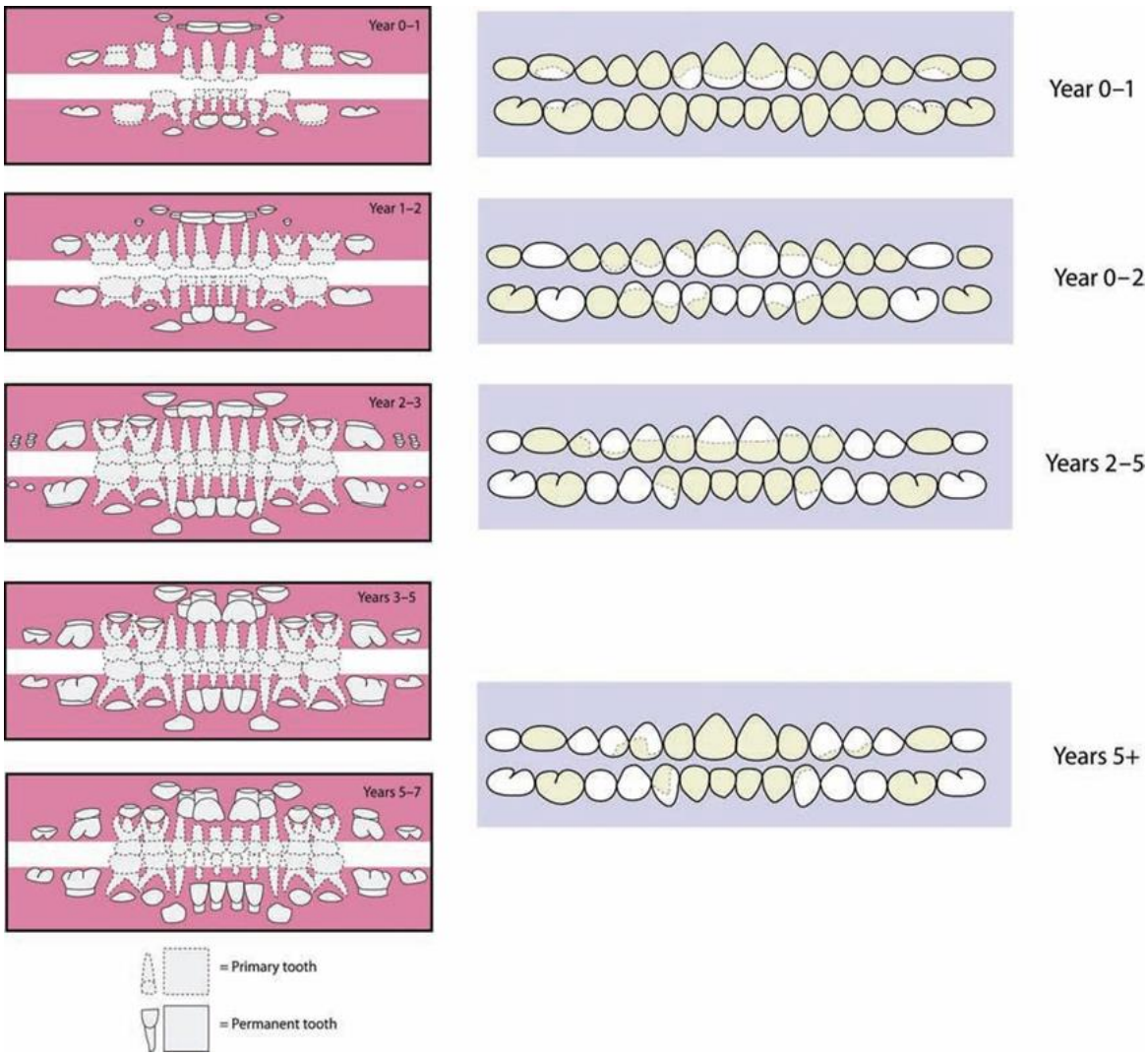
described in few reports from areas with endemic skeletal fluorosis have indicated a change in parathyroid gland activity, that are secondary in nature (F cause iodine deficiency, suppress the thyroid function and cause hypothyroidism).

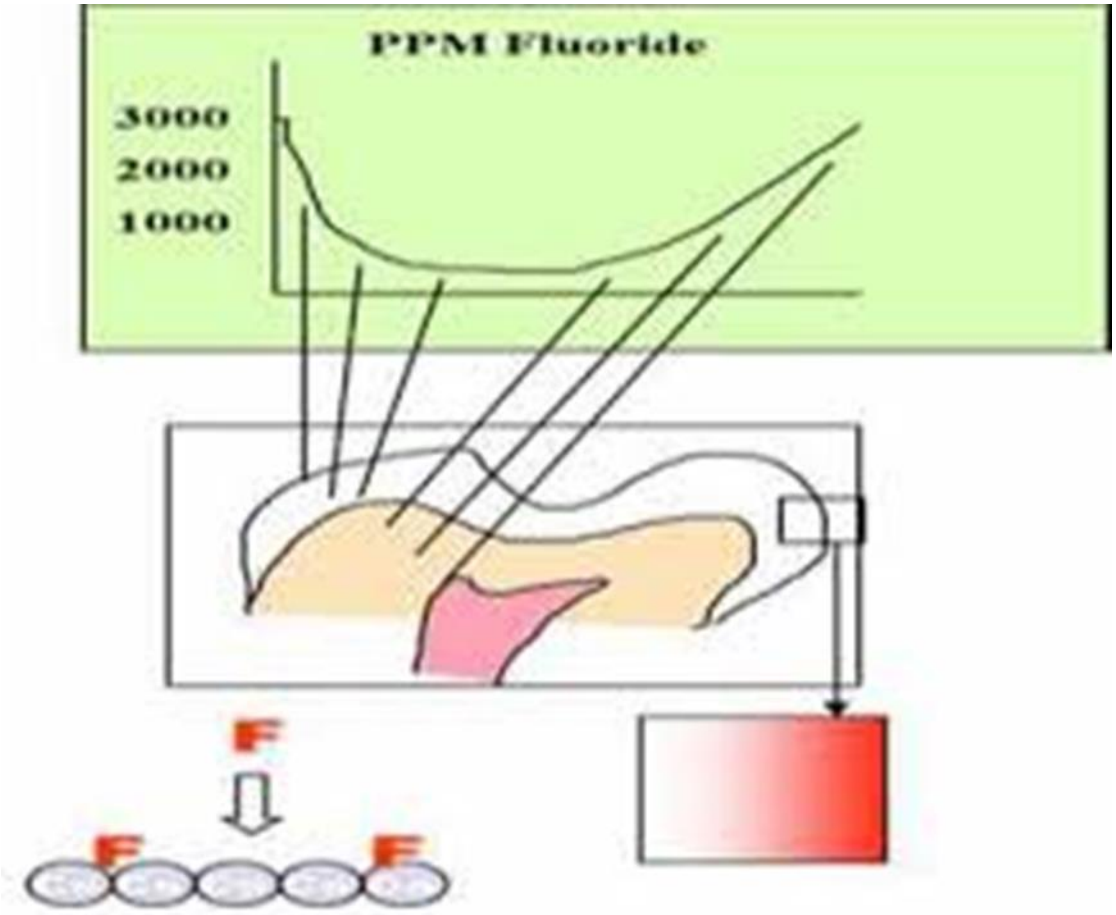
Effect of fluoride through various media Smith & Hodge 1959

Conc or dose of F	medium	effect
1ppm	air	dental caries reduction
2ppm	water	mottled enamel
5ppm	urine	no osteosclerosis
8ppm	water	10% osteosclerosis
20-80mg / day	water or air	crippling fluorosis
50ppm	food or water	thyroid changes
100ppm	food or water	growth retardation
More than 125ppm	food or water	kidney changes
2.5-5 g f	acute dose	death

Toxic effect of chronic excessive fluoride ingesting

Effect	dosage	duration
Dental fluorosis	more than 2 times optimal	until 5 years old
Skeletal fluorosis	10-20 mg/day	10-20 years
Kidney damage (animals)	5-10	6-12 months


Year 0–1
Year 1–2
Year 2–3
Years 3–5
Years 5–7
= Primary tooth
= Permanent tooth
Year 0–1
Year 0–2
Years 2–5
Years 5+


PPM Fluoride
3000
2000
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F

New approach in restorative dentistry

By Assist. Prof Azhar Alkamal

The 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 (MID)** is the modern medical approach to the management of caries, utilizing caries risk assessment and focusing on the knowledge of how caries develops. MID include 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 and it is applied to other areas of oral health such as periodontology, oral rehabilitation, and oral surgery.

The restorative procedures based on the G.V. Black concept; does not keep teeth functional for life for all individual. While the focus of MID is to **reduce the need for cutting** away healthy tooth tissues, this has led to the smaller and less destructive cavity preparations and therefore, smaller restoration. **MID defined as a philosophy of professional care concerned with the first occurrence, earliest detection, and earliest possible cure of disease on micro (molecular) levels, followed by minimally invasive and patient-friendly treatment to repair irreversible damage caused by such disease.**

The objective of MID is tissue preservation, thus performing treatment with as little tissue loss as possible and treated the dental caries as an infectious condition. The extension for prevention practice has changed to constriction with conviction. By early 1990, research had shown that managing dental carious lesions should differ from the traditional surgical approach and move to a

biological or medical approach. The aim of the MID is to keep teeth healthy and functional for life.

Four basic principles for was proposed for minimally invasive dentistry: -

1. Lesion control (early caries detection and risk assessment).
2. Remineralization of early caries
3. Minimal surgical trauma
4. Repair rather than replacement of 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

Re-mineralization of early carious lesion: -

Re-mineralization (regeneration) aim to arrest and reverse incipient lesions, using appropriate topical agents. This procedure is indicated in presence of early dental caries as white spots, initial root caries, and areas susceptible to dental caries. In addition, it is indicated for patients with high risk and increasingly susceptibility to dental caries. Re-mineralizing agents can be applied also in the management of hypersensitivity and cases of tooth wear as dental erosion.

Agents applied include: -

1- fluorides agent.
2- Casein phosphopeptides-amorphous calcium phosphates (CPP-ACP).
3- Combination of CPP-ACP and fluoride.
4- Other materials as, silver diamine fluoride (SDF), light-activated fluoride (LAF), amorphous calcium phosphate (ACP), Dicalcium phosphate dehydrate (DCPD).
   Note: - CPP-ACP may promote re-mineralization of teeth and bone calcification and influences cariogenic bacteria.

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 re-mineralized. 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 others, all these have overcome the excessive loss of the healthy tooth structure caused by traditional dental drilling.

Non-machinery Preparation

1-Air Abrasion The principle of air abrasion is to apply highly pressurized, nontoxic particles carried with the steam of air, 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 fill. It reduces the likelihood of micro-fracturing

The disadvantage: -

1- Total loss of tactile sensation.
2- Ability of alumina particles to remove sound tooth structure rather than the caries substrate.
3- Potential risk of inhalation problem.
4- Increase the risk of air embolism in the soft tissue.
5- Easier removal of dentine than enamel which may cause overhang of enamel and may require 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

2- 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.

3- Ozone therapy: -

Causes re-mineralization of the incipient pit and fissure caries as well as incipient root caries. Its usefulness in open lesions has also been demonstrated. Ozone readily penetrates through decayed tissue, eliminating cariogenic bacteria. The re-mineralization process will then take place with the aid of a topically applied re-mineralizing solution.

4- 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.

Various agents with their methods have been used, as ***carisolv*** which is the most successful and commonly used agent in CMCR. It consists of two-component mixtures (mainly amino acid and hypochlorite), forming an active gel.

Indication of the CMCR for patients: -

1- 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.
2- those in whom anesthetic is contraindicated.
3- needle-phobic patients, those with a dental phobia.
4- pediatric patients and elderly 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 caries, using etching technology and the resin material filling up the early fissure caries, and the occlusal surface coated with the sealant. It is a preventive measure combined between pits and fissure sealing and fissure caries filling.

Because it does not use the traditional extension for prevention, the 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.

Pit and fissure sealant

By Assist. prof. Azhar Alkamal

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 agents to the extent they remain bound 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".

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

Prevalence

In the US, 42% of children aged 6–11 and 48% of adolescents aged 12–19 had fissure sealants on permanent teeth during 2011–2016.

In other European countries, such as In Denmark, 66% of 15-year-old children had at least one sealed molar. In the UK in 2003, 13% of 8-year-old, 25% of 12-year-old and 30% of 15-year-old had at least one fissure sealant. Around 25% of Japanese children have at least one sealed molar.

A study surveying fissure sealants and dental caries in primary school girls in Saudi Arabia in 2017 found that only 1.3% of the children had at least 1 fissure sealant applied.

Types of pit and fissure sealants according to:

1- **Generations.**
   a- First generation sealants which are activated by ultra-violet light, no longer 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 are activated by visible light e.g., Fissurit (Voco) Delton (Johnson & Johnson).
   d- Fluoride containing sealants which have double protection.
2- **Fillers**.
   a- Free filler which flows better.
   b- Semi filled 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.

In 1955 Buonocore wrote about the technique of acid etching as 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 benzoin ethers to activate the peroxide-curing system. The visible light-curing sealants have diketones and aromatic ketones, which are sensitive to visible light in the wavelength region of 470 nm (blue-light). Some sealants contain filler, usually silicon dioxide micro fill or even quartz.

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 resin.
2- Urethane resins.
3- Bis-GMA resins
4- Glass ionomer cements.
5- Resin-modified glass ionomer cement.

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 a prophylaxis cup and pumice.
2- *Isolate and dry the tooth surface: -*
   Rubber dams provide the best isolation of the tooth from salivary contamination.
3- *Etch the tooth surface: -*
   The tooth should be etched with 37% orthophosphoric acid for 15-30 seconds. The etchant should be applied to all the pits and fissures at least a few millimeters beyond the final margin of the sealant (at least 2mm). Do not allow the etchant to contact with soft tissue. If this occurs, rinse the soft tissue thoroughly.
   Not: - if by any chance an etched surface is not covered by a sealant, or poor sealant retention ever present; the normal appearance of enamel returns to normal within one hour to a few weeks. Tooth will re-mineralize again from salivary constituents.

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-etch 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 to prevent defects. After the sealant has set, the operator should wipe the sealant surface with a wet cotton pellet. This allows 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 with articulating paper and check interproximal contact with floss, and if present correct them.

10- *Retention and periodic maintenance: -*
It is necessary to re-evaluate the sealant at recall visits for retention in the first 3 months, then every 6 months. 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 cement. This procedure should be regarded as 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, remove all stain, deposit, and debris. The slurry to use non-fluoridated pumice.

3- Appropriate etching and drying time (acid etch increases the surface area thus increases the adhesive potential). Traditional retention of a sealant on tooth surface is through acid etching.
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 pit 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 to 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 because 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: according to different generations of sealant and adhesive system.
2- Position of teeth in the mouth. Retention of sealant on first molars is better than second molars.
3- Clinical skill of the operator.
4- Age of the child: affecting the behavior of the child in the clinic thus proper procedure as dryness of teeth.
5- Eruption status of the teeth: sealing molars at an early stage provide good protection, sealant is retained better recently erupted teeth than in teeth with more mature surfaces, 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 into the fissure.

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

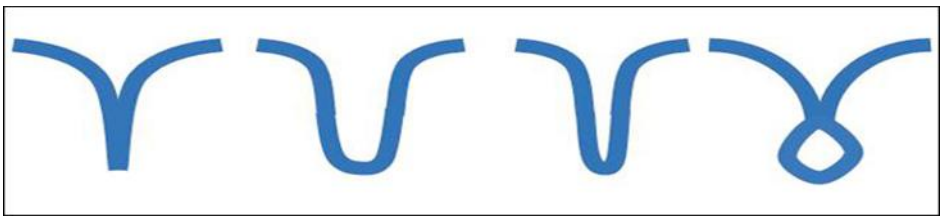
Fluoride containing sealants.

The addition of fluoride to sealants has been 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 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.



Preventive measures for elderly population

By Assist. Prof. Azhar AL-Kamal

The number of elderly populations increased in the last few decades because of improved health services programs in general and oral health. The science which deals with old age refers to **Geriatric dentistry** which is a science deal with the diagnosis, management, and prevention of all types of oral diseases in the elderly population.

The elderly population can be divided into the following categories: -

1- People aged (65-74) years are young elderly who tend to be relatively healthy and active.
2- People aged (75- 84) years are the old or mid-old, who vary from those being health and active to those managing an array of chronic diseases.
3- People 85 years and older are the oldest old, who tend to be physically frailer.

Physiological age changes

A- General body changes

1- Impairment of tissue repair.
2- Loss of muscle mass and strength.
3- A reduction in a metabolic rate.
4- Reduction in cellular reproduction which delay the repair process.
5- Reduction in the blood circulation.
6- Increase in fibrosis.
7- Degeneration of the elastic and nervous tissue.
8- A loss of cartilage and bone.

B- Oral changes

a- Oral soft tissue

1- Decrease in taste bud function.
2- Decrease in the thickness of epithelium and mucosa.
3- Decrease in saliva flow and changes in its composition.
4- Increase in the size and number of Fordyce spots (enlarged ectopic sebaceous gland in the mucosa of the mouth, lips, cheek and tongue), lingual varices and foliate (leaf-like) papillae.

b- Dental hard tissues

1- Tooth wear is a natural age-related factor.
2- Enamel is less permeable.
3- Cementum undergoes continuous deposition with age.
4- The volume of secondary dentine increase.
5- There is reduction in cellularity in the alveolar bone and the surface in contact with the periodontal ligament appears jagged.

c- Dental pulp

1- Increase in fibrosis and decrease in vascularity.
2- Increase in pulp calcification and the pulp stone are more evident.
3- The volume of pulp decreases with age, owing to the deposition of secondary dentine.

d- periodontium

1- An increase in fibrosis.
2- A decrease in cellularity, vascularity and cell turnover is found with an increase in age.
3- Collagen and protein synthesis decrease.

Pathological changes

A- Systemic
B- Oral
C- Dental condition

A- Systemic

1- Endocrine disorders
   a- Hypo function of the adrenal glands (Addison's disease).
   b- Hyper function of the adrenal glands (Cushing's syndrome).
   c- Diabetes mellitus which accelerates the periodontal diseases, high risk of fungal infection, decreased salivary flow and increase caries if uncontrolled.
   d- Hyper parathyroidism: - PTH which responsible for calcium homeostasis, in case of tumor it will results in demineralization of the bone, causing possible bone fractures, and renal calculi may develop because of excretion of high levels of phosphate and calcium.

e- Hypothyroidism will have symptoms associated with a slow metabolism and the patients appear lethargic, have cold dry skin, and may have enlarged tongue.

2- The immune system which may be impaired due to immunosuppressive drugs or malignancy. Those patients are at high risk for fungal and viral infection and oral ulceration.
3- Cardiovascular disorders like hypertension, ischemic heart disease, anemia, and others which more common in elderly.
4- Muscular system like muscular dystrophic diseases which associated with decrease in bulk of muscle, slower contraction, and less precision of control.
5- Neurological disorders like Parkinson's disease (tremor of the hand, drooling due to swallowing difficulties, postural instability, and speech difficulties), Alzheimer's disease (dementia due to wasting of nerve fibers in the brain).
6- Psychiatric disorders like depression; patient may have xerostomia due to drug therapy and may exhibit signs of oral neglect.

Preventive and treatment program for elderly patient with systemic disease.

1- The approach to an elderly patient should be with respect because elderly patient has a lack of self-motivation and low self-esteem.
2- Elderly patient usually requires extensive oral hygiene instruction and supervision with plenty of positive encouragement.
3- Avoidance of waiting for long time before the patient enters to the dentist.
4- Appointments are best kept short.
5- Elderly patient may have difficulty in swallowing and may tolerate treatment better in an upright position; they may have to be treated in their own wheelchair or a domiciliary setting.
6- Patients on steroid therapy are susceptible to a steroid crisis and their physician may advice an increase in dosage when undergoing stressful dental treatment.
7- Dental treatment for the patient with Parkinson's disease can be improved by the provision of moldable head support and mouth

probe and treatment can usually be more successful if undertaken within 2h of taking anti-parkinsonian medication.

B- Oral pathological changes

1- Leucoplakia

- This present as a white patch on the oral mucous membranes which cannot removed by scraping.
- It most commonly occurs between ages of 40-70 years old and more common in males about 65%.
- It usually located around the ducal gingival tissue or the floor of the mouth.
- It can be associated with tobacco, alcohol or chronic persistent irritation, such as ill-fitting dentures.
- It requires investigation since these lesions can be pre-cancerous until proved otherwise.

2- Oral cancer

- Oral cancer is relatively uncommon condition it does increase in occurrence in older adults.
- The most common sites are the lips followed by the tongue.
- Approximately 90% of oral carcinomas are squamous cell carcinomas.

3- Candida infection (candidiasis).

- Acute candidacies or thrush: - is most common in the young, elderly, and immune suppressed patients. It presents as a creamy white slough which can be gently removed to reveal a raw red mucosa, usually on the palate, oropharynx, or cheek.
- Chronic Candida (candidiasis) or denture stomatitis is usually symptomless. It is commonly seen on the palate underneath a full or partial upper denture, as a reddish area with some white patches.

4- Lichen planus

The intraoral presentation can be bilateral and/ or symmetrical white patches affecting the buccal mucosa, tongue and attached gingival.

5- **Herpes zoster**: its acute self-limiting viral disease and its due to reactivation of varicella-zoster virus. More common with the increase in age.

6- **Pemphigus** it is an autoimmune chronic skin disease.

C- **Dental condition**

1- Periodontal disease

Several early studies found a close association between age, periodontal disease, and tooth loss. However, more recent research has questioned the association between age and periodontitis. With age, some gingival shrinkage and loss of periodontal attachment and bony support are expected, but age alone in a healthy adult does not lead to a critical loss of periodontal support. So severe periodontitis should not regard as a natural consequence of ageing. Periodontal disease, although seen more often in older patients, is not actually part of the physiological ageing process, but it is a result of the disease progression in susceptible individuals.

Susceptible however, greater in older people because of: -

1- Increased gingival recession.
2- Poor oral hygiene.
3- Poor diet and potentially reduced salivary flow.

