Tooth Decay:

Active process of tooth damage resulting from interactions between bacteria, teeth and food

Primary Factors for Caries to Occur:
>Host
>Time
>Substrate
>Micro-organisms

Host Factors:

-Tooth	-Age
-Fluoride	-Morph-ology
-Carbohydrate leve	-Nutrition

Position of tooth:
- Upper>Lower, due to salivary action
- Post>Anterior, due to pits and fissures

Morphology:
- Teeth with stagnation area (ie. Malposed teeth, Crowded teeth)

Smooth Surfaces:
- Interproximal area at contact
- Buccal at cervical third
- Lingual at cervical half

Host Factors (Part 2):

Structure of Tooth:
> Pits and fissures increase propensity for caries formation
> So does depth features

Fluoride:
>Acts by reducing enamel solubility

Host Factors (Part 2): (cont)

> Inhibits tooth demineralization
> Enhances remineralization

Genetics

Age:
- 60-90% of school going children
- 100% of adults (65-75years) have missing teeth
- As people age, caries rates and cavity severity increases

Carbohydrate (CHO) level:
-Type of CHO
-Amount of CHO
-Frequency of CHO
-Local effect of CHO
-Consistency and refinement of CHO

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Salivary Flow:

Salivary glycoproteins

Washing effect of saliva

Buffering effect

SIga

Antibacterial non-immunological enzymes

CHO accumulate in poorly rinsed area

Caries Factors

Protective Factors:	Pathological Factors:
-Saliva	-Bacterias
-Antimicr-obials	-Absence of saliva
-Fluoride	-Dietary habits
-Effective diet	

Bacterial Role in Caries

Germ free animals do not develop caries

Antibiotics fed to animals are effective in reducing incidence and severity of caries

Unerupted teeth do not develop caries

Oral bacteria can demineralise enamel and dentin in vitro and produce caries-like lesions

Bacteria can be isolated from carious lesions

Bacterial Role in Caries (cont)

Enamel invasion demonstrates microscopically

Characteristics of Non-Cario-genic Biofilm:

Lower levels of *S.mutans* and lactobacilli

Higher levels of *Actionmyces, S.sanguinis,Veillonella*

Lower concentrations of lactic acid and higher concentrations of acetic and propionic acids

Characteristics of Cariogenic Bacteria:

Rate of sucrose consumption is higher

Rate of lactic acid formation

Synthesis more intercellular glycogentype -type polysacch-arides

Intra- and extracellular -polys-accharides enhance lactic acid production and colonisation

Aetiology of Caries

Streptococcus mutans: primary agent

S.sobrinus

Secondary Invaders:
After initial weakening of enamel
>Lactobacilli
>*Actinomyces*

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Streptococcus mutans:

Faculative anaerobes

Gram positive

Does not colonize teeth uniformly

Weak ability to absorb to teeth

Low salivary concentrations available for attachment

Antagonises growth of *S.sanguinis*:
-Acid production
-bacteriocins

Secondary Invaders:

Divided into 2 main groups:

1. **Homofermentative:**	Fermentation of glucose produces lactic acid (eg. *L.casei, L.acidophilus*)
2. **Heterofermentative:**	Fermentation produces lactic acids plus acetate, ethanol, carbon dioxide (eg *L.fermentum*)

Rarely isolated in initial caries but predominate in deep cavities

Actinomyces:

Gram positive non-sporing bacillus

Microaerophilic

Normal microflora of the oral cavity

Acidogenic

Several species implicated but mostly: *A.naeslundi, A viscosus*

Insoluble Glucans

Gram positive Cocci	Gram negative Cocci**
S. mutans	*A.viscosus*
S.sanguinis	*A.naeslundi*

>Caries free adults:
-*Streptococcus sanguinis* predominates over *Strep.mutans*
>High Caries adults:
-*Strep.mutans* prevail over *Strep.sanguinis*

>*Strep.sanguinis* from caries free individuals showed higher H2O2 production than high caries adults

Role of Strep.mutans:

Several different species:	*S.mutans, S.sobrinus, S.

