Oral pathology

Dental caries

Dental caries is a multifaceted disease involving interplay among the teeth, the oral host factors of saliva and microflora, and the external factor of diet. The disease is a unique form of infection in which specific strains of bacteria accumulate on the enamel surface, where they elaborate acidic and proteolytic products that demineralize the surface and digest its organic matrix.

Once penetration of the enamel has occurred, the disease progress through the dentin to the pulp. If the process is not stopped, the tooth becomes destroyed.

Epidemiology of dental caries

The prevalence and severity of dental caries differs among various populations throughout the world. The caries activity in a particular society or geographic area is closely correlated with the amount of sugar consumed per capita. In the more industrialized countries, where diets have traditionally had a high content of refined carbohydrates, the caries rate has been considerably higher than in less-developed countries. In recent years with the trend toward preventive measures such as fluoridated water, greater access to dental care and better oral hygiene in industrialized countries, and the concurrent rapid increase in caries activity in the less-developed societies, the large difference in caries rate has decreased.

Factors affecting caries prevalence: -

1- race: people living in same geographical area but belonging to different race have differing caries incidence. Generally, chinese, blacks, indians have lesser caries incidence than the caucasian whites.

2. Age: dental caries more prevalent in children up to 12 years. Incidence decreases somewhat in younger and middle age group. Incidence increases again by the older age.

3. Gender: incidence of caries is significantly higher in females than males. This may be due to the fact that teeth in females erupt earlier compared to males.

4. Familial: there appears to be heredity involved. Children of parents with low caries experience also show lesser caries incidence and vice versa.

Loss of tooth substance may result from the action of oral microorganism in dental caries (bacterial causes), or may be due to non- bacterial causes, which include:

A- Mechanical factors associated with attrition and abrasion.
B- Chemical erosion.
C- Pathologicresorption.

Primary causes of dental caries

Dental plaque,dietary carbohydrates, tooth (susceptible tooth surface) and time.

The carious process : bacteria in dental plaque fermentable carbohydrates such as sugars (sucrose & glucose). .production of acids causing the plaque ph to fall below 5 .Repeated fall in ph in time may result in the demineralization of susceptible site on the tooth surface initiating the carious process.

Dental caries follows the interaction of four main factors, the host, bacteria, food (diet) and time for the process to develop.

Food + bacteria ⟶ acid +tooth ⟶ D.C.

Caries is one of the most common of all diseases and still a major cause of loss of teeth.

Dental caries is the most prevalent chronic disease in man throughout the world, 95% of the population have decay or will have it before they die. The only way to control

the disease is through the use of systemic and topical fluoride, furthermore dental \-\\\\ crycaries can be controlled by controlling the four main factors that are related to it: -

1- Host (tooth): administration of fluoride (as tables, or fluoride-containing diet), and fissure sealant (seal deep fissures in tooth surface to prevent accumulation of plaque).

2- Microorganism (bacterial flora): their action is hindered by active and passive immunization, and reduces the intake of sugars.

3- Diet (food):- reduction in consumption of cariogenic sugar like sucrose, fructose, maltose, glucose, both intrinsic sugar (from fruits and vegetables) and extrinsic sugars (added sugars, milk, fruit juices).sucrose is considered as the most cariogenic type of sugar because (1) it is readily fermented by bacterial plaque, and (2) its easily converted to extracellular glucans by bacterial glucosyltransferase. Glucans act as glue for bacteria helping their adherence to tooth surface.

4- time: frequent sugar intake between primary meals, as well as stopping teeth brushing for 12-14 hours will permit formation of bacterial plaque.

There are other indirect factors that have a role in the development of dental caries, such as:-

Tooth: regarding its

- Composition; (less fluoride, iron, zinc, magnesium make tooth more susceptible to dental caries).
- Morphology; (deep pits and fissures can seat more bacterial plaque).
- Position; (malposed tooth can hold more bacterial plaque).

Saliva: regarding its:

- Composition; inorganic constituents are more beneficial than organic constituents.

- ph; the higher the ph the less the action of bacteria.
- Quantity; the more the best washing action of plaque out of embrasures, fissures and pits.
- Viscosity; the more watery the best for the removal of plaque.
- Other antibacterial factors that prevent the proliferation of bacterial flora.

Diet (food): regarding its: Physical factors: quantity of diet.

Local factors: carbohydrate content, fluoride content, vitamin content.

Soft sticky food enhances the formation of plaque, and consequently caries. Refined carbohydrates, especially sucrose, are more likely to cause caries than raw products.

