EARLY DIAGNOSIS OF PERIODONTITIS DEVELOPMENT

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Abstract: Numerous studies have proved that predisposition to periodontitis depends on heredity, which largely determines the nature of the body's immune response to the introduction of periodontopathogenic microflora.
Key words: ADAMC-1; ADAMC-9; TIMP-3; human; mRNA; matrix metalloproteinase; periodontitis; polymerase chain reaction.

Introduction. Major genetic factors are not single mutations that fundamentally alter a gene or its product, but more subtle genetic changes that merely alter gene expression or gene product function. Studies in the etiology of both aggressive and chronic periodontitis have led to the finding of a dramatic increase in the activity of matrixins (matrix proteinases synthesised by fibroblasts and macrophages in particular) in the occurrence of periodontal tissue inflammation of any nature. Investigations of the periodontal transcriptome in norm and pathology should keep in mind the necessity to build up a molecular cellular model of the periodontal defence. Some authors have suggested that TNFa and IL10 are primary inducers of the response to pathogens in the foci, which in turn cause increased synthesis of IL6, IL8, IL10 and IL4. It can be assumed that the synthesis of matrix metalloproteinases, especially MMP8 and MMP9, as well as their endogenous inhibitors TIMP1 and TIMP2 are also under the control of lymphokines, although the main cell type producing MMP and TIMP on the periodontium remains unclear: it may be fibroblasts, macrophages or both these cell types simultaneously. By analogy with the production of Thi-type lymphokines, it may be speculated that the synthesis of MMP during the rapid non-specific response to infection may have a protective effect (promotes pathogen inactivation), whereas their prolonged synthesis leads to degradation of the periodontal ligament matrix. The ratio of the contributions of bacterial proteinases and autologous human MMP in degradation of the periodontal ligament collagen matrix has not been studied experimentally either, although several authors are convinced of the major role of MMP in this process.

The published works we studied do not question the role of antigen-dependent

mechanisms in periodontal protection, although Abiko Y. et al. (2014) refer to the Msp and TmpC proteins of T. denticola studied as 'surface antigens'. Meanwhile, the role ascribed to IL4 as a positive marker of periodontal health suggests the importance of protective antibody production in maintaining periodontal health

Characterizing clinical-diagnostic value of determining the local level of lymphokines and MMP in periodontal tissue (including the indirect method of measuring the concentration of their transcripts) from the point of view of evaluating the current state of the disease and prognosis of its course for the future, it should be noted that these methods are practically not used in practice. The reason for this, in our opinion, is the lack of predictive power of the existing methods.

Conclusions: In spite of the significant gaps in understanding the mechanisms of periodontitis development, the literature analysis leaves no doubt that the selected markers IL8, TNFa, MMP8 and MMP9 are the important components of periodontal homeostasis maintenance systems in norm and pathology. However, the relationship of these cytokines and matrixins genes polymorphism according to the literature is controversial and not conclusively proved.

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