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.

Plaque control

By Assist. prof. Azhar Alkamal

Plaque control is broadly classified in to two groups:-

1. Mechanical plaque control.
2. Chemical plaque control.

Chemical plaque control agents

Dental plaque is a complex microbial population of bacterial and salivary polymers present on the tooth surface and plaque prevention is an efficient way to both treat and avoid periodontal diseases, it is an important component of gingival and periodontal diseases' (primary management). Chemicals plaque control are designed to be used as supplements to mechanical plaque control procedures and not to replace them. They have ability to interfere with metabolic activity or adhesion of dental plaque.

Ideal properties of chemical plaque control agents:

1. Should reduce plaque and gingivitis.
2. Should prevent growth of pathogenic bacteria.
3. Should prevent resistant bacteria.
4. Should be compatible with the oral tissues.
5. Should not stain teeth or alter taste.
6. Should exhibit good retentive properties.
7. Should be inexpensive and easy to use.

Modes of action

1. Inhibition of bacterial colonization by interference with bacterial adsorption.
2. Inhibition of bacterial growth and metabolism (bactericidal and bacteriostatic
    a. effects).
3. Disruption of mature plaque by eliminating of existing plaque.
4. Modification of plaque biochemistry and ecology.

Chemoprophylactic agents are delivered as: -

1. Mouth rinses
2. Dentifrices
3. Gels (contains humectant, but without abrasives and foam agents.
4. Sustained release devices and varnishes

5. Chewing gums and lozenges (they increase the time of clearance of agents from the mouth.

I- Mouth rinses

The most widely used agents.

1- **Chlorhexidine (CHX):** This antiseptic agent has both bactericidal and bacteriostatic activity depending on its concentration. Chlorhexidine is a cationic compound that binds to the hydroxyapatite of tooth enamel, the pellicle, plaque bacteria, the extracellular polysaccharide of the plaque, and especially to the mucous membrane. The chlorhexidine adsorbed to the hydroxyapatite is believed to inhibit bacterial colonization and prevent pellicle formation. Chlorhexidine may also inhibit the enzyme glucosyltransferase, which is essential for microbial accumulation on tooth surfaces, and the metabolic enzyme phosphoenolpyruvate phosphotransferase, which is involved in the transport and phosphorylation of glucose across the membrane. After binding, the agent is slowly released in an active form over 8 to 12 hours. Also, CHX is effective against gram +ve and yeast organisms. Toothpaste should be used before rinsing with chlorhexidine. Two daily rinses with 10 ml of 0.2% solution of chlorhexidine gluconate will completely inhibit the development of dental plaque, calculus, and gingival inflammation. It can be used in concentration of 0.12% in 15 ml twice daily. Unpleasant taste and brown discoloration of teeth and filling are the side effects related to CHX.

2- **Triclosan:** It is phenol derivative included in mouthwashes and toothpaste. It has a broad spectrum of activity against gram positive, negative bacteria and yeasts. Dentifrice products containing a zinc citrate and triclosan combination have shown to be effective in reducing acid production and plaque formation and in preventing gingivitis.

3- **Essential oil mouthwashes or Listerine:** They are effective in controlling plaque and gingivitis because the oil alters the bacterial cell wall. The active ingredients (essential oils) used in these mouth rinses include a combination of thymol, menthol, eucalyptol, and methyl salicylate. Although the safety of essential oils is well established, some patients can have difficulty tolerating the burning sensation associated with the alcohol content. In addition, slight extrinsic staining has been reported with the use of essential oils rinses, which is a possible transient (short- term) side effect of any antimicrobial

agent. Essential oil mouth rinses are indicated for patients who need and are compliant with antiplaque/antigingivitis mouth rinses. Listerine Antiseptic was antiplaque and antigingivitis mouth rinse to be approved by the ADA in 1988. Patients are advised to rinse twice daily with one-half ounce of Listerine for 30 seconds in addition to their usual oral-hygiene regimen. Microorganisms do not develop a resistance to the antibacterial effects of essential oils, such as clove oil (eugenol) and thyme oil (thymol). As with chlorhexidine, just rinsing with an essential oil mouth rinse is unlikely to be effective in treating periodontitis because the solution does not reach the depths of the periodontal pockets. For the dental professional, these mouth rinses are recommended in patients prior to aerosol- generating procedures. Unless the dental professional uses an effective dry-field technique in a 30-second period, the bacterial aerosol generated by an ultrasonic scaler that removes calculus, an air- powered tooth polisher, or a slow-speed or high-speed hand piece can be roughly equivalent to the aerosols received from a patient directly sneezing into the dental provider's face.

4- **Enzymes:** Certain enzymes are bactericidal to microorganisms. They would be able to breakdown already formed matrix of plaque and calculus. Enzymes like Mutanase and amyloglucosidase.

5- **Sanguinarine extracts (SE):** It is an herbal preparation obtained from the blood root of Sanguinaria canadensis plant. They are effective against Gram-positive and Gram-negative microorganisms, including oral microorganisms. SE may increase saliva-mediated aggregation. SE seems to exert a bactericidal effect by interfering with essential steps in the synthesis of the microbial cell wall.

6- **Metal ions:** Salts of zinc and copper are the most used. These are effective plaque inhibitors at high concentration. Metallic salts reduce the glycolytic activity in microorganisms and delay bacterial growth. They have unpleasant taste with dry mouth and staining.

7- **Antibiotics**: Vancomycin, erythromycin and kanamycin have been used as agents for plaque control.

II- Dentifrices

They are substance used with toothbrush for purpose of cleaning the accessible surfaces of teeth. They may contain the followings:

a) The therapeutic agent like fluoride to inhibit dental caries.
b) Antimicrobial agent such as chlorhexidine to reduce microorganisms.
c) An anti-calculus agent as zinc chloride or citrate to dissolve calculus.

The function of toothpaste in conjunction with toothbrush is:

1. Minimizing plaque buildup.
2. Anti-caries action.
3. Removal of stains.
4. Mouth fresher.

Composition of dentifrices: -

A dentifrice contains several ingredients that serve a definite purpose in providing adequate plaque control thus preventing caries and periodontal disease. The following are the common ingredients:

1- **Abrasive agents** (Calcium carbonate, silicas): These agents have a mild abrasive action which aids in eliminating plaque and remove stained pellicle from tooth surface. The degree of dentifrice abrasiveness depends on the inherent hardness of the abrasive, size of the abrasive particle, and the shape of the particle. The most common types of abrasives used are carbonates, phosphates, and silicas.

a- Carbonates include calcium carbonate (chalk) and sodium carbonate (baking soda). Calcium carbonate is highly effective abrasive, although the calcium ion limits the amount of soluble fluoride in toothpaste up to 7 ppm.

b- Phosphate abrasives include calcium pyrophosphate and dicalcium phosphate dihydrate.

c- Silicas, such as silicon oxides, mechanically cleanse the tooth, are chemically inert, and do not react with other dentifrice ingredients. When toothbrush abrasion damage does occur, it usually appears as a V-shaped notch in the cementum apical to the cementoenamel junction. This area is vulnerable because enamel is about 20 times harder than dentin or cementum.

2- **Binding agents** (Water soluble agents):

These agents control stability and consistency of toothpaste and effects ease of dispersion of the paste in the mouth.

3- **Detergents**: They are producing the foam which aid in the removal of food debris and dispersion of the paste in the mouth. Sodium lauryl sulfate is the most widely used detergent. It is stable, possesses some antibacterial properties, and has a low surface tension, which facilitates the flow of the

dentifrice over the teeth. Sodium lauryl sulfate is active at a neutral pH, has a flavor that is easy to mask.

4- **Humectants** (Glycerin, mannitol, glycerol): These agents aid in reducing the loss of moisture from the toothpaste and prevent hardening. These humectants are non-toxic, but bacterial growth can occur in their presence. For this reason, preservatives such as sodium benzoate, dichlorinated phenols, and alcohols are added to prevent their growth. At high concentration (>40%), humectants act as preservatives.

5- **Flavoring agents**: They render the product pleasant to use and leave a fresh taste in the mouth after use. Spearmint, peppermint, wintergreen, cinnamon, and the most recently introduced flavor, vanilla give toothpaste a pleasant taste, aroma, and refreshing aftertaste. It is difficult to formulate a flavor that is universally acceptable because people have different color and taste preferences. Some manufacturers use essential oils such as thymol which can provide a "medicinal" taste to the product.

6- **Anti-calculus agents** (soluble pyrophosphates or zinc citrate): These agents are designed to inhibit the mineralization of plaque. Dentifrices containing these agents are labeled as tartar control toothpastes.

7- **Sweeteners and coloring agents** (Sorbitol, mannitol): They serve a dual role as sweetening agents and humectants. Glycerin also serves as a humectant, adds to the sweet taste. A new sweetener in some dentifrices is xylitol.

8- **Anti-caries agents** (NaF, MFP, SnF2): These agents aid in the control of caries. Essential-oil dentifrices, Listerine as anti-halitosis.

9- **Desensitizing agents:** Potassium nitrate is a commonly used, it reduces the reaction of nerves in the teeth to stimuli such as heat and cold. It is known to desensitize the nerve by penetrating through the length of the dentinal tubules and to depolarize sensory nerve endings located at the dentin–pulpal interface.

10- **Whiteners** (hydrogen peroxide or carbamide peroxide): The dentifrices (contain whiteners) control stain via physical methods (abrasives) and chemical mechanisms (surface active agents or bleaching/oxidizing agents). Carbamide peroxide breaks down to form urea and hydrogen peroxide. Hydrogen peroxide, in turn, forms a free radical that contains oxygen, which is the active bleaching molecule. Papain, a naturally occurring enzyme that destroys protein is rapidly diluted by saliva. Home-bleaching products can contain other chemicals to aid in the delivery of the bleaching agent. Glycerin or propylene glycol is commonly added to thicken the solution and prolong contact with the tooth surface.