Vitamin content of diet: -

Of all vitamins, only vit d and vit k appear to have some role in the caries process. Vit d may have an indirect effect on caries process. Its deficiency can cause enamel hypoplasia which can make the tooth more susceptible to caries. Vit k has enzyme inhibiting action in carbohydrate degradation cycle can be utilized as an anticariogenic agent.

Calcium &phoshorus content:-

Available evidence indicates that there is no relation between dietary calcium and phosphorus and dental caries.

Fluorine content: - while topical and water fluoridation has been known to be effective in caries control, dietary fluorine may have no role as it is unavailable metabolically.

Systemic factors

Heredity: - racial tendency for high or low caries may be explained by heredity. However, local factors like change in dietary habits can change this tendency. Possible that caries tendency may be inherited through tooth form & structure

Pregnancy & lactation: - commonly observed that during pregnancy, women tend to neglect their oral health owing to all her attention being diverted to that of care for the newborn. Thus increased caries incidence during pregnancy & lactation is more a problem of neglect.

Etiology and pathogenesis:

Etiology is still controversial and not clear, due to its being complicated by many direct and indirect factors. Many theories were postulated in order to explain dental caries. Most noticed theories are:

1) ***Acidogeneic theory (miller s chemoparasitic theory 1890):-*** it is the most accepted and supported theory, because it is based on experimental studies; made later by Orland and his workers in 1954, showed that in germ free oral hygiene in some laboratory animals ,even with administration of sugar; there is no dental caries in these animals. Thus, dental caries is produced by chemical action of acids produced by micro flora.

Miller s theory suggests that dental caries develop in two phases. In the first phase, microflora attack the inorganic structure, where decalcification of enamel and dentin is carried out by means of acids produced as a result of fermented sugar accumulating in retaining spots on tooth surface .in the second phase, dissolution of the soft organic part is carried out.

Miller isolated numerous microorganisms from the oral cavity; most important species are lactobacillus acidophilus, streptococcus mutans , streptococcus sanguis , and streptococcus salivarius.

In his hypothesis, miller assigned essential roles to 3 factors:

1. Carbohydrate substrate.

2. Acid which caused dissolution of tooth minerals.

3. Oral microorganisms which produce acid and also cause proteolysis.

The mouth:

The mouth is the beginning of the digestive system chewing (masticating) not only grinds foods but degrades it with enzymes in the saliva. Saliva is a complex mixture of salts, carbohydrates, and enzymes. Some of these enzymes (amylases) break down carbohydrates into sugars that are important for the initiation of dental caries. There are many bacteria in the mouth there are really only a few species of bacteria, each bacterial species has a unique habitat in the mouth mainly streptococci are found in the mouth. *Streptococcus salivarius*, *streptococcus sanguis*, *streptococcus mitis*, streptococcus *mutans*.

The streptococci are gram positive (and have a sticky cell wall) and facultative anaerobes. Facultative organisms can live in both aerobic and anaerobic environments. Lactobacilli and actinomyces are also important oral micro flora.

Objections to the hypothesis: - unable to explain predilection of specific sites on tooth to caries. Initiation of smooth surface caries not explained. Unable to explain why some populations are caries free and some are caries prone. However, this theory is accepted by majority in unchanged form. Also, bulk of evidence does implicate carbohydrates, acids and microorganisms.

Role of dental plaque: - plaque defined as a soft, unmineralized, bacterial deposit or biofilm which forms on teeth and dental prostheses that are not adequately cleaned. Resists cleansing by physiological oral forces like salivary washing and tongue movements but is removable by tooth brushing. Considered as a contributing factor for at least initiation of caries. However mere presence of dental plaque doesn't necessarily mean caries will occur.

Composition of dental plaque => water – 80% solids – 20% dry weight of plaque composed of bacterial & salivary proteins – 50% carbohydrates & lipids - 25% inorganic ions, mainly Ca++ &PO4--- - 10%.

Classification of dental plaque => plaque classified as – supragingival&subgingival.Supragingival plaque – essential role in causing caries, while subgingival plaque – role in periodontal diseases.

Mechanism of formation =>

1- Plaque formation proceeds through following stages deposition of a cell free layer, acquired pellicle which is derived from salivary glycoproteins. This layer acts as nutrient for plaque bacteria.

2. Colonization of pellicle by gram positive bacteria like s.sanguis and s.mutans within 24 hours.

3. Maturation of plaque by further colonization with filamentous and other bacteria. Also there is buildup of plaque substance by polysaccharides produced by plaque bacteria.

