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Functional toll-like receptor 4, interleukin-6, -8 and CCL-20 release, and NF-kB translocation in human periodontal ligament mesenchymal stem cells stimulated with LPS- P. Gingivalis

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Periodontal diseases, the major public health problem of the oral cavity, are clinically characterized by inflammation of the periodontal connective tissue that ultimately induces the destruction of periodontal tissue and the loss of alveolar bone. In chronic periodontitis, as well as aggressive periodontitis, the anaerobic gram-negative bacterium Porphyromonas gingivalis (P. gingivalis) is implicated, and its pathogenicity is exerted by a wide variety of factors, among which the lipopolysaccharides (LPSs).

LPSs activate the innate immune response during Gram-negative bacterial infections through the Toll-like receptor 4 (TLR-4)/myeloid differentiation protein 2 (MD-2) complex.

In this study, the expression of TLR-4, the cell growth, the cytokines release and the nuclear factor-KB (NF-kB) transcription factor expression in response to LPS-P. Gingivalis (LPS-G) were examined in Human Periodontal Ligament Mesenchymal Stem Cells (PDL-MSCs) [1].

The results obtained have demonstrated that, in basal conditions, human PDL-MSCs express high levels of TLR-4. In inflammatory conditions mimicked by LPS-G challenge, the MTT assay carried out at different treatment times evidenced the decrease of the cell growth. Moreover, the recognition of P. gingivalis components by TLR-4 culminated with the activation of secretion of inflammatory mediators such as: IL-6, IL-8 and CCL-20, and with the up-regulation of NF-kB, which was translocated into the nucleus. Our data intended to specify that TLR-4 expressed by PDL-MSCs is functional and plays a key role in inflammation.

References

[1] Trubiani et al. (2010) Expression Profile of the Embryonic Markers Nanog, OCT-4, SSEA-1, SSEA-4, and Frizzled-9 Receptor in Human Periodontal Ligament Mesenchymal Stem Cells. J Cell Physiol 225:123-31.

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