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Bone resorption, matrix metalloproteinases and caffeic acid phenethyl ester

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Dear Editor,

We have read with great interest the original article, "Effect of salmon calcitonin treatment on serum and synovial fluid bone formation and resorption markers in osteoporosis patients" by Atbinici et al published in Acta Orthopaedica et Traumatologica Turcica.[1] The authors determined their aims as to reveal the relationship between joint cartilage metabolism and osteoporosis and osteoblastic and osteoclastic activity parameters. Apart from the usage of calcitonin treatment and exploring a new marker for osteoporosis in synovial fluid, we would like to complete the discussion by introducing a major route through which A Disintegrin-like and Metalloproteinase with Thrombospondin type 1 Motifs (ADAMTS) could have some responsibilities.

Particle-induced bone osteolysis is a common cause of aseptic bone loses. Protein and mRNA expression of receptor activator of nuclear factor kappa B ligand (RANKL) is increased relative to its inhibitor osteoprotegerin in periimplant tissues correlating with increased resorptive activity[2] confirming the role of osteoclasts in the osteolysis.[3,4] These particles may travel to the other regions of implants and induce bone resorption via inflammatory factors that exacerbate bone resorption.[5] ADAMTS enzymes could have some effects on bone resorption/loss and caffeic acid phenethyl ester (CAPE) (Fig. 1) could have some protective effect on this process.

The main question here is do these particles have direct effect on ADAMTS enzymes, which are already found in osteoclasts, osteoblasts and chondrocytes? Because ADAMTS enzymes play a critical role in the degradation/repairing of extracellular matrix and they are likely to be useful in understanding of many disease pathogenesis such as arthritis, and cancer. It should be kept in mind that bone abnormalities such as bone resorption might be connected with ADAMTS enzyme activation/inhibition pathways.[6] Our study on ADAMTS enzymes identified that the pathways MAPK and NFκB were thought to be responsible pathways for the induction of ADAMTS9, an aggrecanase, in OUMS-27 human chondrosarcoma cells.[7] Therefore, any kind of NFκB inhibitor could possibly block the ADAMTS9 induction. We found that several ADAMTS genes including ADAMTS9 were induced upon IL-1β application. Some molecules that have NFκB inhibitory effects such as CAPE, royal jelly and curcumin prevented NFκB-dependent ADAMTS induction. That might possibly prevent inflammation, which is accused for osteoarthritis and its symptoms.[8] Ilhan et al suggested that the anti-inflammatory effect of CAPE is most likely due to the inhibition of ROS production at the transcriptional level, through the suppression of NFκB activation, and direct inhibition of the catalytic activity of iNOS.[9] NFκB and NFATc1 therefore represent potential targets for novel therapeutic agents developed to block the inflammatory response in cases where this process has become chronic or dysregulated.[10]

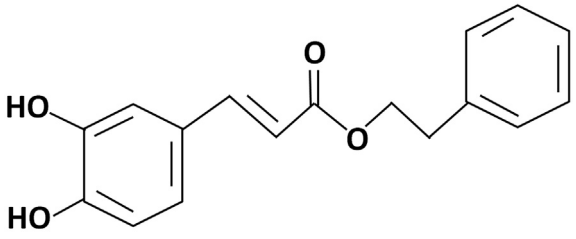

Fig. 1. The chemical structure of caffeic acid phenethyl ester (CAPE).

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