2) proteolytic theory (bodecker 1878) :-

Main suggestion of this theory is that microorganism attack the organic part of enamel, leaving the generated acid responsible for further decalcification of inorganic part. Bodecker suggested that bacteria could penetrate into enamel through lamellae and interprismatic substance.

Objections to the theory: - out of 0.56% of organic matrix, 0.18% is keratin. However, no enzyme systems capable of attacking keratins have been isolated so far. Studies in germ free rats have shown that caries can occur in the absence of proteolytic

organisms. However, even though proteolysis may not play any role in initiation of caries, their role in progression of more advanced carious lesions cannot be ruled out.

3-proteolysis – chelation theory:-

Schatz et al in 1955 proposed that caries occurred as a result of simultaneous degradation of organic substances (proteolysis) and dissolution of tooth minerals by a process called chelation. According to this theory, the initial attack on the tooth is on the organic components of enamel. Breakdown products of the proteolysis have chelating properties which form chelates with mineralized components of enamel and thereby decalcify the enamel even in neutral or even alkaline ph.

Objections to this theory: - direct evidence for proteolysis – chelation as a mechanism for causing caries is lacking. Recent studies have shown that saliva as well as plaque does not contain substances in sufficient concentrations to chelate calcium from enamel. However, although chelation may not be actually responsible for initiating caries, it may still have some role to play in advanced carious lesion where the ph levels return to neutral.

The last two theories are disregarded, simply because they lack support by experimental studies.

In general, the essential requirements for development of dental carries are :

1-cariogenic bacteria.

2-bacterial plaque.

3-stagnation area.

4-fermentable bacterial substrate (sugar).

5-susceptible tooth surface.

6-time for process to develop.

Clinical classification of dental caries:-

It is classified either according to site of attack, or according to rate of attack and according to whether lesion is new or under previous restoration: -

1. Primary (virgin) caries 2. Secondary (recurrent) caries

According to site of attack, it is classified as follows:

1. Pit and fissure caries.
2. Smooth surface caries.
3. Cemental or root caries.

Pit and fissure caries: this is frequent in occlusal surfaces of molars and premolers, buccal surface of molars, lingual and palatal pits of incisors.

Early caries appears as brown or black discoloration in fissures and pits, and when inspected with dental probe, probe stick to it. In some caries in occlusal surface, where caries extends laterally in to dentin, enamel above it appears chalky white in color, because of undermined caries .caries of enamel is usually studied by ground section using a special technique, whereas in decalcified section enamel will be completely lost.

Smooth surface caries: this caries is frequent on proximal surface and gingival third of buccal and lingual surfaces (class v).

Proximal caries occur just below contact point and appear as a well-demarcated chalky white opacity of enamel.

According to rate of attack, dental caries is classified in to:

1. Rampant or acute caries.
2. Slow progressive or chronic caries.
3. Arrested caries.

1- Acute dental caries it is that form of caries that follows a rapid clinical course and results in early pulpal involvement by carious process. Predominantly affects children and young adults probably because their dentinal tubules are larger and show no sclerosis. The point of entry of caries is small even though there is rapid spread of caries at dej, producing large internal cavitation.

The small point of opening doesn't allow the buffering ions of saliva to neutralize acids formed within the cavity. The affected dentin is usually stained light yellow compared to deep brown / black of chronic caries. Pain is more likely to be seen in acute dental caries than chronic caries.

Rampant dental caries characterized by sudden, rapid destruction of teeth affecting even relatively caries free surfaces like proximal and cervical surfaces of mandibular teeth. 10 or more carious lesions over a one year period are characteristic of rampant caries. Prominently observed in deciduous dentition of young children and permanent dentition of teenagers. Dietary factors like high carbohydrate intake as well as physiological factors affecting saliva are major contributors to etiology of rampant caries.

Nursing bottle caries also called baby bottle syndrome and bottle mouth syndrome. It is a type of rampant caries and occurs due to – nursing bottle containing milk, milk formula or sweetened water. Usually, the above aids are used at sleeping time after one year of age.

Clinically seen as widespread caries of the 4 maxillary incisors followed by 1st molars and then canines. Absence of caries in mandibular teeth distinguishes it from ordinary rampant caries. If milk or other carbohydrates are rapidly cleared from mouth, they aren't cariogenic, but if they pool in the mouth, then they can cause rampant caries.

