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Editorial

Osteoimmunology and Microbial Load of Periimplant Tissues

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The search of a link between the cytokine levels and the specific bacterial pathogens is the subject of new studies in osteoimmunology. Bacteria impact similar responses to the host, causing the characteristic effects of signaling pathways associated with a series of proinflammatory cytokines. Conditions provided by the host in the mouth affect the development, composition and metabolic activity of the oral microflora and vary in different areas of the oral cavity. Analogous to this, the composition of the oral microflora varies in different ecosystems: tongue, buccal mucosa and teeth. When the microorganisms are in "health status" they are in dynamic equilibrium with the host, but change in the key parameters that determine microbial growth can disturb equilibrium. In order to better understand the link between oral microorganisms and the host in the state of health or disease, a concept is developed which is based on ecological principles that explain their interactions. The ecological plaque hypothesis describes the dynamic nature of the relationship between the host and the normal microflora [1]. Changes in the environment increase the competitiveness of pathogens and increase the virulence factors. Pathogenic microorganisms may also be present in a good health condition of the oral tissues, but with extremely low representation and clinically unsignificant levels. According to this hypothesis, there is a direct link between local environmental conditions and activity and composition of the biofilm. The local environmental conditions also involve temperature, redox potential, nutrients, genetic characteristics, lifestyle and the age of the host. Thanks to innate and acquired immunity, the host prevents bacterial invasion and colonization. But despite immune defense, the host developed capabilities to support complex microflora. Receptors for host cell recognition (host cell pattern recognition receptors-PRPs) are deployed strategically in tissues to register changes in the external and internal microenvironment and recognize microbe-associated molecular pathways (microbe-associated molecular patterns MAMPs) such as lipopolysaccharides, nucleic acids, etc. They are able to activate multiple signaling pathways in which most of them are cross through nuclear factor (NF-kB). MAMPs get rid of all microbial cells. Host created mechanisms for tolerance of resident microorganisms in order not to initiate harmful inflammatory responses, but does not lose the ability to effectively defend against pathogens [2]. The importance of systems for detection of host manifested by the fact that abnormal expression of PRPs that bind bacterial lipopolysaccharide (TLR2 and CD14) is associated with predisposition to the development of diseases of the anchor apparatus [3]. Resident oral bacteria determine the normal expression of immune mediators, assist in maintaining the health of tissues through regulation of low levels of expression of intracellular adhesion molecules (intracellular adhesion molecule-1), E-selectin, IL-8, which in turn participate in the creation of a protective layer of neurofili strategically positioned between subgingival biofilm and the attached epithelium [4].

In circumstances where the owner fails to prevent initial microbial insult, begins activation of mechanisms of inadequate response that further improve the conditions for the growth of pathogenic species of microorganisms, which in turn deepen the inadequate response of the host. According to Petrovic and Vojdic [5] implants that show early signs of implant failure have microorganisms similar to those teeth which show clinical signs of parodontopathy. Bacteria produce bacterial collagenase and under their influence cells synthesize osteoclasted activated cytokines. According to the research of the authors Mitsugi., *et al.* [6], Odila., *et al.* [7], Nonnenmacher., *et al.* [8], there are a number of periodontopathogenic bacteria, including: *Porphyromonas gingivalis*, Bacteroides forsythus (*Tannerella forsythia*), *Fusobacterium nucleatum*, *Streptococcus intermedius, Peptostreptococcus micros, Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Dialister pneumosintes*, *Micromonas* (*Peptostreptococcus*) *micros*.

Cytokines participate in complex cascades of osseointegration as in healing soft-tissue wounds in three phases: phase of inflammation (IL-1, IL-6, TNF-α, PDGF), proliferative phase (cytokine-activated macrophages secrete angiogenic factors - VEGF and fibroblast - stimulating cytokines) and the phase of remodeling (TGF-β). TGF-β occupies a central role in the healing process of wounds. TGF-β inhibits the function of activated inflammatory cells, accelerate the healing process through the proliferation and engaging of fibroblasts including periosteal mesenchymal cells which promote matrix synthesis. It stimulates the synthesis of collagen (collagen I and III) by fibroblasts, affects integrin expression and thus participate in the composition and interconnection of collagen structures (remodeling phase). This cytokine causes greater predominance of bone formation processes in terms of bone resorption during bone remodeling. Also TGF-β affects the process of angiogenesis. Schierano and Bassi [9], Schierano and Bellone [10] noticed an increase of TGF-β1 and TGF-β2 in peri-implant tissues during post-implantation period and conclude that successful osseointegration depends on the modulation and balance of bone factors for remodeling, through increased expression of TGF-β during the post-implantation period, through the absence of increased levels of pro-inflammatory cytokines and expression of such a cytokine profile that is able to modulate osteoclastic formation and phosphatase activity in primary osteoblasts.

Possible factors that may cause marginal bone loss observed in the first year of implant function, involved bacterial adhesion to implant surface, surgical trauma, occlusal overload, the presence of microgap between the implant and abatement, inadequate biological width implant crestal module etc.

Bibliography

1. Marsh PD. "Are dental diseases examples of ecological catastrophes?" *Microbiology* 149.2 (2003): 279-294.
2. Hooper LV. "Do symbiotic bacteria subvert host immunity?" *Nature Reviews in Microbiology* 7.5 (2009): 367-374.
3. James JA., *et al*. "Polymorphisms of TLR4 but not CD14 are associated with a decreased risk of aggressive periodontitis". *Journal of Clinical Periodontology* 34.2 (2007): 111-117.
4. Dixon DR., *et al*. "Modulation of the innate immune response within the periodontium". *Periodontology 2000* 35 (2004): 53-74.
5. Petrovic A and Vojdic D. "Determination of Inflammatory cytokine concentrations in gingival fluid samples of dental implants and control teeth". *Serbian dental Journal* 47.1 (2000): 15.
6. Mitsugi O., *et al*. "Intrafamilial distribution of nine putative periodonthopathogenes in dental plaque samples anayzed by PCR". *Journal of Oral Science* 46.3 (2004): 149-156.
7. Odila P., *et al*. "Periodonthopathogenes in the saliva and subgingival dental plaque of group of mothers". *Pesquisa Odontológica Brasileira* 16.4 (2002): 313-318.
8. Nonnenmacher C., *et al*. "DNA from periodontopathogenic bacteria is immunostimulatory for mouse and human immune cells". *Infection and Immunity* 71.2 (2003): 850-856.
9. Schierano G and Bassi F. "Cytokine production and bone remodeling in a patient wearing overdentures on oral implants". *Journal of Dental Research* 79.9 (2000): 1675-1682.
10. Schierano G and Bellone G. "TGF-β and IL-10 around implants". *Journal of Dental Research* 82.6 (2003): 428-432.

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