Gingival recession is frequently seen in the older patients. This can result in: -

1- Exposed root surfaces increasing the susceptibility to root caries.
2- Abrasion lesions because of poor tooth brushing techniques.
3- Thermal sensitivity.
4- Exposure of root fractions in molar teeth, leading to increased plaque accumulation.

Prevention of periodontal disease in elderly

1- Oral hygiene
   a- Effective daily brushing, flossing and antimicrobial mouth wash.
   b- Professional scaling and root planning.
2- The introduction of an electric toothbrush and or chemical plaque control.
3- Fluoride therapy should be a supplement to ordinary periodontal disease treatment. The agent used is NaF, SnF_2, and APF.
4- Smoking cessation.
5- Nutrition counseling.

6- Address systemic diseases/ condition.

2-**Root caries**

Root caries has been described as the adult dental problem of future because of the increasing ageing population and the increased retention of the natural dentition into old age. The root surfaces become exposed due to gingival recession and these root surfaces can become susceptible to root caries. Root caries is associated with periodontal disease, as this is the major cause of gingival recession. It does not mean, however, that all patients with exposed root surfaces will experience root caries. The root surfaces are more vulnerable to caries than enamel since the critical pH of demineralization for dentine is 6.0-6.5 whereas it is 5.2-5.5 for enamel caries.

The primary causative factors for root caries are -

1- A susceptible root surface: gingival recession is the predominant factor but as root caries occur in a pocket, so it is more accurate to use the term loss of attachment.
2- Fermentable carbohydrate: these are metabolized by oral bacteria to produce acid.
3- Dental plaque biofilm: like Streptococcus mutans and Actinomyces.
4- Time: when pH decreases the root surface is more susceptible than enamel to demineralization over the same period of time.

Secondary factors are: -

1- Saliva: the saliva has a protective effect by neutralizing the pH and providing an antibacterial and buffering effect. It influences the rate of clearance of acid and sugar.
2- Fluoride: fluoride has toxic effect to bacteria; it's also inhibited glycolysis (formation of acid from sugar by bacteria) as well as increasing remineralization of the root surface.
3- Root surface factors: the surface roughness influence plaque formation.
4- Location: maxillary teeth are believed to be more susceptible to root caries. Incisors are the least vulnerable followed by canines, premolar and molars.

Prevention of root caries

1- Use of fluorides (rinses, gels, varnishes).
   a- Lifelong exposure to water-containing optimum levels of fluoride reduces the prevalence of root caries.
   b- Self-applied: toothpaste, mouth rinses
   c- Professionally applied: gels, varnishes, fluoride releasing restorative material.
2- Consider salivary substitutes for dry mouth or if salivary flow is reduced.

   A variety of regimes have been described for the prevention and remineralization of root caries in individual with reduced salivary flow which include: -
   a- Topical fluorides, either as mouth rinse or in gel form.
   b- Also supersaturated calcium phosphate mouth wash, which can be used to enhance remineralization (also useful in patient with normal salivary flow). These treatments act by reducing the S. mutans colonization on the root surfaces as well as enhancing remineralization.
3- Plaque and microbiological control.
   a- Brushing and flossing: vigorous and regular individual and professional tooth cleaning for prevention of root caries.
   b- Mouth rinses:

      chlorhexidine used as 15 ml of 0.2% for 30 seconds 2 times a day for no more than 7 days can be used to reduce oral colonization with S. mutans, and hence assist in reducing caries activity. CHX varnish remains active for 3-6 months. It is also used as a component of chewing gum; regular used of this chewing gum has been shown to reduce plaque and caries level and increase salivary flow debilitated older people.
   c- Dietary control:

      The relation between sugar in diet and caries is well established and extends to root caries. Prevention by managing diet through requires a substantial behavioral change and in the context of the older patients which is not easy. It has been demonstrated that the risk of root caries being present was approximately doubled where the frequency of sugars intake exceeds nine episodes per day. High frequencies of sugars intake are often the result of sugar in tea and coffee, biscuit

eating, or sometimes sucking sweets, perhaps to relieve their effects of dry mouth. Patient will often be prepared to replace sugar with saccharine in hot drinks, and to alter their habits when the strategic importance is fully explained. The dietary problem in the institutional setting where food is mass produced and the diet often tailored to the lowest level of masticatory function, resulting in a high carbohydrate diet, often with frequent sugar intakes. So, the rates of root caries in institution are much higher than in free living older people.

4- More frequent dental examinations.
5- Combined strategies.

3- Xerostomia (dray mouth)

With increasing age there is a normal physiological reduction in saliva flow**.** The normal stimulated saliva secretion rate is 1-2 ml/minute, while in patient with xerostomia, this rate may be reduced between 0.7-0.1 and less than 0.1 ml/minute in patient with severe salivary gland malfunction. Xerostomia can cause by:

1- Radiation therapy to head and neck.
2- Systemic disease, like diabetes mellitus, Sjörgen's syndrome, liver disease, immune deficiency diseases and depression.
3- Local factor like Chronic Sialadentitis, obstractions and Sialoadenoma.
4- Hormonal disturbances.
5- Eating disorder and dehydration; the elderly tends to have a lower fluid intake.
6- Side-effect of medication; drugs may alter salivary flow and composition e.g. beta blockers for hypertension, diuretics for hypertension and chronic heart failure, hypnotic for anxiety, anti-parkinsonian drugs, and anti-depressants.
7- Non-therapeutic drugs e.g. illegal drugs, such as ecstasy medication.
8- Atrophy: a physiological decrease in cellular function with age.

Signs and symptoms

1- Dryness of oral tissues.
2- Difficulties with speaking, eating of dry foods, and swallowing.

3- Increased thirst.
4- Difficulty in wearing removable dentures.
5- Increase in fungal infections.

Prevention of xerostomia

1- Change in medications or dosages.
2- Stimulation of salivary glands (sugar-free gums, lozenges, drugs which have cholinergic agonist).
3- Salivary substitutes.
4- Meticulous oral hygiene.
5- Periodic dental examination.
6- Non-alcohol antimicrobial mouth rinses.
7- Fluoride therapy to prevent tooth decay.

4-**Tooth wear**

Prevention of tooth wear

1- Prevention of erosive wear.
   a- Dietary counseling which should arrest the rapid progression of tissue loss.
   b- Stop the medications with low pH which have been reported as producing erosive damage (abnormal use of aspirin, chewable vitamin C, and iron tonic).
   c- Medical /surgical advice should be sought if the cause is gastric regurgitation.
   d- It may be possible to protect dentition using a soft splint. This should extend well onto the palatal mucosal in the upper arch, and it may be of benefit to place a fluoride gel or antacid preparation inside the splint before use. There is evidence that fluoride therapy is a benefit in the control of erosive tooth tissue loss.

2- Prevention of attrition

Mechanical wear because of normal masticatory function cannot be eliminated completely and is part of normal aging.

3- Prevention of abrasion

Damage from porcelain restorations is a problem in an elderly people. Regular contact with opposing natural tooth tissue will result in very rapid wear of natural tooth producing a very complicated management problem. It is advisable to produce tooth on artificial crown contacts on a metallic surface or on a high glazed porcelain surface.

5- Denture related problems

1- Loose denture.
2- Denture stomatitis.
3- Papillary hyperplasia.
4- Denture sores.
5- Epulis fissuratum.

Denture stomatitis causes

1- Fungal infection (C. albicans).
2- Poor denture hygiene, denture fit, nutrition.
3- Immunosuppression.
4- Wearing dentures continuously day and night.

Treatment of denture stomatitis

1- Daily denture cleaning.
2- Wear dentures only during the day.
3- Rinse mouth with nystatin.
4- Soak dentures in nystatin mixed with water.
5- Address denture fit (reline) and systemic issue.

Domiciliary care

Physical and mental disability or chronic disease may make it difficult or impossible for elderly patients to attend a dental surgery or clinic for their routine or emergence care.

The aim of domiciliary care is to provide comprehensive dental care to patients who are unable to access a dental clinic, surgery, or mobile dental unit for their dental care. However, it is useful to make an initial assessment of a patient's eligibility.

The advantage of providing domiciliary care include: -

1- Better access to dental care for patient.
2- Providing a better understanding of a patient's home/living environment.
3- Providing better understanding of patient's ability to carry out oral hygiene advice.
4- Reducing the likelihood of failed appointments.
5- Frequently achieving better patient compliance because the patient is usually very appreciative of the individual care provided.
6- Providing added interest for the operator.

Topical fluoride therapy Lec -3-

By Assist. prof. Azhar Alkamal

Topical fluoride therapy 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.**

The use of topical F was old sin e 1940_s. It can be applied at any age of life that is from tooth eruption till the whole life but the best time for application in the post eruptive maturation period (two years after eruption). The tooth is not fully maturated ionic exchange will be occurred between outer enamel surfaces and oral environment, element like F, Zn, Tin & others continue to incorporate the enamel surfaces.

The main type of reaction is the formation **of calcium fluoride** that is dissolved rapidly there to increase F fixation it needs to be applied frequently and continually.

Types of topical fluoride application: -

1- **Self-application of fluoride.**
   a- Dentifrices.
   b- Mouth rinses.
   c- Fluoridated gel.
2- **Professional fluoride applied.**
   a- Solutions.
   b- Gel.
   c- Varnishes.
   d- Prophylactic paste.

Self-applied of fluoride used with relatively low conc & applied by individuals themselves. The conc of F about **1000 ppm,** while professional applied periodically by the dentist with high conc (**9000-19000 ppm**). Different types of professionals are used such as sodium fluoride, stannous fluoride, acidulated phosphate fluoride, amine fluoride & others.

Advantages of topical fluoride:

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

Disadvantages of topical fluoride:

1. People 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.

The efficacy of topical fluoride depends on:

a. The concentration of fluoride used.
b. The frequency with which it is applied and the duration of application.
c. The specific fluoride compound used.

I-Self applied fluoride:

1 - Fluoridated dentifrices:

The first clinical trial of fluoridated dentifrice by bibby 1942, and the active agent was sodium fluoride (NaF). It was added to dentifrices using di calcium phosphate (DCP) as an abrasive.

The mechanical functions of these dentifrices are:

1- Physio-mechanical function: the action of abrasive material & toothbrush.
2- chemical function that it reacts with outer enamel surfaces & effect on dental plaque (antimicrobial effect).

Types of fluoride used in dentifrices: -

1- Sodium fluoride (NaF)
2- Stannous fluoride ($SnF_{2)}$
3- Sodium monofluorophosphat (MPF)
4- Amine fluoride
5- Combination of NaF and MPF

The conc of F range 525- 1450 ppm. With increase the time of storage conc of F decrease with the time of the storage i.e., 6 month or

more. The type of F used should be compatible with the constituents of the dentifrices, especially the abrasive system.

Following brushing there will be retention of F in oral fluid & dental plaque which will act as reservoir for F ions. These ions will be released gradually in the Saliva region & there by maintaining a degree of protection against caries. Fluoride in toothpaste is taken up directly by demineralize enamel and it also increases the fluoride concentration in dental plaque, thus leaving a store of fluoride available for remineralization when pH drops. The increase in frequency of brushing will increase the benefits of fluoride. The caries reduction by using fluoridated toothpaste is about 25- 30%.

Guideline in using toothpaste: -

1- Children under 5 years:

A brush full of 1000 ppm F paste may contain 1 mg F ions. Child may swallow paste accidentally as he/ she cannot control muscles of swallowing. Thus, brushing twice a day with 1000 ppm F tooth paste the child may swallow about 0.5 mg F / day. The child may be at risk of dental fluorosis especially if the child is living in fluoridated area or taking F supplements, so a small pea sized amount of toothpaste used, or child should be brush under supervision or use low conc of F dentifrice.

1- Children above 5- year and adults:

For children, instruction should be given not to swallow toothpaste & a high F conc can be applied.

2- Fluoridated mouth rinse

The use of fluoridated mouth rinse started in the early 60_s of the last centuries. Rinses can be used in home or school programs.

Fluoride mouth rinse can be used in the following cases:

1- Primary prevention programs for children & adults.
2- In subjects with high risk to dental caries.
3- Patients with rampant caries.
4- Patients with hypo salivation or xerostomia.

5- Patients with sensitive teeth as cases of abrasion, attrition & erosion.
6- Patients with periodontal diseases & present of root caries.
7- Patients with orthodontic appliances.

Types of fluoridated mouth rinse:

1- Sodium fluoride NaF

0.2 % (900 ppm F) for weekly use.
0.05 % (225 ppm F) for daily use.

2- Stannous fluoride 100, 200, 250 ppm

3-Amine fluoride or Ammonium fluoride.

Fluoridated mouth rinse ***should not be*** used for children under 6 years of age as they cannot control muscle of swallowing. Also, it is not preferable to be given to children living in fluoridated areas or receiving F supplements. Fluoride mouth rinse is not a substitute for F dentifrices, it is usually used as a supplement to toothpaste. Studies reported caries reduction about 30%.

A 10 ml of rinse is used by forcefully swishing of liquid around the mouth for one minute then expectorate.

3 – Fluoridated Gel

It **is home** programs in two types:

a- NaF or APF (5000 ppm).
b- SnF_2 (0.4% ppm).

These can be applied using special tray or directly applied by toothbrush. Used for 1-5 minute then expectorate. Patients advised not to rinse by water & not to eat or drink for at least 30 minutes.

Indication for use:

1- Patients with rampant caries.
2- Patients with xerostomia.
3- Sensitive teeth.
4- Root caries.

It can be used for 4 weeks course. When the onset of the disease is stopped, we can switch back to mouth rinse. It is **not** recommended for children under 6 years. It is used in combination with dentifrices, and not preferable to be used in combination with mouth rinses.

II - Professional applied fluoride

It was introduced by Bibby in 1942 that showed that repeated application of NaF to teeth of children can significantly reduce dental caries.

Types of professional applied fluoride: -

It can be used in the form of solutions, gel, foam, varnishes, or pumices. Different agents can be used as sodium fluoride, stannous fluoride, potassium fluoride, zirconium fluoride, titanium fluoride & others.

Method of applications

1- Paint technique: by which fluoride material applied to teeth by cotton applicator or brush.
2- Tray technique: a small amount of fluoride is added to a try then inserted in the patient mouth. Trays come in different shapes and types as foam lined or paper, custom vinyl, and others.

In two techniques:

1- Teeth should be clean firstly (scaling and polishing) to remove dental plaque, calculus, stain, and debris to increase the effect of the fluoride ions.
2- Teeth should be isolated by using cotton roll and saliva ejector.
3- The head of the patient tilted forward to avoid accidental swallowing of the materials.
4- The fluoridated agent applied following dryness of teeth for 4 min and the amount of agent used must not exceed 4 ml to prevent acute fluoride toxicity.
5- Following treatment ask the patient to expectoration several times.
6- Avoid eating or drinking for at least 30 minutes.ma

Indication of use: (in general material can be used at any age)

1- Prevention of dental caries
2- Control of rampant caries
3- Sensitive teeth and root caries.

1- Sodium fluoridated agents (NaF).

- It was used for first time by Bibby in 1947.

- It is now used in conc of fluoride 2% (over 9000 ppm).

- It can be used in form of solutions or gel; flavoring & coloring agent can be added.

- It can be used in primary preventive programs for children & adults. It can be applied every 6 months or once a year.

When the agent (NaF) is added to outer enamel surface calcium fluoride is formed. The following reaction take place: -

$$Ca_{10}(PO_4)_6(OH)_2 + 20F \longrightarrow 10CaF_2 + 2(OH) + 6(po_2)$$

These agents have a basic pH, chemically stable when stored in plastic or polythene containers. It should not be stored in a glass bottle as a fluoride ion will react to the silica of the glass forming silica di oxide reducing the free ionic fluoride necessary for caries reduction. A flavoring and sweetening agent can be added.

Knutson's technique: - method of application of NaF where the agent added in a series of 4-weeks at ages of 3, 7, 11, and 13 years. Coinciding the eruption time of permanent teeth and for protection of primary teeth. The disadvantage of this method is that the patient needs to visit the dentist four times to accomplish this technique.

Advantage of NaF:

1- Not irritant to gingival tissues.
2- Does not cause discoloration to the teeth.
3- Chemically stable when stored in plastic or polythene containers.
4- Dental caries reduction about 30%.

2- Stannous fluoride (SnF_2):

It was used for the first time in the early 50_s by Muhler & coworkers. SnF_2 contains cat ion (stannous) & anion (fluoride), both react with enamel surfaces forming calcium fluoride, stannous fluorophosphates & hydrated tin oxide. These complex agents may increase the resistance of surfaces to acid dissolutions. Caries reduction by SnF_2 was reported to be greater than NaF. These reactions depend on the conc of F ions, acidity (pH) & reaction times.

$$Ca_{10}(PO_4)_6(OH)_2 + 19SnF \longrightarrow 10CaF_2 + 6Sn_3F_3PO_4 + SnOH_2O$$

Stannous fluoride is used in the form of solutions or powder. Powder one should be prepared by dissolving appropriate weight in distilled water. For children, the recommended conc is 8% (dissolving 0.8 mg in 10 ml distilled water). For adolescents & adults the recommended conc is 10% (dissolving 1 mg of powder in 10 ml of distilled water). The pH is about 2.4-2.8. Another conc are used in form of gel in a conc of 30%.

Muhler's technique applied SnF_2 once a year.

Advantage:

1- Effective in primary prevention of dental caries and increasing of outer enamel surfaces by rapid penetration of fluoride in deeper layer of enamel.
2- Re mineralization of initial dental caries.
3- De sensitizations of teeth.
4- Anti-plaque agents: specific antibacterial effect agent cariogenic bacteria & nonspecific effect agents on other types of bacteria.
5- Additive effect by tin ions in addition of F ions. Highly insoluble tin-fluoro-phosphate complex acts as a protective layer against acid attachment.

Disadvantage:

1- Not stable in aqueous solution, it undergoes rapid hydrolysis & oxidation to form stannous hydroxide & stannic ions, these may

reduce the effectiveness of fluoride. Therefore, stannous fluoride solution needs to be freshly prepared.

2- Un pleasant taste, it has metallic & astringent taste. In past flavoring agents were not added as cause's reduction in the effectiveness of F, but now day they add a flavored solution with glycerin & sorbitol to retard hydrolysis of stannous fluoride.
3- Reversible irritation to gingival, gingival bleaching may occur especially in patients with poor oral hygiene. Therefore, stannous fluoride is not preferable to be used in present of sever gingival inflammation.
4- Pigmentation & staining of teeth especially in areas with carious lesion, hypo calcification, & around margins of restorations.

Indication of used:

It can be applied to children as well as adults.

1- Primary preventive programs (once a year).
2- High risk groups & rampant carious (every 3 or 6 months).
3- Initial caries (3 or 6 month).
4- De sensitizing agents (applied once a week, then every 3 months or 6 months).
5- Patients with xerostomia (3-6 months).
6- Hypoplasia & hypo calcifications (as cases of amelogensis & dentinogensis imperfect).
7- Root caries (grade one & two).

3- Acidulated phosphate fluoride (APF):

It was introduced by Brudevld et al (1963). It is sodium fluoride at which **acid** is added. **The success of any topical fluoridated agents depends on its capability of depositing fluoride ions in the enamel as fluroapatite & not only as calcium fluoride.** Fluoroapatite crystals are stable & not like calcium fluoride that can easily dissociated to calcium & fluoride ions.

$$Ca_{10}(PO_4)_6(OH)_2 + 2F \longrightarrow Ca_{10}(PO_4)_6F_2 + OH^-$$

There are two ways of speeding up the reaction that led to the formation of fluoroapatite:

1- Increase conc of F ions in the agent.
2- Lowering the pH, that is making the solution more acidic**.**

Increase the conc of F ions lead to formation of calcium fluoride & phosphate. The presence of acid leads to the breakdown of the outer enamel surfaces (hydrolysis of hydroxyl apatite crystal & release of Ca & phosphate and formation of dicalcium phosphate dehydrate (DCPD).

$$Ca_{10}(PO_4)_6(OH)_2 + 20F \longrightarrow 10CaF_2 + 2(OH) + 6(po_2)$$

$$Ca_{10}(PO_4)_6(OH)_2 + acid \longrightarrow CaHPO_4 + 4Ca^{+2} + 2H_2o$$

In both reactions phosphate formed. The increase in phosphate concentration causes the shift in the equilibrium of the reaction to the right side that is in the direction of formation of fluoroapatite as well as hydroxyapatite crystals. In another word, the increase in the concentration of fluoride ions and lowering the pH in the presence of phosphate lead to increased deposition of ions in form of fluoroapatite crystals (i.e., increase fixation of fluoride ion in the enamel surface).

Acidulated phosphate fluoride (APF) is composed of NaF; the conc of F is 1.23% in addition to acid in form of orthophosphoric acid. It comes in the form of solution, gel, & foam to which coloring & flavoring agents can be added.

APF solution can be prepared by dissolving 20 gm of NaF in one liter of 0.1 m phosphoric acid, this is known as **Brudevold's** solution.

The gel is preferable to the solution as it increases the time of retention of the materials on the tooth surfaces. The gelling material added is in form of carboxy methyl cellulose. Another type of gelling material can be added known as thixotropic gel; this material is highly viscose at low shear rates & low viscose at high shear rate. When used by tray under pressure the thixotropic material will flow between teeth & remain in situ & remain stationary. Therefor it will not flow behind the tray to enter the patient's throat. Both the conventional & thixotropic APF contain the same conc of F 1.23% the difference between them is only in the gelling material.

Indication: - primary preventive programs for children & adults.

Advantages

1- Chemically stable when stored in plastic or polythene containers.
2- Tolerable taste as flavoring agents can be added.
3- Does not cause staining of teeth.

Disadvantage:

1- cannot store in glass containers as reducing the free F ions
2- Repeated exposure of APF to the teeth with porcelain and composite restoration to it may cause loss of material and surface roughening with cosmetic change because of its high acidity.

4-Sliver diamine fluoride: - (SDF)

It is a topical medication used to treat and prevent dental caries and relieve dental hypersensitivity. The trade name of SDF is Saforide and the chemical formula was $AgF(NH_3)_2$.

SDF is colorless and odorless; it looks like water. SDF has a pH of 10 and can cause staining or irritation of skin, soft tissue defects in the tooth structure, such as caries lesion and restorative margins with dark brown or black color, sound tooth structure will not be stained by SDF.