2- Chronic caries: ordinary caries that develops slowly, and appears fully damaging in old ages, because it requires time.

3- Arrested caries: is type of caries where a reminalization of dentin occurred, thus hindering further caries. .

Reminalization is achieved by fluoride in saliva, and if the caries is in a self – cleansing area. This happen in case of badly carious teeth, where enamel is grossly damaged and fractured, thus dentin is reminalized. Also in proximal surfaces caries, after one of the teeth is extracted, the spot of caries will be in self – cleansing area and get reminalized by fluoride in saliva.

4- Recurrent caries.

Histopathology of caries:

Histopathology of caries of enamel

Enamel forms the main protective covering of the grown. Enamel is composed of 96% inorganic material, and 4%organic material and water. Enamel structure is constructed by enamel rods or prisms, rod sheath and interprismatic substance. Enamel rods appear as a body and tail directed from dentinoenamel junction; dej, outward to root surface.

Enamel consists of crystals of hydroxyapatite packed tightly together in orderly arrangement. Each crystal is separated from its neighbors by tiny intercrystalline spaces or .pores. The spaces are filled with water and organic material. When enamel is exposed to acids produced by dental plaque, minerals is removed from the surface of the crystals which shrinks in size. The intercrystalline spaces enlarge and the tissue becomes more porous. “At this stage the carious lesion can be detected clinically and called white spot lesion “.

White spot lesion

The earliest microscopic evidence of caries in enamel best seen on dried tooth as a small, opaque, white area.Sometime the lesion may appear brown in color due to exogenous materials .absorbed into its porosities. If the early enamel lesion progress, the intact surface breaks down (cavity formation).

The carious lesions in smooth surface are slightly different when compared to that in pits and fissure caries.

Microscopic appearance of the white spot lesion on a smooth surface:

Smooth surface = proximal surfaces and buccal surfaces of the teeth. Usually cone shaped: the apex of the cone pointing toward the dentino enamel junction (dej). The lesion takes this shape because it follows the direction of the enamel prisms.

Several zones can be distinguished before complete destruction of the enamel

Zone i: translucent zone: In this zone, demineralization has taken place (magnesium and carbonates are dissolved) not seen in all lesions lies at the advancing front of the lesion More porous than sound enamel. Pores have been created by the demineralization process.

Zone ii: dark Zone: Someremineralization happens due to reprecipetation of minerals, lost from the translucent zone just superficial to the translucent zone. More porous than the translucent zone.

Zone iii: body of the lesion: It is the area of greatest demineralization and having a higher fluoride level and lower magnesium level. The largest portion of the lesion superficial to the dark zone. Increase in porosity from the peripheries to the center.

Zone iv: the surface zone unaffected surface layer that cover the small lesion. High degree of mineralization than subsurface enamel.

If the lesion progress the surface layer will be destroyed.Leads to cavity formation.

Histopathogenesis of the early lesion

The development of enamel caries can be traced through the following stages when ground sections are examined by transmitted light

1-development of a surface translucent zone, which is unrecognizable clinically and radiographically.

2-the subsurface translucent zone enlarges and a dark zone develops in its center.

3-as the lesion enlarges more mineral is lost and the center of the dark zone becomes the body of the lesion. This is relatively translucent compared with sound enamel and show enhancement of the striae of retzius. Interprismaticmarkings and cross striation of the prisms. The lesion is now clinically recognizable as a white spot.

4-the body of the lesion may become stained by exogenous pigments from food, tobacco, and bacteria. The lesion is now clinically recognizable as a brown spot

5-when the caries reaches the amelodentinal junction it spreads laterally, and in this way the enamel may become widely undermined, giving the bluish-white appearance of the enamel seen clinically, extension along the amelodentinal junction may result in secondary undermining enamel caries. The time for caries to progress through enamel on the approximal surfaces of permanent teeth has been reported to be about 4 years but may be up to 8 years.

6-break down of the surface zone with formation of a cavity. This stage may precede stage 5.

In some lesions dentin may be involved late in stage 3. While in other lesions it is not involved until stage 5 or 6. Bitewing radiograph do not show lesions until stage 4 or possibly late stage 3

No unequivocal evidence for specific points of entry of the carious attack into the enamel has been found with light microscopy. Acid appear to diffuse in over a broad front. Ultrastructural studies suggest preferential dissolution initially along prism

boundaries, but there is also a diffuse demineralization with an increase in intercrystallite distance affecting areas both within and between the prisms. These intercrystallite spaces presumably reflect the variation in pore volume in different areas of the lesion; changes in crystal structure are thought to be due to both demineralization and reprecipitation of mineral.