Baking soda dentifrices: baking soda (sodium bicarbonate) had a long history of use as an oral-hygiene aid. They are known to reduce plaque and gingivitis, remove extrinsic stain, and reduce malodor. Baking soda dentifrices contain only a small amount of baking soda in addition to the standard fluoride compatible abrasives.

Dietary counseling in dental practices Lec-5-

By assist. prof. Azhar AL-Kamal

Dietary counseling: - A process by which a health professional with special training in nutrition helps people make healthy food choices and form healthy eating habits.

There are two ways in which the diet may act upon the oral tissue and play its role in the prevention of the diseases:

- First through the oral environment.
- Second through general nutrition.

The relationship between dental caries and frequent consumption of carbohydrate fermentable by oral micro-organism is well known. However, caries development becomes severe when intake is excessive or, the resistance is lowered (disease, medication, or poor nutritional status). Therefore, assessment of dietary habits is crucial for: -

1- understanding of the actual caries situation.
2- predicting the caries risk in the individual.
3- designing treatment plans and preventive programs.

Nutritional status assessment using body mass index (BMI)

BMI: - Is a number calculated from a child's weight and height. For children, BMI is used to screen for overweight, at risk of overweight, or underweight. It's easy to perform method screening for weight categories that may lead to a health problem. For children or teens, BMI is age-and gender-specific and is often referred to as **BMI-for-age.**

After BMI is calculated for children and teens, the BMI number is plotted on the CDC BMI-for-age growth charts (for either girls or boys) to obtain a percentile ranking. Percentiles are the most used indicator to assess the size and growth patterns of individual children. The percentile indicates the relative position of the child's BMI number among children of the same gender and age. The growth charts show the weight status categories used with children and teens (underweight, healthy weight, at risk of overweight, and overweight). The CDC BMI-for-age growth charts for girls and boys consider these differences and allow translation of a BMI number into a percentile for a child’s or teen’s gender and age.

Calculating and interpreting BMI using BMI percentile. Calculator involves the following steps: -

1- Obtain accurate height and weight measurements.
2- Calculate the BMI and percentile using the child and teens BMI calculator. The BMI number is calculated using standard formulas **weight (kg) / height^2 (m^2).**
3- Review the estimated BMI-for-age percentile and results. The BMI-for-age percentile is used to interpret the BMI number because of BMI in both age and gender specific for children and teens. These criteria are different from those used to interpret BMI for adults who do not consider age and gender. Age and gender are considered for children and teens for two reasons:
    a-The amount of body fat changes with age.
    b-The amount of body fat differs between girls and boys.
4- Find the weight status category for the calculated BMI-for-age percentile as shown in the following table.

The interpretation of BMI-for-age varies by age and gender so if the children are not the same age and of the same gender, the BMI numbers have a different meaning. Calculation BMI-for-age for children of different ages and gender may yield the same numeric result, but that number will fall at a different percentile for each child for one or both of the following reasons:

1- The regular BMI-related changes that take place as children age and as growth occur.
2- The normal BMI-related differences between genders.

Weight status category	Percentile range
underweight	Less than the 5th percentile
Healthy weight	5th percentile to less than the 85th percentile
At risk of overweight	85th to less than the 95th percentile
Overweight	Equal to or greater than the 95th percentile.

Assessment of dietary intake

In dentistry, nutritional evaluation and counseling are essential for success in treatment and prevention in some oral diseases; this means the information on eating pattern and consumption of energy and nutrient would be connected and evaluated.

Objectives of dietary assessment

1- To provide an opportunity for a patient to study personal dietary habits objectively.
2- To obtain an overall picture of the types of food in the patient's diet food preferences and quality of food eaten.
3- To study food habits and snacking patterns.
4- To record the frequency of use and when the cariogenic food is consumed to determine the overall consistency of the diet and the fibrous food that are regularly included.
5- To identify the nutritional status of an individual about total requirements.
6- To provide a basis for making the individual recommendation for changes in the diet that is important to the health of the oral mucosa and the periodontium and to the prevention of dental caries.

Nutrition Counseling in Dental Practice Started even before teeth eruption and in the following years: -

- Pregnant women
- Infants, toddlers, and young children

- Adolescence and adulthood

The methods that used for dietary assessment: -

1- 24 – hour recall

The 24-hour recall method is a widely used method to assess dietary intake. A trained person from the dental team interviewed the patient on the consumption of food and beverages during the latest 24-h period. Consistency in the technique and skill of the interviewer are essential since this influences the communication and cooperation of the patient and thereby the result.

Food models and life-size illustrations are recommended by most research as support to estimate eaten quantities. The portion size can also be given in household measures, such as glass, cup, tablespoon, deciliters, and grams. To reduce bias, the 24-h recall is requested without providing the patient with prior notice. For nutrients with large day-to-day variations, the number of days increased. The day should be selected to represent an ordinary day.

2- Dietary record

In the dietary record, which is also called food diaries, the patient records type and amount of everything consumed during the prescribed period, usually 3-7 days. Estimates of portion size and selection of days are done in the same way as for the 24-h recall method. The patient is thoroughly instructed and motivated to fill in the record and to keep to normal dietary habits during the record period. The dietary record and 24-h method are both reported to underestimate the intake.

3- Food frequency questionnaires

A food frequency questionnaire contains a list of food items, usually 50-150 items. They are selected to illustrate the whole diet or a specific nutrient (sucrose). The patient marks his / her consumption on a scale ranging from never to several times per day. It can be used to estimate nutrient intake. It's uncomplicated and cheap to perform and may be useful as a screening instrument or to collect dietary data on a group level.

4- Semiquantitative food frequency questionnaire

- collect portion size information as standardized portions or as a choice of portion sizes.

- It used to indicate a general food frequency questionnaire that allows for quantification of serving size.
- There are many food frequency tools with a long list of food that tend to produce an estimate of nutrient intake; these were developed for different populations and different purposes.

Evaluation of dietary assessment

After completion of the dietary registration, the intake is evaluated.

Evaluation of cariogenic potential

The frequency of intake and the consistency of refined carbohydrate are the key factor in the initiation and continuation of the caries process, to identify the type of cariogenic food; we should look at this form public health viewpoint not a purely biochemical viewpoint even bread contain sugar, but overall bread is no threat to teeth. Therefore, evaluation of cariogenic potential includes an estimation of factors such the number of intakes containing fermentable carbohydrate, taking snacks during the night, and retentiveness of cariogenic products (length of time food might remain on the tooth surface).

Sugars that are added to provide sweetness are classified into: -

1- **Solid and sticky** cakes, sweet rolls, pastry, canned fruit candy, toffee, sugared chewing gum.
2- **Liquid soft drink sugar** and honey in beverage, ice cream, custard.
3- **Slow dissolved** canned

Other properties of the food, known to modify the caries process, should be regarded. An example of this chewing stimulation provides food. The period with lowered pH in plaque are reduced by a diet, which increases saliva secretion.

Evaluation of nutritive value

Several inexpensive software for evaluation of energy and nutrients in the diet are available, and computer, based analysis of diet registrations are also common dental practices, this is a convenient way to evaluate the nutritive value of the intake. Computing the nutrients of each food eaten by each person into its different nutrient was following food composition table, this table converts food that are most prepared and eaten into their major nutrient components such as protein, vit, and minerals.

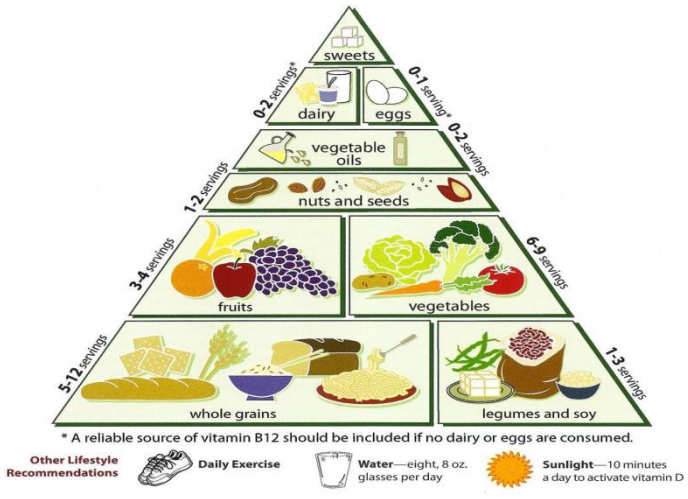
Compare the result with Recommended Dietary Allowance (RDAs)

The standard dietary adequacy was prepared by the National Academy of Science for certain nutrients. RDAs reflect adequate intake for healthy individual to prevent deficiency state, the value are adjusted for age and gender.

Estimate the number of intakes representing specific groups.

It is another way to estimate the nutrient value of the diet. Its attempt to translate scientific knowledge of nutrient need into clear guideline to help people select an adequate diet; therefore, five foods were required for health these are: -

1- Bread and cereal group --------- 6-11 serving day.
2- Vegetable group---------- 3-5 serving day.
3- Fruit group---------- 2-4 serving day.
4- Milk group---------- 2-3 serving day.
5- Meat, poultry, fish, egg---------- 2-3 serving day.



The ideal snack

Many of the physical and chemical attributes of an ideal snack from a dental health viewpoint are: -

1. Its physical form should stimulate saliva.

2. It should produce a minimal amount of intra-oral retention.

3. Its chemical composition should include: a relatively high protein and low-fat content; minimal fermentable carbohydrates; a moderate mineral content (particularly calcium, phosphate, and fluoride); an inherent pH above 5.5 so as not to increase oral acidity; a large inherent acid buffer capacity during mastication; and a low sodium content. With present-day food technology these attributes should not pose an insurmountable problem.

Dietary counseling

After assessment of the dietary registration, the advised plan for the individual is formulated.

Approach to counseling.