FDA (Food and Drug Administration) provides 38% of SDF for reduction of dental hypersensitivity then the FDA approved SDF antibiotic liquid which clinically applied to control active dental caries and prevent further progression of caries. While the ideal way to treat teeth with decay is by removing the decay and placing a restoration, this alternative treatment allows us to stop decay with noninvasive methods, particularly with young children that have baby teeth. Treatment with SDF will not eliminate the need for restorative dentistry to repair function or aesthetics but has been effective at prevention of further decay.

Mechanism of action

SDF is used for caries arrest and treatment of dentin hypersensitivity. In the treatment of exposed sensitive dentin surfaces, topical application results in development of a squamous layer on the exposed dentin, partially plugging the dentinal tubules.

Upon application of SDF to a decayed surface, the squamous layer of silver-protein conjugates forms, increasing resistance to acid dissolution and enzymatic digestion. The treated lesion increases in mineral density and hardness while the lesion depth decreases. Silver ions act directly against bacteria in lesions by breaking membranes, denaturing protein, and inhibiting DNA replication (killing cariogenic bacteria in dentinal tubules). More sliver and fluoride are deposited in demineralizes than non-demineralizes dentin; treated demineralized dentin is more resistant to caries bacteria than treated sound dentin. When bacteria killed by silver ions are added to living bacteria, the silver is re-activated, so that effectively the dead bacteria kill the living bacteria in a " Zombie effect". This reservoir effect helps explain why silver deposited on bacteria and dentin proteins within a cavity has sustained antimicrobial effect.

Method of application

1- Isolation of the teeth that need to apply of SDF.
2- Brush and rinse the teeth without paste.
3- Dried and any debris such as plaque should be removed.
4- SDF is applied to the affected teeth with a micro brush and the solution can cure on the tooth for two minutes.
5- Advise patients not to eat or drink for at least an hour to allow material to cure.

Indications

1- Children who may have excessive decay.
2- Young children who may have difficulty cooperating for treatment.
3- Children with carious lesions may not all be treated in one visit.
4- Children with disabilities or those who are unable to sit for longer cavity treatment.

Contraindication

1- Allergic to silver

2- There are painful soreness, ulceration, gingivitis, or stomatitis.

Advantages

1- Non-invasive
2- Painless
3- Quick treatment time
4- Effective cavity prevention
5- May stop decay.
6- Relieves tooth sensitivity.
7- Very cost effect as one drop can be used for multiple teeth.

Disadvantages

1-Stain defects in the tooth structure, carious lesion, and restorative margins; sound tooth structure will not be stained

2-The patient may notice a metallic taste after application

3-There is a risk that the procedure will not stop the decay and not guarantee success. In this case the tooth will require further application of SDF, dental filling root canal therapy or extraction.

5- Fluoridate varnishes

It was used for the first time in Germany in 1972. The agent used is Duraphat. It is Sodium fluoride in conc of 2.26% (22600 ppm). It is a viscose yellow material of NaF in an alcoholic solution of natural varnish. It is water tolerant thus can be used even for moist teeth. Varnishes are added to increase time of retention of F to tooth surfaces i.e. slow release or semi-slow-release agents. Prolonged exposure time and high fluoride concentrations result in the formation of a large calcium fluoride reservoir. Fluoride release continues for a long time, as for at least 8 hours or even for several weeks according to the type used. Studies showed that the use of fluoridated varnishes resulted in the most significant caries reduction among topical fluoride agents (30- 70 % caries reduction).

Advantages:

1- Increase retention time of agents on tooth surfaces.
2- It can be added to certain surfaces & not all teeth.
3- It can be applied even in the presence of moist teeth.

Indication: -

1- High risk groups (to be applied 2-4 times a year).
2- Initial carious lesion even for children under 6 years (can be applied on affected tooth surfaces only).
3- Sensitive teeth.
4- Root caries.

6- Fluoride containing prophylactic paste: -

Before application of F agents, it is recommended to clean the teeth by polishing with rubber cup using fluoridate pumice, to remove all exogenous deposit.

Different types are used as: -

1- Zirconium silicate contains stannous fluoride.
2- Silicon dioxide contains acidulated phosphate fluoride.

This paste is not a substitute for the topical agents they are used to increase the accessibility of F ion by tooth surfaces. Through prophylaxis will remove a thin layer of enamel of 1-4 μm, thus it is always recommended to use F pumice.

7-fluoride and restorative material:

Different restorative material may contain fluoride to be released slowly to prevent recurrent caries such as glass ionomer cement, resin modified glass ionomer cement, resin composite, amalgam. Fluoride may be added to fissure sealant.

8-fluoride release devices:

Devices allow for a slow release of F ions as glass beads, copolymer membrane type and others. Indicated for patients with high-risk group and those wearing orthodontic appliance. Not to be given for a child receiving fluoride supplements.

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

Fluoride and 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 diagnosed early in children and adults to stop its progress. Fluoride use to formation of a protective layer on dental hard tissue, which compose calcium fluoride (CaF_2) 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.

Laser and preventive dentistry

By assist. Prof. Azhar Al-Kamal

Although dental caries is a preventable disease, but it is still common and remains a public health problem, especially in developing countries, and certain populations in economically developed countries. Therefore, there is still a need to prevent dental caries and search for alternative methods to disease prevention, or new ways of augmenting current preventive programs. One of the potentially effective preventive measures is the use of lasers. The acronym laser stands for **light amplification by stimulated emission of radiation**.

Laser mean

L = Light

A = Amplification

S = Stimulation

E = Emission of

R = Radiation

In 1704 Newton characterized light as steam of particles in spaces and in 1880 Maxwell demonstrated that electric and magnetic field are propagated in space in the form of waves. In 1900 Max planck introduced the quantum theory (smallest unit of energy), 1916 Einstin postulated stimulated emission, until 1960 Maiman was the first working laser as ruby.

Light is a form of electromagnetic energy (particles in wave). The basic unit of this energy is called a **photon**. Normal light and laser energy are significantly different, ordinary light usually appearing white which is the sum of the many colors of visible spectrum violet, blue, green, yellow, orange, and red this mean that the light have polychromatic characteristic. light it is incoherent mean non identical in size and shape also, light its un collimated (waves not parallel). Laser energy is one specific color, a property called monochromatic; in dental application that color may be visible or invisible. This energy also possesses a property known as

coherency, meaning that the waves produced in the laser instrument are all in phase with one another and have identical shapes when plotted on a graph. In laser the beam itself is collimated; in other words, their rays or beams are parallel within the laser instrument

Absorption: -

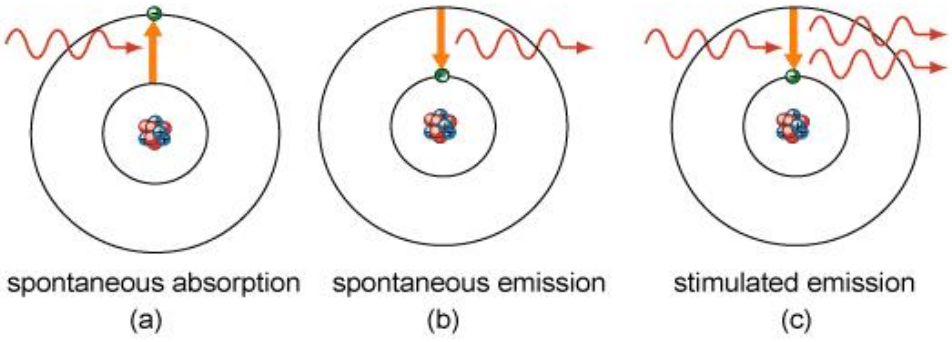
when electron move from low energy level (E1) to high energy level (E2) when a photon hits on the atom of unexcited state.

Spontaneous emission: - (like sun light)

1- Electron moves back from high energy level (E2) to low energy level (E1).
2- Emission of a photon happens in different phase and different direction.

Stimulated emission: -

1- Photon hits on an atom of excited state.
2- Electron moves back from high energy level (E2) to low energy level (E1).
3- Emitting two photons in same phase and same direction.



Laser works because of resonant effect. They produce heat by converting electromagnetic energy into thermal energy. Their working principle is generation of monochromatic, coherent, and collimated radiation by suitable laser medium in an optical resonator. Laser-tissue interaction is controlled by irradiation parameters: wavelength, repetition

rate, pulse energy, pulse duration, continuous or pulsed emission, beam size, delivery method, and optical and thermal properties of the tissue.

Laser emission modes play an important role in increasing the tissue temperature. The thermal effect of laser energy on tissue primarily involves the water content of tissue and the temperature rise of tissue. A laser beam has a natural sterilization effect: it evaporates bacteria, viruses, and fungi, which leads to a decrease in local infections. Probably most important, the laser decreases post-operative pain by sealing nerve endings.

As early 1966, Stern and Sognnaes, using an Nd: YAG (Neodymium-Doped Yttrium Aluminium Garnet) laser showed that irradiated enamel specimens were resistant to acid demineralization, while in 1993, Hicks, et al. concluded that exposure of sound enamel surfaces to argon laser resulted in a significant reducing in lesion depth after acidic challenge.

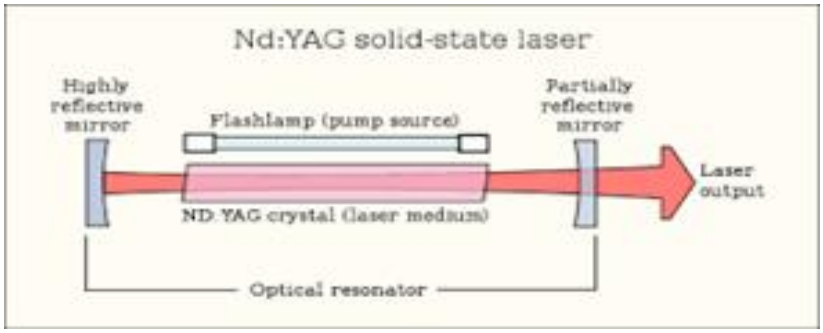
Basic terms: -

1- Amplitude (potential work) (intensity)
2- Wavelength
3- Frequency (oscillation / time)
4- Ability to perform work -----joules
5- Measurement of work per time ---- watt

Laser can produce multiple pulses of energy in one second. The length of each pulse is called pulse duration or width.

Laser component

1- Active medium
2- Pumping mechanism (excitation source)
3- Optical resonator.



Visible light ranges from 400-700 nm any light below 400 nm or above 700 nm become invisible

Dental laser emits visible light.

1- Argon laser
2- KTP laser
3- Low level laser therapy

Dental laser emits invisible laser light

1- Diode laser
2- Er: YAG laser
3- Nd: YAG laser
4- CO_2 laser

Laser available for utilization in dentistry include: -

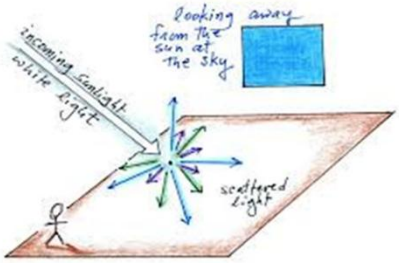
1- Hard lasers Co_2, Nd: YAG and argon lasers.
2- Soft lasers He Ne lasers, Diode lasers.

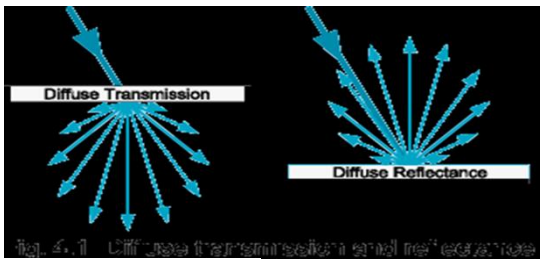
All lasers except the Co_2 laser are transmitted by glass fiber. The Co_2 lasers are transmitted via mirror systems. Laser is being used in almost all the specialties of dentistry.

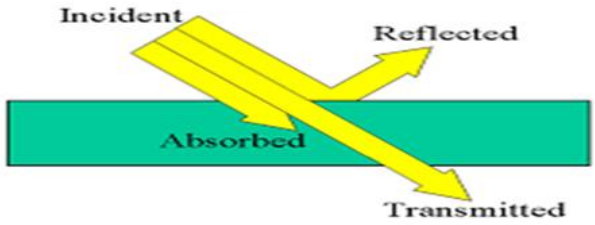
Laser effects on tissues: -

Depending on the optical properties of the tissue, the light energy from a laser may have four different interactions with the target tissue, as follows: -

1- Reflection.
2- Transmission.
3- Scattering.
4- Absorption.







The ways that dental lasers can be used:

1- **Tooth preparation** – Prior to laser dentistry, a drill would be required to prepare the tooth for a filling. Lasers can now eliminate the need for drilling and anesthesia. Lasers also successfully kill oral bacteria around the surgical site.
2- **Reshaping soft tissue** – Dental lasers can dissolve soft tissue to expose more of the natural tooth (crown lengthening), reshape soft tissue to make "gummy smiles" more attractive, and remove uncomfortable soft tissue folds caused by denture wear.
3- **Frenectomy** – Lasers can improve speech and the feeding habits of babies, children, and adults by untying the tongue.
4- **Tumor removal** – When benign tumors have formed in the soft tissue areas of the mouth, a dental laser can completely remove them without causing pain.
5- **Whitening** – Lasers can greatly expedite the tooth whitening process by increasing the activity of the particles in the peroxide bleaching solution.
6- **Biopsy** – Lasers are sometimes used to perform a biopsy on suspicious areas of soft tissue.

Role of laser in preventive dentistry

Laser dentistry when combined with conventional preventive dentistry techniques has enhanced the effect of preventive measure. Action of preventive techniques likes fluoride applications; pit and fissure sealants etc. have more successful results after combining them with lasers.

Studies shown: -

1- Significant reduction in enamel solubility following Co_2 laser irradiation and reported that there was significant synergism between that laser and 0.2 ppm fluoride solution. The combined laser-fluoride treatment led to 98% reduction in mineral loss.
2- Combining acidulated phosphate fluoride with argon laser irradiation resulted in a 50% reduction in lesion depth compared with control lesions which did not receive any treatment.
3- The use of fluoride before and after laser irradiation has been shown to increase the fluoride uptake and decreases the amount of solubility in acidic solution.

<u>Certain roles of laser in preventive dentistry of caries as follows: -</u>

1- Low-energy density lasers partially inhibit caries process without causing any damage to pulp and oral mucosa.
2- Laser alters the tooth surface making it resistance to acid demineralization.
3- Co_2 lasers are more effective than ruby and Nd:YAG laser in decreasing subsurface demineralization during caries process. Co_2 laser treatment of dental enamel can markedly inhibit subsequent caries-like progression. Optimum caries inhibition in enamel appears to be achieved by pretreatments that produce surface temperatures in the range of 800-1200 degrees C. For clinical application the surface enamel heating must not lead to consequent pulp chamber temperature rises of > 4 degrees C. to meet these conditions a pulsed laser is required with a sufficiently high absorption coefficient (wavelength = 9.3 or 9.6 microns).
4- Good mouth opining is required so that laser beam through flexible arm can reach the caries susceptible tooth surface.
5- Minimum possible energy density should be applied to prevent injuries to the pulp and soft tissue.
6- More effective for occlusal surface caries and least effective on proximal caries because of difficultly in approach and proximity of the gingival.
7- YAG lasers are impractical for intraoral use as they guide with a conventional optical fiber and require rigid arms which are difficult

in intraoral manipulation. They also increase the pulp temperature by 20 C.

8- Pulse low-energy infra-red laser radiation is better than other types of lasers as a preventive tool in dentistry.

9- Use of laser on root caries helps in decreasing caries and Argon laser used in combination with fluoride is seen to have very good effect against caries.

10- A greater selectivity of wavelengths (9300 nm and 9600 nm) in the targeting and removal of the carbonate group from enamel mineral molecule results in a greatly increased acid-resistant compound. Additionally, the altered mineral has greater uptake of topically applied fluoride.

11- For diagnosis of caries: -

a- Quantitative light-induced fluorescence (QLF) is a highly sensitive method for determining short term changes in hard tissues lesions in the mouth. The excitation wavelength (~ 405 nm) produced by QLF system allow visualization and quantification of intrinsic green fluorescence of dental tissues and the red fluorescence of bacterial origin.

b- DIAGNOdent is laser fluorescence allows for detection of non-cavitated, occlusal pit-and-fissure tooth decay based upon the differences in fluorescence of sound and carious enamel, in addition to smooth surface caries at an earlier stage than visual inspection. The sensitivity is 0.76-0.87 for non-cavitated enamel/or dentin. Recommendations for treatment are: - values between 10-15 require no active care or treatment; values between 15-30 require preventive or operative care, depending on the patient's caries risk; values of 30 + require operative and preventive care.

c- The Argon laser (488 nm) provided the most suitable wavelength for detection of carious lesion using fluorescence. Its effectiveness in distinguishing caries lesion from sound areas was related to the higher absorption of argon laser light by carious tissue, resulting in less fluorescence being emitted by them compared to sound tissues.

Benefits of dental lasers

1- The main benefit is the ability to interact selectively and precisely with diseased tissues.
2- Reduce the number of bacteria and other pathogens.
3- Faster healing and tissue regeneration.
4- Preservation of more of the natural tooth.
5- Reduced bleeding during and after treatment.
6- Reduced need for anesthesia.
7- Reduced need for stitches and sutures.
8- Reduced risk of bacterial infections after procedures.

Disadvantage of dental lasers: -

1- The disadvantages of dental laser are the relatively high cost and the required training.
2- Another drawback of erbium laser is the inability to remove metallic restorations.
3- No single wavelength will optimally treat all dental tissues.

Laser safety

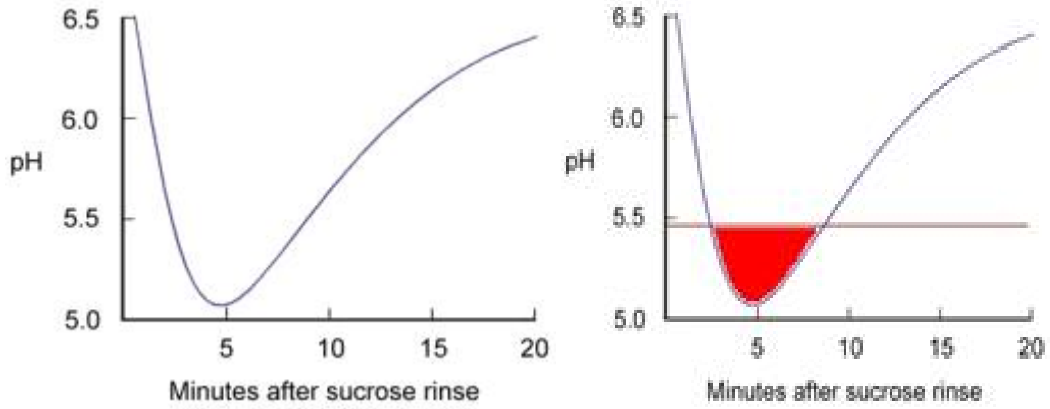
1- All laser devices have complete instruction on the safe use of the machine.
2- Appropriate protective eyewear must be worn by patient and dental team.
3- Masks must be of appropriate filtering capacity to prevent inhalation of plume.

Stephan curve

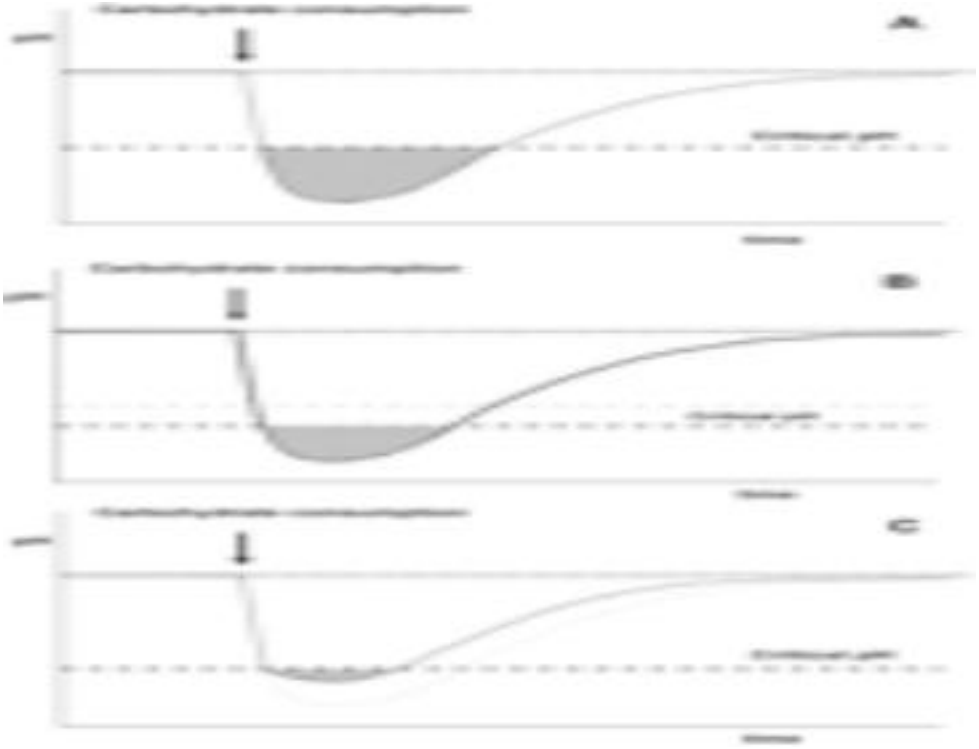
Lec -3-

By assist. prof. Azhar AL-Kamal

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 30-60 min.



The usual form of Stephan curve obtains when changes in reaction to bacteria plaque are measured directly. Patients with active caries tend to show a lower fall in PH, as in the lower curve, feature is very rapid fall in PH and the slow recovery to normal level in spite of the very short time the sugar is in the mouth. Carbohydrates which are retained on the teeth will have a more prolonged effect.



The plot of plaque PH against time has become known as **the Stephan curve**. The rapidity with which the PH fall was reflected to the speed on which sucrose come diffuse into plaque and the activity of the concentration of enzymes. The slow rate of recovery to the resting pH and critical factor in caries production depend mainly on:

1- Rapid production of high conc of acids within the plaque, temporarily over comes local buffering.
2- Escape of acids into saliva, delayed by the diffusion limiting properties of plaque and its thickness.
3- Diffusion of saliva buffers into plaque hampered by the diffusion limiting properties of plaque and its thickness.
4- Continued sugar production from bacterial intracellular polysaccharides.

