Periodontology- fourth stage

First semester-Microbiology of dental plaque
Lec-6-Part-2

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Content

- Microbiologic Specificity of Periodontal Diseases
- Criteria for the Identification of Periodontopathogens
- Experimental gingivitis.

Microbiologic Specificity of Periodontal Diseases

1. Nonspecific Plaque Hypothesis

During the mid-1900s, periodontal diseases were thought to result from an accumulation of plaque over time, eventually in conjunction with a diminished host response and increased host susceptibility with age. This theory, which is called the nonspecific plaque hypothesis

- According to the nonspecific plaque hypothesis, periodontal disease results from the "**elaboration of noxious products by the entire plaque flora.**" When only small amounts of plaque are present, the noxious products are neutralized by the host. Similarly, large amounts of plaque would cause a higher production of noxious products, which would essentially overwhelm the host's defenses.

➢ Several observations contradicted these conclusions:.

- First, some individuals with considerable amounts of plaque and calculus, as well as gingivitis, never developed destructive periodontitis.
- Furthermore, individuals who did present with periodontitis demonstrated considerable site specificity with regard to the pattern of disease.

Inherent in the nonspecific plaque hypothesis is the concept that the control of periodontal disease depends on the reduction of the total amount of plaque. The current standard treatment of periodontitis by debridement (nonsurgical or surgical) and oral hygiene measures still focuses on the removal of plaque and its products.

2. Specific Plaque Hypothesis

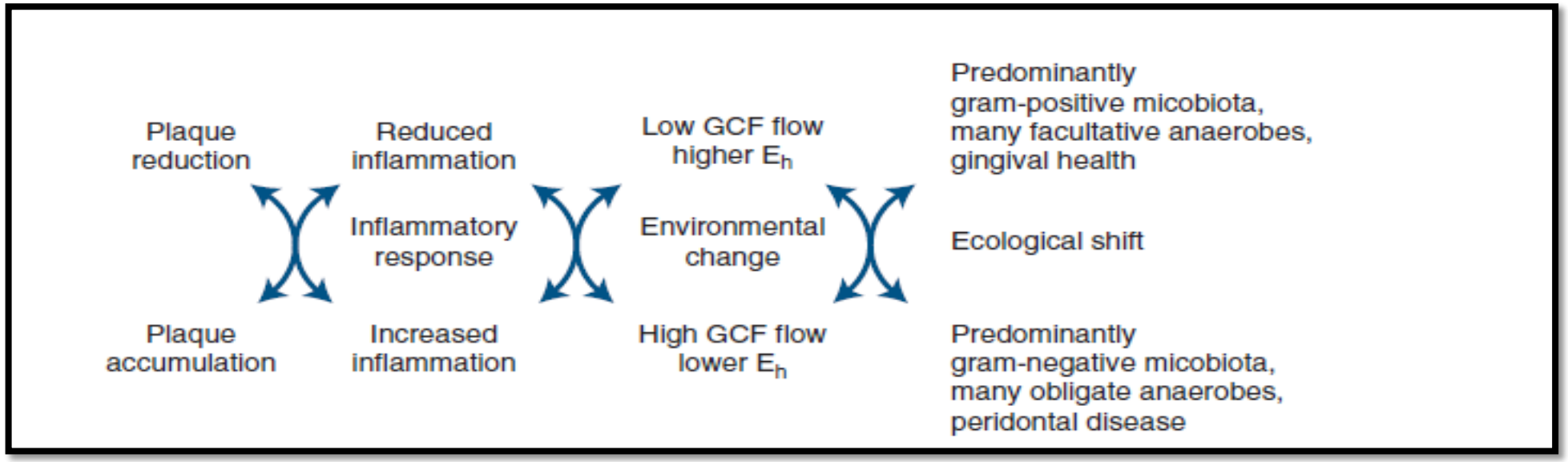
- The specific plaque hypothesis underlines the importance of the qualitative composition of the resident microbiota. The pathogenicity of dental plaque depends on the presence of or an increase in specific microorganisms.