1- **Non-directive**

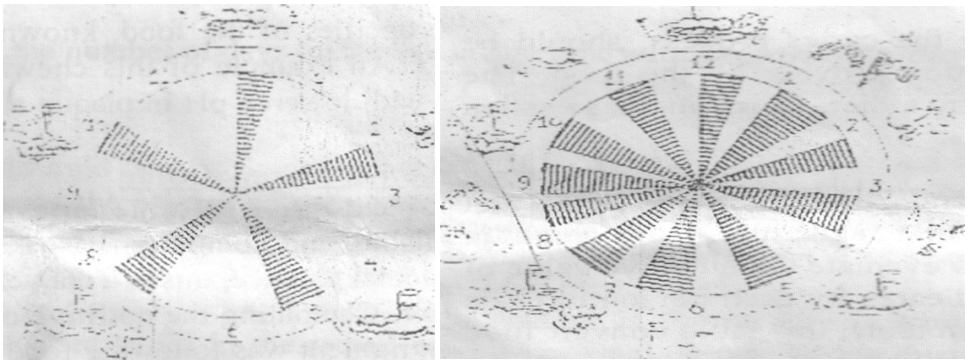
A counselor's role is merely to aid the patient in clarifying and understanding his or her own situation and provide guidance so that the patient can make his or her final decision.

2- **Directive**

The role of the patient is passive, and the decisions are made by the counselor for the patient.

A useful tool may be the sugar-clocks demonstrating the high caries risk of eating frequently.

In some patients, a single habit explains the caries activity, e.g., frequent eating of sugar-containing lozenges or taking a snack at night, and this may easily be corrected. In others, a complicated eating situation is found. The eating pattern may be characterized by snacks when virtually no ordinary meals give satiety or proper nutrient intake. In cases like these, a change in basic behavior is required.



Change in behavior.

It is the process affected by the established fact that humans are neophobic. This means we have a fear of new things. Therefore, forced dietary changes cannot be successful unless the benefit accrues rapidly and is of demonstrable advantage. This can be seen for some weight-reducing program. Otherwise, a successful shift in diet behavior relies on a program with repeated, small steps. This is shown to be true for the introduction of a new food item and habits to small children as well as adults.

Guideline for counseling

1- The patient should accept the responsibility for the dietary modification.
2- Gather information on personal like dislikes.
3- Evaluation and interpret data, relative adequacy of the diet eating habits to find reasons for the patient dental problem.
4- Develop and implement a plan of action by prescribing consisting primarily gradual qualitative modification using acceptable food exchanges.
5- Seek active participation of the patient's family in all aspects of dietary change.
6- Follow-up to assess the progress.
7- The primary purposes of this session are to clarify the problem and to reinforce and encourage and to maintain the change.

Motivation patient to modify food habits.

1- **Awareness** is recognition that the problem exists but without an inclination to solve it, (hard candies produce acid which can cause my teeth decay).

2- **Interest** is a greater degree of awareness but still without the inclination to act (may should give up the hard candies, I do not want any more sensitive or painful teeth).
3- **Involvement** is an interest and definite intention to act (I will give up hard candied).
4- **Action** is trial performance (I have given up hard candies and chew sugarless gum instead to prevent the dry feeling in my mouth).
5- **My habit** is the commitment to perform this action regularly over sustain period (I have not had a hard candy in six months).

Motivation

To modify a patient, diet the clinician can only seek and encourage the patient's own
motivation. The basic factors that motivate people

- ***self-preservation***
- ***recognition***
- ***religion***
- ***money***

All factors influence the desire of each person. If clinician can help patients understand that healthy mouth and teeth and a nice-looking smile can help them to achieve one or more of these four goals, the patient will be inclined to adopt diet that will promote better oral health.

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 also 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. Not 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 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.

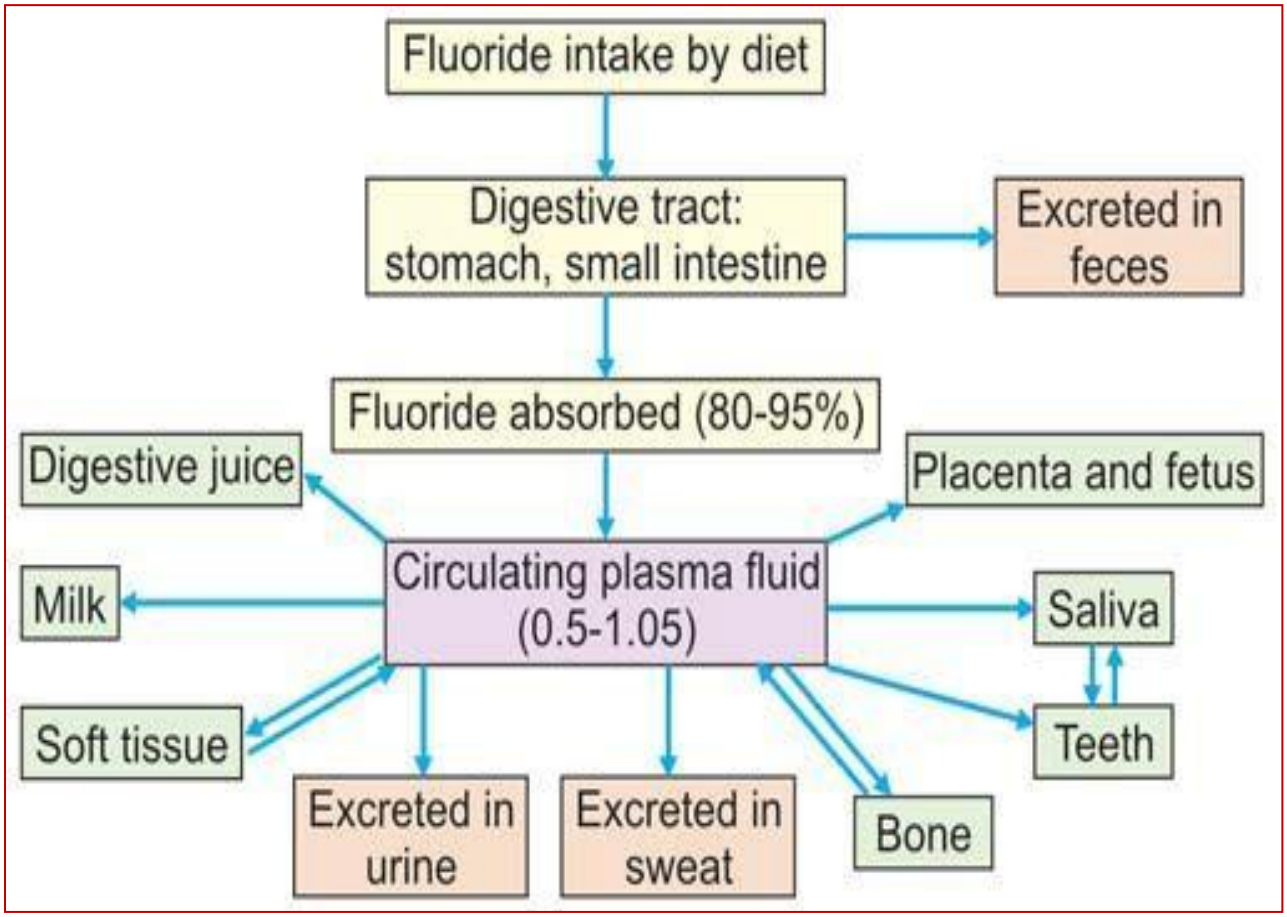


Fig Metabolism of fluoride in human body

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, there rays or beams are parallel within the laser instrument

Absorption: -

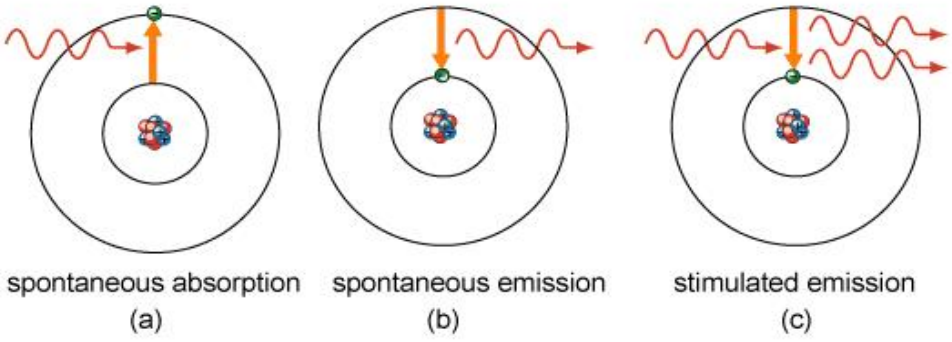
when electron move from low energy level (E1) to high energy level (E2) when a photon hits on the atom of unexcited state.

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.

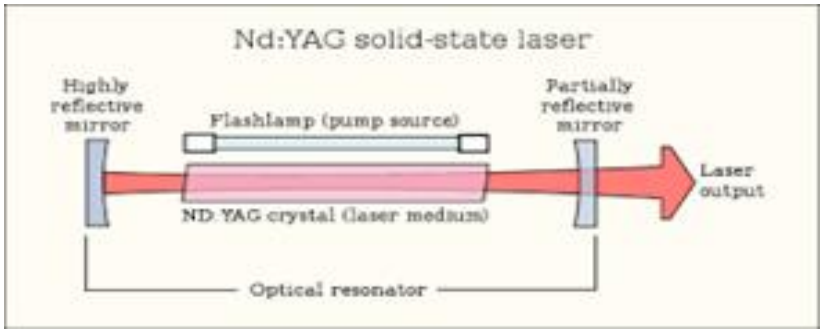
Basic terms: -

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: -

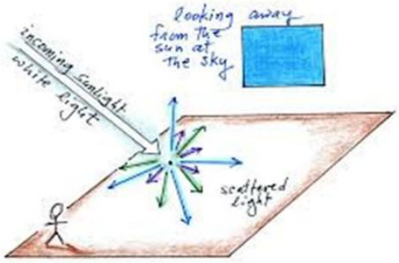
1- Hard lasers Co_2, Nd: YAG and argon lasers.
2- Soft lasers He Ne lasers, Diode lasers.