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

The cariogenicity of sugar increases with its frequent intake, it leads to drop of the pH of plaque and not given enough time for salivary buffer to neutralize the acidity. It has been reported that taking sweets within meal may decrease cariogenicity due to increase salivary flow rate attributed to mechanical stimulation of saliva by food (and increase of buffer system).

Non- sugars sweeteners

Much research has been carried out to fined sugar substitutes. The usefulness of these compounds must be judged from a nutritional, dental, toxicological, economic, and technical point of view. Sugars substitutes can be separated into:

1- Bulk (Nutritive) sweeteners (caloric).
2- Intense (Non-nutritive) sweeteners (non- caloric)

Bulk sweeteners (caloric)

Many of the bulk sweeteners are sugar alcohols, and being chemically like sugars, they have a similar caloric content to sucrose, the most known include **Sorbitol, Mannitol, and Xylitol.** Because sorbitol and mannitol are only half as sweet as sucrose, they may be a tendency to increase caloric intake with the use of these two compounds. Xylitol has the same sweetness as sucrose.

Bulk sweeteners have similar physical characteristics as sucrose, and their substitution does not change the customary size and weight of a product. These bulk sweeteners do not require insulin for their metabolism and are therefore incorporated into food for diabetics. One of the disadvantages of the bulk sweeteners is that they are only partially absorbed in the small intestine and pass the colon where they may induce osmotic diarrhea. Bulk sweeteners are therefore not recommended for children under three years of age and care must take with sugar free medicines containing bulk- sweeteners, since high intakes cause gastrointestinal disturbance.

1-Sorbitol

It is used extensively as a non –sugar sweeteners in confectionery, chewing gum, liquid oral medicine, and toothpaste. It is a derivative of

glucose, occurs naturally in such fruits as apples, pears, and peaches and in several vegetables.

It is not actively absorbed from the gastrointestinal tract and absorbed at about one third of the rate of glucose absorption. This means that eating food rich in sorbitol allow blood glucose level to remain above the fasting level for a longer time than dose eating food corresponding rich in glucose. Thus, eating sorbitol may delay the onset of hunger. For this reason, sorbitol is an ingredient in some foods designed for use in weight-reducing diets and has been used clinically as non-insulin stimulating carbohydrate, so used to be in diabetic food.

Sorbitol is less cariogenic than sucrose, as it fermented slowly by plaque organisms, and the rate is very much slower than that for glucose and sucrose. Sorbitol & sorbitol – containing products are considered safe for teeth. The oral microflora may adapt to sorbitol so that it loses its safe for teeth property.

2- Mannitol

It is a sugar alcohol (polyol). It has a good flavor with little or no after taste. Less popular than sorbitol, partly because of its higher price, and they have a similar dental property. It is used in toothpaste, mouth rinses and as a dusting agent for chewing gum.

3- Xylitol

It is sugar alcohol (polyol) derived from birch trees, corncobs, and oats as well as from bananas. It is the best nutritive sucrose substitute with respect to caries prevention. Xylitol is more expensive than sucrose and sorbitol and it is used in chewing gum, tooth paste and confectioner. It can be considered as non-cariogenic and anti-cariogenic that prevent dental caries. Its non-fermentability in plaque and its saliva stimulating effect may support this statement.

Xylitol may have an antimicrobial effect since the plaque accumulation after xylitol consumption is reduced and there is a good evidence that the ability of plaque to reduce acids by metabolism of sugar reduced by xylitol. This seems to be explained adequately by the decrease in S. mutans in plaque exposed to xylitol and possibly a decreased in plaque quality, xylitol has ability to inhibit growth and metabolism of S.

mutans to the same degree as some antimicrobial. Microbiological studies have shown clearly that plaque organism did not adapt to metabolize xylitol. Using of xylitol in chewing gum to assess the reduction in S. mutans and hence the amount of plaque because it stimulates salivary flow. Taking xylitol may increase the concentration of basic amino acids and ammonia in saliva and plaque (increase the plaque pH i.e. alkalinity). Thus, enhance re-precipitation of calcium and phosphate on tooth surfaces.

Intense sweeteners (non-caloric)

The need for intense sweeteners is acute. An intense sweetener should permit caloric reduction without sacrificing palatability. For primary preventive dentistry practices, a non-carious product that could be used in oral medication, mouth rinse, toothpaste, and all form of candy is highly desirable.

Intense sweeteners are chemically very heterogeneous group and are not chemically related to sugar. They have an intense sweet taste and contain no energy (have a negligible energy value, or too little to have any clinical importance). They are hundreds to thousands of times sweeter than sucrose and they are not metabolized to acids by the oral micro-organisms so they cannot cause dental caries. However, they have disadvantage in taste, stability, lack of volume, although a sweetener with low physical weight is also highly desirable for reducing the size of product packages.

The most popular intense sweeteners are: -

1- Saccharin

It is considered approximately 300 times sweeter than sucrose. Due to its intense sweetness, the use of saccharin is only about 4% as costly as an equivalent sweetness derived from sucrose; it is compatible with most food and drug ingredients. It has a bitter taste in over 0.1% although the perception of this varies between individuals. Saccharin has been reported to inhibit bacterial growth metabolism but its caries inhibiting effect are small.

2- Aspartame

It is a dipeptide consisting of aspartic and phenylalanine. It is approximately 200 times sweeter than sucrose with a similar taste to sucrose. Individual with phenylketonuria, who have a genetic defect of phenylalanine metabolism, should avoid ingestion of aspartame.

3-Acesulfame K

It is approximately 200 times sweeter than sucrose. It has a pleasant, sweet taste. Its sweetness is quickly perceptible and diminishes gradually without any unpleasant after taste. It has had a good potential as a sweetener in most classes of food and drinks and useful sweeteners in boiled sweet and preserves.

4-**thaumatin**

It is approximately 300 times sweeter than sucrose, and it is used with other sweeteners in soft drinks. Disadvantage of thaumatin that its liquorice after taste.

Factors in diet that protect against dental caries

Foods and food component that have anti-cariogenic properties are sometimes referred to as cariostatic factors like fluoride. However, dairy products, plant foods, tea, and even chocolate contain factors that protect against decay.

Milk

Despite being one of the main sources of sugar in the diet of small children, normal milk consumption does not cause dental caries'; and an inverse relation between the consumption of milk and carried increment has been reported. Cow's milk contains lactose, which is less acidogenic than other mono and disaccharides, and it also contains Ca, Ph, and casein, all are cariostatic. Ca & ph are present in cow's milk in high concentration and can prevent enamel demineralization. Several studies have shown that fall in plaque pH following milk consumption is negligible.

Human breast milk is higher in lactose and lower in ca& ph than cow's milk and so, in theory, may be more cariogenic. Breast feeding

provides no opportunity to add additional sugar to milk feeds and breast-fed infants are perhaps less likely to use baby bottles containing sugar liquids. However, prolonged, and nocturnal suckling have been associated with increased caries risk.

Many studies shown that cow's milk a non-cariogenic drink suitable for use as artificial saliva in caries prone xerostomic patients, since it appears to have caries protective properties.

Cheese

Experimental studies have indicated that cheese is anticariogeic. Cheese stimulates salivary secretion due to its sharp test and increases plaque Ca and Ph concentration. The Ca concentration within dental plaque strongly influences the balance between de and re-mineralization of the enamel (even in cooked, cheese-containing meals). Chewing cheese may reduce the levels of cariogenic bacteria. Cheese contains significant amount of tyramine, which could be used by microorganism to rise the pH value of plaque.

Fats

Fats seems to reduce the cariogenicity of foods, they may act merely by replacing carbohydrate in the diet. Fat may also form a protective barrier on the enamel, or surround the carbohydrates, making these less available and speeding up their removal from the mouth. Bacterial surface properties involved on plaque formation could also be altered by fats. Certain fatty acids have antimicrobial effect and have been shown to inhibit glycolysis in human dental plaque.

Plant’s foods

There are many protective factors in the foods of plant origin including phosphates, inorganic phosphates, and phytate.

Phytate is anticariogenic and acts by adsorbing onto the enamel surface to form a physical barrier that protects against plaque acids. When isolated from foods, phytate is an effective anti-caries factor, but as an intrinsic food component, is not effective. Therefore, to be effective, phytate would need to be extracted from grains and then used as food

additive. However, this would not be desirable since phytate binds minerals like calcium, magnesium, iron, and zinc reduced their abortion from the gut, it probably that this side effect will make it unwise to recommend the use of phytate as a food additive.

Some investigators have found statistically significant reduction in caries increment by adding 1-3% of sodium phosphates or calcium sucrose phosphate to various foods or chewing gum, one major problem is that phosphate when added to sucrose, are cleared from saliva faster than sugar and fail to produce substantial increases in the phosphate concentration of human plaque. Other problems could possibly risk of increased dental calculus formation or pathological calcification of the internal organ.

Tea and apples

Apples contain polyphenols which have antibacterial properties and are a good stimulus to salivary flow also apple contain condensed tannins which have anti-adhesion properties that may inhibit some bacteria from bonding to each other and producing dental plaque. Tea contains polyphenols in addition to fluoride.

Fruit and dental caries

Bananas appear to have a greater potential than citrus or apples to cause dental caries, but this does not appear to have occurred in man. Based on the present evidence, increasing consumption of whole fresh fruit in order to replace non-milk extrinsic sugars (free sugars) in the diet, as recommended by the Department of Health, is likely to decrease the level of dental caries in the population. Fruits as lemons, fruit juices are sufficiently acidic when in prolong contact with tooth, may cause dental erosion.

Fluoride and dental caries lec 2

By Assist. Prof Azhar Alkamal

Since 1930 fluoride was demonstrated for its anti caries effect. Fluoride in small dose has a remarkable influence on dental system. It causes a strong inhibition of dental caries, while in large concentration it may cause dental fluorosis.

Fluoride has been found to be a very important element in the prevention of dental caries this is through epidemiological and clinical studies. Fluoride is derived from Latin term fluore meaning “to flow”, at room temp fluoride is a pale yellow–green gas. Fluoride may occur in combined form in a wide variety of minerals such as: -

CaF_2, Ca_{10} (Po_4) o6F2, Na_3ALF_6.

Fluoride is highly reactive it forms salts of almost all metals, it is rarely occurring as fluoride ions in nature. Fluoride contents is expressed in parts per million (ppm) i.e., 1 mg fluoride per Kg or liter water. It is widely distributed in the atmosphere

When Frederick began dental practical in Colorado spring in 1901 has been noticed a feature of dental enamel among residents which he had previously encountered elsewhere this condition was known as **Colorado Brown Stain** and exhibited itself as a stain of varying intensity ranging from fine pale to dark brown mottling which could be quite ugly. Frederick become interest in the condition and his investigation showed that the stain in area around Colorado and these led him to conclusion that causative agent was found in the drinking water of communities and, he observed in this area where mottled enamel condition was found the prevalence of dental caries appear lower**. Dean** began by devising an index of mottled enamel in 1934 then modified in 1942 and this index is used today in six grades of severity.

Fluoride is widely distributed in the atmosphere **originating** from: -

1- Dusts of fluoride containing soils
2- Gaseous industrial wastes
3- Gases emitted in areas of volcanic activity

Fluoride enters vegetation by:

1- Uptake from soils. 2- Water.

4- Absorption from air. 4- Deposition from atmosphere. 5- Rain.

Sources of Fluoride intake in man: -

1-water: the greater part of fluoride intake originates from water ingested each day. Fluoride is naturally present in rivers, oceans, and ground water. The concentration may range from 0.1 – 10 ppm. 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 although concentrations as high as 95 mg /L have been recorded in Tanzania. The highest natural fluoride concentration ever found in water was 2800 mg/L, recorded in Lake in Kenya.

2- Food: It may present in various concentration in soft drinks, different infants, and adult food. Fluoride concentration in various foods reflects its concentration in water where the product has been prepared. For infants, the daily fluoride intake is determined by feeding pattern, as breast milk or formula milk. In human breast milk fluoride concentration is 0.3-0.4μm this is in fluoridated and non-fluoridated area. In cow's milk the concentration is less than 0.019 ppm. In formula and cereals, the conc depends on the product and fluoride conc in water these are prepared. For adult's food fluoride conc vary, but usually less than 0.5 ppm. A higher conc may be found in tea 0.5- 4 ppm & in fish and shellfish.

3- Drugs and dental products: some drugs contain a high conc of fluoride as diuretics and anesthetics, while dental products as dentifrices & mouth rinses.

4- Pollution: fluoride is present in high conc in the vicinity of metal industries, about 25-1000 times the normal. A high concentration of fluoride is a recorded in area with volcanic activity.

Fluoride metabolism:

1- Absorption:

After intake of F it will be absorbed in the GIT, it is also absorbed via lungs. Approximately 75-90 % of fluoride ingested each day is

absorbed form the GIT, with higher proportion from liquids than from solid.

There are factors affecting the rate of F absorption, these are:

*** Solubility and degree of ionization of the components**: This will determine the amounts of F ions released. Only F **in ionic** form is of importance to health. NaF is more soluble than CaF_2 thus the rate & degree of absorption of NaF is more than CaF_2.

*** Dose & F conc**: following absorption there will be an elevation in plasma F level. The height of plasma peak is proportional to F dose ingested & rate & degree of absorption in addition to body weight (with increase body weight there will be a lower plasma peak).

*** Presence of food in the stomach** presence of certain dietary items as Ca may lead to formation of insoluble salt with F. Food acts a physical barrier that retards absorption of F from GIT.

*** Gastric acidity.** There is an inverse relation between gastric acidity & absorption of F from GIT. Milk may retard absorption of F from the stomach in the first hour, later absorption will continue at higher levels for longer period.

2-Retention and distribution in the body:

The maximum plasma conc of F is reached in 30 min. The plasma peak will be reduced as F distributed in the body. Fluoride is a calcified tissue seeker, more than 99% of F in the body is found in calcified tissue. F is rapidly distributed to bone, teeth, heart, kidney & liver, while it is slowly distributed to skeletal muscles & adipose tissue. The uptake of F is affected by age factor, younger the age the greater will be the uptake of F. **In bone** fluoride distribution is not even, in long bone for example, the highest concentration of fluoride is in periosteal region with a slight increase in endosteal region. Cancellous bone possesses high concentration of fluoride compared to compact bone.

Note: all F in bone is firmly held as some of F is subsequently lost again by the osteoclastic resorption of the bone.

In pregnant women, uptake of fluorides in the placenta is dependent on the fluoride concentration in the mother`s bloodstream. When the concentration is low, fluoride is transmitted into the placenta. On average, the concentration in the placenta is about 60% of the concentration in the mother`s bloodstream. If the fluoride concentration increases over 0.4 ppm, the placenta works as a barrier, preventing the fluoride from passing through and thus protecting the fetus from a high fluoride concentration. Fluoride can also be transmitted through the plasma into the mother's milk; however, the concentration is low.

Two forms of fluoride are present the ionic fluoride (free fluoride), and the non-ionic or bounded fluoride. The only ionic form is of importance to health. **In soft tissue** fluoride concentration depends on the pH of extracellular fluid, as higher acidity increases fluoride ionic exchange through tissue plasma. Thus, in treatment of fluoride toxicity alkalization of body fluid is useful treatment.

In teeth: - in enamel, the highest fluoride concentration is near outer enamel surfaces compared to deep enamel layers. In dentine the highest concentration is at pulpal wall compared to DEJ. The precipitation of fluoride increases with age in outer enamel surfaces. However, with aging and wearing of teeth fluoride will be lost and decrease. Fluoride in dentine seems to be in similar concentration as bones, reflecting the amount of fluoride ingested and adsorbed.

Fluoride in plaque and saliva: fluoride is present in saliva in a low concentration 1-2 µmol/ L, the source of this fluoride ion is from water, food, topical agents as dentifrices. the concentration of fluoride in the dental plaque varies from 0.01-50 ppm wet weight in form of free ions, calcium fluoride and / or fluoroapatite. The source is water and food, saliva, and gingiva, in addition to fluoride product, especially toothpaste. Ions in plaque will be released and transferred to enamel surfaces underneath the plaque and redeposit in form of calcium fluoride or fluoroapatite or fluorohydroxyapatite. 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

3-Excretion

The major route of excretion is by kidney. After entering the renal tubules some of F ions will be reabsorbed and return to the circulatory system, while the reminder of ions will be excreted by the urine. Degree of reabsorption depends on the pH of the tubular fluid, urinary flow, and renal function. The renal clearance of fluoride is 30-50 ml / min, the clearance increases with increase in urine pH which is in turn is affected by diet and medication. Patient with renal failure showed a reduced fluoride excretion. About 10% of F is removed by feces this amount is never absorbed, also a less quantity is excreted by sweat, tear, saliva & gingival exudates.

Mechanism of action of fluoride in caries reduction: -

1- Increase enamel resistance (reduction in enamel solubility).
2- Aid post eruptive maturation of enamel surface.
3- Remineralization of incipient lesions.
4- Fluoride as an inhibitor of demineralization.
5- Interference with plaque micro.
6- Modification in tooth morphology.

1-Increase enamel resistance

It has been well established that dental caries involves dissolution of enamel by acids from bacterial plaque and that dissolution is inhibited by the presence of fluoride. Because fluoride forms fluorapatite, which is less soluble mineral, it has been thought that the anti caries effect of fluoride is the result of reduced solubility. The dissolution of enamel during a caries attack is a complicated process when enamel is exposed to a pH of about 5.5 or lower, it will dissolve to its ionic form (Ca, HPO_4, OH) this occurs beneath a bacterial plaque. The concentration of Calcium, phosphate and other ions in the solution will increase. When plaque stop producing acid, the pH will raise and the dissolved minerals get precipitated in the outer enamel surfaces Thus, carious dissolution of enamel is a cyclic phenomenon consisting of phases of demineralization and remineralization. The presence of fluoride reduces the solubility of

enamel by promoting the precipitation of hydroxyapatite and phosphate mineral.

When hydroxyapatite is exposed (during period of tooth formation) to low fluoride concentrations (about 1ppm) = (1mg/1L) a layer of fluorapatite forms on the hydroxyapatite crystals. This reaction is irreversible i.e., once fluoroapatite crystal is formed it will remain so for the lifetime and this reaction is irreversible. Ingestion of fluoride in the pre-eruptive stage will allow the incorporation of fluoride in the whole enamel and dentin. This will increase the resistance against dental caries in addition reduces the progression of dental caries.

$$Ca_{10}(PO_4)_6(OH)_2 + 2F \rightarrow \mathbf{Ca10\ (PO_4)_6F_2} + 2OH$$

When F is present in saliva and dental plaque it will react with the outer enamel surface to enhance remineralization. Two types of reaction may develop the main reaction is formation of **calcium fluoride**, while second reaction is the formation of **fluoroapatite** crystal.

$$Ca_{10}(po_4)_6(OH)_2 + 20F^- \longrightarrow \mathbf{CaF_2} + 6PO_4^{-3} + 2OH^-$$

$$Ca_{10}(po_4)_6(OH)_2 + 2F^- \longrightarrow \mathbf{Ca_{10}\ (po_4)_6\ F_2} + 2OH^-$$

Calcium fluoride (CaF_2) **is not permanent** as it dissociated to calcium and F ions. Therefore, fluoridated products need to be **applied continually** & at a **high conc**, otherwise the enamel will be opened to renewed demineralization. *This type of reaction can be initiated at any time of subject life.*

2-Aid post eruptive maturation of enamel surface

Newly erupted teeth often have hypomineralized areas that are prone to dental caries. In addition, the entire enamel surface is at its maximum degree of susceptibility to caries as soon as it appears in the mouth. Fluoride increases the rate of mineralization, or post eruptive maturation of these areas.

3 -Remineralization of incipient lesions

Remineralization, the deposition of minerals into previously damaged areas of the tooth is a dynamic process that results in reduced enamel solubility. This increase in enamel resistance is achieved through the growth of crystals which become larger than those in either demineralized or sound enamel. These larger crystals are more resistant to acid attack.

4-Fluoride as an inhibitor of demineralization

With lower Ca^{+2}, PO^{4-}/ $+OH^{-}$ ion conc (ph↓). A higher dissolution rate was observed. Addition of fluoride to this, reduce the rate of the lesion.

5-Interference with microorganisms

The presence of fluoride in high concentration (more than 40 ppm) in dental plaque may affected the growth and fermentation of bacteria. These by: -

* Interference with bacterial adherence by retardation of extra cellular poly saccharide.

* Inhibition of intercellular enzymes as enolase and phosphatase. Thus, inhibit the bacterial glycolysis and metabolism.

* In a high concentration, fluoride is toxic to bacteria.

6-Modification in tooth morphology

There is a direct relationship between the amounts of fluoride ingested during tooth development and the incidence of dental caries. If fluoride is ingested during tooth development, there is some evidence to suggest the formation of a more caries resistance tooth, slightly smaller with shallow fissures. The size and morphology of teeth in humans and experimental animals can be influenced by ingestion of fluorides. The reduced level of occlusal caries found in fluoridated areas may be partly attributed to the improved morphology of the occlusal surface.

