Topic: Basic Research: Aetiology and Pathogenesis

P0060

Effect of rhPDGF-BB on periodontal regeneration

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Aim: We investigated the cell kinetics during bone regeneration after guided tissue regeneration (GTR) in combination with application of recombinant human platelet-derived growth factor (rhPDGF-BB).

Material and Methods: Buccal dehiscence defects were surgically prepared on the mesial root surface of the premolars of 10 beagle dogs. The defects were divided into two groups: e-PTFE membrane and rhPDGF-BB were applied to the GTR/rhPDGF-BB group (n=10), and e-PTFE membrane were applied to the GTR group (n=10). The animals were sacrificed at 1, 2 or 8 weeks. To observe the location of proliferating cells, we performed immunohistological staining for proliferating cell nuclear antigen (PCNA). The distribution of osteogenetic cells was evaluated by assessing alkaline phosphatase (ALP) activity.

Results: In the GTR group at two weeks, a few PCNA-positive cells were observed in the tissues near blood vessels, and ALP activity was localized at the bottom of the defects. In the GTR/rhPDGF-BB group at two weeks, many PCNA-positive cells and ALP activity were located at the bottom of the defects and they were also spreading to the coronal portion of the defects. The percentage of PCNA-positive cells at two weeks was significantly higher in the GTR/rhPDGF-BB group than in the GTR group ($p<0.01$).

Conclusion: These results suggest that GTR in combination with application of rhPDGF-BB promotes the proliferation of cells involved in bone regeneration in the early stages of wound healing.

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P0061

The role of TGF Beta 1 and VEGF in the pathogenesis of scleroderma

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Aim: Systemic sclerosis (SSc) or scleroderma is a rheumatic acquired disorder that typically results in the fibrosis of the skin and internal organs. The pathogenesis of this disorder includes inflammation, autoimmune attack, and vascular damage, leading to fibroblast activation. The aim of this study was to compare the gene expression profile, by immunohistochemical analysis, of TGFB-1 and VEGF mediators in 44 patients divided in two groups: Scleroderma and Control (CO).

Material and Methods: 44 patients were enrolled in this prospective clinical study. The collection of gingiva biopsies (2x2 mm) and periodontal ligament specimens was carried out during routine extraction for oral surgery therapy and processed for immunohistochemistry. The following primary antibodies were used: anti-TGF Beta1 and VEGF. Also, frequency distributions, media and standard deviation (SD) were determined at baseline in each group to describe the clinical parameters (PD, CAL, CPITN, PI and BOP). The Kruskal Wallis and the Mann Whitney U and Wilcoxon Singed Rank Tests were carried out when comparing the clinical parameters between two groups.

Results: Gingival samples clearly showed a normal staining pattern for TGF-B1 in CO, whereas it appeared severely reduced in samples of patients with SSc. Immunofluorescence reactions performed using VEGF antibodies, staining patterns showed a higher intensity in SSc that observed in CO. Similar results were obtained on periodontal ligament.

Conclusion: The findings presented here make it clear that biomarker such as TGF B1 and VEGF have an important role in the orchestration of the immune response, which in turn influence the outcome of disease establishment and evolution.

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Wnt5a expression by Porphyromonas gingivalis LPS via NF-kappaB and STAT1

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Aim: Wnt signaling molecules play important roles in bone biology, apoptosis, chronic inflammation and wound healing. Recent studies have suggested the associations of these molecules with various diseases including cardiovascular diseases, rheumatoid arthritis and osteoarthritis. But there have been no reports on Wnt5a expression in periodontitis tissue. We previously demonstrated that Wnt5a mRNA expression was up-regulated in chronic periodontitis tissue when compared to healthy control tissue. In this study, we investigate what molecular mechanisms are involved in regulation of Wnt5a expression.

Material and Methods: Human monocytic cell line THP-1 were stimulated with Porphyromonas gingivalis (P. gingivalis) LPS. To investigate the involvement of NF-κB and JAK/STAT pathways in the modulation of Wnt5a expression, we performed inhibition assay, transfection, western blotting, luciferase assay and EMSA. The levels of Wnt5a mRNA were determined by real-time RT-PCR. To investigate the involvement of NF-κB and STAT1 in the modulation of Wnt5a expression, we performed Western blotting (WB) detection of proteins obtained through immunoprecipitation (IP).

Results: Wnt5a mRNA was expressed by THP-1 cells in response to P. gingivalis LPS. P. gingivalis LPS-mediated Wnt5a mRNA expression was inhibited by IKK inhibitor and dominant-negative IκBα. Binding of NF-κB to DNA was increased by P. gingivalis LPS stimulation. Wnt5a expression was inhibited by JAK/STAT and STAT1 inhibitor. Immunoprecipitation analysis demonstrated that STAT1 interacted with p65 by P. gingivalis LPS stimulation.

Conclusion: STAT1 may activate NF-κB and work in concert to promote Wnt5a production. These findings will help reveal the mechanism of molecular pathogenesis of periodontal disease.

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