Periodontal diseases

Disorders of supporting structures of the teeth: gingivae, periodontal ligaments, supporting alveolar bone

Microbial involvement:

Shift in the balance of the **endogenous** microflora – usual situation *(similar situation to dental caries)*

Much less ferquently **exogenous** pathogens: *S. pyogenes*, *N. gonorrhoeae*, HIV, herpetic gingivitis

Classification of periodontal diseases

Multifactorial etiology

regression if etiologic factors are reduced

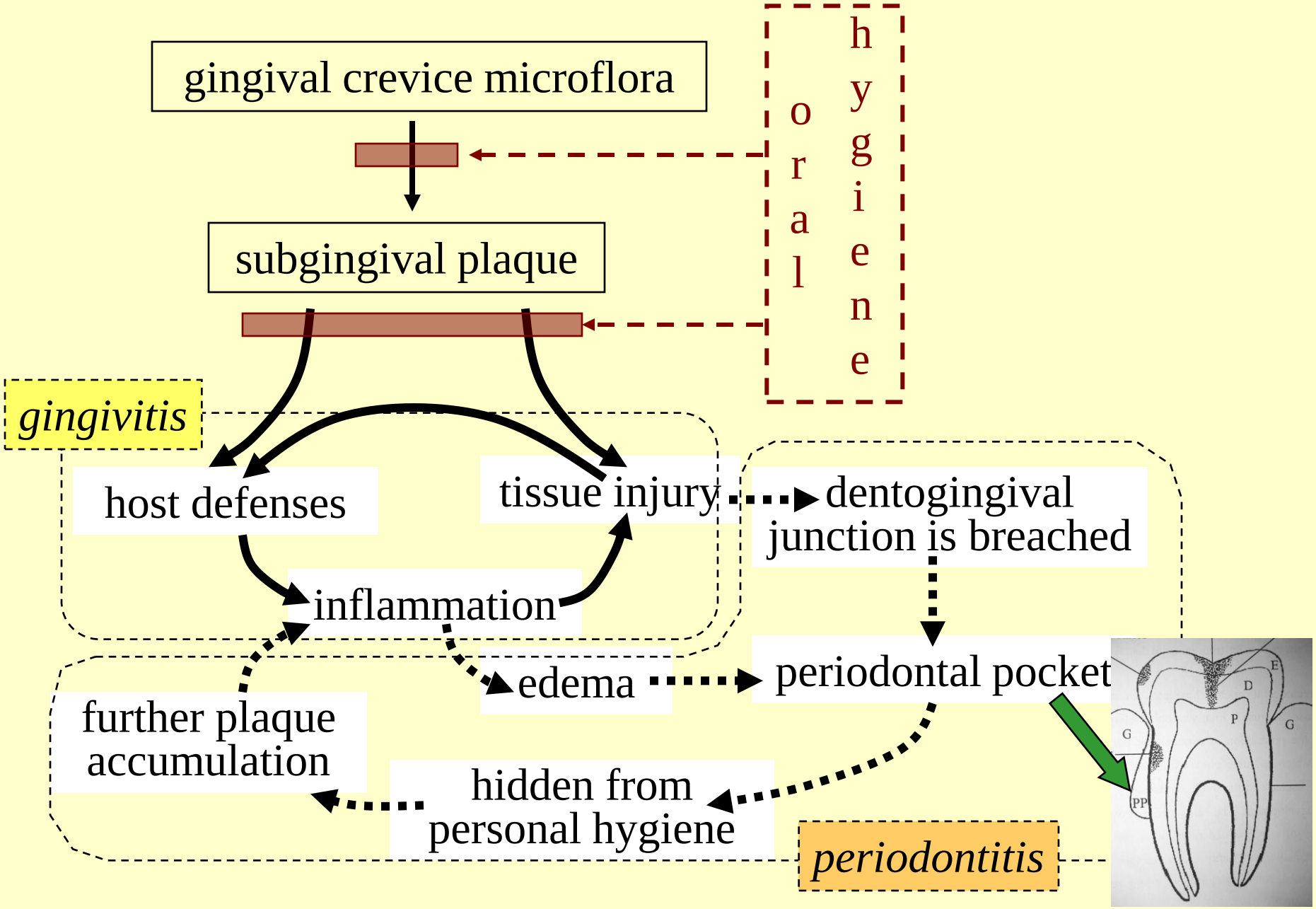
gingivitis – reversible in natural ways, tooth attachment to the supporting structures is not altered