Pit and fissure caries (occlusal caries):-

The caries follow the direction of the enamel rod, and a triangular or cone shaped lesions with the apex at the outer surface, and its base toward the dentino-enamel junction.

Light microscope appearance of occlusal caries:-

The lesion forms around the fissure walls. As the lesion increases in size, it coalesces at the fissures. The enamel lesion enlarges as it approaches the underlying dentin guided by prism direction. The lesion takes the shape of a cone with its base toward the enamel-dentin .junction. The tooth enamel (outer layer of crown) is very hard and doesn't dissolve easily even with acids, but the underlying dentin dissolves easily with lactic acid eventually a cavity is formed in the dentin below the enamel , when the cavity is large enough, the enamel will crack, exposing the dentin, bacteria find the dentin a rich environment for growth and quickly eat through the dentin into the underlying pulp(where the blood vessels and nerves are) this is now a rampant carries and the tooth will likely be removed.

Fluoride makes the tooth mineral harder to dissolve, this is why fluoride treatment is effective in preventing dental caries.

Note: - more cavitations in pits and fissure than smooth surface caries. Why? Because:-

1- The enamel at the bottom of the pit and fissure may be very thin, so caries reach faster.

2- In pits and fissure, the enamel rods are directly directed laterally (diverge), when caries occur it follows the direction of these rods leading to the formation of cone shaped or triangle.

3- The enamel at the surface become undermined and starts to collapse under the stress of mastication and to fragment around the edge of the cavity. By this stage, bacterial attack on the dentin is well established. Cavitations are greater than that of the proximal surface.

Caries of dentin

Dentin composed of 30% organic material and water, 70% inorganic material.

Dentin is composed of dentinal tubules, inside which is the odontoblastic process. Odontoblastic process is the extension of odontoblas inside the dentin, these process have lateral branches anatomizing with each other, and form a network.

As caries reaches the enamel-dentin junction, caries spread laterally along the junction. Sound enamel appears to be undermined by the carious process in dentin. Undermined enamel is brittle and can be fractured producing a large cavity.

When caries reached the dentin, there is a lateral spread of the lesion, involve more tubule which act as pathway or tract along which the microorganism will spread to the deeper areas and then to the pulp in a conical or triangular pattern with the apex toward the pulp and the base to the dentine.

At the first, the decalcified dentin retains its normal morphology and no bacteria can be seen.

Once the dentine has been reached, pioneer bacteria extend down the tubule, soon fill them and spread along any lateral branches.

The tubules become distended into spindle shapes by the expanding masses of bacteria and their product, as a result, adjacent tubule which are less heavily infected become bent, later the intervening tubule wall are destroyed and collections of bacteria in adjacent tubule coalesce (united) to form irregular liquefaction foci (these are ovoid areas of dentinal destruction and it is parallel to the direction of dentinal tubule. It is filled with necrotic debris which increases gradually in size by expansion; in some areas, bacteria also spread laterally and occasionally large bacteria filled, clefts formed at right angles to the tubules. Clinically, these clefts may allow carious dentin to be excavated easily.

We can summaries dentin carious lesion from the pulpal aspect outward into following zones:-

1- Zone of fatty degeneration of protoplasmic process: effect of bacterial enzyme on the cell membrane of the organic component.

2- Zone of dentinal sclerosis (translucent zone): regarded as vital reaction of odontoblast to irritation (deposition of calcifying salts from the demineralized zone).

3- Zone of decalcification: soft dentin due to the action of bacterial enzyme

4- Zone of bacterial invasion.

5- Zone of decomposition of dentin: cavitation (become no mineralized remain and the organic component dissolved by the bacteria).

Protection reaction of dentin and pulp under caries:

The reaction in dentin are mainly due to odontoblast activity, these reactions are not specific to dental caries, but may be occur as a result of other irritant cause such as attrition, abrasion and restorative procedure.

At early stage of dentin caries, a defensive mechanism of dentinal tubule and the vital pulp occur by:

1- Development of dentinal sclerosis or translucent dentin which mean calcification of dentinal tubules which will seal them to prevent bacterial penetration, this form in a band about half way between the pulp and adj. This process is minimal in rapidly advancing caries and prominent in slow dentin caries.

2- the odontoblast in the pulp react to changes in dentin by formation of reparative dentin (tertiary dentin – a tubular dentin) this dentin is localized to the irritant odontoblast irregular or a tubular dentin.

