DOWNLOAD PDF BACKGROUND TEXT : ATTACHMENT OF ORAL MICROBIAL TO DENTAL SURFACES SMITH

Chapter 1 : List of periodontal diseases - Wikipedia

INTRODUCTION. The microbial diversity in the oral cavity is among the largest so far characterized in the human body ().Of specific interest is the dental biofilm, which forms first by selective adsorption of bacteria from saliva onto the tooth surface, followed by bacterial growth.

The aim of this study was to detect the prevalence of selected bacterial species in intraoral sites of patients with chronic periodontitis CP using multiplex polymerase chain reaction PCR. Samples were collected from the tongue dorsum, buccal mucosa, supragingival and subgingival plaque and saliva of 30 patients with untreated CP. Multiplex PCR was used to determine prevalence rates, which were then compared using a chi-square test. Mean and standard deviation values were used to evaluate variations in prevalence according to site. The prevalence of S. The prevalence of E. The highest prevalence of P. The prevalence of bacteria did not vary significantly among the intraoral sites. All studied bacteria were identified in intraoral sites. Multiplex PCR proved to be an adequate method for epidemiological studies. Microorganisms are distributed in four oral ecosystems - tongue dorsum, buccal mucosa, supragingival and subgingival plaques. Saliva keeps direct contact with oral tissues; it contains cells from different sites of the mouth that may spread as plaque or planktonic suspension7. Streptococcus mutans, Enterococcus faecalis, Porphyromonas gingivalis, Prevotella intermedia are bacterial species that knowingly cause several oral diseases, such as dental caries, endodontic infections and periodontal diseases7,14,19, One of the important virulence properties of these microorganisms is the ability to form a biofilm, known as dental plaque, on tooth surfaces13, Some of the bacterial components associated with the adhesion phase of S. The number and distribution of genotypes of S. There were differences in the distribution of genotypes of S. A number of factors are associated with the virulence of this oral anaerobe, including a variety of proteases, endotoxins, and collagenase, as well as the production of surface structures such as fimbriae and capsular polysaccharides This species has several of the virulent properties of P. Its virulence factor is determined by its capability of adhesion to hard surfaces and soft tissues of the oral cavity6. Several studies used polymerase chain reaction PCR to detect selected bacterial species in the oral cavity7,12,20, The multiplex variant of PCR can be used for the simultaneous amplification of two or more loci in the same reaction Success in the treatment of intraoral infectious diseases depends on the knowledge of the prevalence of the bacterial species associated with etiological factors. Therefore, this study detected S. All participants were in good general health. Patients were included in the study if they had a clinical diagnosis of chronic periodontitis and no history of odontogenic pain. None of the patients included in the study had previously received any type of periodontal treatment. Clinical examination Clinical measurements were performed at six intraoral sites mesiobuccal, buccal, distobuccal, distolingual, lingual and mesiolingual positions on each tooth except the third molar at a baseline visit. Clinical parameters measured were: Saliva, oral mucosa and supra- and subgingival plaque samples for microbiological assessment were collected before clinical measurements. All patients provided samples of soft tissue and supra- and subgingival plaque. This model of clinical examination was described in previous studies17, Sampling procedures Samples were collected from saliva, buccal mucosa, tongue dorsum and supra- and subgingival plaques, in this sequence. Aseptic techniques were used during all the study. For the other intraoral sites, samples were collected by gently rubbing epithelial and dental surfaces for 1 minute with two 50 sterile absorbent paper points. Supra- and subgingival plaque samples were collected from 2 different sites. Samples from supragingival plaque were collected with gently rubbing dental surfaces with two paper points and samples from subgingival plaque were collected with a two sterile 50 absorbent paper points introduced into the periodontal pocket maintained for 1 min. For DNA extraction, the samples were thawed under refrigeration, vortex-mixed for 1 min, and then centrifuged at rpm for 5 min. Reference DNA for the microorganisms under analysis S. Thermal cycling parameters for E. Reactions were classified as positive when bands of expected sizes were detected. Mean and standard deviation values were used to evaluate the