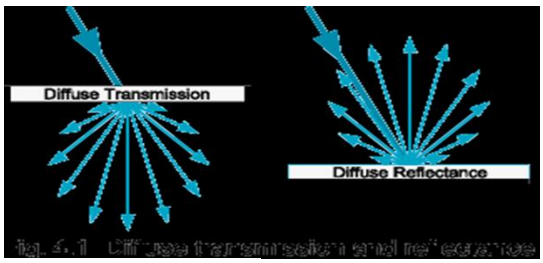
All lasers except the Co_2 laser are transmitted by glass fiber. The Co_2 lasers are transmitted via mirror systems. Laser is being used in almost all the specialties of dentistry.

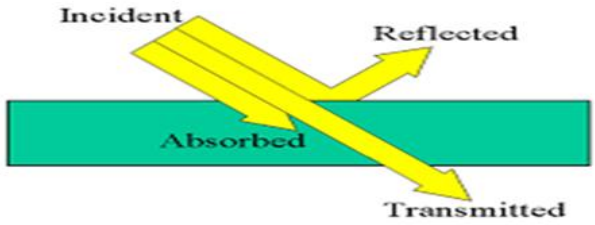
Laser effects on tissues: -

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.

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.

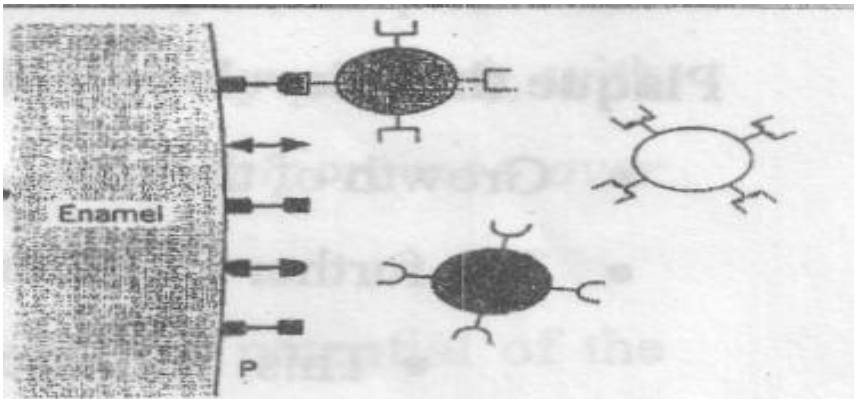
In the initial stage (pioneer) species of bacteria colonize the tooth surface by binding to component of the salivary pellicle, among these initial colonizers are believed to constitute a highly selective part of the oral micro flora, mainly

Sterpotococcus 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 surfaces:

- **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.

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.

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

Nutrition and oral mucosal disease Lec -4-

By assist. prof. Azhar AL-Kamal

The type of the food and the amount eaten is an environmental factor in an etiology of several diseases and variations in the morbidity and mortality between the world's populations.

Malnutrition where one or more of the essential nutrients are absent in the diet or there is inadequate absorption from the gastrointestinal tract. Nutrition deficiencies can impair oral mucosal health & oral immune defense, & component of some diet may be harmful to mucosa. Oral disease can interfere with feeding & nutrition because of compromised mastication & swallowing, pain, or discomfort.

Common oral mucosal manifestations of nutritional defects

1- Candidacies
2- Oral ulceration
3- Angular stomatitis
4- Burning mouth syndrome
5- Gingival bleeding
6- Post extraction hemorrhage

The current national dietary recommendations

1- Proteins ---12% of the diet
2- Fats ----no more than 30%
3- Carbohydrates
4- Vitamins
5- Minerals
6- Water

oral disease can interfere with feeding and nutrition because of compromised mastication and swallowing, pain, or discomfort. **Protein-energy** deficiency may in children vary in its effect from

mild growth retardation to marasmus and kwashiorkor (sever protein malnutrition). Protein malnutrition decrease collagen synthesis in oral mucosa and oral lesion has been described in kwashiorkor; these include edema of the tongue and papillary atrophy, angular stomatitis, hypopigmentation circumorally and Xerostomia. Interestingly, tolerance of dentures appears to be increased if the dietary protein intake is improved in edentulous patients.

Minerals

Minerals are inorganic substances, which are required in the structural composition of the hard and soft body tissues. They also participate in the contraction of muscles, nerve conduction & blood clotting.

Vitamins

Vitamins are group of substances that are required in small amounts and are essential for growth and development. They exist in natural foods, but most have been produced synthetically. Many vit are co-enzymes which have an essential function in a chemical reaction catalyzed by a specific enzyme. Vitamins are classified into fat-soluble A, D, E, K, and water-soluble B- Complex and C.

Vitamins and antioxidant activity

Carotenes, including beta-carotene, vit C and vit E are among the many substances in food that have antioxidant properties. This attribute can help to counter the effects of reactive oxygen species e.g., free radicals which is highly reactive molecules carrying unpaired electron which are produced by the body's normal metabolic processes or enter the body from atmosphere or from environmental pollution such as cigarette smoke. If the free radical accumulates, they can damage key cellular molecules, such as DNA and proteins. Cells with damaged DNA may be more prone to developing cancer. Free radicals readily oxidize polyunsaturated fatty acids in food and in cell membranes in the body to give lipid peroxides which can also damage cells. People who consume large amounts of yellow fruits or dark green or orange or vegetables seem to be less prone to some forms of cancer.

Vitamin C contributes to immune defense by supporting various cellular function of both the innate and adaptive immune system. Vit C supports epithelial barrier function against pathogens. Vit C accumulates in phagocytic cell, such as neutrophils and enhance chemotaxis, phagocytosis generation of reactive oxygen species and ultimately microbial killing.

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

Vitamin E: -

Vitamin E found in peanuts, sunflower seeds, green leafy vegetable such as spinach, broccoli and in soybean oil. Dietary substitute with vitamin-E can prevent oral cancer at a very early stage that is in premalignant lesions, in premalignant conditions. Main action of vitamin E includes increase immunity, controls free radicals mediated cell disturbances, maintains membrane integrity, inhibit cancer cell growth, cytotoxicity. Many past studies suggest the role of antioxidant (vitamin E) in treatment of oral mucosal lesions particularly includes oral leukoplakia, oral lichen planus, oral submucous fibrosis and oral cancer.

Notes: -

- Nutrient with antioxidant action help maintain cell integrity by reducing the free radical damage to host tissue that is initiation by the host inflammatory and immune reaction. They also serve to protect the host from bacterial damage.
- It is known that several antioxidant nutrient and enzymes are present in the cervical fluid & in the oral epithelium & secretion considering the responses elicited by the host against pathogenic oral bacteria.
- Some of the nutrient that influences an individual oxidative status includes vit C, E, Zinc copper, Manganese & Selenium. These antioxidant compounds are essential for helping to maintain cell integrity.

Nutrition and oral cancer

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

Prevention of oral cancer exerted as: -

1- Primary prevention focused on elimination of risk factor & inhibition of tumor initiation & activation.
2- Secondary prevention focused on inhibition of tumor promotion & progression.

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

Carcinogenic agents may be of two types:

1- Exogenous agent
A- physical: - ultraviolet rays, gamma rays
B- biological: - viruses
C- Chemical: e.g., Nitrosamines, which a class of carcinogenic amine that are form from nitrate & nitrites in food, either during drying and cooking or when then the food in the gastrointestinal tract, also found in cigarette smoke. This Nitrosamine is known carcinogens are also used as food additives to preserve the color of meat, inhibit oxidation, and discourage the growth of microorganism in meat. Ascorbic acid and sulfur dioxide are used to inhibit nitrosamine formation in foods.

2- **Endogenous** (normal product of oxidative metabolism that can cause damage to DNA and covert normal cell to cancer cell). Oxygen is essential for sustaining life but once it transforms into a free radical, it assumes a destructive power. Free radicals are ***unstable form of oxygen*** they have lost an electron from their molecular structure, (normally these electrons exist in pairs). To replace the lost electron free radicals actively seek out electron from other substances in the body. When these materials give up an electron to the free radicals their structure, become damaged. The favorite targets of free radical are cell proteins, enzymes, fatty acids in cell membrane & genetic material DNA. Damage to these structures can trigger the development of cancer. The prevalence of cancer was reported to be less among people eating a large quantity of fresh fruits and vegetable. These dietary items provide the body with vitamin and minerals which act as antioxidants and protect the body from cancer. ***antioxidants*****,** these substances can neutralize free radicals, and include:

1- vitamins: Vitamin C, E, carotenoids, beta-carotene (provitamin A) & some poly-phenolic compounds found in green tea, fruit & vegetables have been shown to be effective in inhibition of tumor promotion.
2- Minerals: selenium, manganese, and zinc.

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

Retinoids are inhibitors of substances specific to tumor promotion. powerful antioxidants and protecting cellular lipids from oxidation.

Retinoids prevent tumor promotion &progression in many ways: -

- Sequester free radicals, thus limiting their production (tumor initiation).
- The retinoid prevent cancer is by activation of cellular differentiation. that may be responsible for some cancer.
- The retinoid can join with the nuclear receptors and activating the genes responsible for the maintenance of cellular differentiation (regulate gene expression).

Soybeans

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

The relation between diet and erosion

Erosion is the physical result of a pathological, chronic localized loss of dental hard tissue that is chemically etched away from the tooth surface by acid and / or chelation without bacterial involvement.