- Acceptance of the specific plaque hypothesis was spurred by the recognition of A. actinomycetemcomitans as a pathogen in localized aggressive periodontitis

3. Ecologic Plaque Hypothesis

- During the 1990s, Marsh and coworkers developed the “ecologic plaque hypothesis” as an attempt to unify the existing theories regarding the role of dental plaque in oral disease

- According to the ecologic plaque hypothesis, both the **total amount of dental plaque** and the **specific microbial composition of plaque** may contribute to the transition from health to disease.



Ecological plaque hypothesis in relation to periodontal disease

4. Keystone Pathogen Hypothesis and Polymicrobial Synergy and Dysbiosis Model

- The keystone pathogen hypothesis indicates that certain low-abundance microbial pathogens can orchestrate inflammatory disease by remodeling a normally benign microbiota into a dysbiotic one.

- The data indicated that ***P. gingivalis*** subverts the host immune system and changes the microbial composition of dental plaque, ultimately leading to periodontal bone loss. On this basis, *P. gingivalis* was labeled a **"keystone"** pathogen; this means that it is an organism that is central to the disease process, even when it is at a relatively low abundance.

Criteria for the Identification of Periodontopathogens

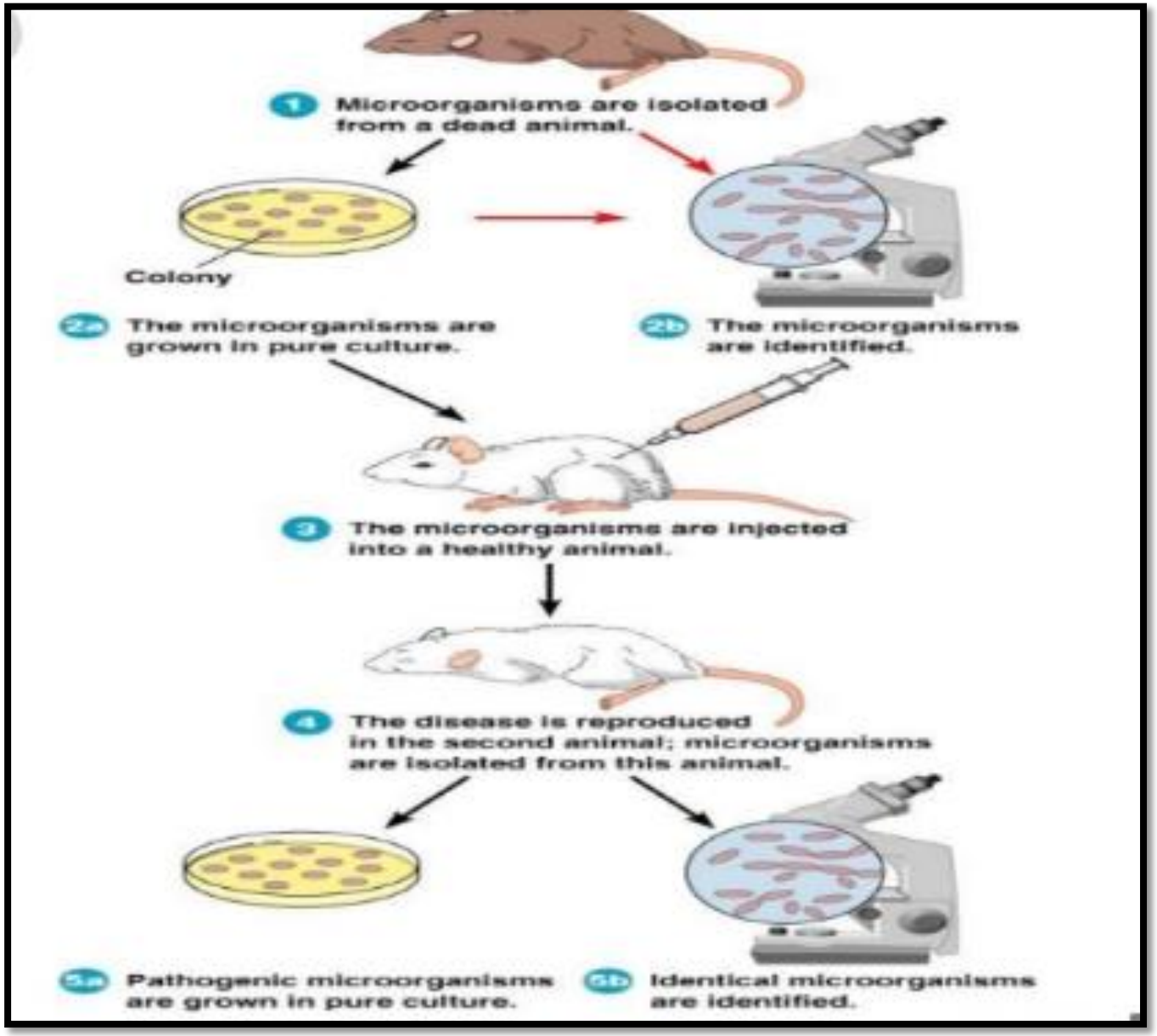
- During the 1870s, Robert Koch developed the classic criteria by which a microorganism can be judged to be a causative agent in human infections. These criteria, known as **Koch's postulates**.

Be routinely isolated from diseased individuals.

Be grown in pure culture in the laboratory.

Produce a similar disease when inoculated into susceptible laboratory animals.

Be recovered from lesions in a diseased laboratory animal.



Koch's postulates

Sigmund Socransky, proposed criteria by which periodontal microorganisms may be judged to be potential pathogens.

- Must be **associated with disease**, as evidenced by increases in the number of organisms at diseased sites
- Must be **eliminated or decreased** in sites that demonstrate the clinical resolution of disease with treatment.
- Must **Induce a host response** in the form of an alteration in the host cellular or humoral immune response.
- Must be capable of **causing disease** in experimental animal models.
- Must **demonstrable virulence factors** that are responsible for enabling the microorganism to cause the destruction of the periodontal tissues.

Microbial flora are associated with:

1. Clinically healthy gingiva

- If the teeth are kept clean with proper oral hygiene measures, the gingiva remains healthy and few bacteria are found along the gingival margin.

- If the person with such gingiva stops cleaning his teeth, bacteria will be accumulated on his teeth within few hours, the most predominant bacteria are streptococcus (G+ve cocci) and actinomyces (G+ve rods),also G-ve rods and facultative anaerobic rods are found in small proportions.

2. Gingivitis:

a-in mild to moderate gingivitis:

- 25% of microbial flora of subgingival plaque are *streptococci*, another 25% composed of *Actinomyces* species, another 25%G-ve anaerobic rods, other 25%are miscellaneous bacteria.

b-acute necrotizing ulcerative gingivitis:

- The microflora is composed of fusiform bacteria and spirochetes to form fusospirochetal complex.

3. Chronic periodontitis:

- The microflora is dominated by anaerobic microorganisms, G-ve rods form about 75%, spirochetes form about 50% of flora.

4. Aggressive periodontitis:

- Anaerobic G-ve rods form about 60% and 7%spirochetes.