variations of bacterial prevalence rates according to intraoral sites. The prevalence of P. Bacterial prevalence remained independent from site analyzed, which was demonstrated by the relatively constant value of standard deviations for the studied sites according to type of bacteria. Mean prevalence values for P. The determination of bacterial prevalence rates in all sites under study at the same time and in association with CP was an important characteristic of this study because saliva establishes direct contact between four oral ecosystems, which are important reservoirs for infection. The bacteria selected for this study play an important role in oral microbial ecology. The microorganisms that colonize the oral environment produce plaques of different complexities depending on intraoral site, genetic background and individual environmental factors7. Socransky and Haffajee25 reported that once periodontal tissues are colonized, evidence suggests that only a subsequent species causes destructive periodontal disease. To colonize subgingival sites, bacteria must attach to one or more of the available surfaces, multiply, compete successfully against other species for that habitat, and defend itself from host defense mechanisms. This bacterial species is the one most often studied in the human mounth14, The prevalence rate in our study was lower This type of bacteria is common in gastrointestinal infections and secondary endodontic infections. In the present study, teeth from which samples of sub- and supragingival plaques were collected showed pulp vitality and absence of dental caries. These results are in agreement with previous findings. Periodontal pathogens were detected in supragingival plaque from sites in which subgingival samples were negative for the same species. Supragingival plaque can harbor putative periodontal pathogens, which suggests that this environment may be a reservoir of such species and for the spread of infection or reinfection of subgingival sites. In average, all of the species tested were found in all of the sampled surfaces. The major differences were in the proportion of colonization of the different surfaces, which suggests that receptors, co-aggregation or local habitat differences may play major roles in defining community structure. The microbial composition of saliva was most similar to that found on the lateral and dorsal surfaces of the tongue, suggesting that these surfaces may be the major sources of salivary bacteria. The microbiotas colonizing the remaining surfaces showed great similarities to each other, but differences were detected between surface sites. Plaques on teeth were somewhat similar to each other, but quite different from the microbiotas on soft tissue surfaces and in saliva. However, as pointed out above, tooth-colonizing species were detected on soft tissues. The results of the present study are in agreement with values found in other investigations, particularly when some characteristics of variables and methods are taken into consideration2,11,12,14,19,20,23,27,28, The method used in this study confirmed results obtained by other authors1,20, which indicated that the PCR method can be used in epidemiological studies to analyze the prevalence of selected bacteria in different populations. According to Oho, et al. According to their study, these organisms should be detected by more sensitive means, such as PCR, to examine their roles in periodontal diseases. The risks and benefits of molecular techniques to achieve the main objective of this study indicate that multiplex PCR is adequate to determine the prevalence of bacteria in several intraoral sites. The prevalence rates for S. Clinical and microbiological features of refractory periodontitis subjects. Subgingival microbiota of Brazilian subjects with untreated chronic periodontitis. Detection of Actinobacillus actinomycetemcomitans in unstimulated saliva of patients with chronic periodontitis. Molecular genetics analyses of biofilm formation in oral isolates. Prevalence of periodontopathic bacteria in aggressive periodontitis patients in a Chilean population. Molecular pathogenesis of periodontal disease. American Society for Microbiology Press; Introduction to microbial aspects of periodontal biofilm, communities, development and treatment. Microbial complexes in supragingival plaque. Pathogenicity of Prevotella intermedia and Prevotella nigrescens isolates in a wound chamber models in rabbits. Virulence factors of Porphyromonas gingivalis. New bacterial species associated with chronic periodontitis. Molecular analysis of the subgingival microbiota in health and disease. Genotypic and phenotypic analysis of Streptococcus mutans from different oral cavity sites of caries-free and caries-active children. Role of Streptococcus mutans in human dental decay. The bacteriology of acute necrotizing ulcerative gingivitis. Occurrence of Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, and Prevotela intermedia in progressive adult