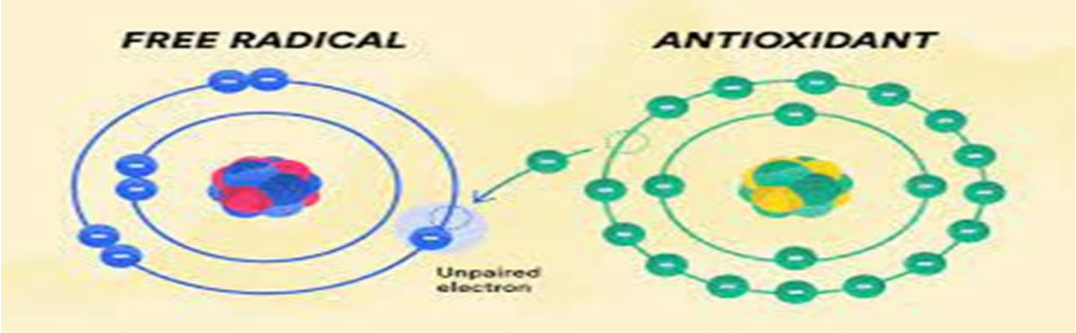
Causes

1- Diet (extrinsic) e.g., citrus fruits, fruit juices, acid carbonated drink, acidic sports drinks, wine, flavored sweets, iron medicines, vit C, acidic mouth washes.
2- Stomach (intrinsic).
3- Environmental (extrinsic).

The type of acid varies. For example, phosphoric, citric, and malic acids are found in fruit and fruit juices, ascorbic acid is added to sweets and sports drinks and has been identified as a significant cause of erosion. In destruction from intrinsic cause the hydrochloric acid from the stomach causes the erosion. The effect is due to the low pH of the acid and its chelation effect which is the binding to calcium and removing it from the tooth surface. Enamel is gradually lost in layers and dentin is lost at a greater rate. The loss is irreversible, giving a characteristic appearance to the lesions and the remaining tissue is softened so avoidance of tooth brushing after an acid attack is based on preservation this softened tooth tissue.

Table shown the relation between some vitamins and minerals in oral manifestation.

	Vitamin/ mineral	Function	Oral relevance
1	A /Retinol	Epithelial differentiation.	Keratosis of mucous membrane, impaired salivary flow, enamels hypoplasia.
2	B_1 /Thiamine	Co-enzyme in energy metabolism.	Burning mouth, reduce taste perception.
3	B_2 /Riboflavin	Co-enzyme in energy metabolism.	Angular cheilitis, glossitis, aphthous ulceration.
4	B_{12}	Co-enzyme, needed for hemoglobin and essential for cellular function.	Atrophy, glossitis, aphthous ulcers, angular cheilitis.
5	B Complex Folic Acid	As B_{12}	Aphthous ulceration, papillae atrophy of tongue.
6	C /Ascorbic Acid	Antioxidant, collagen production.	Gingivitis/periodontitis, aphthous ulcers.
7	Ca	Formation of bones and teeth.	Hypoplasia, and deficiency during tooth formation.
8	Iron	Hemoglobin formation	Glossitis, angular cheilitis, candidiasis



Oral health of disabled patients

By Assist. Prof. Azhar AL-Kamal

Disability is any physical, developmental, mental, sensory, behavioral, cognitive, or emotional impairment or limiting condition that requires medical management, health care intervention, and/or use of specialized services or programs. The condition may be congenital, developmental, or acquired through disease, trauma, or environmental cause and may impose limitations in performing daily self-maintenance activities or substantial limitations in major life activity.

Impairment: Any loss or abnormality of a physiological, psychological, or anatomical structure or function.

Disability: any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being.

Handicap: a disadvantage for a given individual that limits or prevents the fulfillment of a role that is normal.

Mental retardation is a general term used when an individual's intellectual development is significantly lower than average and whose ability to adapt to their environment is consequently limited.

Recommendations for the disabled patient: -

1- Improved oral health.
2- Affordable comprehensive oral health care in high-quality dental homes
3- Early oral health interventions (e.g., risk assessment by age 6 months)
4- Improved oral health status upon transition to adulthood.

People with disabilities should be recognized for their ability, not their disabilities, and dental care should be offered to the same standard as the general population.

There **are barriers** to dental care for this group of people which are: -

1- Oral health may have a low priority in the family.
2- Dental care may be restricted by attitudes and access.

3- Treatment may be difficult to provide because of fear, and anxiety, lack of understanding and inability to cooperate.
4- Involuntary movement may restrict oral care of dental treatments.
5- Difficulty with communication complicates the situation.

Promotion of oral health disable groups requires the following: -

Disabled people have the same right to good quality health care as all other groups in the population.

1- Understanding and concern of the caregivers, to provide the disabled patient with the required preventive measures and health service needed.
2- Health services in the community, need to be accessible and reached disabled patient in flexibly.
3- The financial barriers for health services need to be removed.

Personal predisposing factors for oral health diseases:

Several factors are present explaining the increase in the prevalence and severity of oral diseases like dental caries and periodontal diseases:

1- Age
2- Psychological status
3- Socio-economic status
4- Behavioral risk factors
5- Nature and severity of the individual's learning disability
6- Medications
7- Ability to undertake regular oral hygiene procedures or receive care from other caregivers.
8- Importance placed on oral health by caregiver whether family or paid.
9- Previous dental history including treatment and attendance pattern.

General health services to the disabled patients

Oral health may little difference between people with disability and normal. Reports show that disabled individuals have tended to have more teeth missing, more untreated decay, and fewer teeth restored. Dental care is often generalized as an emergency. With few exceptions, prevention care has not been emphasized in the way it should. The two most important oral health problems among disabled patients are dental caries and periodontal diseases.

One of the challenges for a service providing care for people with impairment or disability is the time that is required to plan, organize, and deliver care for patients. Problems concerning the delivery of care to disable the need to be overcome and solved. Health services should focus on primary prevention first, and then on secondary and tertiary prevention. Prevention program for disabled groups depends on the age of the subjects, type of disability (learning or physical disability) and attitudes of caregiver.

General preventive programs involve: -

1- Dietary consideration: -

Consistency of the diet for disabled groups is either in liquid or semi-liquid form. Other may receive a diet or supplements with high calorie. Medication for certain disabled groups may contain high-level sugar.

For severely impaired people, food is often liquidized or fed in a semi-solid state after mashing. Pureed diets are recommended for cerebral palsy patients who have difficulty swallowing. Some very disabled children and adults need to take high-calorie supplements to maintain their nutritional status. Liquid oral medicines taken can be damaging dentition in chronic users. A proper diet is essential to a good preventive program for the disabled child, to reduce the cariogenic potential it is necessary to; -

a- Restrict between the meal snacking.

b- Limit the use of highly cariogenic food.

2- Oral hygiene: -

It is greatly depending on the age and type of disability. Some patients are either lacking dexterity or unable to perform good brushing and flossing. A modification of the toothbrush or brushing technique may be needed.

A regular mouth cleaning by using fluoride toothpaste was preferable. If the patient will not tolerate the use of the toothpaste; mouth rinse with fluoride (0.2% NaF) can be used as a part of the mouth cleaning routine.

For the patient who has difficulty grasping a conventional toothbrush; he can use a slim-handled brush. A larger handle can make mouth cleaning more easily, many modifications resemble a bicycle grip and are made in rubber or plastic to fit over the toothbrush handle. Electric toothbrushes are not recommended for those disabled individuals due to their increased weight, difficulty in using on/off switched as well as these devices can cause

considerable damage to the hard and soft tissue in a short time. Concerning toothpaste, the role of the caregiver or supervision is very important.

Patients with mental retardation or severe disability may suffer from periodontal diseases due to difficulties in proper oral hygiene. For those patients, chlorhexidine can be prescribed (0.06%). The disclosing agent may be needed for those having difficulties in visualize plaque to be removed.

3- Fluoride therapy: -

Fluoride supplements (systemically) need to be given to young children with disabilities. As for older children and adults, topical fluoridation is necessary in form of dentifrices and/or mouth rinse to control dental caries.

4- Professional health care: -

Dental visit on a regular schedule needs to be designed for those people. They may need professional scaling and polishing, fluoride application, fissure sealants to prevent and control dental caries and periodontal disease. Treatment and follow up can be conducted sometimes for the disabled patient in their wheelchair.

5- Immobilizations of uncooperative children: -

physically disabled patients need a papoose board for stabilization of the body, adjustment of the head-by-head positioner, mouth props, strap, and tap for extremities.

Children with hearing disability: -

These are deaf people. In communities each state or region should have the health centers for these disabled persons, the training programs for the handicapped should be established by health center, dental school health departments, and they select dentists or dentists to attend that health center for all handicapped groups programs ranging from oral hygiene programs.

Communication with those patients was very difficult. The dentist or educator should seat directly in front of the child, at the same eye level and face to face. The facial expression of the dentist is most important for children with a hearing problem, like smiling and gentle handholding. Instructions concerning oral hygiene are achieved using models or clipboards and a colored pen. For a patient wearing a hearing aid, a caution not accidentally dislodges these hearing aids by

their hands during treatment, this may happen when the dentist stands behind the patient in the 12 o'clock position. Feedback may occur for those patients with hearing aid due to handpieces. It is advised to remove this aid before to dental treatment.

Children with physical impairment (neurological): -

Patients with cerebral palsy or muscular atrophy may need prevention programs from their first of life including fluoride supplements, oral hygiene under supervision, modification of toothbrushes, and periodic dental visit for professional fluoride application. Fissure sealants may be difficult for those patients due to difficulties in controlling the moisture contamination. Salivary pooling is often seen among those patients due to swallowing difficulties. Mouth rinse should not be prescribed to such patients, and a very little amount of dentifrice should be used. In the dental clinic, the patient should be seated in upright position rather than a reclining position because of difficulties in swallowing.

Children with visual deficits

The severity of visual deficits may be varying from correctable to total blindness. Those patients may need chair instruction concerning oral hygiene using large size models and giant toothbrushes. Colored dental floss may be needed (red or green) instead of white color to be visualized more easily by those visual impairments. Dental floss with holder makes it easier to be them.

Medically compromised children

Children or persons with medically handicapped fall into two groups:

1- That general health affected if they were to develop dental diseases.
2- Those who need dental care in it, are at risk, for example: - persons with cardiovascular disease and leukemia.

