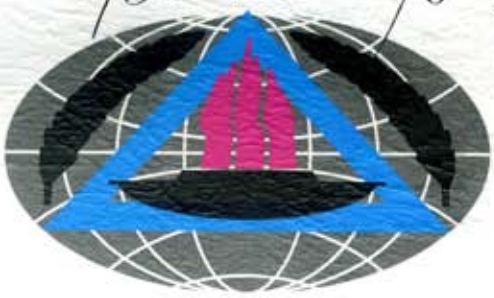
PROGRAM AND ABSTRACTS OF PAPERS

JAPANESE ASSOCIATION FOR DENTAL RESEARCH



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081 Down-regulation of RANKL/NF-κB by β-TCP implant in dog jaw

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Objectives: Successful remodeling in the bone surrounding dental implants requires the coordinated activities of both osteoblasts and osteoclasts. Bone induction by different calcium phosphate biomaterials has been previously reported, and β-tricalcium phosphate ceramic (β-TCP) has shown osteoinductive activity in implant therapy. However, little is known about the molecular basis for mechanisms responsible for β-TCP in bone formation. To study the effects of β-TCP on mineralization, the gene expression profiles of dog jawbone tissue implanted with β-TCP were examined. Methods: The premolars of beagle dogs were extracted. After 3 months, β-TCP was implanted into cylindrical artificial bone defects (4.5 x 8 mm). After 7 and 14 days, all specimens were taken out, and total RNA was isolated from bone tissues using Multi-Beads Shocker. After amplification of mRNA, gene expression profiles were examined using Affymetrix GeneChip (Canine Genome 2.0 Array, ca. 38,000 genes) system. GeneChip data was analyzed using GeneSpring software and imported into Ingenuity Pathways Analysis (IPA). Results: At 7 days, 2,224 up-regulated genes and 2,503 down-regulated genes, at 14 days, 2,108 up-regulated genes and 5,304 down-regulated genes were observed ($p<0.05$). β-TCP altered many gene expressions, and decreased RANKL, IL-1, MYD88, TRAF2, RIP, and NF-κB1 gene expressions, which were involved in NF-κB signaling pathway. Conclusion: IL-1 is proinflammatory cytokine that is a potent stimulator of bone resorption and an inhibitor of bone formation. RANKL is a key mediator of osteoclast formation, function, and survival. Our findings suggest that β-TCP may inhibit the differentiation of osteoblast precursors to osteoclasts through reduction of RANKL and NF-κB expressions and promote matrix bone mineralization and calcification.