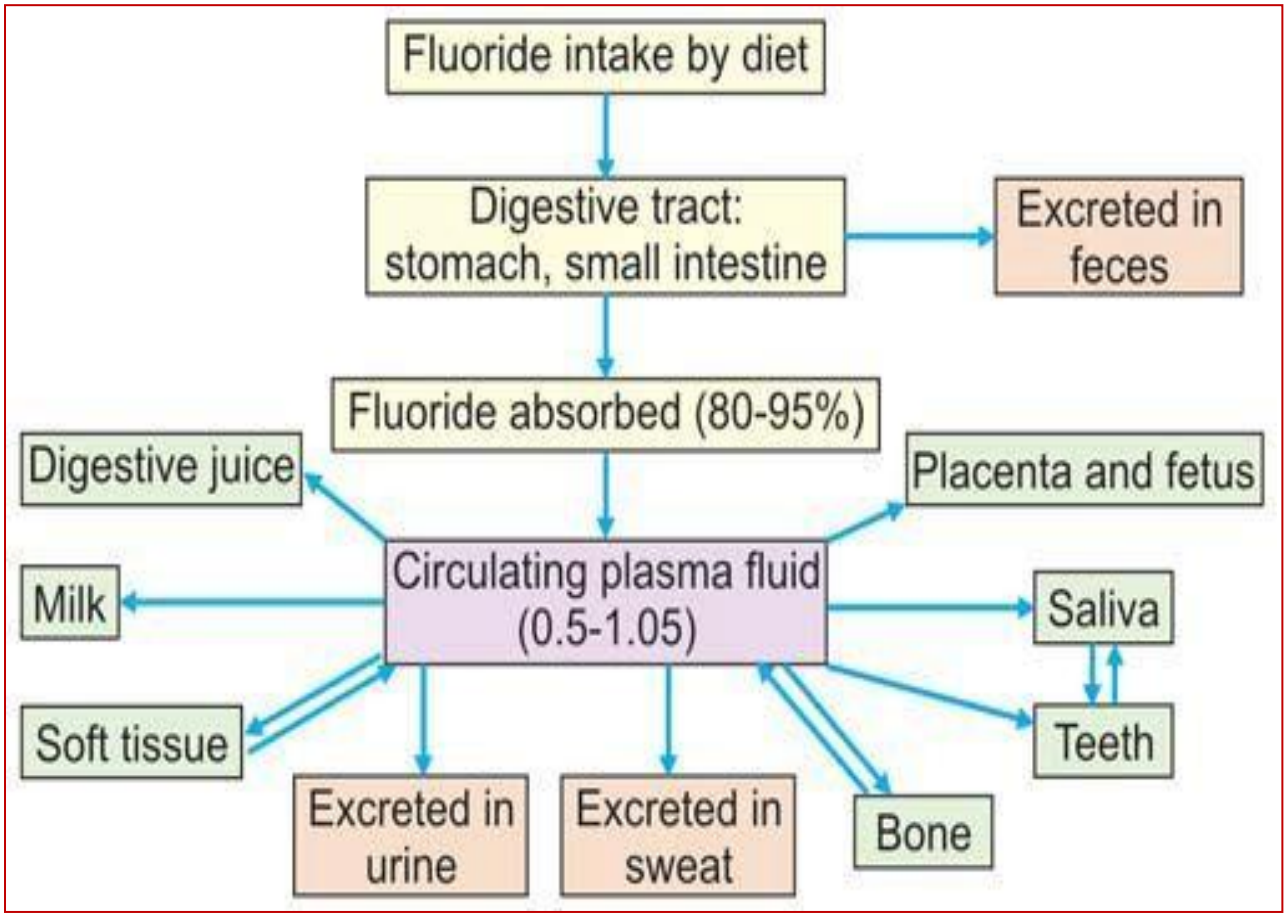


Fig Metabolism of fluoride in human body

Microbiologic aspect of dental caries

By Assist. Prof. Azhar Alkamal

One milliliter of whole saliva may contain more than million organisms representing more than 250 different bacterial species. These microorganisms constitute a very complex microorganism which itself does not result in disease as they exist in equilibrium and in balance with the host.

Oral cavity is a unique ecological system, which is warm, moist, and relatively opens to the outer environment. Tooth surfaces as well as dental plaque constantly encounter different challenges from food intake, speech, and so on. Bacteria grow in two different ways: planktonic and biofilm forms. Because biofilm is composed of various species of organisms, interactions with other members of the multispecies community in the oral cavity can influence the behavior of dental bacterial plaque. The mixed-species bacteria engulfed within the biofilm population behave differently from planktonic, liquid-phase, mono-species cells. This difference in behavior has significant clinical implications.

The resident microflora has a diverse composition, consisting of a wide range of Gram-positive and Gram-negative bacterial species, as well as yeasts and other types of microorganism. In addition, the composition of the oral microflora will change as the biology of the mouth alters over time, the oral cavity, for example, the tooth surfaces provide distinct binding factors for microorganisms. Moreover, the mouth is continuously bathed with saliva at a temperature of 35–36 °C and a pH of 6.75–7.25. The nutritional condition of the oral cavity is often described as “feast or famine”, further exerting far-reaching influence on the composition of microflora.

The oral cavity is sterile in uteri, but although during birth the neonate is exposited to all the complex micro flora of the birth canal; these organisms fail to colonize illustrating the highly selective environment of the mouth. A distinctive oral flora is rapidly established soon after birth. Streptococci are numerically dominant, particularly S. salivarius, S. mitis, and S. oralis, which colonize the mucosal surfaces and dorsum of the tongue. Since the normal habitat of all these species is

most likely that the source of these will be an adult, most probably the mother or other primary career. S. mutans preferentially colonizes hard surfaces and hence, its appearance is delayed until the eruption of the first molar teeth. Most infants, therefore, acquire S. mutans during the age of two. It was possible to accept the conclusion that matching patterns in mothers and children had not occurred chance, and the maternal transmission must be the source of S. mutans in 80% of the children while, reducing mutans levels in mothers can delay infection of their child, whereas no evidence of father-child transmission was found. It may not be feasible to achieve a lifetime free of S. mutans but delaying the age at which a child becomes infected can reduce their subsequent caries risk.

All surfaces within the oral cavity will constantly be subject to microbial colonization, the pattern of which will be partly dependent on the surface properties. As the squamous epithelium of the mucous membranes of the oral cavity is constantly being renewed because of turnover in the epithelium, the surface epithelial cells will be constantly being desquamated, together with the colonizing microorganisms and swallowed.

On tooth surface no surface renewal takes place and therefore microorganisms are able colonize and grow on these surfaces unless removed mechanically or chemically. Colonization may be somewhat different on tooth surface that are exposed to chewing forces and abrasion from foods on lips and cheeks than in areas which are not easily accessible, such as proximal site, tooth surfaces along the gingival margin, and in irregularities of tooth surface, such as occlusal fissures. These areas are often designated stagnation areas.

The dental plaque was formed in two steps; firstly, formation of acquired pellicle then plaque formation after deposition of bacteria

1- Acquired pellicle

Microorganism do not deposit or adhere directly an appetite crystal surface. Within seconds after saliva first contact the external tooth surface, a coating of salivary materials called **the acquired pellicle** begins to develop on the tooth. In uncolonized areas the pellicle reaches a thickness of 0.01–1 μm within 24 h.

The saliva compartment is separated from the tooth by a thin layer of material called the acquired pellicle. This layer of salivary protein adheres strongly to the enamel that is not removed during tooth brushing **the pellicle is** defined as a cellular layer of adsorbed salivary proteins and other macromolecules on the dental mineral surface that is continually deposited on the soft tissue and on surfaces of the tooth exposed to the oral cavity The formation rate varies between individuals probably due to differences in salivary composition. If the pellicle is displaced by prophylaxis, it begins to reform immediately. The pellicle on the tooth surface consists of more than 180 peptides, proteins, glycoprotein including keratins, mucin, histidine-rich, proline-rich proteins, phospho-protein and, to a lesser extent components from the gingival crevicular fluid (GCF).

The pellicle layer even if thin has an important role: -

1- Protecting the enamel against mechanical and chemical insults (abrasion and attrition), moreover, this film can take up **stain** and in other way influence the coating of the teeth.
2- Pellicle in other wards can be defined as protective diffusion barrier formed on enamel from salivary protein. Because of its perm selective nature, restricting transport of ions ***in*** and ***out*** the dental hard tissues, through this matrix network surface, fluids and small sized molecules can slowly diffuse throughout the enamel; therefore, the pellicle may play an important modifying role in caries process. Experiments has shown that the pellicle delay the initiation of caries and the dissolution of the enamel when teeth are placed in low pH soft drink.

There is a competition for the binding sites on the pellicle, not only by receptors on the bacteria, but also from host protein, such as ***immunoglobulins, i.e., antibodies, proteins of the complement system, and the enzyme.*** These host proteins originate from the saliva and the gingival crevicular fluid. Once one of the competing entities occupies a pellicle site, occupancy by another is interdicted. Not only dose competition arises for occupancy of binding sites, but an antagonistic relationship often exists between different types of bacteria occupying

the binding sites. For example, it has been shown that some streptococci synthesize and release bacteriocins, which can inhibit some strains of Actinomyces. The bacterial colonization of the acquired pellicle can be beneficial for the bacteria because the pellicle components can serve as nutrients

Dental plaque

It is gelatinous, sticky material which accumulates around the teeth composed of mucin, bacteria, and bacterial products. It forms the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable and fixed restoration. Dental plaque is readily visualized on teeth after 1-2 days with no oral hygiene measures.

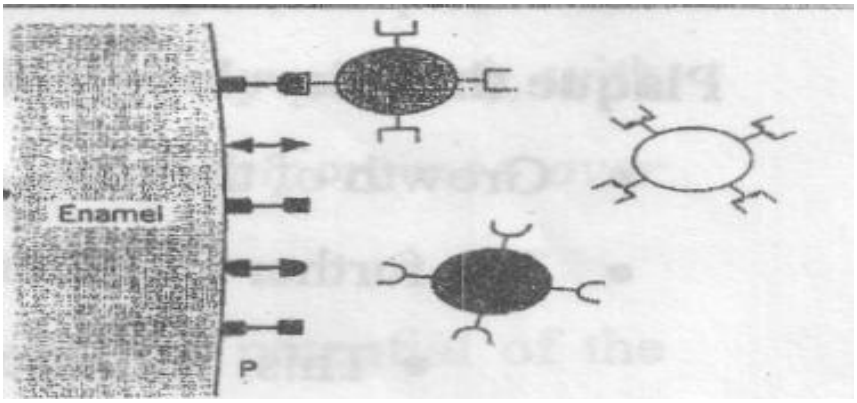
In the initial stage (pioneer) species of bacteria colonize the tooth surface by binding to component of the salivary pellicle, among these initial colonizers are believed to constitute a highly selective part of the oral micro flora, mainly

Streptococcus sanguis, streptococcus oralis and Streptococcus mitis. In addition, the initial microflora comprises minor proportion of Actinomyces and gram- negative bacteria, e.g., Haemophilus spp.

The selective way the bacteria attach to the tooth surface in two ways

1- The fact bacteria on their surfaces contain a recognition system which enables component on the bacterial surface (adhesions) to bind to complementary molecules (receptors) in the pellicle.

2- Bacterial adhesion may be done through the phenomenon termed **calcium bridging,** which links negatively charged bacterial cell surfaces to the negatively charged acquired pellicle via interposed, positively charged divalent calcium ions from the saliva.



There are six stages of plaque biofilm development.

Stage 1: - Formation of a cellular layer. Called the acquired pellicle, this layer of salivary glycoproteins, phosphoproteins, and lipids, but no bacteria, forms almost immediately on naked enamel surfaces.

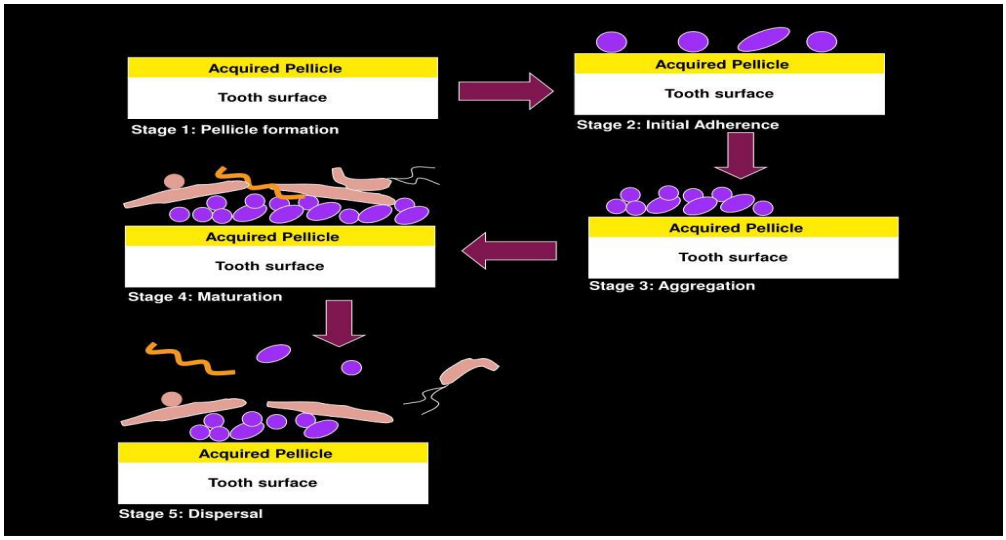
Stage 2: - Initial attachment. Free-floating early colonizers of the teeth, such as Streptococcus sanguinis, which are normal inhabitants on the mouth, form an initial attachment to the pellicle by weak and reversible van der Waals forces. If these bacteria are not removed, they eventually anchor themselves with adhesive structures, such as pili.

Stage 3: - Irreversible attachment. Organisms that were unable to attach to the pellicle begin to adhere to the first layer of colonizers with irreversible attachments via specific adhesion-receptor interactions. The bacteria replicate and form microcolonies embedded in an extracellular matrix.

Stage 4: Early Maturation (also called Maturation I). As a result of the previous steps in which bacteria form attachments, early colonizers become established. This leads to increased dental plaque complexity due to allogenic factors, such as oxygen consumption within plaque creating anaerobic zones, food chains becoming established, and an increased range of receptor sites for bacterial attachments. Cell division and induction of new bacteria also allows the bacterial population to increase.

Stage 5: Late Maturation (also called Maturation II). In this stage, microbial diversity continues to increase, while rates of cell division decrease. The heterogeneous nature of plaque becomes apparent as a mosaic of microenvironments develop, particularly areas of different pH, oxygen concentrations, and secondary metabolite accumulations around and within microcolonies. The plaque microbial ecology reaches a pseudo-steady-state **climax community**, where there is a constant turnover of cells, but the overall composition remains roughly the same. At this point, a thick, three-dimensional layer of dental plaque biofilm has formed.

Stage 6: Dispersion. Enzymes that degrade the biofilm (such as dispersion B) allow some bacteria to detach themselves from the biofilm–sometimes in response to harmful environmental conditions–to spread and colonize new surfaces in the oral cavity.



Dental caries is the result of the metabolic activities of bacteria in microbial communities on teeth termed dental biofilms (often referred to as dental plaque).

There are three hypotheses for plaque formation: -

1- **The specific plaque hypothesis: -** proposed that, out of the diverse collection of organisms comprising the resident plaque microflora, only a single or very small number of species were activity involved in disease. This proposal has been easy to promote because its focused efforts on controlling disease by targeting preventive measures and treatment against a limited number of organisms, such as by vaccination or gene therapy or by antimicrobial treatment. This may be due to the structure of the biofilm and the localization of mutans streptococci in plaque.
2- **The non-specific plaque hypothesis: -** considered that disease is the outcome of the overall activity of the total plaque microflora, so not just those that make acid, but also species that produce alkali or consume locate need to be considered. Thus, a heterogeneous mixture of microorganisms could play a role in disease. mutans

streptococci are found in high numbers in plaque but in the apparent absence of any demineralization of the underlying enamel.

Explain of nonspecific theory

1- There is extreme variation in supra gingival plaque.
2- Other plaque bacteria have the same biochemical characteristics
3- Bacteria associated with caries other than mutans streptococci and lactobacillus.
4- The presence of lactate-consuming species (e.g., Veillonella).
5- The production of alkali to raise the local pH (e.g., by ammonia production from urea arginine by S. salivarius and S. sanguinis, respectively).

3- **The Ecological plaque hypothesis**: - considered that the disease is a consequence of imbalances in the resident microflora resulting from changes in the local surrounding environmental conditions. Caries is a result of changes in the environmental due to acid production from the fermentation of dietary carbohydrates, which selects for acidogenic and acid-tolerating species such as mutans streptococci and lactobacilli.

Disease could be prevented not only by targeting the putative pathogens directly, but also, by interfering with the key environmental factors driving the deleterious ecological shifts in the composition of the

Dental calculus

A last stage in the maturation of some dental plaques is characterized by the appearance of mineralization in the deeper portions of the plaque to form dental calculus. Calculus formation is related to the fact that saliva is saturated with respect to calcium and phosphate ions. Supragingival calculus forming on the tooth coronal to the gingival margin frequently develops opposite the duct orifices of the major salivary glands. Subgingival calculus forms from calcium phosphate and organic materials derived from blood serum, which contribute to mineralization of subgingival plaque. Alkaline conditions in dental plaque may be an important predisposing factor for calculus formation. Bacterial phospholipids and other cell wall constituents may act as initiators of mineralization, in which case it may begin in the cell wall

and subsequently extend to the rest of the cell and into the surrounding matrix. Calculus is generally covered by actively metabolizing bacteria, which can cause caries, gingivitis, and periodontitis.

Strategies of caries prevention according to plaque hypothesis: -

1- Inhibition of plaque acid production: by fluoride-containing products or other metabolic inhibits (like chlorhexidine and others). Fluoride improves enamel chemistry and inhibits several key enzymes especially those involved in glycolysis and in maintaining intracellular pH.
2- Avoidance between main meals of foods and drinks containing fermentable sugars thereby reducing repeated conditions of low pH in plaque.
3- Stimulation of saliva flow after main meals, saliva will introduce components of the host response, increase buffering capacity, remove fermentable substrate, promote re-mineralization, and more quickly return the pH of plaque to resting levels.
4- Probiotics are live microorganisms which, when applied in adequate amounts, will benefit the health of the host.

Role of mutans streptococci (M.S) in dental caries including the fallowing: -

1- Correlations of M.S counts in saliva and plaque with the prevalence and incidence of caries.
2- M.S can often be isolated from the tooth surface immediately before development of caries.
3- Positive correlation between the progression of carious lesions and S.M counts.
4- Production of extracellular polysaccharides from sucrose (which help to cement the plaque organisms together and to the tooth surface).
5- Most effective streptococci in caries studies in animals (rodents and non-human primates).
6- Ability to initiate and maintain microbial growth and to continue acid production at low pH values.
7- Rapid to metabolism of sugars to lactic and other organic acids

(such as butyric and propionic).

8- Ability to attain the critical pH for enamel demineralization more than other common plaque bacteria.
9- Ability to produce intracellular polysaccharides as glycogen, which may act as a food store for use when dietary carbohydrates are low.
10- Immunization of animals with specific S.M serotype significantly reduces the incidence of caries.

Streptococcal adhesions

Mutans streptococci can attach to the tooth surface by either of two mechanisms: -

1-Sucrose - independent adsorption, in which the bacteria attach to the acquired pellicle through specific extracellular proteins (adhesions) located on the fimbriae (fuzzy coat) of these organisms that bind to salivary glycoprotein on tooth surface.

2 -Sucrose dependent mechanisms, in which bacteria was require the sucrose to produce sticky extra cellular polysaccharides, or glucan which allow attachment and accumulation.

Many fermentable carbohydrates may be utilized by plaque bacteria to generate the acids which attack enamel substance, **sucrose** is recognized as being particularly important in caries process because not only can it be fermented, but it also serves as a substrate for extracellular enzymes of plaque bacteria which synthesize sucrose - derived polymers.

These polymers are of central important:

1- Adhesive interaction in plaque where they mediate attachment of bacteria to the tooth surface and to other bacteria.
2- They stabilize the plaque biofilm
3- Serve as energy stores aiding the survival of plaque bacteria,
4- Volume of dental plaque is increased through its content of glucans, which provide a barrier to diffusion of acids away from the tooth surface.

The role of lactobacilli

It is gram-positive bacilli and aero tolerant or anaerobic bacteria. Lactobacilli were previously believed to be the causative agents of dental caries. They were candidate organism for caries because of: -

1- Their high numbers in most carious lesion affecting enamel (many studies

have now shown its high prevalence in root surface too).

2- The positive correlation between their numbers in plaque and saliva and caries activity.
3- Their ability to grow in low-pH environment below pH 5 (can carry out glycolysis at pH values as low as 3) and to produce lactic acid.
4- Their ability to synthesize both extracellular and intracellular polysaccharides from sucrose.
5- The ability of some strain to produce caries in gnotobiotic (germ-free) rats.
6- The fact their number in dental plaque derived from healthy sites are usually low.
7- On the negative side, however, lactobacilli are rarely isolated from plaque before the development of caries, and they are often absent from incipient lesion.
8- lactobacilli are poor colonizer on the smooth surfaces of the teeth.

Although the role of lactobacilli in the carious process is not well defined, it is believed that they are involved more in the progression of the deep enamel lesion (rather than the initiation); they are the pioneer organisms in the advancing front of the carious process, especially in dentine.

The role of Actinomyces spp

Root lesions differ from enamel caries in that the calcified tissues are softened without obvious cavitation. The association studies in vivo; in vitro experiment work with pure cultures; experimental work in gnotobiotic rodents even though Actinomyces spp (especially A. viscosus) predominate in most plaque samples taken from root surface lesions, some studies have reported both mutans streptococci and Lactobacillus spp. The sites from which these organisms were isolated appeared to have risk of developing root surface caries than other sites, so the role of Actinomyces spp in caries was not clear.

The role of Veillonella

Gram -ve anaerobic coccus present in significant number in supra gingival plaque samples. Require lactate for growth but unable to metabolize it, so they use lactate produced by other bacteria and convert it into weaker and less cariogenic organic acids, so it has beneficial effect demonstrated in vivo and in animal experiment but not in human.

From studies, there is specific type of bacteria to develop dental caries following the type of tooth surface:

- **Smooth surfaces**

S. mutans, S. salivaris, Actinomyces.

- **Occlusal fissures**

S. mutans, S. sanguinis, *lactobacilli*, *Actinomyces* spp. S. mitior

- **Approximal surfaces**

Actinomyces spp., Gram negative bacteria., Fewer streptococci.

- **Cervical surfaces**

Actinomyces spp., Anaerobic bacteria.

- **Root caries** (cervical)

Actinomyces, another anaerobic G -ve bacteria, and S. mutans.

Metabolism of plaque

Dental plaque: - it is a tenacious microbial community which develops on soft and hard tissue surfaces of the mouth, comprising living, dead and dying bacteria and their extracellular products, together with host compound mainly derived from saliva.

For metabolism to occur, a source of energy is required, for streptococcus. Mutants and many other acid forming organisms this energy source can be sucrose. Almost immediately following exposure of these organisms to sucrose, they produce

1-acids

2- An intracellular polysaccharide which provide a reserve source of energy for each bacterium.

3- Extracellular polysaccharide glucan (e.g., dextran) and fructan (levan).