S.mutan serotypes c,e,f and *S.sobrinus* serotypes d and g are species most commonly found in humans.
>Serotype c most prevalant followed by d and e

Role of Strep.mutans in Caries:

Cariogenic and initiate caries on smooth tooth surfaces

Characteristics of *S.mutans*:
> Potent acidogenic
>Highly aciduric
>Synthesize extracellular polysaccharides: Glucan and Levan
> Synthesize and store intercellular glycogen-like polysaccharides known as amylopectins

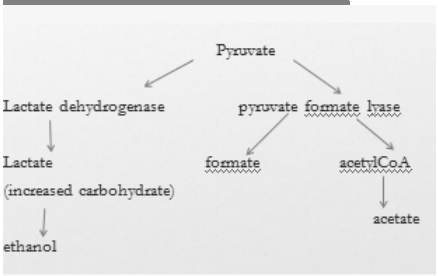
Glucan vs. Levan

Glucan:	**Levan:**
Extracellular polysaccharide	Extracellular polysaccharide
Glucose polymer	Fructose Polymer
Water insoluble	Water soluble
Adhesive	Less adhesive

Glucan Binding Protein:
>S.mutans secretes 3 distinct proteins w/glucan binding activity (GBP-A,GBP-B and GBP-C)
>Helps in binding of glucan to S.mutans

Dextranases:
>important constituent of dental plaque
>enzyme produced by strp.mutans
>destroy and thus bacteria can invade dextran-rich early plaque
>when used as an antigen,can prevent colonization of organism in early dental plaque

Glucose Degraded by Bacteria:



Embden-Myerhof Pathway

Molecular Pathogenesis: S.mutans

Mutans streptococci participate in the formation of biofilms on tooth surfaces. These biofilms are known as dental plaque(s). Sucrose is required for the accumulation of mutans streptococci. Also required for this accumulation are the enzymes glucosyltransferases (GTFs), which are constitutively synthesized by all mutans streptococci.

a | Initial attachment of mutans streptococci to tooth surfaces. This attachment is thought to be the first event in the formation of dental plaque. The mutans streptococcal adhesin (known as antigen I/II) interacts with α-galactosides in the saliva-derived glycoprotein constituents of the tooth pellicle. Other moieties at the surface of mutans streptococci include glucan-binding protein (GBP), serotype carbohydrate and GTFs.

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Molecular Pathogensis: S.mutans

b | Accumulation of mutans streptococci on tooth surfaces in the presence of sucrose. In the presence of sucrose, GTFs synthesize extracellular glucans from glucose (after the breakdown of sucrose into glucose and fructose), and this is thought to be the second event in the formation of dental plaque. The mutans streptococcal protein GBP is a receptor-like protein that is distinct from GTFs, and it specifically binds glucans. GTFs themselves also have a glucan-binding domain and can therefore also function as receptors for glucans. So, mutans streptococci bind preformed glucans through GBP and GTFs, and this gives rise to aggregates of mutans streptococci..

Stages of Caries Development:

1. Enamel becomes decalcified
2. Small white spot appear
3. Discolouration becomes pronounced
4. Tooth surface softens and decay penetrates through enamel into dentine
5. Caries spreads laterally and in depth
6. Cavitation occurs

Smooth Surface Caries:

Rarely on buccal and lingual surfaces

Mostly on approximal tooth surfaces just below contact points

S.mutans found mostly on white spots

S.sobrinus found on caries active sites

Pit or Fissure Caries

Most caries prone sites: molars, premolars and lingual surface of maxillary incisors

S.mutans: -strongest association

S.sobrinus - more frequently on molars than anterior teeth

Pit or Fissure Caries (cont)

S.salivarius, S.sanguinis, L.acidophilus, L.casei, Actiomyces also found

Recurrent Caries:
> Associated with existing restoration
>S. mutans and lactobacilli

Root Surface Caries:

Seen on cementum and/or dentine when the root is exposed to oral environment

Mostly middle-aged and older adults affected

Prevalent in primitive communities

Associated bacteria: Actinomyces, Rothia dentocariosa, S. mutans, lactobacilli

Deeper caries: Propionibacterium, Bifidobacteria, Eubacteria

Rampant Caries:
Risk Groups:
>Xerostomic patients (S. mutans, lactobacilli)
>“nursing bottle” (S. mutans, L. fermentum, L. plantarum)

Caries prevention:

1. Healthy Diet
2. Plaque control
3. Teeth brushing
4. Application of fluoride on tooth surfaces

Caries prevention: (cont)

5.Applications of sealants on tooth surfaces

Fluoride in Saliva:
Speeds up crystal precipitation, forming a fluorapatite-like coating more resistant to caries than original tooth structure

Food W/ Anticariogenic Effect:

Milk	Contains lactose
Cheese	Casien Phosphatase
Fibrous Foods	Raw veg and grains
Sugar substitutes	xylitol, mannitol, sorbitol
Tea	green and black tea

Vitamin D

Reduces risk of cavities by producing **cathelicidin** and **denfensisn**.
> These proteins have antibacterial effects to fight bacteria that causes caries

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