persisting disease

progression

periodontitis *is preceded by gingivitis* – infection spreads to the supporting structures of the teeth – *periodontal pocket, alveolar bone resorption, eventual tooth mobility*

Microbial pathogenesis in periodontal diseases

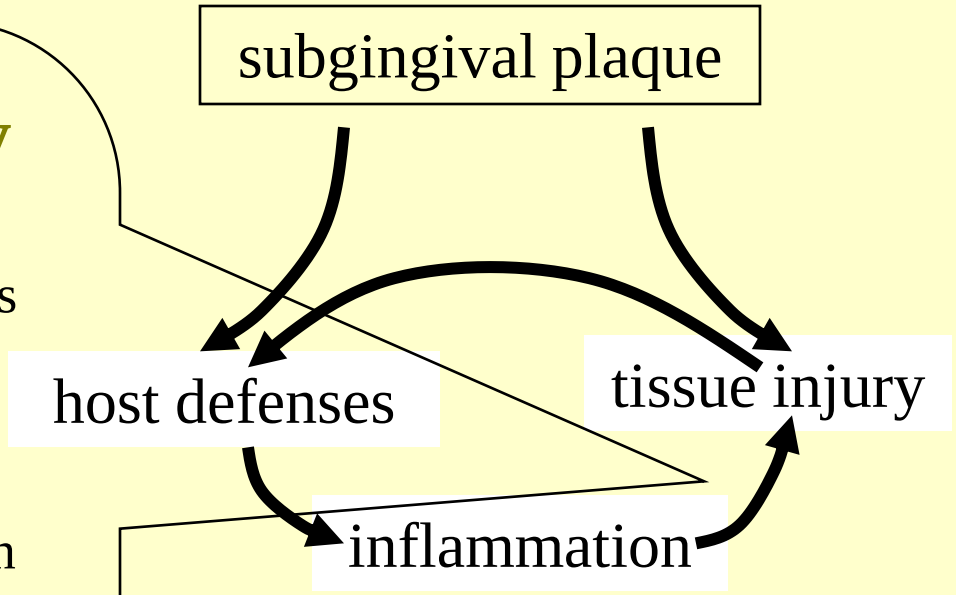


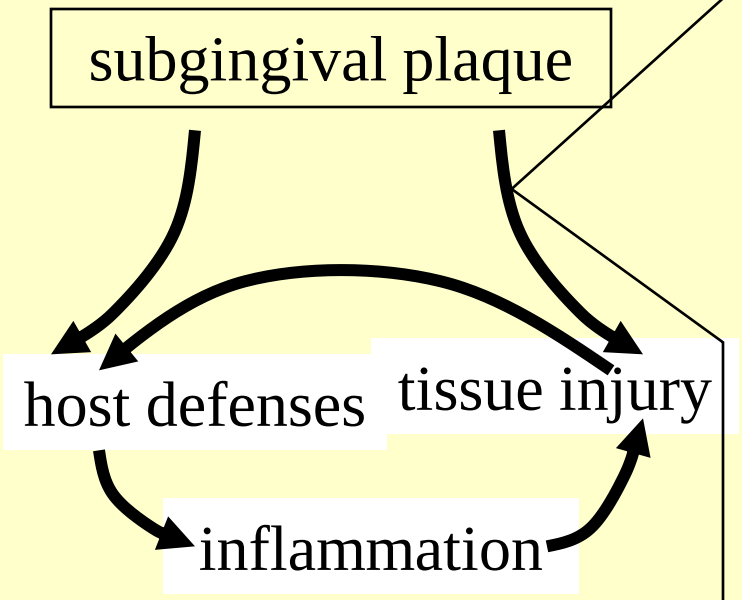
Indirect pathogenicity

•Interaction of PML and bacteria results also in release of lysosomal enzymes

•IgG, IgM, IgA specific to plaque bacteria also contribute to inflammation via: complement and PML activation and prostaglandin release resulting in bone resorption

•T-lymphoyte activated macrophages release also inflammatory cytokines (IL-1, TNF-alpha) inducing tissue collagenase activity and bone resorption





Direct pathogenicity of plaque bacteria

•Colonization: periodontal pathogens coaggregate with Streptococci and Actinomyces and produce bacteriocins to suppress the others

•Evasion of host defenses:
-leukotoxin by *C. rectus*, *A actinomycetemcom.*
-Inhibit PML chemotaxis, intracellular killing
-proteases degrade Igs, complement, cytokines

•Tissue damage:
-enzymes: protease, collagenase, hyaluronidase, chondroitic sulphatase
-bone resorption by LPS, lipoteichoic acids
-cytotoxic bacterial metabolites: butyric and propionic acids, amines, indole, ammonia,

