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Piceatannol stimulates osteoblast differentiation that may be mediated by increased bone morphogenetic protein-2 production
Piceatannol 可能透過增加 BMP-2 的產物的方式刺激調節成骨細胞的分化作用

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Abstract

Background and Purpose: We examined the effect of piceatannol on the proliferation and differentiation of osteoblast cells. Piceatannol stimulates osteoblast differentiation that may be mediated by increased bone morphogenetic protein-2 production. **Methods:** Inhibition of cell proliferation by piceatannol was measured by XTT assay; By means of alkaline phosphatase activity, osteocalcin enzyme-linked immunosorbent assay (ELISA) and we have shown that piceatannol exhibits a significant induction of differentiation; The degree of mineralization was determined using Alizarin Red staining; The type I procollagen assay, which measures the propeptide portion of the molecule and reflects the synthesis of the mature form of the protein; The role of BMP-2 in piceatannol-mediated cell differentiation was determined using assaying the levels of BMP-2;
Expression levels of BMP-2, alkaline phosphatase and osteocaclin mRNA were examined using RT-PCR. **Results:** Piceatannol stimulate osteoblast differentiation at various stages (from maturation to terminally differentiated osteoblasts). Induction of differentiation by piceatannol was associated with increased bone morphogenetic protein-2 (BMP-2) production. Addition of purified BMP-2 protein did not increase the upregulation of alkaline phosphatase activity and osteocalcin secretion by piceatannol, whereas the BMP-2 antagonist noggin blocked piceatannol and BMP-2-mediated alkaline phosphatase activity, and osteocalcin secretion enhancement, indicating that BMP-2 production is required in piceatannol-mediated osteoblast maturation and differentiation. **Conclusions:** Piceatannol increased BMP-2 synthesis, and this effect may contribute to its action on the induction of osteoblasts maturation and differentiation, followed by an increase of bone mass. Decreases in new bone formation, followed by estrogen deficiency or various pathologic factors, may contribute to the mechanisms involved in Postmenopausal osteoporosis.