Medicinal & Aromatic Plants, Volume 7
DOI: 10.4172/2167-0412-C1-018

Monotropein from *Morinda officinalis* attenuates bone loss of mice induced by combination treatment of ovariectomy and LPS and decrease inflammatory impairment on osteoblast through blocking activation of NF-κB pathway

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Both estrogen deficiency and inflammation play important roles in bone metabolism and occurrence of osteoporosis. Monotropein, iridoid glycosides from *Morinda officinalis*, has been reported to possess the properties of decreasing bone loss induced by estrogen deficiency and inhibiting inflammatory responses in LPS-induced RAW 264.7 macrophages. However, the effect of monotropein on bone loss in chronic inflammatory condition remains unclear. In present study, we found that monotropein significantly inhibited the reduction of bone mass, improved the bone micro-architecture through enhancing bone formation and blocking the increase of inflammatory cytokines in osteoporotic mice induced by the combination treatment of ovariectomy and LPS. Further *in vitro* experiment indicated that monotropein increased the proliferation, activity of Alkaline Phosphatase (ALP), bone matrix mineralization, and the expression of bone matrix protein Osteopontin (OPN) of osteoblastic MC3T3-E1 cells injured by LPS. In addition, monotropein significantly decreased the production of interleukin-6 (IL-6) and interleukin-1beta (IL-1β), inhibited the nuclei translocation of p65 and nuclear factor kappa B p50 (NF-κB P50) and markedly down-regulated the phosphorylation levels of nuclear factor kappa B p65 (NF-κB p65) and IκB kinase (IKK), indicating that monotropein could attenuate the impairments of inflammation on MC3T3-E1 cells through suppressing the activation of NF-κB pathway. Therefore, monotropein might be a promising candidate for prevention and treatment for inflammatory bone loss.

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