3- Secondary dentin: tubular dentin separated from primary dentin by hyperchromatic line or demarcated zone. It is formed following eruption throughout the life of the tooth.

Root surface caries:

Cementalcaries:

Cementum, hard tissue covering dentin in root region. It is composed of 45-50% inorganic material, and50-55% organic material. Cementum is of two types:

A- Cellular cementum , covering root from cej to apical one third of the root. Cells are termed cementocytes and are spider shaped cells.

B- AcellularOccur chiefly in old people in whom the gingiva has retracted.

At first, plaque forms in the cemental surfaces, then the microorganism penetrate the cementum along or across calcified sharpies fibers. Then the bacteria seem to spread vertically in layers following the pattern of cementum formation

The cementums soften beneath the plaque over a wide area producing a saucer-shaped cavity. The decalcification of the cementum is formed by further destruction as in dentin.

Arrest of lesions

Inactive or arrested white spot lesions have a shiny surface and may be brown in color, having picked up exogenous stains from the mouth. These lesions cannot be detected by gently drawing a sharp probe across them because they feel the same as normal enamel. Histologically these lesions show wide, well-developed dark zones at the front of the lesion within the body of the lesion and at the surface of the *lesion*.

It is very important to realize that the carious process can be arrested by simple clinical measures such as improved plaque control with fluoride toothpaste and altered diet. It is therefore the clinician's responsibility to detect enamel caries in its earliest form by careful visual inspection of teeth after cleaning and drying. The clinician can now help the patient tip the balance in favor of arrest rather than progression of lesions. An arrested white spot is more resistant to acid attack than sound enamel. It may be regarded as scar tissue and should not be attacked with a dental drill.

Arrested caries and remineralizationprecavity (white spot) may become arrested when the adjacent tooth is removed so that the stagnation area is removed, the lesion may become remineralized by mineral from the saliva.

Dentin caries may occasionally be arrested as a result of destruction of so much enamel, that a wide area of dentin become exposed, if this surface is then subject to attrition , plaque deposition may be prevented by use of fluoride and consumption of a less cariogenic diet may cause a surface lesion in enamel to heal entirely.

Immunological aspects of dental caries

Caries in man is associated with the development of serum and salivary antibodies against s. Mutans, but in all most all individuals this natural active immunity appears to have little effect as caries is virtually universal in western populations. This may be because s. Mutans in only weakly antigenic

However, artificial active immunity following experimental immunization of rats and monkeys with s. Mutans using both live and dead organisms as well as cell wall preparation has been shown to produce a significant reduction in caries. Immunization evokes a humoral response characterized by Igg, Igm, and Iga classes of antibodies and also induces cell-mediated immune responses, these effectors system can gain access to the oral cavity via the gingival crevice and saliva, but the immunological mechanisms involved in prevention of dental caries are unclear, however, in these experimental systems the reduction in the number of carious lesions appears to be associated with the reduction in the number of s. Mutans organisms in plaque.

The salivary immune mechanism would presumably act through secretary Iga and might prevent s. Mutans from adhering to the tooth surface crevicular immune mechanism could involve any or all of humeral and cellular components of systemic immunity.

Immunoglobulin complement: Neutrophil leukocytes sensitize lymphocytes and macrophages may pass through the base of sulcus and so reach tooth surface. Although the relative importance of salivary and crevicular mechanism has not been fully elucidated, the powerful immune component of the later suggest it is the more important.

Evidence suggest that protection is associated with Igg mediated reaction and that Iga, andIgm antibodies confer little or no protection and may interfere with the protective effect of Igg. The Igg antibody may act as opsonin facilitating phagocytosis and ultimately death of s.mutans by neutrophils and macrophages. The role of cell mediated immune response is uncertain but helper T cell function is important. Vaccines composed of whole cell of s.mutans may induce an antibodies that cross react with hard tissue, and so sub component of organsim, have been investigated which return the capacity against caries but which do not contain cross reacting heart antigens. These include proteins purified from the cell wall of s. Mutans that are

involved in the attachment of the organism to the tooth surface. Several over antigens preparation have been tested in animal studies including glucosyltransferase, these bacterial enzymes convert sucrose into glucans which are important for accumulation of s. Mutans on tooth surface. Although antibodies to glycosyltransferase can reduce the accumulation of the plaque and the incident of caries to rodents, they appear to have a little protective value in primates; this may be because such antibodies are probably most effective against the development of smooth surface rather than pits and fissure caries.