Robert Koch's Germ Theory:

All subjects must present with the same symptoms

Causative organism must be isolated in **pure culture**

When inoculated into a 2nd host, it must produce the same symptoms

Identical pure culture must be obtained from second host

Modification of Koch's postulates for oral opportunistic infections:

> Should be present in sufficient numbers to cause disease

> Should have access to the affected tissues

> Should be in an environment that permits its survival and multiplication

> Inhibitory organisms should be absent or not affect it

> The host must be susceptible

Difficulties:Koch's Postulate in Oral Infections:

> No overt pathogen - mostly mixed infections

> More than 700 identified oral species

> Not all members of biofilm community are cultivable

> Presence may be as a result rather than cause of disease

> Sites don't appear to be actively progressing at all times

> Different sites in mouth may break down as a result of different species

> Strains of putative pathogens may vary in virulence

Difficulties:Koch's Postulate in Oral Infections: (cont)

> Some strains may harbour bateriophages or plasmids that confer virulence properties

Non-specific Plaque Hypothesis

Overgrowth of indigenous microbiota

Same organisms observed in health and disease

Shifts in microbial proportions rather than specific pathogens

Any plaque biofilm can cause disease

Problems with NSPH:

- Focus is on quantitative changes only
- Disease in animals not the same as in humans
- Impractical to compare virulence in different host species
- Doesn't explain why individuals with: 1) longstanding plaque don't develop disease and 2) minimal plaque have lower resistance to disease

Specific Plaque Hypothesis

More sophisticated studies demonstrated:

> Improved cultural and sampling methods

> Composition of plaque biofilm differs both inter-orally and intra-orally

> Increase at a sight of infection

> Decrease in health or following treatment

> Qualitative changes in plaque biofilm

Exogenous Theory:

Exogenous pathogens and **not endogenous microbiota** caused disease

Fails to explain:

> Mode of transmission

> Acquisition

> Means of colonization

> Effect of treatment on indigenous species

Contraindications:

- Over simplification
- Overlaps often occurred (Negated SPH and NSPH)
- Eradication of exogenous pathogens (Incorporated both SPH and NSPH)

Ecological Plaque Hypothesis:

Opportunistic endogenous infection

Ecological shift from predominatly G+ cocci to G- rods/cocci-bacilli

Any bacterial species may be pathogenic

Ecological changes in environment dictate virulence mechanisms

Disease prevented by elimination or interruption of ecological succession

Role of Biofilms in Infection:

Most common:

1. Dental caries (supragingival plaque)
2. Periodontal disease (subgingival plaque)

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Dental Caries Theories

1) Tooth worm:

5000BC, 1803 - Diagrams and 1825- Case histories

2) Humoral Theory:

Blood, phlegm, black bile, yellow bile - *Imbalance= disease*

3) Chemical Theory:

Fermentation of food remains

4) Parasitic Theory:

Decomposition resulting from action of organisms in the mouth

Millers (1882) Chemo-parasitic Theory:

2 stage process:

i) decalcification of enamel resulting in destruction of dentin

ii) dissolution of softened residue of enamel and dentin

Dietary carbohydrates -> convert into acid -> calcium and phosphate diffuse out of enamel -> a caries lesion

Proteolytic Theory (Gotliebb 1946)

Invasion of enamel by m/o's -> proteolytic activity -> alteration of pH -> resulting in liquidification of organic matrix of enamel -> inorganic salts dissolved by acidogenic bacteria

The proteolytic-chelation Theory

Schutz and Martin (1955)

Simultaneous attack on organic and inorganic compounds of tooth

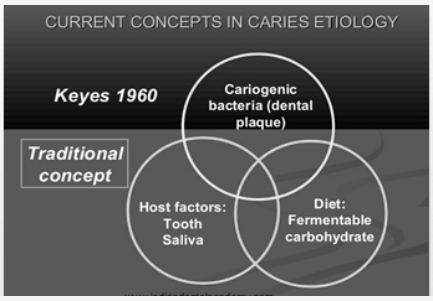
Kaeratinolytic bacteria attack enamel ->

Breakdown of protein and other organic components of enamel (keratin) ->

Formation of complexes with calcium from plaque which chelates with mineral component of the tooth ->

Increased solubility, decalcification of enamel ay neutral or alkaline pH

Current Concepts in Caries Etiology



Keyes Triad & Newbeun's Tetrad