The streptococcal are of a central importance in the metabolism of plaque because they are well equipped to survive in the fluctuating conditions. Within plaque, it has been estimated that the glucose concentration may vary over a 10000-fold range; the pH may shift from7.5-4.0, while oxygen is freely available at the plaque surface,

condition is entirely anaerobic close to the tooth surface. The streptococcal are well suited to flourishing under varying condition of' oxygen availability (facultative anaerobic). About acid, S. mutans is notable in combining the properties of being extremely **acidogenic** and **aciduric**. As consequence when there is a good supply of carbohydrate, S. mutans will produce many acids mainly **lactic acids** that will lower the plaque pH, continues to metabolize under the low pH condition while other competing species are disadvantage (different plaque species differ in the range of pH that they can tolerate), with the net result that the relative proportion of S. mutans in plaque population increase.

Prevention of dental caries

Patient evaluation

1- **Microbiological test in caries assessment**

Saliva samples can be used establish the numbers of Streptococcus mutans and Lactobacilli spp in the oral cavity. This can be done as follows: -

A paraffin wax for stimulation sample of mix saliva is collected, then in the laboratory the saliva is appropriately diluted and cultured on selective media (mitis salivarius bacitracin agar for S. mutans; Rogosa SLagar for lactobacillus spp). The number of typical colonies is then quantified and extrapolated to obtain the count per milliliter of saliva: -

a- High caries activity: $>10^6$ml S.mutans and /or >100 000/ ml Lactobacillus ssp.

b- Low caries activity: < 100 000/ml S.mutans and < 10 000 ml Lactobacillus.

2- **Assessment of dietary habits**

The major approaches to prevention of caries by stopping or reducing between meal consumption of carbohydrates or substituting non-cariogenic artificial sweeteners (sugar substitutes) like sorbitol, xylitol.

3- **Increase resistance of teeth**

Making the tooth structures was less soluble to acid attack by using fluoride and pit & fissure sealants to protect susceptible area of the tooth.

4- **Reducing cariogenic flora**

When the cariogenic flora decreases even in the present of sucrose the acid production will be decrease, this can be achieved by using oral hygiene aids, antibacterial agents, and possible immunization.

Preventing of infection

Disinfection of the sources (reduce the level of the bacteria in the mother) can be as far as possible reduce the risk of cross infection of the infant. That this approach is practicable was first demonstrated by treating expectant mothers to reduce their carriage of S. mutans by intensive professional oral hygiene, including chlorhexidine treatment, and giving dietary advice during pregnancy and after the birth of the child. A reduction in the salivary level of mutans streptococci could be demonstrated in mothers and this dramatically reduced the likelihood of their babies becoming colonized with S. mutans.

Mechanical Plaque Control

By Assist. Prof. Azhar AL-Kamal

Plaque control is the removal of microbial plaque and the prevention of its accumulation on the teeth and adjacent gingival tissues. It also deals with the prevention of calculus formation and leads to resolution of gingival inflammation. Thus, Plaque control is an effective way of treating gingivitis and therefore is a critical part of all the procedures involved in the treatment and prevention of periodontal disease. Plaque control includes mechanical procedures (includes tooth brushing and interdental cleaning aids and professional prophylaxis) and chemical agents which retard plaque formation.

In periodontal therapy, plaque control serves two purposes:

1. To minimize gingival inflammation.
2. To prevent recurrence or progression of periodontal disease in treated mouth.

The process of plaque control requires motivation on the part of the patient, education and instruction, followed by encouragement and reinforcement.

Mechanical plaque control aids include:

Toothbrushes: They were first introduced in China as early as 1600 B.C. Through years toothbrushes have undergone changes in many ways as possible. By early nineteenth century the handles were constructed from gold, Ivory, or ebony in which replaceable brush heads could be fitted. Nylon bristles came into use in 1938 to replace the natural bristles. Nylon bristles flex as many as 10 times more often than natural bristles before breaking; they do not split or abrade and are 1easier to clean. The shape and stiffness of nylon bristles can be standardized.

Objectives of toothbrushing:

1. To clean teeth and interdental spaces.

2. To prevent plaque formation.

3. To disturb and remove plaque.

4. To stimulate and massage gingival tissues.

5. To clean the tongue.

Types of toothbrushes:

- Manual toothbrush.
- Powered toothbrush.
- Sonic and ultrasonic toothbrush.
- Ionic toothbrush.

Manual toothbrush**:** It should be easily and effectively manipulated, inexpensive.

Parts of toothbrush:

1. **Handle:** The part grasped in the hand during tooth brushing.
2. **Head:** The working end of a toothbrush that holds the bristles.
3. **Tufts:** Clusters of bristles secured into head.
4. **Shank:** The section that connects the head and handle.

Toothbrush bristles either natural from hair of hogs or synthetic from nylon (not larger than 0.23 mm in diameter) which are uniform in size and elasticity, resistant to fracture. Rounded bristles end cause fewer scratches on the gingiva. The type of brush is a matter of individual preference. A toothbrush should be able to reach and clean most areas of teeth. For maintenance of toothbrush, most brushes wear out in three months and should be replaced, it should be stored in dry areas and cleaned in antiseptic mouthwashes.

Tooth brushing methods:

➢ **Bass method:** It is the most widely accepted and most effective method for dental plaque removal, adjacent and directly beneath the gingival margin. The technique is placing the bristles at 45° angle to the gingiva and move in technique is place the bristles at 45° angle to the gingiva and move in back-and-forth motions. Strokes are repeated around 20 times.

Advantages: Effective method for removing plaque from the cervical area beneath the height of contour of enamel, easy to learn, provides good gingival stimulation and recommended for patients with or without periodontitis.

➢ **Modified Bass technique:** This technique combines the circular motions of Bass technique with the sweeping motion of the Roll technique. It has sweeping motion from cervical to incisal or occlusal surface. The bristles are gently vibrated by moving the brush handle in a back-and-forth motion.

Advantages: Good interproximal, gingival and sulcus cleaning as well as good gingival stimulation.

➢ **Stillman's method:** The bristles are positioned apically along the long axis of the tooth. The edge of the brush head should be touching the facial or lingual aspect of the tooth. The brush is slightly rotated at a 45-degree angle and vibrated over the crown.

Advantages: It is used for massage and stimulation of gingiva and for cleaning the cervical area of the teeth.

➢ **Modified Stillman's method:** The bristles are positioned partly on the cervical portion of teeth and partly on the adjacent gingiva in an apical direction with an oblique angle to the long axis of the tooth. Roll the brush down to the crown of the tooth.

Advantages: It is recommended for cleaning in areas with progressing gingival recession and root exposure to prevent abrasive tissue destruction.

➢ **Charters method:** The bristles are placed at a 45-degree angle toward the occlusal or incisal surface of the tooth. The bristles should touch at the junction of the free gingival margin and tooth. A circular vibratory motion is then activated.

Advantages: It is recommended for temporary cleaning in areas of healing wounds after periodontal surgery and effective for cleaning around devices used to correct improper contact of opposing teeth and plaque under abutment teeth.

➢ **Scrub brush method:** The teeth are placed edge to edge while the brush maintains a 90-degree angle to the long axis of the tooth. The brush is then moved in a horizontal stroke. This technique is known to cause excessive toothbrush abrasion.

➢ **Roll technique or sweep method:** The bristles are placed at 45° angle and lightly rolled across the tooth surface toward the occlusal surface. The edge of the brush head should be touching the facial or lingual aspect of the tooth. Then with light pressure, the bristles are rolled against the tooth from the apical position toward the occlusal plane. This motion is repeated several times; then the brush is repositioned on the next teeth with bristles overlapping a portion of the teeth previously cleaned. The heel or toe of the brush is used on the lingual aspect of the anterior teeth. It is indicated for children and for individuals with limited dexterity.

Advantages: It works well for patients with anatomically normal gingival tissues.

➢ **Fones method or circular scrub method:** The teeth are clenched, and the brush is placed inside the cheeks. The brush is moved in a circular motion over both maxillary and manibular teeth. In the anterior region, the teeth are placed in an edge-to-edge position and the circular motion is continued. On 4the lingual aspect, an in-and-out stroke is used against all surfaces. This technique can be damaging if done too vigorously.

Advantages: It is recommended for children and physically or emotionally handicapped individuals.

➢ **Vertical method or Leonard's method:** The bristles of toothbrush are placed at 90° angle to the facial surface of the teeth. The teeth are held in an edge-to- edge position. Next the toothbrush is moved in a vertical, vigorous motion up and down the teeth. The maxillary and mandibular teeth are brushed separately.

Advantages: Most convenient and effective for small children.

Powered toothbrushes: These were introduced in 1960's. Powered toothbrushes are not superior to manual. Most powered toothbrush manufacturers do not recommend a specific brushing method. However, some guidelines for using a powered brush are available. It is recommended that the brush be positioned slightly differently for each surface of the tooth. Each tooth and corresponding gingival areas should be brushed separately, always with light, steady pressure. Pressure should never be exerted on the bristles of a powered toothbrush because this could damage the tissues.

The indications for uses are:

- Young children.
- Disabled patients.
- Individuals lacking manual dexterity.
- Patients with prosthodontics, or orthodontic treatment as well as implants.
- Patients on supportive periodontal treatment.
- Institutionalized elderly people.

Sonic and ultrasonic toothbrush: These types produce high frequency vibrations (200-400 HZ for sonic and 1.6 MHZ for ultrasonic), which lead to the phenomenon of disruption of bacterial cell wall (bactericidal) and aids in stain removal.

Ionic toothbrush: This type changes the surface charge of a tooth by influx of positively charged ions. The plaque with similar charge is repelled from the tooth surface and is attracted by the negatively charged bristles of the toothbrush. It

indicates a brush that aims to impart an electrical charge to the tooth surface with the intent of disrupting the attachment of dental plaque.

Effects of improper tooth brushing:

1. Gingival alterations include:

- Acute lacerations.
- Chronic alterations.
- Recession.
- Change in gingival contour.

Corrective measures: Use of soft toothbrush and change of brushing method.

2. Abrasion of the teeth: It means the loss of tooth substance produced by mechanical wear other than by mastication. The contributing factors are: hard toothbrush, horizontal brushing, abrasive agents in dentifrice, excessive pressure during brushing and prominence of the tooth surface labially or buccally. The abraded areas are on the cervical areas of exposed root but may occur on enamel.

Corrective measures: Recommend a less abrasive dentifrice, change the toothbrush method, and advise the patient to use soft texture bristles.

Interdental Cleaning aids: The toothbrush does not completely remove interdental plaque either in healthy or periodontal involved patients. Interdental cleaning is crucial to augment the effect of tooth brushing.

Factors affecting the selection of interdental cleaning aids:

- Type of gingival embrasures.
- Alignment of teeth.
- Fixed prosthesis or orthodontic appliances.
- Open furcation areas.
- Contact areas.

Dental floss: It is used to remove plaque from interproximal surfaces in which the

embrasure is completely occupied by healthy interdental papilla. There are many types either waxed, unwaxed, flavored or tape.

Function of dental floss:

1. Remove of adherent plaque and food debris to teeth and others.
2. Reducing gingival bleeding.
3. Improving oral hygiene.
4. Massaging the interdental papillae.
5. Helping in locating calculus, overhanging restorations, and proximal carious lesion.
6. Polishing of tooth surfaces during plaque removal.

Wooden tips: They are placed in the interdental space in such a way that the base of triangle toward the gingiva and the sides are in contact with the proximal surfaces.

Interdental brushes: These brushes are suitable for cleaning large, irregular tooth surfaces adjacent to wide interdental spaces and may also be used to clean furcation areas.

Miswak (Siwak): It provides both mechanical (bristles) and chemical (antimicrobial agents) measures for plaque control.

Oral irrigation devices:

These devices are beneficial in the removal of unattached plaque and debris. They may also be used to deliver antimicrobial agents such as chlorhexidine.

Gingival massage: Massaging the gingiva with toothbrush produces epithelial thickening and increased keratinization.

Tongue brushing: The tongue is anatomically perfect for harboring bacteria. The tongue can transmit organisms during toothbrushing and infection or reinfection of a periodontal pocket. For these reasons, the tongue, especially those with fissuring or prominent papilla, should be regularly cleaned. Commercial tongue cleaners,

made of plastic or flexible metal, are also available. They are curved so they can be placed over the tongue without touching the teeth. These instruments are swept over the dorsum of the tongue to remove bacterial plaque and debris.

The Carbohydrate

By assist. prof. Azhar AL-Kamal

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

1- **Monosaccharide (simple sugars): -** includes glucose and fructose which are found naturally in fruit, vegetables, and honey while galactose occurs only as a result of breakdown during digestion of lactose.
2- **Disaccharides** three main disaccharides are found are found in food include: -
   a- **sucrose** which formed when one molecules glucose combines with one molecules of fructose. It is refined from sugar cane sugar beets, and it is a major part of dietary sugar.
   b- **Lactose** (milk sugar) is formed from one molecule of glucose combine with one molecule of galactose.
   c- **Maltose** is formed when two molecules of glucose combine; it is mainly derived from hydrolysis of the starch.
3- **Polysaccharides**: - include Starch, which is composed of glucose found in rice, potatoes, peas, and about half of dietary carbohydrate are composed of starch.

Note:

- The term **sugar** comprises all mono and disaccharides while polysaccharides are not sugar.
- Fermentable carbohydrate: any carbohydrate that can be hydrolyzed by salivary amylase and subsequently fermented by bacteria.

Types of study providing evidence for the relationship between diet and caries development

1- **Observational** (epidemiological) studies in which relationships between disease and possible causative and confounding factors are observed.

   Studies concluded that caries prevalence and incidence is low among people consuming food with low sucrose. On the other hand,

an increase in dental caries were reported with increase sugar intake. During World Wars I and II, there was a significant drop in dental caries in numerous countries, because of reduction in the availability of sugar, as the government-imposed sugar rationing. Incidence of dental caries among native population (as Eskimos), was reported to be low, but increased dramatically after exposure to modern type of food.

2- **Interventional studies** in which diets of group of people are purposefully altered and the effect of this intervention observed. Such studies would not be possible to repeat today because of ethical constraints.

1-The Vipeholm study

The Vipebolm study was conducted shortly after the Second World on 436 individuals. War in an adult mental institution in Sweden between 1945 and 1953 (Gustafsson et al. 1954). The study investigated the effect of consuming sugar foods of varying stickiness (different oral retention times) and at different times throughout the day on the development of caries by measuring caries increment in subjects who consumed.

(1) Refined sugars with a slight tendency to be retained in the mouth at mealtimes only (e.g., sucrose solution, chocolate)

(2) Refined sugars with a strong tendency to be retained in the mouth at mealtimes only (e.g., sweetened bread)

(3) Refined sugars with a strong tendency to be retained in the mouth, in between meals (e.g., toffee).

The significance of mealtime consumption of sugar is also that salivary flow rate is greater at mealtimes due to stimulation by other meal component and therefore plaque acid may be more rapidly neutralized.

The main conclusions of the Vipeholm study

1- 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.

2- Consumption of sugar in–between meals was associated with a marked increase in dental caries.
3- The increase in dental caries activity disappears on withdrawal of sugar-rich food.
4- Dental caries experience showed wide individual variation.

2-Hopewood House study

It is a home in rural (orphanage) New South Wales Australia, in which about 80 children of low socioeconomic background were bought up from birth to 12 years. They had a lacto-vegetation diet, and sugar and flour products were virtually absent. The fluoride intake was low and oral hygiene measures absent. Once the period of close supervision ended the children developed the same caries rate as children in the state schools. The study concluded that the diet received up to 12 years did not protect the children from developing dental caries in subsequent years.

3- The Turku sugar study

In a 2-year dietary study, 125 young adults were divided into three experimental groups: sucrose, fructose, and xylitol. The purpose was to study differences in the caries increment rate as influenced by various sugars. Results showed a massive reduction caries in relation to xylitol consumption. Fructose was found to be less cariogenic than sucrose. It was suggested that anti-cariogenic properties of xylitol depend on its lack of suitability for microbial metabolism and physico-chemical effects in plaque and saliva.

4- Animal experiments

Animal experiments designed to investigate the relationship between dietary sugars and dental caries most used the rat model. Animal studies have added to the knowledge of the sugars/ caries relationship by showing: -

1- A clear relationship between frequency of consumption of a cariogenic diet and severity of dental caries.
2- Increasing caries with increasing sugar concentration.
3- Little difference in the cariogenicity of glucose, fructose, and maltose and increased cariogenicity of sucrose only when animals are super infection with S. mutans.

5- Enamel slab experiments

Enamel slab experiments use oral appliances that hold slab of bovine or human enamel. Plaque forms on the enamel slabs that remain in the mouth for 1-6 weeks. The slabs are exposed to the dietary factor being tested and the changes in enamel hardness or degree of demineralization may be measured. Enamel slab experiments have shown that sugars cause demineralization, while non-sugar sweeteners aid remineralization. Increasing the concentration of sugars and frequency of exposure to sugars increases demineralization.

6- Plaque pH studies

Plaque pH studies measure changes in the pH of plaque following consumption of a carbohydrate or carbohydrate-containing food. They measure acidogenic potential, which is taken as an indirect measure of cariogenic potential.

7- Incubation experiments

Incubation studies are simple in vitro test that measure if plaque bacteria can metabolize carbohydrate in a test food to produce acid. Rapid acid production and / or a low final pH is interpreted to mean that a food is potentially cariogenic, while a slow rate of acid production or higher final pH is likely to be of little clinical significance. All mono and disaccharides produce a final pH of below 4.5 when incubated with plaque.

In some incubation experiments, teeth, sectioned or powdered tooth enamel or hydroxyl apatites are incubated with the test substance and the plaque micro-organism to simulate the caries process. Potential cariogenicity is estimated from the extent of calcium and phosphorus release following incubation i.e., determinant the amount of mineral dissolved.

The sweetness of sugars

All sugar contributes sweetness to food, but the relative sweetening powder varies among sugars. In general, the more easily the sugar dissolves in water the greater its sweetening powder. For example,

fructose is 75% sweeter than any other sugar. It is soluble in water; difficult to crystallize, as a result it's expensive, and it's useful in syrup. At the other extreme, the least sweet, least soluble sugar is lactose. Lactose is seldom if ever used as sweeteners because it is almost impossible to dissolve in the food to be sweetened.

It is difficult to determine whether taste is genetically linked, acquired in uterus, neonatal, or influenced by visual, auditory, or taste stimuli during infancy, early childhood or even adulthood. Theoretically an individual can initially acquire and refine taste desire in any of the following stages:

1- in uteri 2- during breast or bottle feeding 3- while passively being fed solid 4- while more actively seeking different nonspecific foods 5- while purposely seeking specific food.

Taste buds are present and functioning before birth, a fact demonstrating by injection sweetening agents into the amniotic fluid results in an increased rate of swallowing by the fetus. At birth, infants show a taste preference for sucrose, and their taste cells are more responsive to sucrose than other sugar and the newborn baby respond unfavorably to a bitter substance.

Factors affecting food cariogenicity

Cariogenicity is ability of producing or promoting the development of caries lesion. The cariogenic potential of food is to foster caries in humans under conditions conductive to caries formation. The cariogenic potential of foods and beverages is based primarily on ability of food to decrease plaque pH by acid produce by cariogenic bacteria. This property of food is term acidogenesis.

1- Types of carbohydrates

There seems to be little difference in the cariogenicity of glucose, fructose, and maltose, if evidence from animal, enamel slab, plaque pH is considered, but the same source of evidence show that **lactose** is less cariogenic.

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

1- Low caries prevalence in population with low sucrose intake.
2- The decline in caries prevalence during wartime sucrose shortages.
3- The rise of caries prevalence with increasing availability of sucrose
4- Archaeological evidence of low caries prevalence in eras before sucrose became freely available.
5- Low caries prevalence in disorders of sucrose metabolism (hereditary fructose intolerance).

Sucrose is unique because it is a substrate to produce extracellular storage of polysaccharides (fructan and glucan) and insoluble matrix polysaccharides (mutans) by cariogenic bacteria S. mutans. Thus, sucrose favors colonization by oral micro-organisms and increases the stickiness of the plaque allowing it to adhere in large quantities on the tooth. In the absence of sucrose S. mutans cannot colonize in the mouth, therefore its plaque count appears to depend on the sucrose content of the diet. Sever reduction in the dietary sucrose S. mutans to decline in number or disappear from plaque.

Starch

The starch (granules of plants) is slowly attacked by salivary amylase because its insoluble form and protected by cellulose membrane, therefore the cariogeniciyty of uncooked starch is very low. Heating of starch in cooking, cause a partial degradation to a soluble form, this can be further broken down by saliva amylase to maltose and glucose. it is a slow conversion and hence dietary starch alone is much less important than dietary sugars as a cause of dental caries.

- Cooking starchy food such as rice, potatoes are of low cariogenicity in humans
- The cariogenicity of uncooked starch is very low.
- Finely ground and heat-treated starch may cause dental caries, but the amount of caries is less than caused by sugar.
- The addition of sugars increases the cariogenicity of cooked starchy foods. Foods containing cooked starch and substantial

amount of sucrose appear to be as cariogenic as similar quantity of sucrose.

- Polysaccharide molecules are too large to diffuse into the plaque, low molecular weight of carbohydrate after degradation by amylase become available for bacteria fermentation.
- 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.

2-Physical form of food and clearance time

Physical and organoleptic properties (particle size, solubility, adhesiveness, texture, and test) are important for carogenicity, they influence eating pattern and oral retention of the foods. Diet that results in the greater retention of refined carbohydrate over the longest period are the most cariogenic.

The texture of the diet is also important for both salivary secretion and elimination of fermentable carbohydrate from the oral cavity. A diet that requires through 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 tend to be retained in the oral cavity and eliminated slowly.

The carbohydrate in various drink is 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 and for chewing gum to15-20 minutes for other sweets.

Key pints

1- The frequency and timing of intake of sugary foods and drinks are important determining factors in caries levels. Such foods should be consumed with main meal because of oral clearance time is quicker, the salivary flow is increased, and dilution increased (pH is minimized). Salivary flow rate reduces during sleep and therefore bedtime is worst time consume sugary products.
2- Sugar-free medicines should be presented for children whenever possible.

3- To speed up carbohydrate clearance toothbrush should be immediately done after meal, or induction of rapid salivary flow by mechanical stimulation through eating tough or highly flavored foods at 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 while mouth rinsing with water has a very limited effect.
4- Frequent intake sugar will induce a prolonged and intense acid attack on the tooth surface' moreover, the time available for remineralization is thus decreased i.e., the pH of dental plaque falls rapidly when sugar is 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.