Chronic marginal gingivitis

•Most frequently diagnosed form of gingivitis

•Inflamed marginal and papillary gingiva

•**Initial lesion** – acute inflammatory reaction with a predominant Gram-positive microflora

•**Early lesion** – clinically recognizable, local collagen destruction, Actinomyces and Capnocytophaga tends to overgrow

•**Established lesion** – infiltration by B- and plasma cells, predominance of black pigmented obligate anaerobes

Chronic periodontitis

•Most frequently diagnosed form of periodontitis developing from progressing chronic gingivitis

•Dentogingival junction is drifting in apical direction, periodontal pocket develops with highly anaerobic habitat:

•Predominant bacteria: *black pigmented anaerobes*, *Fusobacterium*, *oral treponemes*, *A. actinomycetemcomitans*,

•Additional bacteria: *Capnocytophaga*, *Wolinella*, *Eikenella*

Aggressive periodontitis

•Rather related to impaired lymphocyte function than subginigval plaques

•Predominant bacterium: *A. actinomycetemcomitans* capable of local immunosuppression and aggressive tissue destruction

•Additional bacteria: *Capnocytophaga*, *Porphyromonas gingivalis*

Acute necrotizing ulcerative gingivitis

•Acutely inflamed, very painful gingiva bleeding upon pressure

•Ulcerating lesions are covered by pseudomembrane (exsudate)