Oral hygiene measures for those patients depend on the severity of the disability. The oral care may be taken at the bed and depend on the caregiver. In the presence of difficulty in swallowing, aspirating type of toothbrush is required, or dipping toothbrush in mouth wash instead of using toothpaste. Patients with severe periodontal diseases. Chlorhexidine gel by gauze or brush can be swapped around their teeth in contact with gingiva to disrupt dental plaque.

Children with **Down syndrome** experience a high incidence of rapid destructive periodontal disease that may be related to local factors, such as tooth morphology, bruxism, malocclusion, and poor oral hygiene. Certain systemic factors are also believed to contribute to periodontal disease, including poor circulation, decreased humoral response, general physical deterioration at an early age, and genetic influences. Delayed tooth eruption frequently occurs in children with down syndrome. Many of these patients are affectionate and cooperative, and dental procedures can be provided without compromise. Light sedation and immobilization may be indicated for these children. severally resistive patients may need nitrous oxide or general anesthesia.

Dental care for the institutionalized disabled individual: -

For those disabled group, the oral hygiene instruction needs to be directed to the nursing staff, through the educational program using training aids as videos, slide shows etc. disabled patients need routine oral health care and follow up an observation by dentists or hygienist. Educational programs should include dietary counseling, and referral to the dental clinic if treatment is required.

Domiciliary care

Domiciliary care is to provide comprehensive dental care to patients who are unable to access a dental clinic for their dental care. Physical and mental disability or chronic disease may make it difficult or impossible for those patients to attend a dental clinic for their routine or emergence care. However, it is useful to make an initial assessment of a patient's eligibility.

Preventive Dentistry

Lec-1

By assist. Prof. Azhar Alkamal

Preventive dentistry is the employment of all measures that can attain and maintain a maximum oral health.

The **four** levels of preventive care: - primordial, primary, secondary, and tertiary care are detailed below:

1- Primordial prevention

It is the prevention of emergence or development of risk factors (beginning with change in social and environmental conditions) in countries or population group in which they have not yet appeared. Individual and mass education is main intervention method in primordial prevention.

2- Primary prevention employs techniques and agents to forestall the onset of diseases, to reverse the progress of initial stages of disease, or to arrest the disease process before treatment becomes necessary.

3- Secondary prevention employs routine treatment methods to terminate a disease process and to restore tissues to as near normal as possible.

4- Tertiary prevention employs measure necessary to replace lost tissues and to rehabilitate patients to the point that function is as near normal as possible after the failure of secondary prevention.

In preventive dentistry there are five phases suggested for sequence of treatment planning:

Phase 1- Urgent care

It is the treatment of emergency conditions such as extraction or endodontic.

Phase 2- Gingival and Periodontal therapy

It is attempted to control soft tissue diseases.

Phase 3- Prophylaxis and anti caries therapy.

It is the periodic dental prophylaxis, fluoride therapy, and fissure sealants.

Phase 4- Occlusal adjustment

It is done by surgery, restorative procedure & construction of prosthesis

Phase 5- Counseling in self-care

Involves a complete educational program in oral health, started from the first visit and continue throughout the entire period of patient care even in recall period.

Oral diseases

The most prevalent oral diseases are dental caries and periodontal disease these are known **as plaque related diseases**. These are infectious diseases caused by bacteria of dental plaque, so to prevent or arrest or reverse the plaque disease are based on:

1- Reducing numbers of challenge oral pathogens.
2- Building up the defenses of teeth.
3- Enhancing the repair process.

These three points can be achieved by:

1- Mechanical plaque control by using of toothbrush and inter dental cleaning devices as toothpicks, dental floss, inter dental brush.
2- Chemical plaque control by using chemo prophylactic agent as tooth paste & mouth rinse like chlorhexidine.
3- Using fluoridated products either systemic or topical fluoride agents.
4- Diet and sweet restriction by controlling the frequency and consistency of sugar intake.
5- Uses of fissure sealant, this will mechanically prevent accumulation of bacteria on tooth surfaces.
6- Health education a- Patient's education and motivation.
   b- Well planned programs as school programs.
   c- Public education programs.

DENTAL CARIES

Dental caries is a chronic disease affecting many populations. it is a microbial affecting the calcified tissues of teeth, beginning with a localized dissolution of the in organic structure of the tooth surface by acids of bacterial origin leading to disintegration of the organic matrix. Dental caries result from interaction between oral bacteria and dental tissues, its progressive and if not treated may expand in size and progress to the pulp leading to pulp inflammation thus pain and discomfort, and end with vitality lost then loss of the tooth.

Dental caries is a multifactorial disease; it is result of complex interaction between **host (tooth), bacteria of dental plaque, diet & time.** Theoretically dental caries developed when these all factors present, or we can say dental caries is an interaction between pH, mineral flux, and solubility at tooth surface.

Dental caries: - it is a multifactorial disease mainly bacterial etiology, characterized by demineralization of the inorganic portion and destruction of organic substance of the tooth

The etiology of dental caries

1- Host factor
   a- Tooth – enamel composition, morphology of the tooth, position of the tooth and nutrition.
   b- Saliva ---its role in cariogenic effect on the teeth, buffering action, salivary flow rate, salivary cleaning effect and inorganic compound that contain.
2- Microbiology
   a- Bacteria involved streptococci e.g., mutans, sobrinus. lactobacilli e.g., acidophilus.
   b- Possibly associated bacteria: - streptococci e.g., mitis; Actinomyces e.g., viscous.
3- Carbohydrate.

1- Host factors

This involves susceptible **tooth** and **saliva,** in addition to the **subject (patient).** Teeth vary in their susceptibility to dental caries from one surface to other and from subject to other.

A- Tooth susceptibility as:

1- **Positions of the tooth**, posterior teeth are more labial to be affected by caries than anterior teeth.
2- **Morphology of the tooth**, deep pits & fissures allow for accumulation of plaque & food debris thus increases the susceptibility to dental caries.
3- **Composition of the teeth**, teeth composed of inorganic elements (96% in enamel, 70% in dentin), organic element & water. Composition of teeth is affected by environmental factors such as water, diet, and nutrition.

Inorganic composition: involve major elements as calcium, phosphate, hydroxyl group these are the constituents of hydroxyl apatite crystal $Ca_{10}(PO_4)_6(OH)_2$. The Ca/P ratio is 2.15. Any change in this ratio is an indication of presence of other types of crystals. There are minor elements may incorporate the enamel crystal in substitution of Ca ions by Mg, $Ca_9Mg(PO_4)_6(OH)_2$ or substitution of the OH by Fluoride ion $Ca_{10}(PO_4)_6F_2$. These minor or trace elements may adsorb on the surface of the crystals. This incorporation may take place either in the pre-eruptive stage including all layers of enamel & dentin, or post eruptive stage involving the outer enamel surface only. Some of these elements when incorporated may increase the resistance of the teeth to dental caries as fluoride ion, tin ions, zinc, strontium, and molybdenum, while other elements may increase the susceptibility to dental caries as magnesium. However, the role of other elements may not well understand as K, Mn, & Al.

Physical and microscopic features of incipient caries

The development of caries occurs in three distinct stages: -

1st stage: - incipient lesion histological changes of enamel

2nd stage: - progression of demineralization toward the DEJ and dentin.

3rd stage: - development of frank lesion (cavitation).

When the time between onset and development of cavity too rapid then it is called rampant caries.

The speed of progression of caries depends on: -

1- Ion concentration
2- pH of saliva
3- buffering actions
4- salivary flow

The initial acid attack dissolve Mg and carbonate ion and later less soluble Ca ion, pH and other ions that part of the crystal.

Enamel sieve concept

After sugar consumption there will be an increase in hydrogen ions in dental plaque causing an increase in pressure ingredients, this will lead to dissolution of hydroxyl apatite crystal to their ionic components. These ionic wills diffuse toward dental plaque leaving behind microscopically spaces. By the increase of pH due to the action of buffer system and termination of carbohydrate a remineralization episode will be started, calcium, phosphorous ions and others will diffuse back to enamel from dental plaque. The precipitation of ions will be in form of a verity of complex salt crystals. In a succession of demineralization and remineralization cycle, if the sum of the demineralization is greater than the remineralization there will be a continuous loss of minerals thus porosity then cavitation i.e., dental caries.

Types of dental caries

1- Pit and fissure caries
2- Smooth surface caries
3- Root or cemental caries
4- Recurrent caries
5- Baby (nursing) bottle caries
6- Rampant caries

Caries of enamel

1- Pit and fissure caries in enamel

Lesion begins beneath plaque, with decalcification of enamel. Pit and fissures are often deep, with food stagnation. Enamel in the bottom of pit or fissure is very thin, so early dentin involvement frequently occurs. Here the caries follows the direction of the enamel rods; it is triangular with the apex facing the surface of the tooth and the base towards the DEJ. When reach the DEJ the greater number of dentinal tubules are involved and produce greater cavitation than the smooth surface caries and there is more undermining of the enamel.

2- Smooth surface caries

Due to plaque formation on enamel, the earliest manifestation of incipient caries (early caries) of enamel is usually seen beneath dental plaque as areas of decalcification (white spots). The first change seen histological is the loss of inter-rod substance of enamel with increased prominence of the rods; this is followed by the loss of mucopolysaccharides in the organic substance presence of transverse striation of the enamel rods. As it goes deeper, the caries forms a triangular pattern or cone shaped lesion with the apex towards DEJ and base towards the tooth surface. Finally, there is loss of enamel structure, which gets roughened due to demineralization and disintegration of enamel prisms. Once cavitation occur the zones of incipient lesion become not clear less defined because of (1)less mineral present, (2) presence of bacteria and (3) bacterial end product, plaque, residual substance which support further lesion development.

