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BOOK OF ABSTRACTS

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IL-33/ST2 SIGNALING IN HUMAN PERIAPICAL LESIONS

A. Lukic[1], M. Velickovic[2], I. Jeftic[3], T. Kanjevac[3], S. Mitrovic[3]

[1]Faculty of Medical Sciences, University of Kragujevac, Serbia, Kragujevac, Serbia
[2]Center for Molecular Medicine and Stem Cell Research, Kragujevac, Serbia
[3]Faculty of Medical Sciences, Kragujevac, Serbia

Background: Interleukin-33 (IL-33) is a novel member of the IL-1 cytokine family and ligand for the IL-1 receptor-related protein, ST2. Recent evidence suggests that IL-33/ST2 signaling regulates Th1/Th2 immune responses in several chronic inflammatory disorders, but its role in periapical lesions is unknown. We aimed to investigate the expression pattern of IL-33 and ST2 in chronic apical periodontitis and normal periapical tissues. Methods and materials: Healthy periapical tissues (n=10), symptomatic (n=12) and asymptomatic periapical lesions (n=20) were evaluated by immunohistochemistry using anti-IL-33 and anti-ST2 antibodies. Tissue samples were analyzed according to the immunostaining density condition. The intensity of staining was scored from 0 to 3 as followed: (0) absent; (1) discrete; (2) moderate; and (3) intense staining. Results: All periapical tissue samples showed positive immunoreactivity to IL-33. Nuclear expression of IL-33 was detected in the cells morphologically consistent with fibroblasts and endothelial cells in normal periapical tissues and granulomas, and in the epithelium of epithelialized granulomas and cysts. There was highly significant difference in IL-33 expression between symptomatic and asymptomatic lesions (p0.05). Conclusions: We concluded that IL-33/ST2 signaling is involved in the development of human periapical lesions. Increased expression of IL-33 in asymptomatic lesions might be important for the restriction of unwanted immune responses in the periapical region. This work was supported by grants from the Serbian Ministry of Science and Technological Development (OP 175071), Serbia.

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THE EFFECT OF ST2 DELETION ON THE FORMATION OF EXPERIMENTALLY-INDUCED PERIAPICAL LESIONS IN MICE

M. Velickovic[1], N. Pejnovic[2], N. Jovicic[2], T. Kanjevac[2], A. Lukic[2]

[1]Center for Molecular Medicine and Stem Cell Research, Kragujevac, Serbia
[2]Faculty of Medical Sciences, Kragujevac, Serbia

Background: ST2 is a member of the IL-1 receptor family and IL-33 was recently identified as its natural ligand. IL-33/ST2 axis promotes Th2 immune responses and has an important role in allergy, autoimmunity and inflammation. The role of ST2 signaling in the pathogenesis of periapical lesions has not been studied. The aim of this study was to investigate whether in an experimental model in mice the lack of ST2 gene affects development and characteristics of periapical lesions. Methods and materials: Pulps of mandibular first molars from „wild type" (WT) (n=15) and ST2 „knock-out" mice (ST2-/-) on the BALB/c background (n=10) were exposed and left open to the oral environment for 2 weeks. Mice with non-exposed teeth served as controls (n=5/group). After sacrifice, hemi-mandibles were isolated and prepared for histological, immunohistochemical and flow cytometric analysis. Expression of IL-33 was determined in periapical lesions and normal periodontal ligaments from WT mice (n=5/group). Results: ST2 deficient mice showed an increase of periapical bone loss compared to WT mice. Periapical bone loss in ST2-/- mice was associated with enhanced influx of neutrophils, CD4+ T cells, CD3+CXCR3+ and CD3+CCR6+ T cells in the periapical tissue, as well as with increased percentages of CD4+ T cells expressing proinflammatory cytokines TNF-α, IL-6, IFN-γ and IL-17 ($p<0.05$). The expression of IL-33 was markedly increased in periapical lesions induced in WT mice compared to control WT mice ($p<0.05$). Conclusions: The obtained findings suggest that ST2 signaling negatively regulates periapical tissue destruction by preventing Th1/Th17 cell mediated periapical immune responses. This work was supported by grants from the Serbian Ministry of Science and Technological Development (OP 175071), Serbia.