•Predominant bacteria: *Fusospirochetal complex*

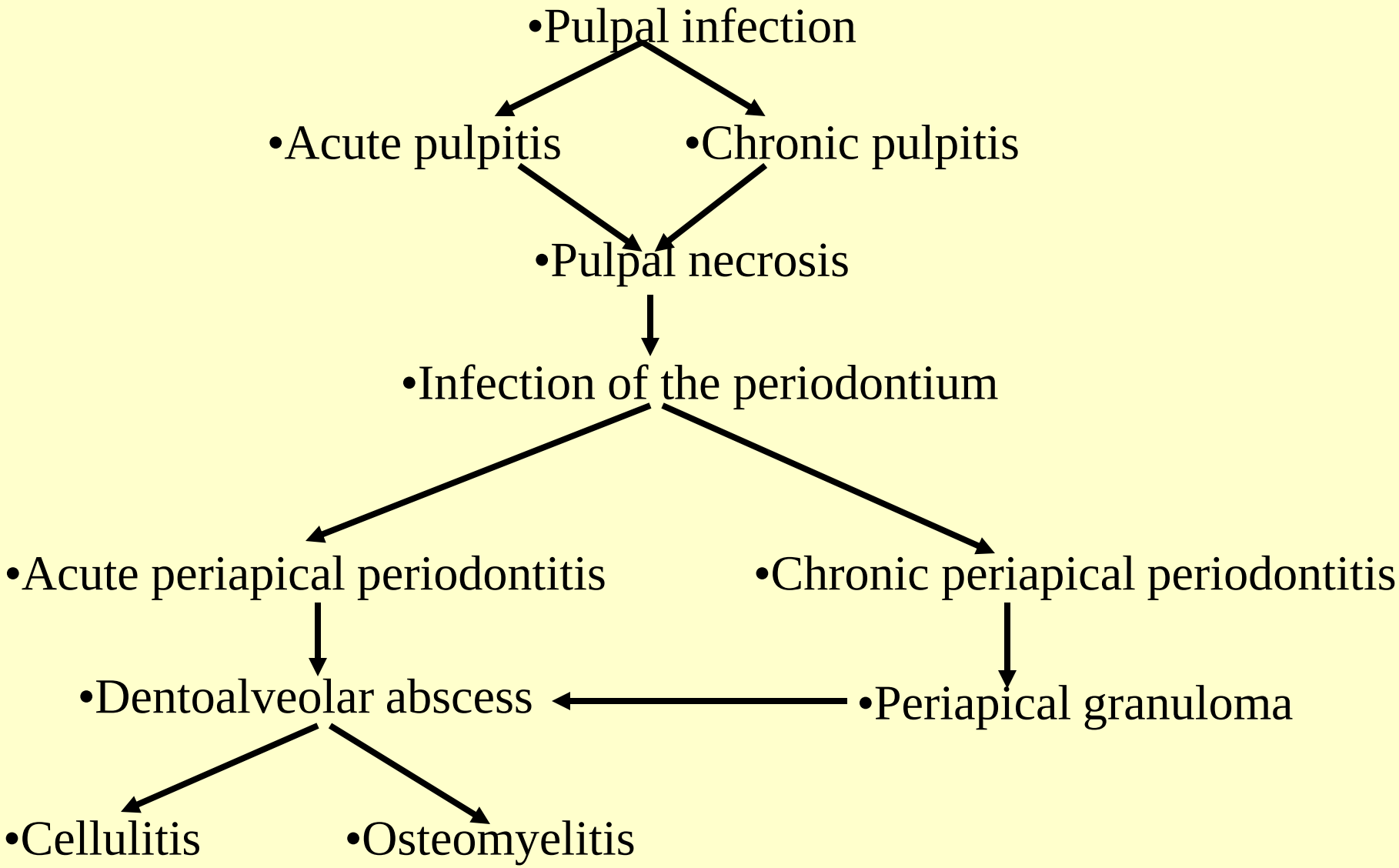
•If untreated,

- it may enter a necrotizing ulcerative periodontitis

- and it may lead to pronounced loss of mouth tissues

(see *cancrum oris*)

Dentoalveolar infections



Dentoalveolar infections

•Pulpitis can develop through dental caries, pulpal exposure to mouth and through the apical foramen

•It may lead to pulpal necrosis

•<u>Microbial involvement:</u>

Streptococcus, *Lactobacillus*, *Eikenella*, *Capnocytophaga* *Peptostreptococcus*, *Porphyromonas*, *Prevotella*, *Fusobacterium*, *Eubacterium*, *Propionibacterium*

Dentoalveolar infections

•Dentoalveolar abscess – predominance of strict anaerobes

•Ludwig's angina – typical soft tissue cellulitis (pyogenic infection) – *any resident or transient bacterium with invasive ability can be involved form the oral flora*

•Osteomyelitis - same mechanisms as in other body sites – bacteria reach medullary cavity – acute purulent inflammation – *any resident or transient bacterium with invasive ability can be involved form the oral flora*