Saliva and Dental Caries

By assist. prof. Azhar AL-Kamal

The fact that the teeth are in constant contact with and bathed in saliva would suggest that this environmental agent could profoundly influence the state of the oral health of a person, including the carious process.

Saliva is a unique complex and important body fluid. It contains several systems to protect oral mucosa and the whole body from oral infections. Saliva in daily use is synonymous with spittle, whole saliva, mixed saliva, and oral fluid.

Saliva is the glandular secretion which constantly bathes the teeth and the oral mucosa. Pure saliva secreted by the oral glands is sterile until it is discharged onto the mouth. Salivary glands are exocrine glands and are classified according to the nature of their secretion or their duct system. Of the major salivary glands, parotid glands are the largest; the sub mandibular gland is smaller than the parotid glands. The sublingual gland is composed of several smaller glands. The minor glands are situated on the tongue, palate, and buccal and labial mucosa. These glands produce 10% of the total volume of saliva. The function of the minor salivary glands is also important since about 70 % of the total volume of salivary proteins is secreted by them. They also contribute fluoride that bathes the teeth and enhances caries resistance. The parotid glands are serous glands; upon stimulation, they produce watery saliva with high content of enzymes like amylase and lipase, while the secretions of sublingual glands are predominantly mucous, mucin-rich fluids, as same as of minor salivary glands. While the secretions of submandibular glands are a mixture of mucous and serous fluids.

Oral fluid is made of secretions of salivary glands (major & minor), gingival exudates, food debris desquamated epithelial cells and microorganisms, transudate of the mucous membrane and mucous from nasal cavity and pharynx, sometimes it may include acid from the stomach in cases of gastric reflex.

Salivary constituents- :

It is mainly composed of water 99.4% - 99.5% in addition to the - :

1 -Inorganic constituents- : Positive ions ex- Ca, H, Mg, k.

Negative ions ex- Co2, C, CL, F, Phosphate & Thiocyanate.

2 -Organic constituents: - CH & Glucose.

3 -Lipids: - Cholesterol, Lecithin.

4 -Nitrogen – Non-protein: - Ammonia, Nitrites, Urea & Amino acids

5 -Nitrogen – Protein: - Globulin, Mucin.

6 -Miscellaneous: - Peroxide, enzymes (Oxidases, Proteases, Amylase & Maltase) .

The function of saliva - :

1 -Physiological function (lubrication, digestion, oral clearance, speech & taste).

2 -Buffer capacity.

3 -Maintenance of health of oral tissues.

4 -Antimicrobial prosperities.

5 -Reservoir for ions facilitating remineralization of teeth.

Salivary flow rate- :

In healthy adults about (0.5 – 1 ml) of saliva is present at any time in the mouth. A total volume of (1 – 1.5 L) of saliva is secreted daily. The normal salivary flow rate for unstimulated (resting) saliva is (0.3 ml/min), while for stimulated saliva is (2 ml/min). However, there is a wide individual variation.

Factors affecting salivary constituents- :

1- **Saliva flow rate:** - the type and conc of organic and inorganic constituents of saliva differ between stimulated and unstimulated saliva. Ex: - the level of bicarbonate increases from 1 mmol / L in unstimulated saliva to 60 mmol / L in stimulated. Levels of Ca, Na & CL will increase also, and other minerals fall in conc in stimulated saliva such as Mg, K, Phosphate.
   Note: 50% of stimulated saliva is secreted from parotid gland, while for unstimulated saliva 69% secreted from the submandibular gland followed by the parotid and other gland.
2- **Natural of stimulation**: - salivary gland innervated by parasympathetic and sympathetic. Parasympathetic stimulation increases in water and electrolyte levels, while sympathetic stimuli increase mucous secretion. We have three types of stimulation chemical, mechanical & psychological. Each may affect the composition of saliva ex: - the taste of salt stimulates protein secretion. Acids is a potent stimulus for secretion and leads to the production of alkaline saliva.
3- **Duration of stimulation**: - increase time of stimuli cause an increase in water, bicarbonate & protein but decrease chloride.

4- **Age**: - with age, there is an increase in Ca & phosphate conc.
5- **Others**: - medication, diseases & exercises.

Factors affecting salivary flow rate- :

1- Nature of stimulation: - e.g.: mechanical stimulation causes more watery saliva.
2- Gland size: - salivary flow rate is more in males than female due to larger gland size,
3- Age: - salivary flow increases with the aging process.
4- Drugs: - antidepressants & analgesics reduce flow rate .
6- Radiation: - decrease salivary flow following head & neck radiation.
7- Others: - body posture (flow is greater while standing than when sitting; the flow is greater when sitting than when recumbent), time of the day (light deprivation, such as blindfolded, decreases flow) & seasons (flow being lower in warm weather & higher in the cold).
8- Smoking: - decrease flow rate especially in long term smoking.
9- Fear: - the flow of saliva can be greatly depressed by fear.

Saliva and dental caries:

Many studies on animals showed that de-salivated experiment animals fed cariogenic diet develop rampant caries and many observational human studies also showed an increase in dental caries severity with reduced salivary flow rate.

There are many reasons explain the increase in incidence and severity of dental caries associated with the reduction of saliva:

1- **Reduction in the quantity of saliva**; thus, its protective constituents in addition to the reduction in the important physiological function that is the oral clearance. Salivary constituents involve Calcium & Phosphate, the important elements in remineralization of teeth. In addition to elements as fluoride. Zinc, Strontium which may be present in saliva and aid in remineralization of outer enamel surfaces. An important function of saliva is to dilute substances introduced in the oral cavity through the salivary oral clearance. Saliva enhances the elimination of food debris, CH, & acid produced by cariogenic bacteria.
2- **Reduction in buffer system**; the salivary buffer system in the saliva composed of bicarbonate / carbonic acid & phosphate. In addition to urea & protein. The buffer means a solution that tends to maintain constant pH. The acid produced by bacteria plaque leads to fall in the PH to even less than 5.5. This pH is known as the critical pH of saliva (at which there will be a dissolution of enamel surface). By buffer action of saliva, the pH increases to normal which is around 7 PH.

3- **Alteration in amount and bacteriological composition**; the reduction in saliva leads to the reduction in the oral immune system. The reduction in the buffer capacity leads to an acidic medium in the mouth favoring the growth of bacteria especially cariogenic bacteria as mutans streptococci .

Buffer system of saliva:

Buffer systems mean a solution that tends to maintain constant pH. The salivary buffer system is composed of bicarbonate/ carbonic acid and phosphate. In addition to urea and protein. Buffers differ between stimulated and unstimulated saliva. In stimulated saliva the main buffer is the bicarbonate. When an acid is added bicarbonate (which is present because of carbohydrate metabolism) releases a weak carbonic acid, which rapidly decompose to water and carbon dioxide that leave the solution. The increase in the carbonic acid concentration led to more carbon dioxide escape from saliva making it possible for more bicarbonate to bind to hydrogen ions (H^+) which will end in the removal of the acid.

$$HCO^{-3} + H^+ \longleftrightarrow H_2CO_3 \longleftrightarrow H_2O + CO_2$$

In the unstimulated saliva the main buffer is phosphate. When the pH decreases below 5.5, dissolution of apatite crystal takes place freeing phosphate. Phosphate attempts to restore the pH balance, increasing pH level and allowing for the remineralization of enamel. Other buffers are present as urea, protein, and peptides, all play a role in buffering saliva.

Note: pH is the negative logarithm of the hydrogen ion concentration. The pH of saliva ranges 6.7-7.4 with great individual variation.

The reduction in the salivary flow rate is known **as hyposalivation** in severe cases it is known as **Xerostomia**. Xerostomia may lead to increased severity of dental caries and even sometimes rampant caries .

Management:

1- Oral hygiene measures and dietary restriction
2- Avoid drugs lead to Xerostomia.
3- Use fluoridated products.
4- Use of artificial saliva.

Artificial saliva is not a perfect substitute for natural saliva (which is very complex physically and chemically), it does—when used regularly—help moisten the oral tissues, relieving the discomfort caused by dry mouth. Speaking, chewing, and swallowing are made easier when the mouth is moist. Artificial saliva normally contains a

mixture of buffering agents, cellulose derivatives (to increase stickiness and moistening ability) and flavoring agents (such as sorbitol). However, they do not contain the digestive and antibacterial enzymes and other proteins, or minerals present in real saliva. Saliva substitutes are quickly swallowed and, therefore, the moistening and lubricating action has a limited duration and repeat applications may be needed. Although saliva substitutes will not cure dry mouth, they can provide temporary relief of some symptoms. Products are available in an aerosol or a liquid that is squirted into the mouth.

Oral Immune System

Immunity is the sum of all naturally occurring defense mechanisms that protect humans from infectious and other diseases .

There are two types of resistance mechanisms - :

1 -Non-specific (innate).

2 -Specific (acquired).

The soft and hard tissues of the oral cavity are under protection by both non-specific and specific immune factors. The function of these protective factors is to- :

1- Limit the microbial colonization of the oral surfaces.
2- Prevent the penetration of noxious substances through the surfaces and ensuing damage to the underlining tissues.

1-Non-specific immune factors

These are factors present in saliva that include lysozyme, the lactoperoxidase system, lactoferrin, high molecular weight glycoprotein and other salivary components that may act as bacterial agglutinins. Unlike antibodies, these nonspecific factors lack any aspect of immunological memory and are not subject to specific stimulation . The non-specific immune may interact with specific salivary immune factors (immunoglobulin) resulting in amplification in respect to their activities.

1- Lysozyme

It arises from both major and minor salivary gland, gingival crevicular fluid and salivary leukocytes. It is present in newborn babies at a level equal to those of adults and may exert antimicrobial function already before tooth emergence. It can hydrolyze the specific bonds in exposed bacterial cell walls causing cell lysis and death. Lysozyme has been proposed as a lytic factor for bacteria to which immunoglobulin have bound. Lysozyme and other antibacterial systems in saliva exclude susceptible invading pathogens, which are

not adapted to oral conditions. It also, aggregate cell suspension of some bacteria species in addition this enzyme may enhance the action of IgA.

2- Peroxidase system

Peroxidase in saliva is produced in acinar cells of parotid and submandibular glands but not in minor salivary glands. Peroxidase enzymes catalyze the reaction of bacterial metabolic product, hydrogen peroxide (H_2O_2) with salivary thiocyanate (SCN^-) originates from serum to produce hypothiocyanite ($OSCN^-$). This system protects human host protein and cells from hydrogen peroxide toxicity, it is highly toxic to bacterial enzymes required energy production and the bacterial activity is inhibited. The more hypothiocyanite present in saliva, the less can dental plaque produce acids after stimulation with glucose. Peroxidase systems are effective against a variety of microorganisms, such as mutans streptococci, lactobacilli, yeasts, many anaerobes, and some viruses.

3- Lactoferrin

It has antibacterial activity. LF is secreted by serous cells of major and minor salivary glands, also polymorphonuclear leukocytes are rich in LF. Ferric iron (Fe^{3-}) is an essential microbial nutrient. Lactoferrin is iron-binding glycoprotein making ferric iron unavailable for microbial use. This phenomenon is known as nutritional immunity. Lactoferrin in its unbounded states has direct bactericidal effect on some microorganisms including mutans streptococci.

4- Mucins

They can agglutinate bacteria. It inhibits the adhesion of bacterial cells to soft tissue surfaces by interacting with adhesions. Mucins also interact with hard tissue surfaces and mediate specific bacterial adhesion to the tooth surface. Some studies showed that increased viscosity of saliva might reduce the clearance activity of saliva thus increase caries severity.

5- Statherrin

It presents in both submandibular and parotid saliva and the key to activity of statherrin is to prevent precipitation of calcium phosphate in the salivary duct and oral fluid to maintain supersaturating, to prevent the formation of duct stones and calcium phosphate crystal growth on tooth surfaces. It aids in remineralization of teeth by inhibition of calcium-phosphate crystal growth.

6- Proline rich protein (PRPS)

Like statherrin is needed to inhibit spontaneous precipitation of calcium phosphate salts in the salivary glands and their secretion. The inhibitory activity of (PRPS) can be explained

by their adsorption onto hydroxyapatite. They are present in the initially formed acquired pellicles .

7- Histatins

This group of small histidine-rich proteins is potent inhibitors of candida albicans growth and has activity against streptococcus mutans. It modulates the precipitation of behavior of calcium phosphate.

II- Specific immune factors

These factors immunoglobulin may be directed at specific bacterial molecules which may be important in the biological activity of the target organisms. The different Ig classes present in saliva which are: - IgA, IgG, IgM, IgD & IgE.

Immunoglobulin IgA

Salivary IgA is produced by plasma cells located in the major salivary glands. It is the predominant salivary immunoglobulin which under normal condition is the only immunoglobulin secreted in saliva. In an individual with gingivitis or periodontitis, the inflammation in periodontal tissue will result in the leakage of serum proteins which include IgG, IgA, IgM and complement factors. IgA present in saliva differs from serum IgA with molecular structure, while serum IgA occurs mainly in the monomeric form, IgA in saliva composed of dimeric associated with J chain and glycoprotein referred as secretory component (SC). The s-IgA is present also found in tears, genital urinary secretions, bronchial secretions, and breast milk. The ability of s-IgA to inhibit adherence of bacteria to dental enamel appears to be related to its ability to bind to surface adhesion of bacteria as well as to neutralize their negative surface charge. On the other hand, IgA has been shown to bind to mutans streptococci facilitating bacterial aggregation and removal from the oral cavity. Secretary IgA molecules are multivalent antibodies and can prevent the adverse effect of bacterial toxins and enzymes .

Note: - the IgA is synthesized by immunocytes cells (plasma cells and mast cells) while the Sc components originated in the glandular secretary epithelium.

Immunoglobulin IgG

It is the predominant immunoglobulin in the blood, lymph, peritoneal fluid, and cerebrospinal fluid. It accounts for approximately 75% of the total serum immunoglobulin in

normal adults and is the most abundant antibody produced during the secondary humoral immune response in the blood. IgG is the only class of immunoglobulin that can cross the placenta in humans, and it is responsible for the protection of the newborn during the first months of life. The direct exposure of the tooth surfaces to gingival fluid, dental plaque, in addition to salivary immunoglobulin, be exposed to significant amounts of serum immunoglobulin. It is, therefore, believed that serum IgG has the potential to modulate the oral colonization by plaque-forming bacteria, especially during tooth eruption. A significant proportion of the immunoglobulin molecules, particularly IgG, that become incorporated in dental plaque occur as fragments because of proteolysis degradation by enzymes excreted by plaque bacteria, IgG is effective against bacteria, viruses, and fungi.

Effects of IgA & IgG response in relations to the protection of the tooth surfaces- :

1- Inhibition of bacterial adherence by (a) Blockage of bacteria adhesions or (b) Bacterial aggregation.
2- Inhibition of bacterial enzymes.
3- IgA has anti-inflammatory activity in gingival tissue, while IgG has the property of induction of inflammatory in gingival tissues and opsonization of bacteria thus facilitating bacterial phagocytosis and killing.

Ig may transfer to the infants by breast feeding. The infant may receive as 1 g/day of s-IgA. It interferes with the establishment of MS in the early part of life.

Immunization of dental caries

Three different approaches have been used to achieve caries immunity - :

1 -The classical route of immunization which elicits serum IgG and to a lesser extent IgM & IgA.

2 -Various immunization procedures aimed at eliciting S-IgA antibody response in saliva.

3 -Passive immunization with orally applied antibody.

Protection against dental caries by immunization would be achieved by immune components from serum by IgA antibodies in salivary secretion or by a combined effect of serum and salivary components.

Different types of antigens can be used as; the whole bacterial cell as S mutans, glucosyltransferase enzyme, cell wall protein, dextran-binding protein, etc. In experimental animals, there are different routes of immunization either by: -

1- **direct stimulation** of s-IgA through repeated injections of the animal vicinity of each parotid and/or submandibular gland with an antigen vaccine.
2- **Indirect stimulation** of s-IgA by feeding the animal, the antigen causes an indirect stimulation of s-IgA. The antigen when meet the gut associated lymphoid tissue in particular Payer's patches in the small intestine will stimulate antibodies (s-IgA), secreted in saliva, tears, and milk.
3- **Stimulation of serum immunoglobulin** by subcutaneous injection of the animal by selected antigen to stimulate serum IM like IgG and to lesser extent IgM, IgA. The IM reach oral cavity mainly by transduction with crevicular fluid.

Vaccination

Vaccines are immune-biological substance designed to produce specific protection against a given disease. It stimulates the production of a protective antibody and other immune mechanisms. Vaccines are prepared from live organisms, inactive or killed organisms, extracted cellular fraction, toxoids, or a combination .

Two points must be considered in relation to caries vaccine- :

1- For developing a vaccine against any infectious disease, the responsible microorganisms must be identified. Lactobacilli were used as the immunogen in the 1930s while after the reduction of Streptococcus mutans in 1960, this bacterium becomes the target of all immunization experiment.
2- The identification of any antigen preparation that combines maximal immunogenic activity with minimal undesirable side effects .

The most important thing in selecting the vaccine, i.e., antigen is its safety, effectiveness, and prolonged effect. Serum antibodies (IM) may be an effective approach to vaccinations, but there is a possibility of cross-reactions with the heart muscle. S mutans possess an antigen component that stimulates the cross-react with the heart muscle. The stimulation of IgG may increase inflammatory reaction thus may increase penetrability for the antigen which may jeopardize gingival protection against plaque antigen. However, the secretory immune response lasts for a short time. Vaccination is a cheap method for protection against dental caries.

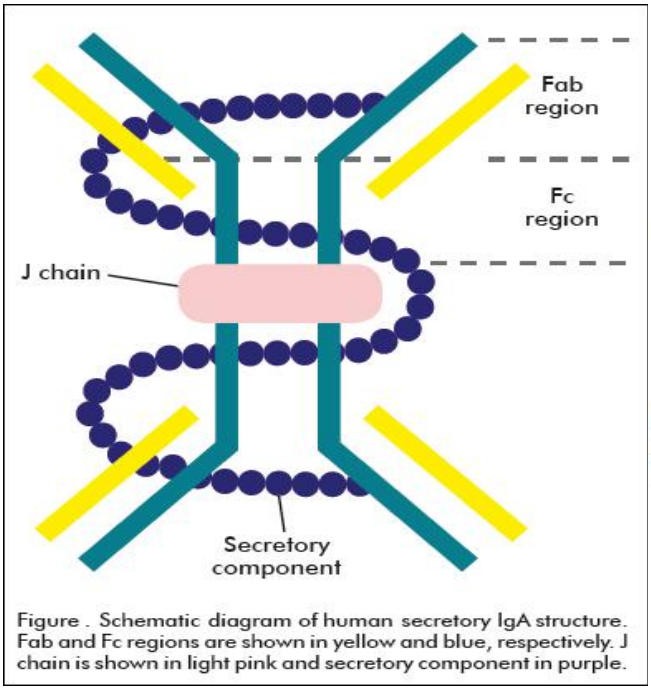
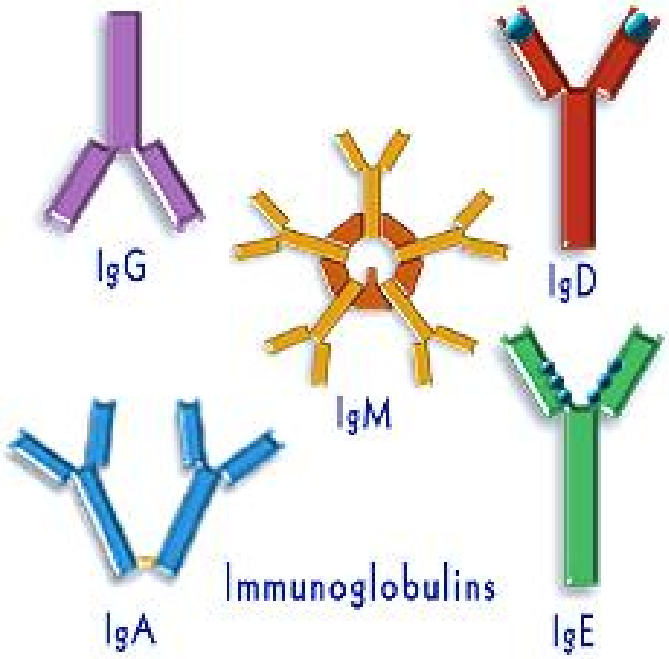


Figure . Schematic diagram of human secretory IgA structure. Fab and Fc regions are shown in yellow and blue, respectively. J chain is shown in light pink and secretory component in purple.



Systemic fluoridation Lec-3

By Assist. prof Azhar Alkamal

Community water fluoridation, also referred to as **fluoridation**, is defined as **the upward adjustment of the natural fluoride level in a community's water supply to a level optimal for dental health**. It is a population‐based method of primary prevention that uses piped water systems to deliver a low concentration of fluoride over frequent intervals during the day. By consuming the water directly or indirectly through incorporation in foods and beverages, consumers accrue preventive benefits regardless of age or socioeconomic status. Fluoridation has been cited by the Centers for Disease Control and Prevention (CDC) as one of the 10 great public health achievements of the 20th century.

Communal water Artificial Fluoridation

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. The studies of Dean and others up to 1943; shown that fluoride was associated with a lower prevalence of caries, and that there was a sound basis for hypothesis that the introduction of fluoride into a water supply would result in a lower communal prevalence of 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.**

In 1901 Dr Fredrick noticed that many of his patients who had lived in Colorado (USA) had a permanent stain on their teeth named Colorado stain also he noticed a low caries severity among those people. Dean discovered that F conc in drinking water affected the severity of mottling & dental caries severity. Two important conclusions were reached:

1- The number of carious teeth & conc of F is inversely related.
2- The amount of F in water & mottled enamel are directly related.

Dean conducts his study among 7257, 12–14-year-old in seven cities in USA. This was to explore the association between F level in drinking water & severity of both dental caries and dental fluorosis. Results revealed a maximum reduction of dental caries at level of F of 1 ppm, at

this level dental fluorosis will involve 10% of the population, but it is of the very mild type with no practical aesthetic significant. Increasing F level in drinking water will cause a dramatic increase in dental fluorosis but with no further reduction of dental caries. Thus, the optimal level is **the level of F in drinking water causing maximum reduction of dental caries but with no clinical signs of dental fluorosis**. Epidemiological & observation studies showed that a more sever dental fluorosis do develop sometimes in certain area of hot climate at 1ppm.

