Greenstein G, Lamster I. Bacterial Transmission in periodontal diseases: a critical review. J Periodontol 1997; 68: 421-31.

Purpose: To review recent data (case reports) which have added insight into possible routes of transmission for periodontal pathogens.

Materials and Methods: Review Article

Findings: Dental instruments have the potential to transmit microbes from one site to another within the oral cavity. There is also evidence for and against the transmission of bacteria between spouses. Colonization with putative pathogens did not necessarily lead to infection and subsequent tissue destruction. The data support the contention that spouses have an increased risk of being colonized by pathogens if a mate is infected, but the incidence of confirmed transmission using molecular genetic techniques is usually low. In regards to transmission of bacteria from parent to child, the data strongly indicates that if a child harbors a pathogen, then at least one of the parents will usually exhibit the same genotype of bacteria. However, the incidence of bacterial transfer to children varies. As far as cross-infection among children, this is difficult to verify between siblings because parents usually possess the suspected pathogen. It has been shown that animals can also be vectors of bacterial transfer as well as saliva. There are several factors that limiting the transmission of pathogens. The new pathogen must find conditions that it can tolerate and it cannot stimulate a bacterial or a host response injurious to its survival. Ultimately, the response to a particular organism in a specific ecological niche will determine if a bacterium can colonize and if disease will occur.

Conclusions: Specific subgingival bacterial agents are the primary risk factor for developing disease. However, the mere presence of pathogens does not mean that an individual with develop periodontitis. To initiate or cause disease progression at a specific site, at a given time, the host must be susceptible, there must be a sufficient number of virulent pathogens, the local environment must be conducive to expression of pathogen virulence factors, and co-existing bacterial species unable to inhibit pathogen activity.

Kubar A, Saygun I, Ozdemir A. Real-time polymerase chain reaction quantification of human cytomegalovirus and Epstein - Barr virus in periodontal pockets and the adjacent gingiva of periodontitis lesions. J Perio Res 2005; 40(2): 97-104.

Purpose: To determine levels of human cytomegalovirus (HCMV) and Epstein - Barr virus (EBV) DNA in the periodontal pocket and in the adjacent gingiva of periodontitis lesions using a real-time polymerase chain reaction (PCR) assay.

Materials and Methods: Study subjects included three women and six men (ages 21–34 years) with aggressive periodontitis, and four women and seven men (ages 36–56 years) with chronic periodontitis. All patients were systemically healthy and had not received periodontal treatment or antibiotics for at least 6 months prior to the study. Clinical periodontal evaluation included the plaque index, the gingival index, probing pocket depth, and probing attachment loss. At 2 weeks prior to periodontal surgery, supragingival plaque and calculus were removed by ultrasonic scaling. A sterile periodontal curette was gently inserted to the bottom of the test periodontal pocket, and subgingival material was removed by a single stroke. A 5'-nuclease real-time PCR assay was used to identify and quantify genomic copies of periodontal HCMV and EBV.

Findings: HCMV DNA was detected in 78% of subgingival and 33% of gingival tissue samples from aggressive periodontitis lesions, but only in 46% of subgingival and 9% of gingival tissue samples from chronic periodontitis lesions. EBV DNA was identified in 89% of subgingival and 78% of gingival tissue samples from aggressive periodontitis lesions, but only in 46% of both subgingival and gingival tissue samples from chronic periodontitis lesions. HCMV and EBV counts in periodontal pockets and in gingival tissue were positively correlated with level of gingival inflammation and periodontitis disease severity, as assessed by pocket depth and probing attachment loss. Positive correlations were found between herpesviruses and several clinical variables of aggressive periodontitis, whereas little relationship existed between the study viruses and clinical features of chronic periodontitis.

Conclusion: The high HCMV and EBV loads in progressive periodontitis lesions support the notion that the two herpesviruses participate in the initiation or progression of the disease. Active herpesvirus infection may serve as an important new marker to indicate progressive periodontitis and, in combination with other disease variables, form the basis for improved assessment of future disease risk.

Ling LJ, Chuan-Chen H, Wu CY, et al. Association between human herpesviruses and severity of periodontitis. J Periodontol 2004: 75(11): 1479-85.

Purpose: To evaluate the relationship between herpesvirus including herpes simplex virus (HSV), human cytomegalovirus (HCMV) and epstein–barr virus type I (EBV-I) and the severity of the periodontal diseases.