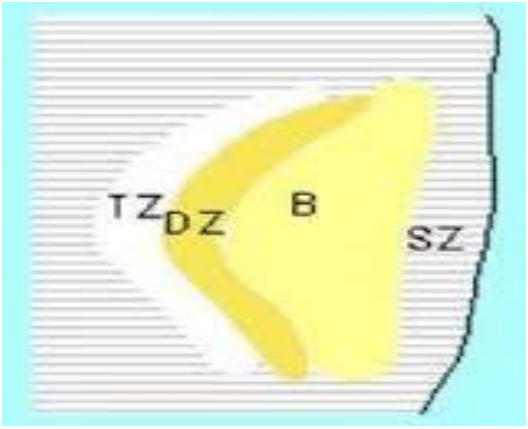
Caries zones

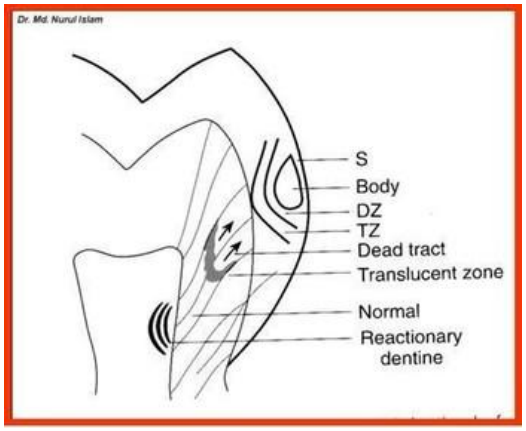
Zone 1: - **Translucent zone**, 50% of the lesion, lies at the advancing front of the lesion, slightly more porous than sound enamel 1.1%. It is not always present.

Zone 2: - **Dark zone**, 95% occur in lesion, this zone is usually present and refer to as positive zone, 2-4 % pore vol. It is formed due to demineralization.

Zone 3: - **Body** of the lesion, 5-25 % pore vol. It is found between the surface and the dark zone, it is the area of greatest demineralization.

Zone 4: - **Surface zone** 1 % pore, relatively unaffected area, greater resistance probably due to greater degree of mineralization and greater F concentration.





Caries of dentin

Begin with the normal spread of the process along the DEJ and rapid involvement of the dentinal tubules. The dentinal tubules act as tracts leading to the pulp, during caries infection more fluid is forced into tubule also act as path for microorganisms.

Early dentinal changes

1- Initial penetration of the dentin by caries leading to dentinal sclerosis because odontoblast loss their vitality and become dead tract and began to calcify.
2- Calcification of dentinal tubules and sealing off from further penetration by microorganisms (protective barrier) more prominent in slow chronic caries.
3- The pulp begins to form amorphous reparative dentin for further protection of the pulp.
4- If surface intact in slow caries theoretically can be remineralized.

Shape of the lesion is triangular with the apex toward the pulp and the base toward to the enamel.

Root caries

Root caries defied as a soft progressive lesion that is found anywhere on the root surfaces that has lost its connective tissue attachment and it's exposed to the oral environment. The prevalence of root caries was shown in men 1.1-2.5 more than women. The root surface must be exposed to the oral environment before caries develop also studies founded that in age 30_s 1/1000, while in 50_s 1/5 may have root caries. Plaque and microorganisms are essential for the cause and progression of the lesion, mostly Actinomyces. Microorganisms invade the cementum either along the Sharper's fibers or between the bundles of fibers. After decalcification of cementum, destruction of matrix occurs like dentin ultimate softening and destruction of the tissue. Finally, invasion of microorganisms into the dentinal tubule leading to pulp involvement and the rate is slower due to fewer dentinal tubules than in crown area.

The root caries appears clinically as yellowish-brown coloration, non-cavitation, soft, may assume any outline, depth 0.5-1 mm, complete loss of cementum. The expose of dentin may undergo remineralization or arrest.

Arrested root caries

It demonstrated three physical characteristics: -

1- Outer barrier layer of hyper mineralized dentin.
2- Sclerotic inner barrier between carious and sound dentin.
3- Mineralization occurring within the dentinal tubules.

The mineralized lesion appears glossy, smooth, hard, while hyper mineralized the active lesion has leathery feeling.

Recurrent caries

It is defined as caries recurring in an area due to inadequate removal of the initial decay, usually beneath a restoration or new decay at a site where caries has previously occurred.

Nursing Bottle caries

It is a type of rampant caries which is demarcated because of the age factor, nursing bottle caries is seen in infants and toddlers and rampant caries is seen in any age and seen in both primary and permanent teeth. As the name suggest nursing bottle caries is caused mainly due to the feeding habits seen in bottle feeding, it was named and renamed many time before ending up with nursing bottle caries-nursing caries, nursing bottle mouth, nursing bottle syndrome, night bottle syndrome, baby bottle caries, milk bottle syndrome and others names.

Mandibular anterior teeth are not affected in nursing bottle caries. Bovine milk and human breast milk have lactose contents which are the cause foe nursing bottle caries, nursing bottle directly blocks the salivary access to the tooth surfaces, thereby increasing the cariogenicity of the oral flora.

1- **Type / nature**: -
   Specific from of rampant caries, acute generalized spread of caries and pulp involvement in selected teeth of dentition
2- **Age:** -
   Infant and toddlers
3- **Cause/ etiology**
   a- Feeding children with milk bottle while the child is lying down or sleeping breast feeding whenever the child asks and at will for prolonged duration of time.
   b- Use of pacifiers which are coated with honey or any artificial sweeteners to stop the baby from crying.
   c- It involves only the feeding factor.
4- **Treatment**
   Treatment plan depends on the stage and time of detection and intervention by the parents and dentist,
   a- if diagnosed at an early-stage fluoride application and parent education.
   b- Pulpotomy and pulpectomy and space maintenance are decided based on the signs and symptoms until the transition occurs.
   c- Education the parents about the feeding habits
5- **Prevention**
   a- Education of the parents or new mothers is important to prevent nursing bottle caries. Feeding habits should be explained in detail
   b- Timing should be maintained for feeding
   c- Prolonged feeding is not recommended.
   d- Try to avoid feeding in sleeping position

e- Make the child drink water after feeding to cleanse off the milk.

Why mandibular incisors are not affected in nursing bottle caries? Because of the constant flow of saliva from sub mandibular gland as the salivary duct opens here and the constant cleansing movement of the tongue tip on the lingual surface of the lower incisors.

Rampant caries

It is an acute, generalized spread of caries and pulp involvement in all teeth.

Type / nature

1- Not specific teeth are involved as all the teeth are equal involvement; mandibular incisors are also equal affected as other teeth. Seen at any age, as nursing bottle caries is a type of rampant caries, if seen in toddle and infant it is called NBC and if seen after that age it is called as rampant caries.

2- Age: -

At any age, both primary and permanent teeth are involvement and no specific teeth.

3- Cause/ etiology

a- Frequent intake of sweet, sugar and sticky food substances throughout the day.
b- Water intake through the day and decreased salivary flow.
c- Genetic predilection if seen in parents or family members.
d- It is combination of many factors coming together.

4- **Treatmen**t

a- Treatment plan depends on the stage intervention; early intervention requires removal of caries and restoration.
b- Crowns depending on stage of tooth decay.
c- In case of pulp involvement pulp therapy / root canal treatment is required.
d- Fixed partial denture or extraction and replacements are done in case of permanent teeth are involved.

5- Prevention

a- As rampant caries occurs at any age; patient education to identify any carious lesions and get immediate dentist intervention.
b- Patient education with the help camps or community centers.

B- Saliva

Saliva affects caries etiology through

1- The rate of secretion and composition.
2- Saliva affects the integrity of the teeth by the composition of buffer system, calcium & phosphate.
3- By the cleansing action of saliva, it can affect the number of oral microorganism and food debris from the mouth.
4- The oral immune system (specific & nonspecific) affect to large degree the cariogenic bacteria.

c- Subject:

The behavior, attitude and dental knowledge affect the caries etiology. These can influence the oral hygiene of the person as well as his dietary habits.

2-Dental plaque

Dental plaque & quality greatly influence caries etiology. Bacteria adhere to the tooth surface & ferment carbohydrate causing release of acid thus demineralization of tooth surface. Cariogenic bacteria involve mutans streptococci, lactobacilli & others.

3-Diet

Sweet consumption especially between meals may lead to continuous drop of PH & not allowing the **enough time** for the PH to return to normal, thus lead to demineralization of teeth.

Theoretically when these all factors are present dental caries develop. Dental caries is an interaction between pH, mineral flux & solubility at tooth surface.

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: -

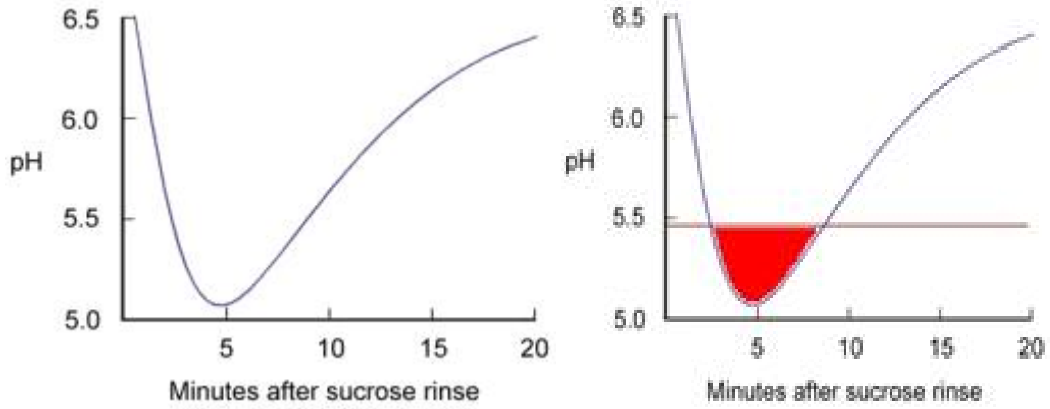
1- Better access to dental care for patient.
2- Providing a better understanding of a patient's home/living environment.
3- Providing better understanding of patient's ability to carry out oral hygiene advice.
4- Reducing the likelihood of failed appointments.
5- Frequently achieving better patient compliance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