periodontitis. Distribution of selected bacterial species on intraoral surfaces. The life and times of the Enterococcus. Simple and rapid detection of Streptococcus mutans and Streptococcus sobrinus in human saliva by polymerase chain reaction. Bacterial diversity in human subgingival plaque. Prevalence of four putative periodontopathic bacteria in saliva of a group of Brazilian children with mixed dentition: Int J Paediatr Dent. Multiplex polymerase chain reaction detection of black-pigmented bacteria in infections of endodontic origin. Subgingival microflora of advanced periodontitis in the Dominican Republic. Microbial mechanisms in the pathogenesis of destructive periodontal diseases: Microbial complexes in subgingival plaque. Life as an endodontic pathogen. Ecological differences between the untreated and the root-filled root canals. Simultaneous detection of Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis by a rapid PCR method.

Chapter 2 : Oral Streptococci Biofilm Formation on Different Implant Surface Topographies

Background. Tea has been suggested to promote oral health by inhibiting bacterial attachment to the oral cavity. Most studies have focused on prevention of bacterial attachment to hard surfaces such as enamel.

Primary occlusal trauma Secondary occlusal trauma The presence of certain developmental or acquired conditions can influence the outcome of periodontitis see table. Transition from plaque induced gingivitis to periodontitis[edit] Plaque-induced gingivitis and the more severe stage plaque induced periodontitis are the most common of the periodontal diseases. While in some individuals gingivitis never progresses to periodontitis, [13] periodontitis is always preceded by gingivitis. Although this new classification does not correlate with clinical signs and symptoms and is admittedly "somewhat arbitrary," it permits a focus of attention pathologic aspects of the disease that were, until recently, not well understood. Initial lesion[edit] Unlike most regions of the body, the oral cavity is perpetually populated by pathogenic microorganisms ; because there is a constant challenge to the mucosa in the form of these microorganisms and their harmful products, it is difficult to truly characterize the boundary between health and disease activity in the periodontal tissues. The oral cavity contains over different microorganisms. It is very hard to distinguish exactly which periodontal pathogen is causing the breakdown of tissues and bone. As such, the initial lesion is said to merely reflect "enhanced levels of activity" of host response mechanisms "normally operative within the gingival tissues. When looking at the gums they look knife like and a very light pink or coral pink. On the contrary, the initial lesion shows increased capillary permeability with "very large numbers" of neutrophils migrating from the dilated gingival plexus into the junctional epithlieum and underlying connective tissue yet remaining within the confines of the region of the sulcus and macrophages and lymphocytes may also appear. Loss of perivascular collagen occurs; it is thought that this is due to the degradative enzymes released by extravasating leukocytes, such that the collagen and other connective tissue fibers surrounding blood vessels in the area dissolve. The initial lesion appears within two to four days of gingival tissue being subjected to plaque accumulation. When not generated through clinical experimentation, the initial lesion may not appear at all, and instead, a detectable infiltrate similar to that of the early lesion, explained below, appears. Immunoblasts are quite common in the area of infiltration, while plasma cells , if present, are only at the edges of the area. The junctional epithelium may even become infiltrated with enough leukocytes so that it resembles a microabscess. Beginning two to three weeks after first plaque formation, the established lesion is widespread in both human and animals populations [22] and can be seen commonly associated with the placement of orthodontic bands on molars. In health, the junctional epithelium creates the most coronal attachment of the gum tissue to the tooth at or near the cementoenamel junction. In the established lesion of periodontal disease, the connective tissue lying subjacent to the junctional epithelium is nearly destroyed, failing to properly support the epithelium and buttress it against the tooth surface. In response to this, the junctional epithelium proliferates and grows into the vacant underlying spaces, effectively causing the level of its attachment to migrate towards apically, revealing more tooth structure than is normally evident supragingivally above the level of the gumline in health. While many established lesions continue to the advanced lesion below , most either remain as established lesions for decades or indefinitely; the mechanisms behind this phenomenon are not well understood. Features of the Established Lesion: