Pathogenesis of periodontal disease

Current hypotheses on the role of plaque bacteria in the etiology of dental diseases

- **Non-specific plaque hypothesis**
- **Specific plaque hypothesis**
- **Ecological plaque hypothesis**
- **Keystone Pathogen Hypothesis**

The nonspecific plaque hypothesis:

- periodontal disease results from the “elaboration of toxic products by the entire plaque flora.” When only small amounts of plaque are present, the toxic products are neutralized by the host. Similarly, large amounts of plaque would cause a higher production of toxic products, which would essentially overwhelm the host's defenses

Specific plaque hypothesis

It is 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. This concept encapsulates that plaque that harbors specific bacterial pathogens may provoke periodontal disease because key organisms produce substances that mediate the destruction of host tissues

Ecological plaque hypothesis

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.

Keystone Pathogen Hypothesis

It indicates that certain low microbial pathogens can orchestrate inflammatory disease by remodeling a normally benign microbiota into a dysbiotic one

Periodontal disease has sometimes been referred to as a "mixed bacterial infection" to denote that multiple microbial species contribute to the development of disease

For a periodontal pathogen to cause disease, it is essential that the pathogen be able to

1. **colonize the subgingival area**
2. **produce factors that either directly damage the host tissue or lead to the host tissue damaging itself**

To colonize subgingival sites, a species must be able to

1. attach to one or more of the available surfaces,
2. multiply
3. compete successfully against other species,

Adhesion

- To establish in a periodontal site, a species must be able to attach to one or more surfaces including the tooth, the sulcular or pocket epithelium or other bacterial species attached to these surfaces.
- fimbriae and cell associated proteins

Coaggregation

While many species attach directly to host surfaces, other species attach to bacteria attached to such surfaces

Multiplication

- The gingival crevice and/or periodontal pocket might be considered a lush area for microbial growth, but is in fact a rather stringent environment for a bacterial species to live. The mean temperature of the area averages about 35°C and ranges from 30°—38°C. The pH is rather restricted (pH 7. 0—8.5).

Host defence mechanisms

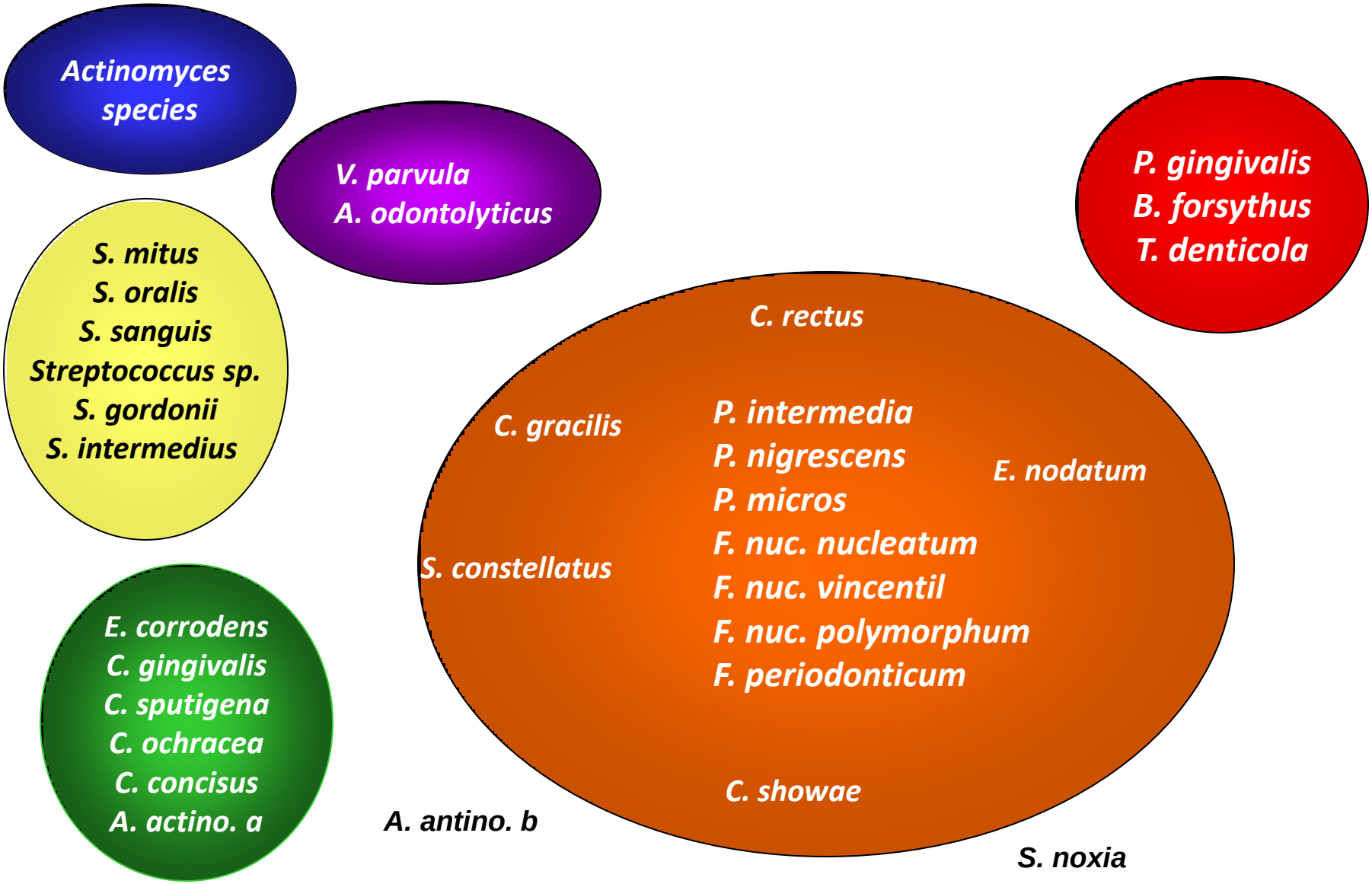
- The intact barrier provided by the junctional epithelium
- The regular shedding of epithelial cells into the oral cavity
- The positive flow of fluid to the gingival crevice which may wash away unattached microorganisms and noxious products
- The presence in GCF of antibodies to microbial products
- The phagocytic function of neutrophils and macrophages
- The detrimental effect of complement on the microbiota.

Virulence factors

can be divided into three categories:

- **substances that damage tissue cells (e.g. H2S)**
- **substances that cause cells to release biologically active substances (e.g. lipopolysaccharide)**
- **substances that affect the intercellular matrix (e.g. collagenase)**

Subgingival Microbial Complex



According to Socransky & Haffajee (1998)- there are 6 major complexes in subgingival plaque, in this paper the red & orange complexes are related strongly to inflammation and periodontal destruction.

The organisms in the "red complex" increase in numbers and prevalence with increasing pocket depth and were shown to be associated with increased BOP, thought to be major etiologic agents.

Members of the "red complex" are rarely found in the absence of members of the "orange complex" and colonization by species of the "orange complex" usually precedes colonization by the "red complex".

Suggest coaggregation activity, may have specific partners in biofilm formation.