Stephan curve

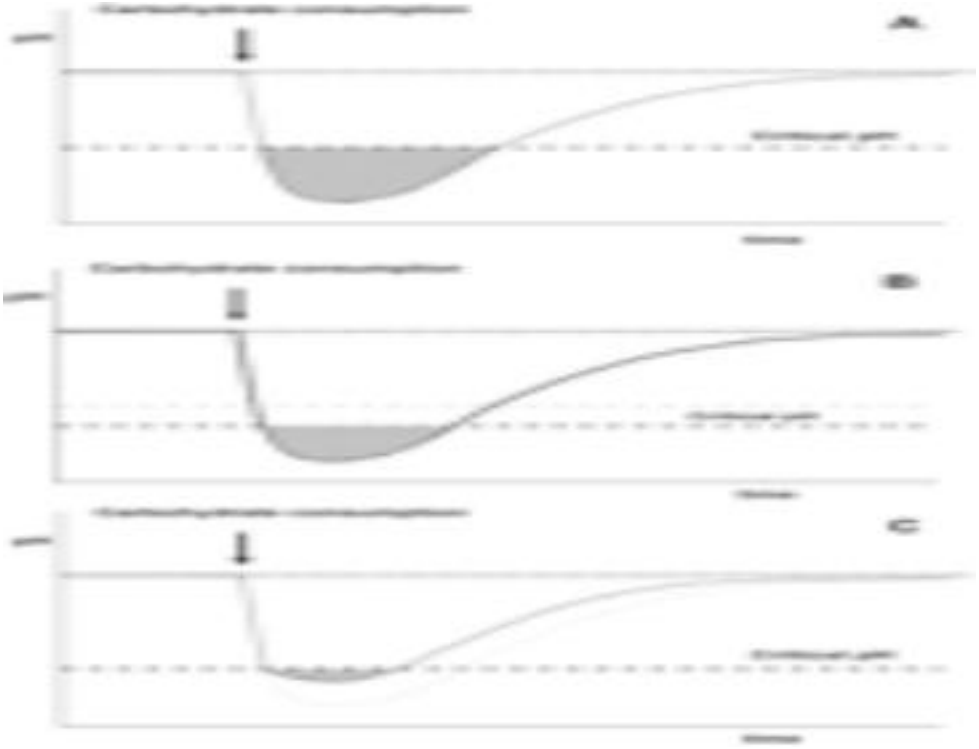
Lec -3-

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 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. Patient 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.

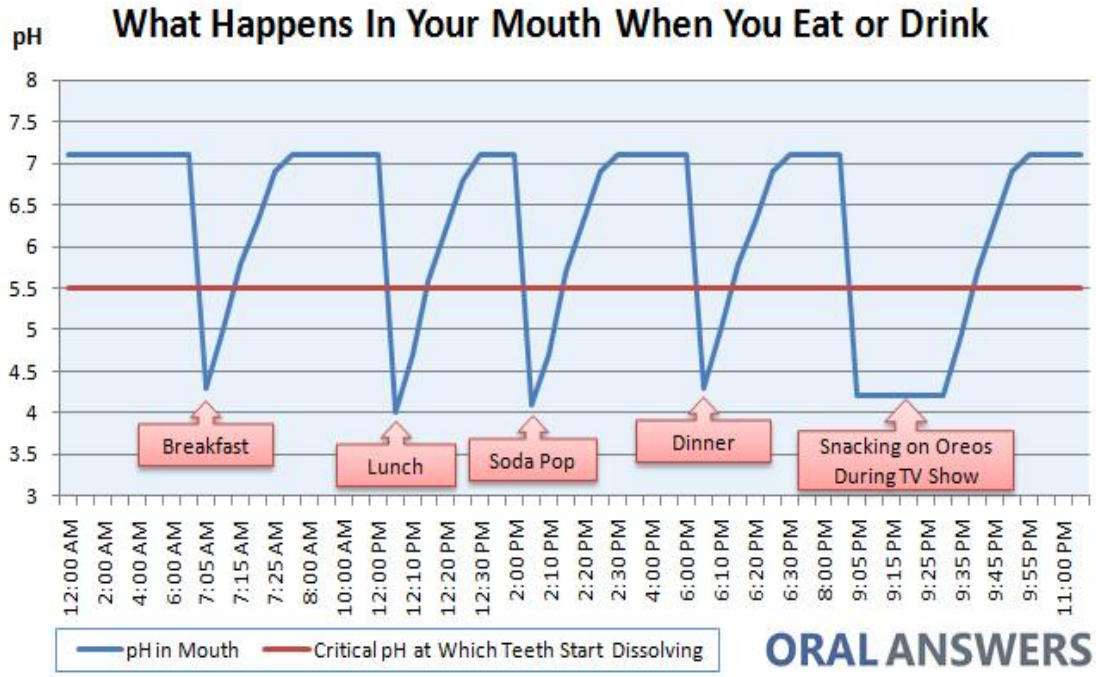


The plot of plaque PH against time has become known as **the Stephan curve**. The rapidity with which the PH fall was reflected to the speed on which sucrose come diffuse into plaque and the activity of the concentration of enzymes. The slow rate of recovery to the resting pH and critical factor in caries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

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.

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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.

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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.

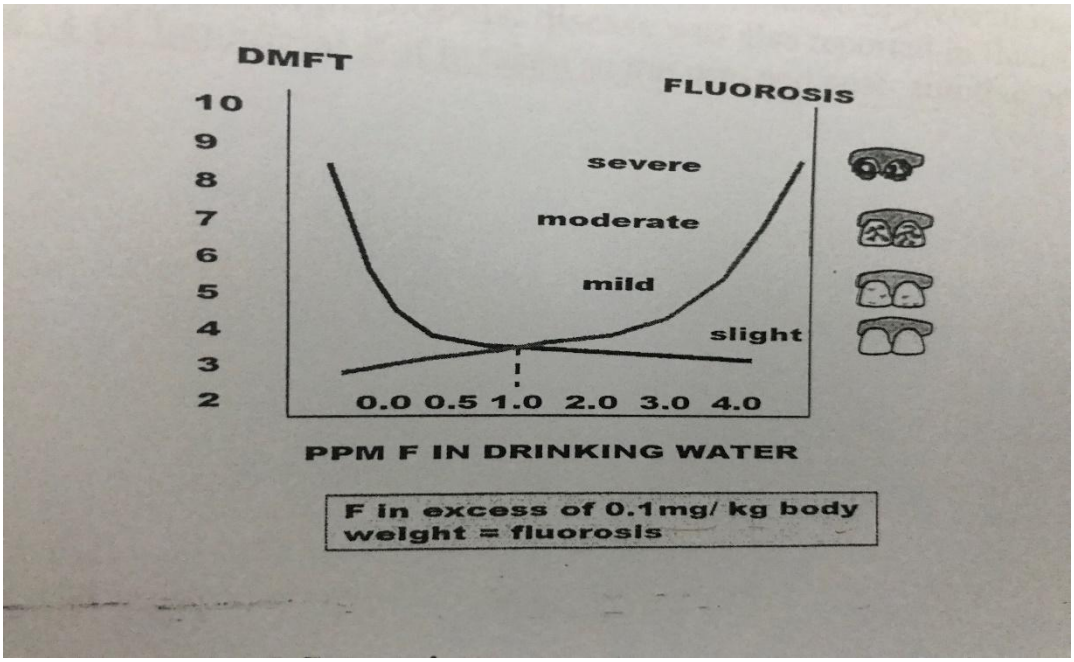

Table (1): Effect of fluoride in water on human health when consumed for longer durations

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)

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- 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.

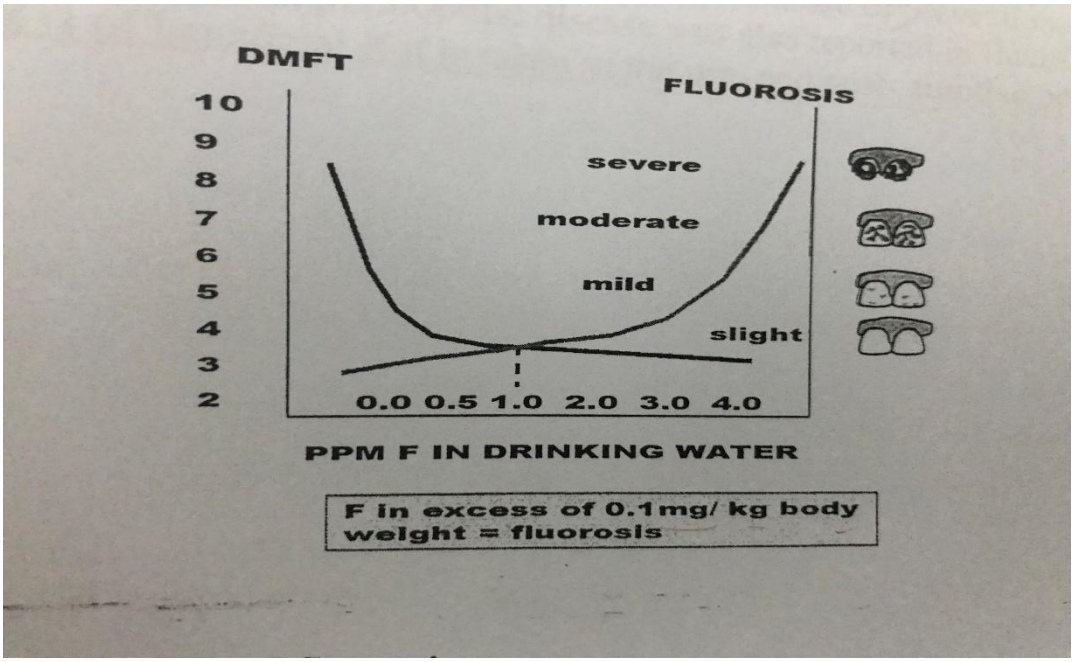

Table (1): Effect of fluoride in water on human health when consumed for longer durations

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)