Materials and Methods: The study consisted of 20 subjects (10male&10 female) who were examined for their periodontal status in terms of gingival inflammation, bleeding on probing, probing depth, and clinical attachment loss. The six ramfjord teeth(3,9,12,19,25 and 28) were selected to evaluate the periodontal status. The presence of HCMV, HSV or EBV-I were tested in the Subgingival plaque samples using polymerase chain reaction technique.

Findings The presence of HCMV, HSV or EBV-I showed no age predilection and was equally distributed between women and men. A total of 120 sites were examined showed the prevalence of HCMV (51.7%) was higher than HSV(30.8%) and EBV(4.2%). The presence of HSV or HCMV were significantly higher in the subgroups that had lower plaque index. The presence of HSV was found to be higher in the subgroup that had higher gingival index, BOP, deeper Probing depth and increased clinical attachment loss. EBV-I was found to be prevalent in subgroup that had higher probing depth. Coinfection of HSV and HCMV was significantly associated with the sites that had higher gingival index or BOP. Coinfection of any 2 herpesviruses was also associated with higher probing depth or higher clinical attachment.

Conclusions: The presence of HSV appeared to be associated with the severity of periodontal diseases on both a site and subject basis. Understanding the role of herpesviruses in human Periodontitis may be of value for improving diagnosis, determining more specific treatment and preventing the diseases.

Mariotti. Dental Plaque-Induced Gingival Diseases. Ann Periodontol 1999;4: 7-19

Purpose: To investigate the clinical characteristics of gingival disease associated with plaque, endogenous hormone fluctuations, drugs, systemic diseases, and malnutrition.

Materials and Methods: Literature review

Findings and Conclusions: The author discusses his findings with regards to gingival disease as they relate to the following sub-topics. Plaque induced gingivitis: It is inflammation that results for bacterial plaque accumulation at the gingival margin. It is the most common form of gingival disease. The common findings are erythema, edema, bleeding, sensitivity, tenderness, and enlargement. There is no bacterial flora that is pathognomonic of plaque-induced gingivitis, however it is differs from the flora associated with health. Puberty associated gingivitis: The rise in steroid hormone during puberty has a transient effect on the inflammatory status of the gingival. It is the propensity to develop gingival inflammation in the presence of a relatively small amount of plaque during the circumpubertal period that distinguishes this disease. Menstrual cycle-associated gingivitis: Cases of overt gingival changes that fluctuate in conjunction with the menstrual cycle are infrequent. Gingival exudates has been shown to increase by at least 20% during ovulation in over 75% of women. Most women with inflammation induced by their menstrual cycle present with a very mild form of the disease. Pregnancy associated gingivitis: Longitudinal and cross-sectional studies have shown that the prevalence and severity of gingival inflammation, is significantly higher in the pregnant versus the post partum patient even thought plaque remained the same between the 2 groups. Pregnancy associated pyogenic granuloma: They occur in 0.5%-5% of pregnant females. They present clinically as a painless protuberant, mushroom like, exophytic mass that is attached by a pedunculated base from the gingival margin or more commonly for an interproximal space. Drug influenced gingival enlargement: Principally seen with 1- Phenytoin, which is used on a chronic regimen for the control of epileptic seizures. Gingival enlargement is seen in approximately 50% of pts. 2- Ca Channel Blockers, prescribed as antihypertensive, antiarrhythmic, and antianginal agents. Gingival lesions with this drug are seen in 20% of patients. 3- Cyclosporine A: a powerful immunoregulatory drug. Enlargement is seen in 25%-30% of the patients taking this class of drugs. Oral contraceptive-associated gingivitis: Studies have shown gingival changes that have developed in pre-menopausal women on oral contraceptives. Today doses are much smaller than previously described, and it is not known if present formulations can induce similar gingival changes. Diabetes mellitus-associated gingivitis: Found consistently in children with poorly controlled type I DM. It is much harder to see the relationship in adults as most studies evaluated gingival inflammation association with attachment loss in diabetics. Leukemia-associated gingivitis: oral manifestations of acute leukemia include cervical adenopathy, petechiae, mucosal ulcers, and gingival enlargement. Gingival disease associated with malnutrition: Vit A, Vit B complex, and Niacin deficiency have been shown to affect gingival tissues in animal studies. In Vit C deficiency (scurvy) the gingival tissue have been described as bright red, swollen, ulcerated, and susceptible to hemorrhage.