❖ When comparing the microbiota among conditions of health, gingivitis, and periodontitis, the following microbial shifts can be identified as health progresses to periodontitis:

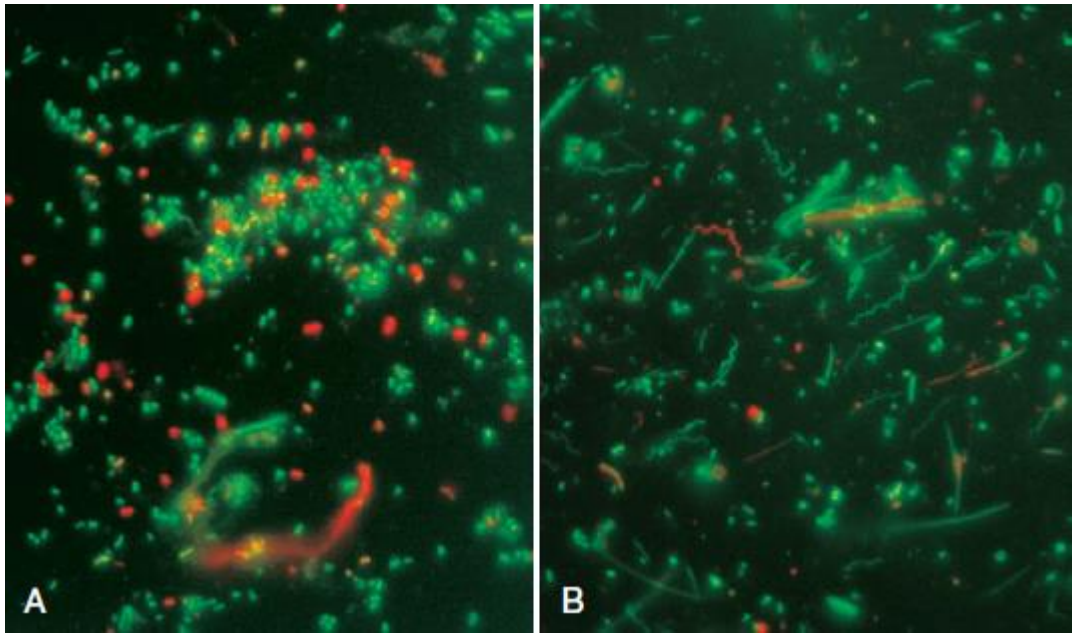
• From gram-positive to gram-negative.

• From cocci to rods (and, at a later stage, to spirochetes).

• From non motile to motile organisms.

•From facultative anaerobes to obligate anaerobes.

• From fermenting to proteolytic species.



Vitality stain of subgingival plaque. (A) Plaque derived from a healthy patient that primarily consists of cocci. (B) Plaque derived from a patient with periodontitis.

Experimental gingivitis

- The cause and effect relationship between dental plaque and gingival inflammation was demonstrated, its called experimental gingivitis in man(Loe et al 1965).

- The oral hygiene of a group of healthy individuals was improved during several weeks of intensive instruction in use of tooth brush and tooth picks this resulted in an excellent gingival condition.

- Then all oral hygiene measures were withdrawn allowing plaque to reaccumulate along the gingival margin.

- all subjects developed gingivitis within 10-21 days the mean gingival index score increased from 0.27 at base line to 1.05 at the end of no brushing gingival period.

- Gingival inflammation resolved in all subjects within 1 week of resuming hygiene measures.

- During the experimental period plaque samples were obtained at regular intervals and subjects to bacteriological examination of gram stained smear the bacteria present in the sample were classified according to their gram reaction and morphology.

- With healthy gingiva, very few bacteria were present on the cervical surfaces of teeth,90%G+ve cocci, the remainder 10% was G-ve bacteria.

✓ When all oral hygiene measures stopped the following phases of plaque development occurred:

❖ **First phase:** Initial 2 days of the experiment not only all types of bacteria increase but their proportional distribution change as well G+ve cocci and rods forming a greater proportion of flora

❖ **Second phase**: Days 3 and 4 are characterized by proliferation of fusobacteria and filamentous bacteria

- **Third phase**: Days 5-9 are characterized by the appearance of spirilla and spirochetes.

- The experiment only gives information about G+ve and G-ve bacteria but not the type of species of bacteria. this experiment proved clinically that dental plaque is the main etiological factor in development of periodontal disease.


Thank you
for Listening!