Fluoride was firstly added to water supply in 1945 in Michigan, caries reduction was reported to be 55%. In USA, now more than 126 million people are receiving systemic fluoridation. The previous year (1944) a baseline study comparing Grand Rapids with the neighboring town of Muskegon had found similar decay levels in deciduous and permanent teeth in both areas. Six years later, surveys indicated that decay levels in 6-year-old children (i.e., those born since fluoridation commenced) in Grand Rapids was almost half of that of Muskegon, in 'non-fluoride' Muskegon the average number of teeth with decay experience was 5.7, compared with 3.0 in 'fluoridated' Grand Rapids.

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).
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

From epi studies concentrating on water fluoridation, it was concluded that: -

1- Artificial water fluoridation is effective in caries reduction in similarity to naturally fluoridated area.

2- Caries reduction involved primary, permanent teeth as well as root caries. The reduction is more in permanent teeth compared to primary because of their shorter maturation period.
3- Communal water fluoridation is public health measure. All people in the community can gain the benefit from water fluoridation. No effort is needed by recipient to prevent caries.
4- It is cheap & successful method of preventive dental caries.
5- A reduction of periodontal disease was also reported in fluoridated area.

Disadvantages of water fluoridation

1. Political and/or emotional objections to water additives.
2. Possibility of mild to moderate fluorosis if other sources of fluoride are ingested
3. 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 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].

Medical aspect of Water Fluoridation

Medical aspect or Safety of water fluoridation was research concern from the time of fluoride's identification in water in 1931.

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.

Dental fluorosis

It is a developmental hypoplastic defect caused by excessive fluoridation during the period of tooth formation. It is the first sign of chronic toxicity appears clinically as a white spots or lines involving incisal edge or cusps of posterior teeth or as a white opaque or brown area, in severe cases a corroded appearance will occur. In principle increased exposure to F during period of tooth formation led to increase in enamel porosity. In severe cases the fluorotic teeth are highly porous because of increase of inter crystalline spaces; these spaces are occupied by water & protein more than enamel. In more sever condition changes involve enamel as well as dentine. After eruption of teeth, although the surface layer is well mineralized it is susceptible to mechanical trauma leading to break down of the outer enamel surfaces.

The exact cause of **hypoplasia** is not clear it may be attributed to:

1- Altered metabolism in any or all phase of the enamel formation.
2- Altered ameloplastic activity.
3- Interference with crystal nucleation or growth.
4- Faulty enzymatic factor.

Factors affecting severity of dental fluorosis:

1- F conc in drinking water: A direct relationship is present between dental fluorosis & level of F ingested.
2- Total amount of F ingested: F ingested from water, food, inhalation because of pollution all affect severity of dental fluorosis. The total amount of water intake is affected by temp. in hot area there is an increase ingestion of F due to increase intake of water thus increasing the risk of dental fluorosis, the opposite is true in cold area.
3- Duration of exposure to F: Excessive intake of F for a long time as eight years during the period of tooth formation may increase the severity of dental fluorosis. Teeth mineralized early in life develop less dental fluorosis, thus posterior are more than anterior teeth. Also, primary teeth are affected less severely compared to permanent teeth, due to shorter maturation period. In addition, enamel maturation & calcification of primary teeth take place in

the intra uterine life, studies showed that the placenta do regulated the amount of F reaching the fetus, also F concentrated in bones of the mother& the fetus more than teeth.

Others: Dental fluorosis was found to increase among children with mal nourishment. The exact cause for this is not clear

Dean's Fluorosis Index – Modified criteria…was introduced by Dean in 1942.

Classification	**Criteria**
Normal	No dental fluorosis
Questionable	The enamel discloses slight aberrations from the translucency of normal enamel ranging from a few white flecks to occasional white spots.
Very mild	Small, opaque, white areas scattered irregularly over the tooth, but not involving 25% of the tooth surfaces.
Mild	The white opaque areas in the enamel of teeth are more extension. But not involve as much as 50% of tooth.
Moderate	All enamel surfaces of teeth are affected and surfaces subject to attrition show wear, brown stain is a disfiguring feature.
Sever	all enamel surfaces of teeth are affected and hypoplasia Is so marked that the general form of the tooth may be affected, pitting surface with brown stain.

Alternative water fluoridation

There are alternative methods to provide F systemically: -

1- School water fluoridation or home water fluoridation.

2- Dietary fluoride supplements by:
   a- Fluoridated tablets or drop or lozenges.
   b- Fluoridated salt.
   c- Fluoridated milk or juice.

School water fluoridation:

This method was first applied in USA (1954). The optimal level of F here is about **4.5 times** the optimal amount in the community. This because of

- Children spend only 5-6 hours in school.
- They enter the school at 6 year- old.
- Only a part of daily water intake is consumed.

Advantage of school water fluoridation:

1- Technically feasible.
2- Low in cost.
3- No effort is needed by recipients.

The disadvantage of this method is that fluoridation started late in life, that is at 6 years of age, & there is an interruption of fluoridation due to holidays and weekends. A maximum benefit of systemic water fluoridation is by early intake of F from first years of life till13-15 years of age.

The home water fluoridation is also of beneficial in caries prevention; however, the level of F in water is like communal water fluoridation.

Fluoridated supplements:

1- Tablets, drops & / lozenges:

This is especially prescribed for children with high risk to dental caries like handicapped children or those with serious illness as blood disorder, cerebral palsy &others. This method is an effective measure to prevent or reduce dental caries. It's taken daily from the first years of life till 13-15 years, reduction of dental caries can be reached 50-80%.

A variety of supplements are present in form of NaF (given daily).

- **Liquid** form for infants and young children, conc are 0.125 mg F/ drop, 0.25 mg F / drop, & 0.5mg F/ drop (10 drops= 1 mg F/ L = 1ppm).
- **Liquid form with vitamins** as A, D, E, B1, B3, B6, B12 & Iron, prescribed to malnourished children only
- **Tablets** without vitamins, it can chew then swallowed.
- For school children more than 6 years of age as **mouth wash** fluoride of 5 ml. the child is asked to rinse his mouth first for one minute then swallow to have a topical & systemic effect.

In prescription of F tablets several **factors should be taken**:

1- F content of the water supply (communal or bottled water). It should be applied only in non-fluoridated area or with low F level.
2- Age of the child.
3- Cooperation of parents.

Fluoridated tablets

NaF = 2.2 mg (1mg F)

1.1 mg (0.5 mg)

Birth –6 month	non
6m.-3y	0.25 mg/day
3-6y	0.5 mg/day (½ of 2.2 mg tab)
6-13y	1 mg/d

Another program

4- Started at birth given 0.25 mg /daily then at 2-4 years given 0.5 mg, then at 4-year-old given 1 mg till 13-15 year- old.
5- Started at 3 years of age given 0.5 mg /day till 13-15 years.

Instructions

1- Given daily (once or twice).
2- Tablets crushed between teeth.
3- Each bottle contains no more than 264 tablets, to avoid acute toxicity after the accidental ingestion of fluoride tablets.
4- Dentifrices used should be without F, or with low F conc.

Fluoridated salt

It was introduced first in Switzerland, 1955. It is considered next to water fluoridation regarding caries reduction. F is added to salt inform of NaF or CaF_2 in different doses 200, 250, 350 mg F /kg of salt for domestic use or bakeries.

Advantage of salt fluoridated are:

6- Low cost
7- Ease of implementation
8- No personal efforts are needed.
9- Effective in caries reduction for permanent & deciduous teeth.

Disadvantage: children would start to use salt too late in life, or they used to take small amount of salt.

Fluoridated milk:

Human and bovine milk contain a low level of fluoride it is about 0.03 ppm. Milk is a good food for infant and children, it is a suitable vehicle for supplementary F to children, it is an excellent source for calcium & phosphorous in addition to vit D. milk is essential for development of bones &teeth.

The concentration of fluoride in the milk is (2.5-6) ppm as calcium in milk may react with F reducing the amount of free ionic fluoride absorbed. The bioavailability of F from milk is in like water, other studies showed that milk may be retard the absorption of F from GIT but does not prevent F absorption. Fluoridated milk can be used in home & school programs, with caries reduction of 70%. The disadvantages of milk fluoridation are the high cost & some children dislike milk, so a fluoridated juice can be used.

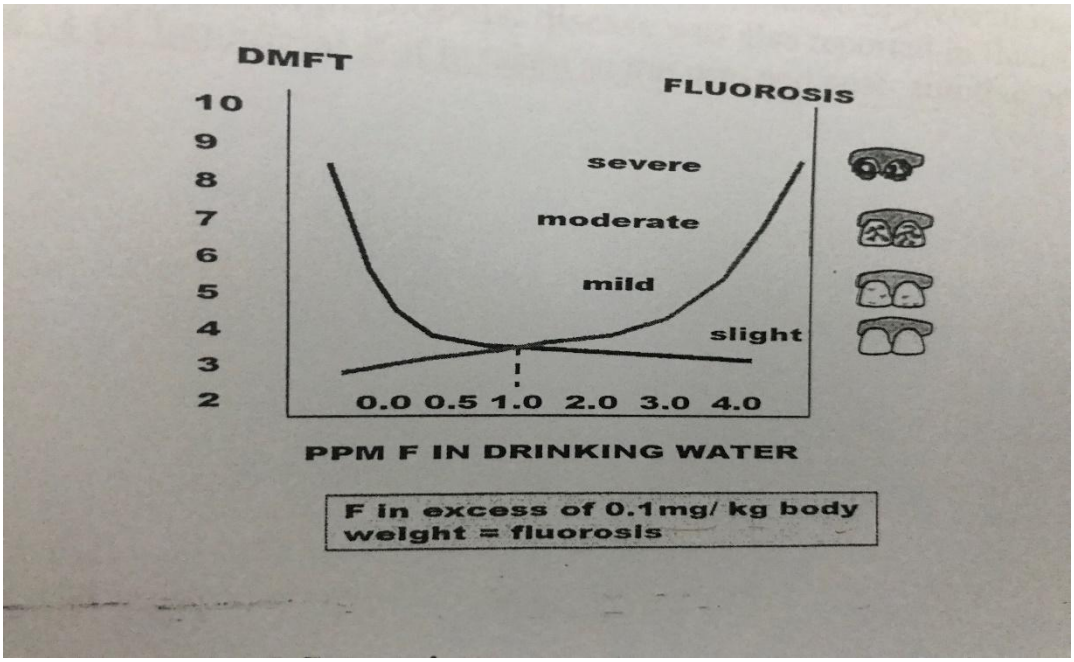


Table (1): Effect of fluoride in water on human health when consumed for longer durations

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)

Nutrition and Oral Health

By assist. prof. Azhar AL-Kamal

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 while diet refers to food and drink consumption. The type of food and the amount eaten is an environmental factor in the etiology of several diseases and variations in the morbidity and mortality.

The oral cavity is a mirror of the nutritional status of the variations in the body. Nutrition is one of the most critical factors that play an essential role in tooth formation as well as in bone development and metabolism; it may involve in disease process affecting the tooth and its supporting structures. Deficiencies in much nutrition are thought to be linked to the development and progression of oral diseases.

Nutrition, diet, and dental caries

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

1- Secretary phase when the organic matrix is formed.
2- Mineralization phase which consists of crystal formation and crystal growth.
3- Maturation phase during which water and organic matter withdrawn and mineral content increase.

These stages are considered a acritical period that defines as; **time interval** when specific nutrient is needed by a particular tissue programmed to develop at prescribed time and rate, inappropriate supply of nutrient at such time can result in severe irreversible changes that affect the growth of the organ these changes, in turn, can result in permanent defect in function and decreased resistance to disease. Malnutrition during these critical periods of growth can in a dentition that

is more susceptible to dental caries, while during the post-eruptive period when teeth fully erupt, the enamel is non-vital tissue, in the sense that after eruption into the oral cavity it does not metabolize energy or nutrient or regenerate subsequent to injury, the food exerts dietary (topical) effect, so the role diet and nutrition in the etiology and pathogenesis of caries may be viewed as: -

1- systemic effect
2- local effect.

1- Systemic dietary effect: -

Nutritional factors may influence on the following: -

1- Morphology and integrity of the teeth.
2- The quality of the hard tissues
3- The quality of saliva.
4- Integrity of periodontium
5- Other effects like wound healing

1- Morphology and integrity of the teeth

Genetic factors largely determined the morphology of the teeth, 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 chronology 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 result.
- If maturation is lacing or in complete hypo calcification of enamel will occur in which deficiency in the mineral content of the enamel is found.

Poor nutrition is one of many causes of the dental defect, the clear relationship between specific dietary nutrients deficiency during

critical periods of developments and the poorly calcified tooth had been demonstrated. Many studies showed that deficiencies of Calcium and Phosphate, Vit A, D & C, and protein-energy, affect tooth tissue formation according to their biological roles.

Protein-energy relation

When dietary energy intake is adequate, the a. a (amino acid) 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), a.a are used as a source of energy.

Vitamin D, Calcium, Phosphorus

It is well known that 96% of enamel is apatite mineral principally ca & ph while vit D involved with ca metabolism and its intestinal absorption and therefore, it has a role in tooth formation.

The study of Lady May Mellanby in the early half of the twentieth century showed that vit D deficiency had a marked effect on the development of the teeth. Dogs reared on diets that were deficient in vit D had delayed construction of teeth and teeth that were poorly calcified and poorly aligned. Many of the teeth showed signs of hypoplasia. Mellanby attributed the improvements in the teeth of children in Britain between 1929 and 1943 to improvements in diet and the status of vit D, including the introduction of cheap milk in 1943, the provision of vit D rich cod liver oil to pregnant and lactating mothers, infants, and young children, and in addition of vit A & D to margarine.

Recent studies have shown that supplementation with vit D to pregnant mothers resulted in higher circulating calcium levels in infants at birth and lower incidence of hypoplasia in infants at age three, compared with controls who did not receive supplements.

The studies of Lady May Mellanby

1- Showed that vit D deficiency impairs tooth development.
2- Concluded that the improved diet during the war year, concerning vit and Ca intake was responsible for enhanced dental health.
3- Showed that enamel hypoplasia increased susceptibility to dental caries.
4- Showed that vit D supplementation reduced the incidence of dental caries in children.

In many studies' deficiency of protein-energy, vit A, Zinc and Iron during the pre-eruptive 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 pre-eruptive period. It has been shown that feeding a diet high in sugar during pregnancy and lactating will result in changes in the offspring dental tissue 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 a lower level of carbonate, mucopolysaccharides in enamel were found to be more resistance to dental caries.

Nutrition and eruption of teeth

Among children with protein energy malnutrition, protein deficiency may be the reason for the delayed eruption of their deciduous teeth while first eruption was noted among children with height and weights were higher than the 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 the lowest rate of dental development.

Disease associated with delay eruption of teeth includes Rickets that is caused by vit 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 usual, found delayed tooth eruption. Undernutrition result in delays shedding of the primary teeth and delayed eruption of the permanent teeth, this may influence the caries prevalence at a given age. Protein deficiency cause Kwashiorkor which caused delay eruption of teeth, hypoplasia, and

retarded cementum deposition, decreased salivary rate, and increase caries susceptibility.

Evidence of the effect of some nutrients on dental caries

Vit A deficiency is known to impair enamel (hypoplasia) and dentin formation, impair immune function, reduce the synthesis of specific glycoprotein such as salivary bacteria agglutinating glycoprotein (BAGP) and in cases of severe deficiency to reduce saliva secretion rate. It was reported that dental caries was increased when they fed vit A deficiency diet. Iron deficiency during the pre-eruptive period of tooth development in an animal caused increase caries development.

Caries susceptibility is increased among children with

1- chronic malnutrition (stunted) means deficient height for age.
2- acute malnutrition (wasting) means insufficient weight for height.
3- underweight (low weight for age)
4- micronutrient deficiencies or insufficiencies of important vitamins and mineral.

   Note: malnutrition refers to deficiencies, excesses, or imbalances in a person's intake of energy and/or nutrients. It could be due to systemic factor as malabsorption or due to local factor.

2- The quality of the hard tissues

Protein nutrition is an essential consideration in growth and development of the oral cavity, if the diet includes too little essential a.a during a critical period of active growth permanent structural damage can occur, synthesis of protein in the cell disrupted, resulting in ill a disturbed tissue growth and development. This will affect the maxillary and mandibular bone, resulting in an adverse effect on tooth alignment and alveolar bone integrity.

3- Quality of saliva

Nutrition may affect the quality of saliva; it has been shown that individual on lacto-vegetarian, high protein or high fat diet produce saliva with high buffering capacity whereas individual on high carbohydrate diet produce saliva with lower buffering ability. Saliva lactoferrin, lysozyme, and statherrin are protein molecules that are part of defense arsenal secreted by salivary glands. All of these can be

diminished in volume or altered in the structure during severe period of malnutrition. However, protein-energy deficiency during the pre-eruptive period impairs the condition for the development of salivary glands, which causes a decreased secretion rate and amount of protein secreted per minutes.

4- Integrity of periodontium

The periodontal tissue is composed of epithelium, collagen fiber, blood vessels, cementum, and bone so nutritional deficiency will affect adversely these tissues. Nutrition deprivation was affected rate and degree of periodontal diseases rather than its initiation, as nutritional deficiency does not initiate periodontal disease but may modify the severity and extent of the lesion by altering the resistance of the affected local tissue. Over-all nutritional deficiency affects the severity and extent of periodontal disease by modulating the responses and repair properties of the tissue. Nutrition affects periodontium **directly** by 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 primary etiological factor in gingivitis is the accumulation of microbial plaque over time. The published data on the effect of nutritional deficiencies on periodontal health indicate that deficiencies of vit A, B, D & E. vit C in the citrus fruit was essential to the prevention of disease. Vit C is necessary for the maturation of collagen and acute deficiency result in edema and hemorrhage of the periodontal ligament and tooth mobility, as the alveolar bone is also affected. Vit C (ascorbic acid) is required for synthesis of collagen, wound healing, prevents oxidative damage by action of free radicals (ROS scavenger). Reports are present regarding vitamin E relation to periodontal health and controlling inflammation. Deficiency of Riboflavin (B_{12}) cause angular stomatitis and cheilosis.

Another nutrient reported to have any beneficial effect in the gingival tissue is folate. Both systemic and topical administration, the mouth rinse produced a significant improvement in the gingival health of pregnant women in months 4-8 of pregnancy. Folic acid is required for

DNA synthesis and tissues with the highest cell turnover, e.g., crevicular epithelium, may be affected by this deficiency.

It is well documented that increasing the carbohydrate especially sucrose content of the diet will increase the bulk of supragingival plaque. This is due to the formation of extracellular polysaccharide by cariogenic bacteria. There is a strong positive correlation between the amount of supragingival plaque and subsequent development of gingivitis. The bacteria dominating in supragingival plaque provide the nutrients and gaseous requirements for the colonization of the subgingival environment by the anaerobic, Gram- negative organisms which initiate gingivitis.

The effect of fibrous foods on gingival health

Studies show that the area of plaque which accumulates along the gingival margins in humans is not subjected to friction from food and during digestion. Whilst chewing apples and carrots are more beneficial to the dentition than eating food high in refined carbohydrate, there is no health gain in the prevention of gingivitis.

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

1- Antimicrobial action: - many diets have antimicrobial activity these may alter the quantity & or quality of dental plaque & thus be associated with a reduction in gingival inflammation. Food like Turmeric, Cinnamon, Yogurt, and Fluoride has antibacterial effect.
2- Anti-inflammatory effect: - a nutrient that decreases the host response to injury may result in a reduction in the severity of gingivitis & or development & progression of periodontitis. These work by affecting the enzymes involved in the production of the anti-inflammatory compounds or by altering which compounds are produced. Food that has anti-inflammatory effect like: Avocados, Tomatoes, Blueberries, Strawberries, Kiwi, and Blackberries
3- Immune system modification: (affecting the immunological response to bacterial antigens). Some nutrient is thought to act as immune system modifiers in that they optimize the host immune response so that the protective immune reaction outweighs the self-destructive ones; this could also be accomplished by alteration of

the permeability of the gingival epithelium thus changing host resistance to the bacterial product.

4- Antioxidant micronutrient: Pathogen (in the dental plaque) stimulate the production of reactive oxidative species (ROS) or called free radicals. Free radicals may play a part in the inflammation of gingival tissue and in activating osteoclast, cell responsible for resorbing of the bone. The antioxidant as vitamin A, C, E, enzymes (as glutathione), mineral as Zn, reduce the free radicals and may overcome the inflammation of periodontal tissue.
Note: oxidation is a chemical reaction that transfers electrons from a substance to an oxidizing agent, producing the free radicals which start chain reaction that damage cells. While antioxidants are molecules capable of slowing or preventing the oxidation of other molecules.

Effect of food texture on periodontal health

It has been assumed that solid fibrous food may be beneficial to periodontal health & that eating soft, sticky food might tend to have an adverse effect.

- ☒ Fibrous food does not remove plaque at the gingival level of the tooth. Chewing on fibrous or firm food stimulate salivary flow &can, therefore, aid the oral clearance of food debris.
- ☒ Chewing fibrous or firm food does not increase gingival keratinization, but it does produce a type of local exercise that can stimulate and strengthen the periodontal ligaments & perhaps may also increase the density of alveolar bone adjacent to the root.
- ☒ Fibrous or solid food can replace sugar rich sweet that retained in the mouth and may provide a substrate for increased formation of supragingival plaque bacteria.

3- The local dietary effect

The disease of dental caries has a multifactorial etiology, requiring a dynamic interaction between the factors to result in the lesion of dental caries affecting the tooth surface. One of these factors is the substrate for the growth and metabolism by the bacteria. Substrate is referred to as **fermentable carbohydrates** because of the ability of

cariogenic bacteria to metabolize them quickly, resulting in rapid drop in plaque pH, with a potential to cause demineralization of tooth enamel over time. Whilst in theory, removal of any of the etiological factors will prevent dental caries; the control of the bacterial substrate is the most feasible and potentially manageable.

Diet has a local effect on oral health primary on: -

1- The integrity of the teeth
2- pH and composition of saliva and plaque