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News on current events in osteoporosis and rheumatology

Nicotine is bad for your bones

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That cigarette smoking is detrimental to bone is now widely acknowledged and causes are not univocal. The cellular mechanisms underlying these effects, however, are not fully understood. Two recent studies, performed in rats [1] and man [2] bring new insights into this field.

In the first study, Hapidin et al. administered nicotine to rats for 4 months. They observed that nicotine administration increased serum concentrations of IL-1 and IL-6, two interleukins known to stimulate bone resorption. Histomorphometric analysis showed that nicotine significantly decreased the trabecular bone volume, trabecular thickness, mineralizing surface, mineral appositional rate, and bone formation rate.

The second study was conducted in humans. The serum concentrations of RANK ligand (RANKL) and osteoprotegerin (OPG) were measured in age- and sex-matched groups of 35 smokers and 35 nonsmokers with almost identical levels of periodontal disease. Cigarette-smoker patients tended to have lower serum concentrations of RANKL and OPG than nonsmoker patients. While no statistically significant difference was observed for RANKL, there serum concentration of OPG was significantly lower in smokers than in nonsmokers whereas the ratio of serum concentrations of RANKL over OPG was higher in smokers. Concentrations of OPG in the smoker patients also had a statistically significant negative correlation with tobacco consumption. These results suggest that bone loss in smoker-related periodontitis patients may be partially explained by suppression of OPG production.

1. Hapidin H. et al. *J Bone Miner Metab* 2007;25:93-98.
2. Lappin DF et al. *J Clin Periodontol* 2007;34:271-277.

Nicotine stimulates bone resorption

The stimulatory effect of nicotine on bone resorption has been demonstrated in vivo and in vitro. Nicotine activates macrophages which produce more interleukins IL-1 and IL-6. These cytokines are known activators of resorption and induce a loss of trabecular bone.

This effect is partly due to a decreased synthesis of osteoprotegerin (OPG), a decoy receptor of RANK ligand (RANKL). More RANKL is thus available to bind RANK expressed on osteoclasts and activate them.

