Microbial Ecology in the Periodontal Pocket
- Impact on Treatment in Juveniles and Adults

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Periodontitis is a microbiologically induced inflammatory disease that affects the tooth supporting tissues, bone and connective tissues. The commensal oral microbiota colonizes the surfaces of the mucosa and teeth including the gingival crevice and forming a subgingival microbiota (biofilm), which induces the inflammatory reaction by release of bacterial products e.g. metabolites, enzymes and toxins. The tissues are in a constant fluctuation between destructive phases and healing and tissue repair (granulation tissue formation). Loss of periodontal ligament and bone resorption are irreversible processes and leads to a net loss of attachment, epithelial down growth and pocket formation. The role of bacteria in the disease process apart from inducing the inflammation is less clear. The magnitude of the inflammatory response is probably dictated by genetic risk factors resulting in individuals that are more susceptible for progression and others. Environmental factors (e.g. smoking) or acquired risk factors (e.g. systemic risk factors such as diabetes) may further modulate the severity of that response. There is also reason to believe that certain bacteria (periodontopathogens) associated with periodontal progression may play an active role in the modulation of the inflammatory response.

From a microbiological point of view periodontitis could be divided into two main categories a) the polymicrobial anaerobic type and b) the Aggregatibacter actinomycetemcomitans associated type.

The absolute majority of adult periodontitis belongs to the polymicrobial anaerobic type associated with a microbiota, characterized by Gram-negative anaerobic bacteria of which many are motile, in a complex ecologically driven microbial community of the deep periodontal pocket. The aggressiveness of the disease is related to an increased metabolic activity, multiplication and invasiveness of the bacteria into the gingival tissues. A burst of activity caused by a collaboration between several bacteria – a team work – which is a characteristic for most anaerobic infections. Some bacteria e.g. Porphyromonas gingivalis, may have a specific role here and has recently been suggested as a candidate for the “key-stone hypothesis”. Antibiotics are rarely indicated since it is difficult to identify ongoing progression, the clinical response is marginal and the effect will only be temporary. Mechanical infection control with or without surgery is the main therapeutic option. Pocket elimination may be necessary for long-term control of the periodontal microbiota.

A. actinomycetemcomitans fulfills the criteria for the specific plaque hypothesis. It is mainly associated with periodontitis in juveniles (primary as well as permanent dentition), which often is rapidly progressing and clinically diagnosed as aggressive periodontitis. For certain highly toxic clones of A. actinomycetemcomitans (e.g. JP2) it is possible to predict a future periodontal breakdown indicating a specific role in this type of periodontitis. Although mechanical infection control is mandatory, adjunct antibiotic treatment may be necessary to reduce the bacterial activity and production of metabolic products and toxins and create a balanced condition between the bacteria and the host. The lesions seem to burn out after some years and only maintenance therapy may be necessary.